Resource Allocation Theory
Applied to Farm Animal Production

Edited by Wendy Rauw

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It is the magician's wand,
by means of which [the agriculturist]
may summon into life
whatever form and mould he pleases

William Youatt (1872) cited in Darwin (1872)

One of the most remarkable features
in our domesticated races
is that we see in them adaptation,
not indeed to the animal's (…) own good,
but to man's use or fancy

Charles Darwin (1872)

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1 Introduction

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1. Life History Theory

When Charles Darwin travelled with the H.M.S. *Beagle* to Tierra del Fuego and the Falkland Islands in 1834, he observed a discrepancy between the number of eggs of a large white Doris (a sea slug) and the abundance of the species. He realized that the abundance of a species does not necessarily depend on the number of offspring produced (Van Straalen and Roelofs, 2006). Life history theory deals with the question of why the power of propagation differs so much between species, and with the way an organism spreads its reproduction over its lifetime and forms an adaptation to the environment it lives in (Brommer, 2000; Van Straalen and Roelofs, 2006).

Darwin adopted Herbert Spencer's (1864) term 'survival of the fittest' in 1866. His concept of fitness arose from his view of the organism and the environment it lives in. He did not actually use the term 'fitness', but referred to individual organisms as being more or less 'fit' than other individuals: different individual members of a species 'fit' into the environment to different degrees as a consequence of phenotypic variation, and those that make the best 'fit' survive and reproduce their kind better than those whose 'fit' is poorer (Ariew and Lewontin, 2004). In other words, fit organisms are better represented in future generations than their relatively unfit competitors (Stearns, 1976). Textbooks, monographs and articles show a wealth of diversity in fitness definitions (De Jong, 1994). Or as Ariew and Lewontin (2004) state: 'No concept in evolutionary biology has been more confusing and has produced such a rich philosophical literature as that of fitness.' Fitness concepts may refer to the functioning of an organism (fitness itself is a cause of natural selection), or may constitute a technical term in population biology summarizing numerical processes (fitness is a description of natural selection; De Jong, 1994). Within the numerical fitness concept, many quantities are proposed as fitness measures: life-history traits may include lifetime reproductive success, survival, viability, fecundity, mating success and age at maturity (Schluter *et al.*, 1991; De Jong, 1994). Theories on the evolution of life history

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focus on the notion that natural selection results in maximal fitness and prunes away less-optimal life histories (Brommer, 2000).

Life histories are shaped by the interaction of extrinsic factors, i.e. environmental impacts on survival and reproduction, and intrinsic factors, i.e. trade-offs among life-history traits and lineage-specific constrains on the expression of genetic variation (Stearns, 2000). Cole (1954) referred to the extrinsic factors when stating: 'It is obvious that the ability of the ancestors of existing species to replace themselves has been sufficient to overcome all environmental exigencies which have been encountered (…) through physiological, morphological, and behavioural adaptations that enable offspring to be produced and to survive in sufficient numbers to insure the persistence of a species.' Thus, in the absence of trade-offs, selection would drive all life-history traits to limits imposed by design and history, i.e. the body plan and the physiological limits posed by the phylogenetic history of the group to which the species belongs (Stearns, 1989; Van Straalen and Roelofs, 2006). From a population genetics point of view, given limits set by trade-offs and lineage-specific effects, survival and fertility of a species are optimized in such a way that population growth rate is at a maximum (Van Straalen and Roelofs, 2006).

Trade-offs depict the situation where the increase of one life-history trait imposes a cost to another, resulting in a negative correlation (Van Straalen and Roelofs, 2006). In a population genetics context, a trade-off is generated by either linkage disequilibrium (different loci influencing separate traits are situated closely together on the same chromosome, preventing the genes from segregating independently at meiosis) or pleiotropy (a single gene affects two or more different traits); trade-offs between life-history traits are more commonly assumed to be the result of the latter (Roff, 2007). Life-history trade-offs are often thought to be caused by the allocation of limited resources among competing traits such as reproduction, somatic growth and maintenance (Leroi, 2001; Roff, 2007). Trade-offs are extensively discussed in Chapter 3.

The idea of trade-offs resulting from energy allocation is very old and can be traced back to Saint Hilaire and Goethe, who pronounced at about the same time their law of compensation or balancement of growth (Darwin, 1872). As Goethe expressed it: 'the budget of nature is fixed; but she is free to dispose of particular sums by any appropriation that may please her. In order to spend on one side, she is forced to economize on the other side' (in Stauffer, 1975). In two volumes of *Philosophie anatomique*, Saint Hilaire identified the principle that all animals are formed of the same units of construction. According to his principle of connections, these units are fixed in number and always maintain the same position relative to each other. Since he argued that the budget of nature is fixed, he applied the principle of balance ('loi de balancement') to show that if one structure is enlarged, another one has to be reduced in order to maintain an exact equilibrium (Kliman, 1982; Mayr, 1983). 'The atrophy of one organ turns to the profit of another; and the reason why this cannot be otherwise is simple, it is because there is not an unlimited supply of the substance required for each part' (Geoffroy Saint Hilaire, 1818). Darwin (1872) agreed: 'I think this holds true to a certain extent with our domestic productions: if nourishment flows to one part or organ in excess, it rarely flows, at least in excess, to another part; thus it is difficult to get a cow to give much milk and to fatten readily.'

Rendel (1963) suggested that both the total amount of developmental resources available and their distribution may be under genetic control and variable. When availability of resources increases, both characters sharing the resources will be exaggerated; if resources are reduced, both will be retarded. This will result in a positive relationship in both cases. However, when the distribution is affected, increase in the share received by the first character must result in a decrease received by the second, generating a negative correlation (Rendel, 1963). Curvilinearity in the relationship between traits competing for resources was discussed by Sölkner and James (1994). At the phenotypic level, the relationship between two traits is often found to be curvilinear: various physiological limitations and feedback mechanisms will potentially produce deviations from linearity at the genotypic level as well. Sölkner and James (1994) presented a genetic model producing non-linearity in the relationship between two traits competing for resources, based on pleiotropy of loci responsible for acquisition and allocation of resources; this model was further developed by Fuerst-Waltl *et al.* (1997).

2. Resources

The Austrian physicist Ludwig Boltzmann (1844–1906), one of the main figures in the development of the atomic theory of matter, used his theories to explain the nature of living beings (Cercignani, 1998). 'Metabolism' is Greek for change or exchange. This exchange is important to enable us to perform activities. But the question is 'exchange of what'? Boltzmann reasoned that we exchange negative entropy for food, to keep our state well ordered, and this food in turn gets its negative entropy from the sun, through photosynthesis:

> The general struggle for existence of living beings is (…) not a fight for the elements – the elements of all organisms are available in abundance in air, and soil –, nor for energy, which is plentiful in the form of heat, unfortunately untransformably, in every body. Rather, it is a struggle for entropy that becomes available through the flow of energy from the hot Sun to the cold Earth. To make the fullest use of this energy, the plants spread out the immeasurable areas of their leaves and harness the Sun's energy by a process as yet unexplored (…). The products of this chemical kitchen are the subject of the struggle in the animal world.
>
> (cited in Cercignani, 1998)

These ideas were further explored by Schrödinger in 'What is life?' (Schrödinger, 1944): 'Thus the device by which an organism maintains itself stationary at a fairly high level of orderliness (= fairly low level of entropy) really consists of continually sucking orderliness from its environment.' According to Schrödinger, it was not energy that keeps us from death, but negative entropy. The biophysicist Morowitz (1968) proposed that the evolutionary process has been driven by the constant pumping of energy flows, mainly from the sun, and even went so far as to call evolution the 'necessary' result of our perpetual sunbath (cited in Corning and Kline, 1998).

Although the work of Schrödinger has inspired many, it has been critiqued by others. According to, for example, Corning (2002), more credit should be given to the energetics of living systems, which have developed highly efficient mechanisms for capturing energy available in various forms and then using it for various

purposes. It is adenosine triphosphate (ATP), not (negative) entropy, which provides much of the available energy that is utilized to build and operate living systems: it is more accurate to say that organisms feed upon available energy and create order than to say that they feed upon order. Available energy is used by living systems to pay for the purposeful biological work (chemical, active transport, mechanical, electrical and thermal work) that must be done to build, sustain and enhance biological structures (Corning and Kline, 1998). Zhuravlev and Avetisov (2006) gave an explicit definition of life, consisting of three parts. The first part was described as follows:

> Life, as we see it, is a specific state of matter (the living state) resulting from the interaction between matter and energy carriers. This interaction starts from the utilization of solar radiation by autotrophic organisms, and spreads over a diversity of organisms via numerous (bio)chemical cycles. A significant part of the utilized energy is retained in organisms by molecular carriers and 'network channels' of high-energy content; lessening of the utilized energy pool up to some critical level entails in death.

Thus, resources can be thought to include light, carbon, water and minerals for plants, or energy and specific nutrients for animals. Their intake is determined by 'foraging', while life-history patterns (survival, reproduction and growth) result from resource expenditure on fitness-related activities (Boggs, 1992). Weiner (1992) proposed the 'barrel model' of an organism's resource allocation pattern. Input constraints (foraging, digestion and absorption) are engaged in series, whereas outputs (maintenance, growth and production) are parallel and independently controlled (Fig. 1.1). If the sum of output rates does not match the input, the balance is buffered by the storage capacity of the system. In the long run, however, energy expenditure must balance energy intake (Weiner, 1992). Resources allocated to

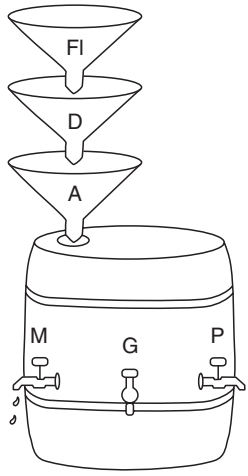

Fig. 1.1. The barrel model of an organism's energy balance (after Weiner, 1992). The first spigot always leaks (basal metabolic rate). FI = food intake, D = digestion, A = absorption, M = maintenance, G = growth, P = (re)production.

outputs are discussed in Chapter 7 (maintenance), Chapter 8 (growth), Chapter 10 (reproduction) and Chapter 11 (immune response).

When stable patterns of energy allocation are affected by changing environmental conditions, energy budgets may be affected in one of three different ways or combinations thereof: (i) the energy input into the system increases or decreases, but the relative pattern of energy allocation remains more or less unaffected, resulting in a proportional effect; (ii) both energy input and relative pattern of energy allocation are affected, resulting in a disproportional effect; and (iii) energy input into the system may or may not be affected, but metabolic energy is allocated preferentially to a selected function at the expense of other functions, resulting in a trade-off (Wieser, 1994). Resource allocation patterns are extensively discussed in Chapter 2.

One would assume trade-offs to be required only when the organism involved approaches a limit to its metabolic capacity. However, this is not necessarily the case (Wieser, 1994). One could think of metabolic sensors initiating the re-allocation of metabolic energy when the load of one process exceeds a critical threshold (Wieser, 1994). Activation of the immune system during infection, tissue injury and stress changes the priority of partitioning of nutrients from growth to host defence (Colditz, 2002). Homeorhesis represents 'the orchestrated or coordinated changes in metabolism of body tissues to support a physiological state' and was initially extensively described for the physiological state of lactation (Bauman and Currie, 1980). At the initiation of lactation, marked alterations in the partitioning of nutrients and metabolism of the whole animal occur to accommodate the demands of the mammary gland and to ensure that lactation proceeds successfully. In addition, the preference of other body tissues for nutrients is altered to allow partitioning of a greater percentage of glucose to the mammary gland (Bauman and Currie, 1980). Metabolic constraints to resource allocation are discussed in Chapter 4. Chapter 5 describes the process of homeorhesis under temperature stress.

3. Resources and Natural Selection

'But if variations useful to any organic being do occur, assuredly individuals thus characterised will have the best chance of being preserved in the struggle for life; and from the strong principle of inheritance they will tend to produce offspring similarly characterised. This principle of preservation, I have called, for the sake of brevity, Natural Selection' (Darwin, 1872). 'Selecting' can be defined as 'to take as a choice from among several', which implies some sort of active action. But nature is not in power to actively select; rather, it deals with those individuals that manage to survive and reproduce because of 'favourable genes' producing 'favourable phenotypes' best suited to survive in a given environment.

> A struggle for existence inevitably follows from the high rate at which all organic beings tend to increase. Every being, which during its natural lifetime produces several eggs or seeds, must suffer destruction during some period of its life, and during some season or occasional year, otherwise, on the principle of geometrical increase, its numbers would quickly become so inordinately great that no country could support the product. Hence, as more individuals are produced than can possibly survive, there must in every case be a struggle for existence. (…) All that we

> can do, is to keep steadily in mind that each organic being is striving to increase at a geometrical ratio; that each at some period of its life, during some season of the year, during each generation or at intervals, has to struggle for life, and to suffer great destruction. When we reflect on this struggle, we may console ourselves with the full belief, that the war of nature is not incessant, that no fear is felt, that death is generally prompt, and that the vigorous, the healthy, and the happy survive and multiply.
>
> (Darwin, 1872)

Thus, Darwin saw natural selection by the environment as responsible for evolutionary and other genetic changes through the resources and challenges that each environment presents (Beilharz, 1998). Does natural selection tend to 'maximize' for any particular value or objective and is there any discoverable trend or general direction to the process (Corning and Kline, 1998)?

3.1 Optimization of resource uptake

Since life forms depend on energy as well as material capture and flow, their evolution does so as well. The initial stages of life and the subsequent process of evolution in response to changes in the chemical environment are wonderfully described by Williams and Fraústo da Silva (2003). Their work explains how changes in life forms resulted from the forms in which energy was available over the period of evolution, how energy was usefully transformed by these life forms and the ways in which the energy sources changed. They suggest that the drive to the most effective set of organisms is such that, together with the environment, it generates an optimal total uptake of energy. In this view, the drive behind evolution is the optimal possible retention of free energy (Williams and Fraústo da Silva, 2003).

These views are in line with those of Alfred Lotka, who in 1922 referred to Boltzmann pointing out that 'in the struggle for existence, the advantage must go to those organisms whose energy-capturing devices are most efficient in directing available energy into channels favourable to the preservation of the species'. Natural selection tends to maximize the energy flux through the system compatible with the constraints, i.e. increases the rate of turnover of the organic matter through the life cycle. When the energy resources that are available 'at the disposal of the organisms for application to their life tasks and contests' increase, 'then an opportunity is furnished for suitably constituted organisms to enlarge the total energy flux through the system'. In the situation that energy resources become restricted, 'the advantage will go to that organism which is most efficient, most economical, in applying to preservative uses such energy as it captures'.

Corning (2002) referred to 'thermoeconomic principles' characterizing living systems as seeking to increase access to energy sources, and increasing the efficiency of energy transformation processes (Raine *et al.*, 2006): 'It is the organized use of available energy in informed structures that constitutes the centre ring in the circus of life' (Corning and Kline, 1998). Brown *et al.* (1993) applied the principle to redefine fitness as 'the rate that resources, in excess of those required for growth and maintenance of the individual, can be harvested from the environment and used for reproduction', i.e. the rate of conversion of energy into useful work for reproduction.

These views were challenged by Illius *et al.* (2002). The ultimate goal of an organism is to maximize its inclusive fitness, and since survival, growth and reproduction must be met by nutrient demands, an important sub-goal must be the optimization of the lifetime pattern of food intake: an animal that can forage more effectively than others will be at an advantage in allocating acquired resources to survival and reproduction (Illius *et al.*, 2002). Such theory suggests that animals will maximize food intake subject to physical and physiological (e.g. digestive) constraints. However, in variable environments, animals are restricted only intermittently, under extreme conditions. For example, Johnson and Speakman (2001) showed that mice experiencing cold stress (at 8°C) were able to increase their (non-reproductive, 21°C) food intake 53%, lactating mice increased their food intake 353% and lactating mice experiencing cold stress increased their food intake 488%. It seems therefore impossible to argue that food intake is constrained by the digestive capacity when they are able to expand that capacity to accommodate vastly greater energy requirements (Illius *et al.*, 2002). Illius *et al.* (2002) argue that there are sound theoretical reasons why animals should have evolved mechanisms to control food intake at sub-maximal quantities as fitness benefits are rather unlikely to be a linear function of food intake, i.e. there are costs as well as benefits to food intake. The costs to food intake are both extrinsic costs associated with the activity of foraging (such as less sleep and rest, reduced anti-predator vigilance and reduced time for territorial defence) and costs intrinsic to food intake itself (accumulation of cellular damage, exposure to parasites and ingestion of toxins). Instead, Illius *et al.* (2002) argue that fitness is maximized by balancing the costs of food intake with the lifetime schedule of benefits (survival, growth and reproduction).

3.2 Optimization of resource allocation: biological structure

Darwin (1872) wrote: 'If under changed conditions of life a structure, before useful, becomes less useful, its diminution will be favoured, for it will profit the individual not to have its nutriment wasted in building up a useless structure.' In 1881, Wilhelm Roux referred to the competition among organs and organisms as the *struggle of parts*: organs, tissues and cells are found in an unceasing struggle for existence with one another for food, space and the utilization of external stimulation (Roux, 1881; Mayr, 1983; Moore, 2002). Only those parts that are better adapted to 'the obtaining condition of existence' can survive, producing the most efficient structure (Moore, 2002): 'Everything that is not capable of surviving disappears; what remains is that which can survive' (cited in Oppenheimer, 1964). The struggle between the parts of an organism was thought to be at least as important as the struggle between organisms. Roux believed that the struggle between the parts of an organism would be modified by a change in the nutritive regime mediated by changes in the environment (Gayon, 1999). The 19th-century German philosopher Friedrich Nietze believed that this 'internal adaptation', resulting from the competition for nutrition over the course of life, was much more important than Darwin's adaptation to the external environment:

> The influence of 'external circumstances' is overestimated by Darwin to a ridiculous extent: the essential thing in the life process is precisely the tremendous shaping, form-creating force working from within (…) The new forms molded from within are not formed with an end in view; but in the struggle of the parts a new form is not left long without being related to a partial usefulness and then, according to its use, develops itself more and more completely.
>
> (Cited by Gayon, 1999)

An example of the struggle between the parts is given by Ariello and Wheeler (1995): only animals with cheap guts can afford expensive, large brains. To fuel the enlarged human brain and still maintain a normal basic metabolic rate, another expensive metabolic tissue must be reduced: the increase in brain size is balanced by an equivalent reduction in the size of the gastrointestinal tract (Arriello and Wheeler, 1995; Ofek, 2001). Emlen (2001) showed that beetle horns grow when resources are limiting, resulting in reduced sizes of nearby, simultaneously growing morphological structures (antennae, eyes and wings), and suggests that similar trade-offs are likely for the multitude of exaggerated insect structures.

One hundred years after Roux, Taylor and Weibel proposed the principle of optimal biological design or symmorphosis, based on 'the firm belief that animals are built reasonably' (Taylor and Weibel, 1981; Bacigalupe and Bozinovic, 2002). Symmorphosis suggests that natural selection favours an economic use of resources by all parts of a biological structure, such that the parts are qualitatively and quantitatively coadjusted to their common role: never used excess in biological capacity in any of the parts of a biological structure is costly in terms of maintenance, materials and space and would therefore not be favoured by nature (Taylor and Weibel, 1981). This hypothesis is supported by data on the mammalian respiratory system, in which oxygen flow, from blood circulation to mitochondrial oxidative phosphorylation, attains similar maximum rates: no single bottleneck exists (Weibel *et al.*, 1981). An important prediction of the principle is that if functional needs change, then structural components must change accordingly (Bacigalupe and Bozinovic, 2002). It is mainly evolutionary biologists who have contested that evolution by natural selection can lead to 'optimal' rather than merely 'adequate, sufficient' design. Valid arguments are that 'organisms are not designed', 'natural selection has no final goals and purposes', 'constraints and trade-offs are pervasive in biological systems', 'selection is constrained to work with pre-existing materials (inherited alleles) and these may not be the best possible materials for a particular function' and 'changes produced by migration and genetic drift may not be adaptive' (Garland, 1998). Indeed, evidence in favour of symmorphosis is as abundant as the evidence against it (Bacigalupe and Bozinovic, 2002). Particular structures (e.g. entire organ systems) often serve multiple functions, making it unlikely that optimization could be achieved for each of them (Weiner, 1992). The hypothesis holds mostly for internal characteristics of the body (such as the internal compartments of the respiratory system), whereas most problems arise in organs at the interface to the environment (such as the lung): these organs must adapt both to the needs and constraints of the internal environment and deal with the pressures and resistance of the variable and unpredictable external environment (Weibel, 1998). In addition, excess capacities should indicate

a suboptimal design and, indeed, organisms often possess capacities somewhat in excess of what they normally use. Such buffer capacities provide environmental flexibility and allow individuals to withstand short-term stresses and adapt to long-term changes in the environment (Hammond *et al.*, 1994). Therefore, adaptation to environmental stresses will never reach a state of perfect optimality, but may be considered as a 'process of becoming', resulting in organisms 'designed' the best that they could be (Garland, 1998).

3.3 Optimization of resource allocation: fitness components

'Every single organic being around us may be said to be striving to the utmost to increase in numbers'; thus, fitness is always under selection upwards (Darwin, 1872). Fitness is composed of several components, such as 'number of parities', 'litter size' and 'survival of progeny', and these components are related multiplicatively. Each component requires metabolic resources for functioning, which are related additively, as resources used for one function are no longer available for any other function. These two sets of rules form the basis of the Resource Allocation Theory developed by Beilharz (Beilharz *et al.*, 1993).

> We behold the face of nature bright with gladness, we often see superabundance of food; we do not see, or we forget, that the birds which are idly singing round us mostly live on insects or seeds, and are thus constantly destroying life; or we forget how largely these songsters, or their eggs, or their nestlings are destroyed by birds and beasts of prey; we do not always bear in mind, that though food may be now superabundant, it is not so at all seasons of each recurring year.
>
> (Darwin, 1872)

Beilharz *et al.* (1993) suggest that, since fitness has continuously been under selection for higher values, it is to be expected that, in general, each species is now exploiting its niche as well as possible, implying that all wild animal species that have adapted to their environment are being limited in their fitness by the environmental resources available in their niches. The processes of the life history (maintenance, growth and reproduction) compete for these limited resources that are available for each individual (consisting of the food it has ingested, the body stores of fat and protein it has assimilated in the past, and its physiological state; Gadgil and Bossert, 1970; Dunnington, 1990). Life-history strategy is a matter of optimal allocation of resources among maintenance, growth and reproduction. Devoting resources to maintenance and growth enhances the reproduction at further stages in the life history. Maintenance is essential to enable an organism to survive to these stages. Growth may enhance both survival and reproductive ability (Gadgil and Bossert, 1970).

Since animals with highest fitness are those that were able to utilize available environmental resources most efficiently, an increase in the amount of resources resulting from a favourable environment (e.g. season) will be used by the organism to raise fitness, while there is additive genetic variation available for any of the fitness components: 'organisms respond to natural selection until fitness can improve no more. That is the point at which organisms utilize all available resources of the

environment most efficiently' (Beilharz, 1998). The environmental resources available determine the phenotype that can be sustained most efficiently, and therefore the genotypes that are selected on the basis of such phenotypes (Beilharz *et al.*, 1993). As a result, different genotypes have been favoured in different environments (e.g. *Bos indicus* cattle in the tropics and *Bos taurus* cattle in Europe; Beilharz and Nitter, 1998). It is the environment that prevents or allows evolutionary changes to occur (Beilharz, 1998).

Because the components of fitness are related multiplicatively, but the uses of metabolic resources by these and other processes are related additively, continuing selection for high fitness by natural selection will lead eventually to intermediate optimal values for the fitness components, giving the highest product of fitness. This is explained as follows: 'Find the maximal product of two positive numbers constrained to add to a total that is no greater than 1. The answer is 0.25, when each of the numbers is 0.5. Moving either number away from 0.5 in either direction lowers the product' (Beilharz *et al.*, 1993). Intermediate optimal values maintain additive variance. This follows Lerner's (1954) discussion on 'genetic homeostasis': the methods by which homeostasis is maintained are heterozygosity, stabilized selection and negative genetic correlations between traits which will result in intermediate optima for many characteristics. Lerner differentiated between 'developmental homeostasis', i.e. the greater ability of the heterozygote to stay within the norms of canalized development, and 'genetic homeostasis', i.e. natural selection favouring intermediate rather than extreme phenotypes. In 1944, Jones proposed the term 'genetic equilibrium' and Darlington and Mather (1949) used the term 'genetic inertia' to include somewhat the same idea: natural selection results in intermediate optima for many traits (e.g. body weight, egg weight and immune response) and in maintenance of heterozygosity, which imparts a buffering capacity to a wide range of environmental conditions (Dunnington, 1990; Siegel and Dunnington, 1997).

The importance of short-term flexibility to fitness, i.e. the short-term ability of an organism to adapt to a changing environment, was emphasized by Holliday (1989). In many mammals that live in variable environments (e.g. with severe winters or dry seasons), the breeding cycle is determined by the variability of the food supply, since successful reproduction can only occur when food is plentiful (Holliday, 2006). During famine, it is less worthwhile to invest energy in progeny that are unlikely to survive to maturity, and energy is predicted to be directed towards extra maintenance and away from reproduction (Shanley and Kirkwood, 2000). In these species, calorie restriction has the effect of increasing longevity: the animal survives and its body is maintained during food shortage. When food becomes again available, they begin to breed again. Holliday (1989) postulated that those animals with genomes that respond to food shortage by diverting energy from reproduction to maintenance are better able to survive until food again becomes abundant. Thus, animals with such genomes would be favoured by natural selection: it is the unpredictable periods of food shortage that drive evolution. Similarly, McNamara and Buchanan (2005) hypothesized that under natural selection, the processes that allow redistribution of physiological resources should distribute resources optimally to maximize fitness, and Parsons (2007) indicated that it is appropriate to say that an energy utilization process directed

towards high energetic, and hence metabolic, efficiency under resource limitations is a fundamental evolutionary expectation.

4. Artificial Selection

Genes provide the instructions controlling the development, maintenance and reproduction of organisms, but this genetic potential can only be realized in an environment in which essential resources are adequately supplied (Beilharz, 1998). Therefore, genetic changes in farm animals with artificial selection for improved levels of production must be supported by increased amounts of resources that allow expression of this improved potential, otherwise, genetic changes cannot result in sustainable levels of growth, milk production or reproduction (Beilharz, 1998). In the same way that the environmental resources available determine the phenotypes that can be sustained most efficiently, different genotypes demand different environmental levels for full expression of their potential. In environments with resource levels that are lower than required for expressing the maximal potential, resource-demanding processes must show trade-offs resulting from the environmental limitation. Environmental insufficiency will result in those animals having a *lower* performance than that of animals that have a lower genetic potential, but which is supported by amounts of resources that are sufficient to support this lower potential (Beilharz and Nitter, 1998).

The result is a typical genotype × environment (G × E) interaction. An example is given by Luiting *et al.* (1995): the genetic correlation between production and feed efficiency changed from zero on a regular diet to negative on a limited protein diet. Also, in broilers, the correlation between 'actual growth rate' and 'potential growth rate', which was positive under normal temperatures, reversed to a significantly negative correlation ($r = -0.411$) during a heat-stress period, indicating that breeding values of families under heat stress have little association with their expected breeding values under normal conditions (Deeb *et al.*, 2002). This was recognized by Falconer (1952): 'Performance in a favourable environment has a different genetic basis from performance in an unfavourable environment: a superior genotype in one environment could not be expected to be superior in a different environment.'

Domestication has resulted in an increase in the resources available to animals because it has removed several resource-demanding situations, such as the need to search for food, to be wary of, or fight off, predators, to endure periods of food shortage, and to compete in sexual competition (Beilharz *et al.*, 1993). In addition, livestock feeds are designed to be more nutrient-dense and require less work than natural forage (Illius *et al.*, 2002; Olsson *et al.*, 2006). This additional amount of resources could thus be invested into production, resulting, together with efficient breeding programmes, in an unprecedented increase in production levels (Rauw *et al.*, 1998). However, it can be assumed that farm animals are again limited by their environment. When farm environments are being improved, selection can improve economic traits without penalties until the environment again becomes limiting (Beilharz, 1998).

Under natural selection, individuals need to be ready at any time to allocate their resources among the demands of maintenance, growth and reproduction, with some amount held in reserve as a buffer. However, in animals that origin from a population that is selected for high production, the weighing given to some other components will be increased. During the domestication process, the relative importance of ability to reproduce in captivity or tameness increased. Subsequently, with active selection, the weighting given to production increased (Rauw *et al.*, 1998; Mignon-Grasteau *et al.*, 2005). Preferential allocation of resources may occur because the animal is 'programmed' to allocate a disproportionally large portion of its resources to a particular one of these demands, leaving it lacking in ability to respond to other demands, such as coping with disease and stress (Siegel and Dunnington, 1997; Rauw *et al.*, 1998). Animals that are genetically driven to produce at high levels may reallocate resources away from other processes, where buffer capacities and traits not defined in the breeding goal may be the first to be affected, because their importance is not specifically recognized (Rauw *et al.* 1998, 1999). In addition, historically, it has been necessary to make changes slowly, giving the whole animal time to equilibrate to both direct and correlated responses. If genetic changes are too radical or sought too rapidly (e.g. with introgression of desirable genes into available populations), new genes may disrupt resource allocation that has evolved gradually, and the homeostatic balance may be at risk (Dunnington, 1990).

Moberg (2000) developed a model of animal stress describing the importance of the cost of the stress to animal welfare. When the cost of coping with the stressor diverts resources away from other biological functions, such as growth, reproduction or the immune function, the animal experiences distress, which places it in a prepathological state and increases its vulnerability to a number of pathologies. Similarly, McNamara and Buchanan (2005) indicate that a reduction in the physiological state resulting from the inability to maintain optimal physiological condition during periods of environmental stress potentially includes reduced physiological reserves of essential vitamins, minerals and amino acids, reduced body condition and immunocompetence, and reduced fat reserves, leading to increased mortality and decreased reproductive potential.

Rauw *et al.* (1998) and Rauw (2007) reviewed the literature on undesirable correlated effects of selection for high production efficiency in broilers, pigs and dairy cattle, and showed that the highly favourable increase in production levels is indeed often compromised by behavioural, physiological and immunological problems. The most striking examples of undesirable correlated responses were reported in broiler chickens with an increasing incidence of heart failure syndrome and leg problems. In poultry, selection has been almost for one trait only, i.e. body weight at a certain age, and selection intensity has been high with a short generation interval. In cattle and pigs, selection has been less intensive, for more traits and during fewer generations, resulting in more controversial results (Rauw *et al.*, 1998). However, several obviously undesirable trends between production, and fertility and health were found. Chapters 12–15 discuss side effects of selection for high production efficiency in pigs, poultry, cattle and implications of biological engineering, respectively.

5. Resource Allocation Quantified

5.1 Residual feed intake and estimated grazing intake

Resource allocation according to Beilharz (1998) can be summarized with the following equation:

$$R_i = (k_A \times A_i) + (k_B \times B_i) + (k_C \times C_i) + \sum (k_Q \times Q_i), \quad (1.1)$$

where R = total amount of resources available to individual i, k = resource conversion factor, $(k_A \times A_i)$ = resources used for maintenance, $(k_B \times B_i)$ = resources used for growth, $(k_C \times C_i)$ = resources used for production and $\Sigma(k_Q \times Q_i)$ = resources used for other processes (Beilharz, 1998). This equation is very similar to the one that is used for the calculation of residual feed intake, which is estimated from a linear regression of feed intake on metabolic body weight, growth and (re)production:

$$FI_i = b_0 + (b_1 \times BW_i^{0.75}) + (b_2 \times BWG_i) + (b_3 \times PROD_i) + e_i \quad (1.2)$$

where FI_i = feed intake of individual i, $BW_i^{0.75}$ = metabolic body weight, BWG_i = body weight gain, $PROD_i$ = level of production (kilograms of milk or wool, number of eggs, etc.), b_0 = population intercept, b_1, b_2 and b_3 = partial regression coefficients representing maintenance requirements, feed requirements for growth and feed requirements for production, respectively, and e_i = the error term, representing residual feed intake. Regression coefficients represent the average 'cost' of body maintenance, growth and production, based on the population on which the model is formed. Individual deviations from this average accumulate in the error term, which is unique for each individual. Residual feed intake is thus defined as the part of the feed intake that is unaccounted for by average feed requirements for maintenance and production. Apart from variation in partial efficiencies for maintenance, growth and production, variation in residual feed intake can be explained by variation in metabolic food demanding processes not included in the model, such as behavioural activities, responses to pathogens and responses to stress (Luiting, 1990). The similarity between these models implies that calculation of residual feed intake can be used to quantify the amount of 'buffer' resources available to an animal, for example, physical activity and the ability to cope with unexpected stresses. Residual feed intake is extensively described in Chapter 6.

Application of residual feed intake estimation to deal with resource allocation matters is not restricted to intensive production systems only. Rauw *et al.* (2006) proposed a model for estimating grazing intake in extensive free-range conditions by rewriting Equation 1.2:

$$EGI_i = FI_i - b_0 - e_i = (b_1 \times BW_i^{0.75}) + (b_2 \times BWG_i) + (b_3 \times PROD_i) \quad (1.3)$$

where EGI_i = estimated grazing intake of individual i, FI_i = feed intake of individual i and other parameters are as in Equation 1.2. Equation 1.3 shows that the amount of resources ingested is confounded with the efficiency of resources

allocated. Preliminary results on a grazing experiment in sheep in the cold Nevada desert showed that 94% of 915 pregnant ewes lost body weight during the grazing period, while pregnant animals in particular must gain weight (Rauw *et al.*, 2006). Therefore, in this situation, it is more important for the rancher to know if the animal has been able to ingest a sufficient amount of resources than if the animal is more or less efficient in allocating those. Since feed intake and the partial regression coefficients cannot be estimated in the field, estimates from literature can be used, or better, estimates from controlled experiments on a subgroup originating from the animal population of interest (Rauw *et al.*, 2006). With these estimates, feed intakes do not need to be estimated: body weights can be estimated before and after animals are allowed to range freely on the rangelands, and metabolic body weight and body weight gain can be calculated. When grazing ability is expressed in metabolizable energy consumed units, comparison of grazing efficiency can be made not only for animals sharing a given environment, but also for animals living in different environments or even for different species. The methods can be used to evaluate the grazing potential or the load of a flock on a given ecosystem.

In the context of resource allocation, EGI presents an estimate of the individual ability to graze at resource-limiting rangelands and the ability to retrieve nutrients from nutrient-limiting environments. For example, sheep have a greater ability to selectively harvest leaves and current annual plants than do cattle or horses (Hanley and Hanley, 1982). Also within species there is variation in the ability to graze selectively. Selection for within-species variation in grazing ability may offer the opportunity to breed for range animals that are better adapted to poor-quality rangelands, resulting in healthier animals and improved production. Several important consequences of undernutrition during the grazing period in sheep have been reviewed by Dwyer *et al.* (2003): maternal nutrition in pregnancy affects both lamb birth weight and the incidence of lamb mortality. Maternal undernutrition is associated with a reduction in udder weight and mammary development, resulting in a reduced colostrum yield and total milk production, and with a delayed onset of lactation and a lower milk secretion rate (Dwyer *et al.*, 2003). From their own study they concluded that even a moderate level of undernutrition impairs the attachment between ewes and lambs by affecting maternal behaviours expressed at birth. In addition, their results suggest that levels of nutrition resulting in a decrease in birth weight will affect neonatal lamb behavioural progress (Dwyer *et al.*, 2003). Therefore, selection for improved EGI in nutrient poor environments would foremost result in healthier animals that can produce offspring without compromising welfare of their own and that of their offspring.

5.2 Genetic size-scaling

When animals of different mature sizes are compared at the same chronological age, a considerable part of the variation in feed intake can be explained by comparison at different stages of physiological development. After adjusting for the environmental variation (e.g. by comparing animals in the same environment), this part of the variation in feed intake can be adjusted for by scaling the chronological

timescale according to the genetic size-scaling rules developed by Taylor (1985; Luiting, 1999). The genetic variance that results can be further separated into variation due to genetic size and a size-independent genotype-specific variance.

Most of the responses in feed intake have been achieved by changes in mature size; differences in specific genetic factors become statistically significant only in the long term, although they generally remain small compared with the differences resulting from the genetic size factor. Luiting (1999) gives two explanations: (i) very small differences in specific genetic factors accumulate over generations and become detectable and statistically significant; and (ii) a further increase in genetic size may be restricted by limiting environmental resources. A selection-induced change in specific genetic factors may thus be an indication for a limiting resource situation (Luiting, 1999). Indeed, in the short-term, increased levels of production are generally accompanied by increased feed intakes. In the longer term, the genetic correlations between production traits and feed intake decrease, and those between production and feed efficiency increase. Limiting resource situations will be reached sooner when selection is for high production efficiency, i.e. high production combined with low feed intakes (Luiting, 1999). We can thus use the techniques of standardized curves to deal with resource allocation matters (Luiting, 1999). Genetic size-scaling is extensively described in Chapter 9.

6. Implications

Undesirable effects of increased production efficiency have raised questions about what is ethically acceptable in animal breeding. A question similar to that asked by Corning and Kline (1998) regarding natural selection can be asked with respect to artificial selection: when we speak of a 'breeding goal' do we actually have any particular value in mind, or is the goal simply 'more'? The answer is given by Groen in Chapter 16: 'Genetic improvement is not aiming at an optimum; genetic improvement is dynamically searching for improvements. Given animal genetic variation (within or between breeds), there is always a means of improvement.' The key ethical question is not whether animal breeding should be abandoned, but how we should breed (Gamborg and Sandøe, 2003). The general opinion is that it is acceptable to use animals as long as it is done 'humanely' and does not result in physical damage, pain or distress (Christiansen and Sandøe, 2000).

Animal well-being and welfare can be *improved* by breeding as well. Increased emphasis on welfare-associated traits in the selection index, such as longevity and health, will result in improved animal welfare and increased public confidence in animal farming (Pryce *et al.*, 1999; Kerr *et al.*, 2001; Stott *et al.*, 2005). Breeding companies can play an important role in addressing welfare problems by defining broader breeding goals that include not only production traits but also functional traits and non-economic values, such as emotional and societal values (Kanis *et al.*, 2005; Olsson *et al.*, 2006). Every trait that matters must be included in the breeding objective, which necessarily means that improvement must go more slowly in each of the many traits in order to achieve progress towards the total goal (Beilharz and Nitter, 1998; Oltenacu and Algers, 2005). Kanis *et al.* (2005) proposed a

selection-index method to obtain the proper weights for societally important traits in the breeding goal, such as welfare and health. It will become clear from Chapters 12 to 14 that breeding programmes are more frequently including functional traits in the breeding goal, and that this is successful. Chapters 16–18 discuss breeding goals, robustness traits and resource allocation models, respectively, as methods to improve the breeding objective.

Falconer (1952) suggested that '[i]t would therefore generally be recommended that selection should be carried out under the environmental conditions in which the improved breed is destined to live'. He also suggested that performance in two different environments (such as on a low versus a high plane of nutrition, or temperate versus tropical climates) can be regarded as two different characters that are genetically correlated. Thus, selection for the character in one environment will bring about a correlated response in the trait in the other environment. But an advantage of selection in the secondary environment would accrue only an advantage through an increased heritability, and this should be great enough to offset the loss of efficiency through selection being made for a character that has not exactly the same genetic basis as the desired character (Falconer, 1952). According to the Resource Allocation Theory, using highly productive animals from populations that were selected in favourable environments, in poorer environments is very likely to cause problems with fitness (Beilharz and Nitter, 1998).

Questions are being asked on the future direction of agriculture in several countries, with special emphasis on the question as to how the agricultural sector can find sustainable ways of being more productive (Garnier *et al.*, 2003; Oltenacu and Algers, 2005). MacArthur Clark *et al.* (2006) recommend the establishment of a committee for the evaluation of welfare problems associated with breeding technologies that would advise on the effectiveness of existing legislations and practices relating to animal breeding procedures to assure animal welfare, and would give consideration to ethical questions associated with animal breeding even where measurable detrimental effects on animal welfare may not be immediately evident. We may expect that increased and combined efforts may result in better animal welfare in the future.

7. This Book

This chapter has shown that many theories on natural selection and evolution have something to do with food resources: acquisition, utilization and allocation. Furthermore, these theories and thoughts date back (at least) to the mid-19th century and have been developed by people from different disciplines. Animal breeders, however, have concentrated mainly on animal genotypes, i.e. the environmental variation is statistically taken out of the equation. The reason for this is obvious: genotypic variation is passed on to the next generation through inheritance, i.e. can be selected for, whereas entire environmental variation cannot. Furthermore, animal genotypes can be bought and sold, whereas environments cannot. Application of animal breeding models that are based on genotypic information, such as Best Linear Unbiased Prediction (BLUP), has been (and is) clearly very successful. However, undesirable side effects of selection for high production efficiency are

becoming apparent. These side effects may result from a limiting resource situation, where improved levels of production with artificial selection are no longer supported by sufficient resource intake.

A Spanish tale goes as follows: A farmer was teaching his donkey not to eat. Little by little, every day, he gave the donkey a little less food to eat. The donkey would eat less and proceed to work. Time passed, and the farmer enjoyed greater benefits, because the donkey would do the same work, but every day it would cost less to maintain him. The farmer was very happy. But he was so very unfortunate that just when he was about to accomplish that the donkey learned to live without food, it died. The very extreme example of food efficiency given in this tale, resulting eventually in the death of the animal, seems little surprising. But also in the case of less extreme food efficiencies and increased production levels it should be intuitively acceptable that the first law of thermodynamics holds, i.e. the law of conservation of energy, which states that energy cannot be created or destroyed, but can only be changed from one form to another: output (production) requires input (food).

The 'Resource Allocation Theory' was first proposed by Goddard and Beilharz (1977) as a congress contribution and first published by Beilharz *et al.* in 1993. Since then, more and more authors have applied resource allocation theory to livestock production (e.g. Rauw *et al.*, 1998, 1999; Luiting, 1999; Schütz and Jensen, 2001; Knol, 2002; Mignon-Grasteau *et al.*, 2005; Oltenacu and Algers, 2005) and developed models incorporating the theory (e.g. Van der Waaij, 2004; Chapter 18). Without an understanding of the underlying physiological processes on which genetic selection acts, cumulative and permanent genetic improvement through selection is essentially a black box technique. Genetic increase in a biological system that is not well understood is likely to lead to unfavourable and improperly understood side effects. An understanding of the biological background will offer the opportunity to understand, anticipate and prevent negative side effects of selection.

It is the intention of this book to present the reader with information on resource allocation matters with the aim to further development thoughts and models on resource allocation applied to livestock production. Due to necessary space limitations, although extensive, the information and views presented in this book are by far exhaustive. Resource allocation is presented in four parts:

Part I: 'Resource Allocation' (Chapters 1–5) describes resources and resource allocation patterns, trade-offs, metabolic constraints to resource allocation and the process of homeorhesis with a special emphasis to homeorhesis during heat stress.

Part II: 'Inputs and Outputs' (Chapters 6–11) describes the relationship between food intake and resources allocated to body maintenance, growth, reproduction and the immune response.

Part III: 'Consequences of Selection for Increased Production Efficiency' (Chapters 12–15) discusses consequences of high production efficiency in pigs, poultry and dairy cattle, and the consequences of improved production by means of biological engineering.

Part IV: 'Animal Breeding and Resource Modelling' (Chapters 16–18) discusses options to include resource allocation matters in the breeding objective and in resource allocation modelling.

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2 Resource Allocation Patterns

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Two major processes vital to all of life are the acquisition and use of resources, and the acquisition and use of information. Both the essence of life and its evolution are critically linked to the nexus of interactions that occur between these fundamental processes. Organisms can be considered 'informed resource users' that perpetuate themselves by reproduction. And today's organisms are those that have evolved the most 'informed' systems of resource acquisition and use, as a result of differential reproduction (i.e. natural selection). Those organisms whose genetic and neuroendocrine information is well suited (i.e. adapted) for directing the transformation of resources into new living substance, in the context of specific environments, have replaced those that were not so well-informed. Since natural environments change both in time and space, organisms have responded by evolving varied systems of information-controlled resource management to meet specific environmental challenges.

1. Resources: What Are They and How Are They Used?

Energy, nutrients and time are the most important resources considered by scientists interested in improving farm animal production. Other kinds of resources often considered by ecologists, such as mates and space (e.g. territories and nesting sites), are important in other contexts, but are not discussed here (see Wiens, 1984; Begon *et al.*, 2006).

1.1 Resource acquisition, allocation and association

This book focuses on resource *allocation*, but two other processes – resource acquisition and resource association – are also important for understanding resource use in animals. Resource allocation involves the partitioning of available energy and

materials into various vital activities or structures. Classical life-history theory was founded on the 'Principle of Allocation' (Cody, 1966; Sibly and Calow, 1986), which has been recognized for centuries, including by Aristotle, as embodied in his idea of 'physiological limitation' (Egerton, 1973), by Goethe and Geoffroy Saint-Hilaire, as represented by their 'law of compensation' or 'balancement of growth' (Darwin, 1899), by Rubner (1910) who stated that it 'is not possible for all organs to be simultaneously in a state of high activity', by Huxley (1932) who discussed 'competition for food (…) in the regulation of relative size of parts', and by Rensch (1959) in his review of the evolutionary significance of 'material compensation'. Given limited resources, if an organism increases its use of resources for one activity or structure, it must proportionately decrease its use of resources for other activities and structures. Trade-offs among reproduction, growth, survival and other life-history traits have often been explained as being the result of resource allocation.

However, resource *acquisition* also plays an important role in determining life-history patterns. Lack (1954) was one of the first ecologists to emphasize this 'RA'. For example, he explained the latitudinal increase in clutch size in some birds as being the result of longer summer days in higher latitudes permitting the feeding of more offspring than was possible at lower latitudes. However, the general importance of resource acquisition was underappreciated until Van Noordwijk and De Jong (1986) developed a model that showed how variation in resource acquisition may explain why trade-offs predicted by resource allocation theory are often not observed (see also Roff and Fairbairn, 2007). At specific average levels of food availability and allocation, their model predicts that if individual variation in resource acquisition exceeds that of resource allocation, then positive correlations should likely occur between life-history traits (e.g. reproduction and somatic growth or survival). By contrast, negative correlations between traits (i.e. trade-offs) should likely occur if individual variation in resource acquisition is less than that of resource allocation. This model potentially has wide applicability, though trade-offs between life-history traits may or may not occur for other reasons (Glazier, 1999, Chapter 3, this volume). In any case, resource acquisition and allocation are interdependent in other fundamental ways as well. For example, allocation of resources (including time, energy and materials) to foraging and food processing affects resource acquisition (Pianka, 1976; Ricklefs, 1991; Boggs, 1992, 1994). Conversely, the level of resource acquisition may affect the pattern of resource allocation (Glazier and Calow, 1992; Glazier, 1999; Niewiarowski, 2001).

Another aspect of resource use that has received even less attention by ecologists is what I term resource *association* (D.S. Glazier, 2002, unpublished data). Ecologists typically assume that, based on the laws of thermodynamics, organisms cannot use the same energy for more than one function or structure, but in fact, physiological systems often 'violate' this assumption. For example, energy used to support digestion, locomotion and production may be used again (as heat) for thermoregulation in various kinds of birds and mammals, a phenomenon known as 'compensation' (Rubner, 1910; Wieser, 1989; Krockenberger, 2003) or 'substitution' (Hart, 1971; Bruinzeel and Piersma, 1998; Kaseloo and Lovvorn, 2006). Although the effects of substitution or 'energy sharing' vary among species and environmental conditions, resource association may lead to significant energy savings. As pointed out in a study of ducks that dive in cold water, ignoring thermal substitution 'may substantially overestimate total energy costs' (Kaseloo and Lovvorn, 2006). A recent model

also shows how thermal substitution may affect the optimal amount of body-energy reserves that birds should accumulate during the winter (McNamara *et al.*, 2004).

To summarize, resource allocation theory is typically based on the concept of a 'zero-sum game'. In its simplest form, this theory makes two fundamental assumptions: (i) the organisms being compared have the same amount of finite resources available to them (i.e. the same total 'sum'); and (ii) resources expended for one function cannot be used for another function (i.e. adding all of the resources gained by various functions and subtracting the total resources thereby lost to other functions sums up to 'zero'; in other words, the costs of various functions are additive and completely non-overlapping). However, these assumptions are not always true. The first assumption is frequently violated whenever resource availability and (or) the ability to acquire resources varies among organisms. The second assumption is violated whenever resources used for one function are also used for another. As a result, resource association and variable resource acquisition may cause resource use to follow a 'nonzero-sum game' (cf. Wright, 2000). This situation is exemplified by the endothermic birds and mammals, whose high-energy lifestyles have allowed them to enhance several functions simultaneously, relative to those of ectothermic animals (cf. Wieser, 1985; Glazier, 2002a). Although resource allocation is emphasized here, a complete understanding of resource use in animals requires explicit consideration of all three 'RAs'.

1.2 Hierarchical resource allocation

To fully understand resource use in animals, one must realize that resource allocation occurs at several hierarchical levels (De Jong, 1993; Worley *et al.*, 2003). For example, ingested resources are partitioned between reproduction and somatic growth and maintenance at the highest level of the hierarchy, whereas, at lower levels, resources allocated to reproduction are partitioned among reproductive organs and offspring, and resources allocated to somatic growth and maintenance are partitioned among several structures and functions, including the various organs of the body, and so forth. Therefore, resources allocated to one compartment represent the resources acquired for allocation among the structures and functions within that compartment. Furthermore, the model of Van Noordwijk and De Jong (1986) can be applied at each of these hierarchical levels (Glazier, 1992; De Jong, 1993; Worley *et al.*, 2003). Therefore, if variation in resource allocation high in the hierarchy exceeds that for allocation lower in the hierarchy, trade-offs between traits may be masked at lower levels. For example, if the allocation of resources to reproduction varies among individuals, populations or species, this may prevent trade-offs from being seen between offspring size and number, as has been observed in cladocerans (Glazier, 1992), birds (Christians, 2000), scorpions (Brown, 2003) and ground squirrels (Risch *et al.*, 2007).

1.3 Priorities of resource allocation

An additional important aspect of resource allocation is that organisms allocate differential amounts of resources to various structures and functions, and protect these

investments to varying degrees when they are subjected to stressful conditions (Hammond, 1947, 1952; Wunder, 1978; Jackson, 1987, Bronson, 1989; Glazier, 2002b). In other words, resource allocation in organisms involves a 'priority system', or 'set of rules' that maximizes evolutionary fitness in specific environments. Theoretical models of resource allocation rules have been developed (e.g. Hammond, 1952; Wunder, 1978; McCauley *et al.*, 1990; Kooijman, 2000), but have rarely been tested (e.g. Glazier and Calow, 1992; Norgueira *et al.*, 2004). The relative priority of various organs and systems in the body can be estimated in several ways. They include estimates of the timing of the first appearance of these systems during development, estimates of the relative rate of energy use by these systems and estimates of the relative variation in the size, energy expenditure, and internal environment of, or blood flow to, these systems among individuals or in response to changing (especially stressful) environments.

2. Environmental Effects on Resource Allocation

The diversity of resource allocation strategies that animals have evolved in various environments is immense and can only be touched on here. I shall briefly describe some examples of various genotypic strategies that have been evolved by different species with different ecological lifestyles, and of various phenotypically plastic strategies expressed by individual organisms in response to environmental change.

2.1 Environmental effects on resource allocation strategies

Resource allocation patterns may be related to various characteristics of an animal's ecological niche or habitat, to age-specific patterns of mortality and to various resource, survival and reproductive costs or benefits of employing various allocation strategies (as reviewed by Sibly and Calow, 1986; Roff, 1992, 2002; Stearns, 1992). For example, according to the classical theory of *r*- and *K*-selection (MacArthur and Wilson, 1967), disturbed, temporary or otherwise unstable habitats should select for high allocation of resources to reproduction (i.e. high r or intrinsic rate of increase), at the expense of competitive ability and survival, whereas crowded, stable habitats (where the carrying capacity K has been reached), should select for high allocation of resources to competitive ability and survival, at the expense of reproduction. In short, the relative resource priority of reproduction in relation to maintenance should decrease as habitat stability increases. This pattern is frequently, but not always, observed (Roff, 2002; Begon *et al.*, 2006). The theory has often been applied to comparisons among distantly related taxonomic groups with distinctly different body sizes, though better tests rely on closely related populations and species to minimize extraneous phylogenetic and allometric effects (Stearns, 1992; Roff, 2002). For example, 'weedy' species of white-footed mice (*Peromyscus*) that occupy various kinds of temporary, disturbed or highly seasonal habitats have larger litter sizes and reproductive efforts, but reduced competitive ability compared to those that chiefly occur in more stable habitats (Glazier, 1980, 1985a; Glazier and Eckert, 2002).

Exceptions to the simplistic theory of *r*- and *K*-selection have prompted ecologists to take other approaches. For example, the *r*–*K* habitat template model has been expanded to include other dimensions, such as habitat adversity (reviewed in Southwood, 1988; Arendt, 1997). Still other ecologists have focused on how different age-specific patterns of mortality may select for particular life-history strategies (Stearns, 1992; Roff, 1992, 2002). For example, a high ratio of adult to juvenile mortality is predicted to favour semelparity (i.e. one episode of extravagant, 'big bang' reproduction per lifetime), whereas, a low ratio of adult to juvenile mortality is predicted to favour iteroparity (i.e. multiple episodes of conservative reproduction per lifetime). As predicted, semelparous species generally invest more in reproduction per breeding episode than do related iteroparous species (e.g. Calow, 1979; Roff, 2002). More generally, resource allocation to first reproduction should be an inverse function of expected life expectancy (Stearns, 1992), as has been observed among mussel species (Bauer, 1998) and populations of the garter snake *Thamnophis elegans* (Sparkman *et al.*, 2007). In addition, low juvenile survival and growth rates are expected to favour relatively few, large offspring because they are more likely to pass through the vulnerable juvenile stage successfully than smaller offspring requiring more time to accumulate more mass to reach adulthood (Sibly and Calow, 1986). As expected, terrestrial isopod crustaceans that suffer relatively high mortality from desiccation tend to produce fewer, but larger offspring than related aquatic species (Glazier *et al.*, 2003).

Optimal patterns of reproduction may depend not only on the costs of diverting resources from other activities, such as growth and maintenance, but also on increased risks of mortality and impaired future reproduction (Calow, 1979; Stearns, 1992; Roff, 1992, 2002; Glazier, 1999). As a result, trade-offs between reproduction and growth or survival, and between early and late reproduction, have been documented in numerous animal and plant species (see above references), though we still have much to learn about the proximate and ultimate causes of these trade-offs.

Other resource allocation patterns have received comparatively little attention, for example, allocation between growth and maintenance (Sibly and Calow, 1986), between body storage and other functions (Calow, 1994), between various body organs (Sibly and Calow, 1986; Perrin, 1992), and between many other life-history traits (Stearns, 1992). Selection or genetic engineering of rapid growth in laboratory mice is accompanied by reduced longevity and resistance to tumours and parasites, suggesting that somatic protection has been impaired (Rollo, 1995; Arendt, 1997). Similarly, food-enhanced growth rates in zebra finches (*Taeniopygia guttata*) are accompanied by reduced red blood cell resistance to oxidative damage (Alonso-Alvarez *et al.*, 2007). Conversely, increased immune challenges depress growth in the Leach's storm petrel (*Oceanodroma leucorhoa*) (Mauck *et al.*, 2005) and the lizard *Ctenophorus fordi* (Uller *et al.*, 2006). A broader comparison of laboratory mice and rats further shows that genetically or environmentally increased growth rates are accompanied by decreases in life span (Rollo, 2002). Similarly, an even broader comparative study of birds and mammals has revealed that rates of embryo growth are positively correlated with rates of ageing (Ricklefs, 2006). In wild animals, survival costs of rapid growth may also be the result of increased feeding causing increased exposure to predators (e.g. Biro *et al.*, 2006), or of diminished locomotor ability causing increased vulnerability to predation (Billerbeck *et al.*, 2001).

Greater body storage may enhance survival during resource-poor times, but by making an animal heavier and more sluggish, it may also increase vulnerability to predation (Witter and Cuthill, 1993). Predation may prevent accumulation of body stores by inhibiting foraging, and by favouring increased diversion of resources away from body storage to early reproduction to ensure offspring production before an animal is likely to be eaten. One or more of these hypotheses may explain why the freshwater amphipod *Gammarus minus* carries less fat in springs with versus without fish predators (Glazier, 1998). Increases in body storage and decreases in reproduction in the white-footed mice, *Peromyscus* spp. (Glazier, 1985a) have been observed in response to habitats with lower food quality and (or) quantity.

We know surprisingly little about the proximate and ultimate causes of resource allocation to various body organs and systems, a fundamental aspect of how organisms are built. Various intrinsic and extrinsic factors affecting the relative growth of organs have been identified (Goss, 1966; Bryant and Simpson, 1984; Emlen and Allen, 2004; Stanger *et al.*, 2007). And the theoretical models of Sibly and Calow (1986) and Perrin (1992) predict that most resources should be allocated to the organs that provide the most benefit in fitness (reproduction and survival) per unit investment, but this reasonable idea has not been tested rigorously, probably because it is technically difficult to do so. However, some extreme cases are consistent with these models. For example, the loss of eyes in cave-dwelling animals is as expected, because in total darkness the allocation of resources to eyes would have no fitness advantage (Sibly and Calow, 1986).

In any case, the principle of allocation suggests that increases in the relative size of one organ should be at the expense of that of others. Both resource and body-size limits may cause trade-offs between organ sizes (Sibly, 1991; Pitnick *et al.*, 2006), but such trade-offs have been little studied. In insectivorous bats, an inverse correlation between brain size and size of the testes may be due to energetic trade-offs because both of these tissues are metabolically expensive, and because primates and fruit-eating bats, which appear to have less-restricted energy budgets, do not show this inverse correlation (Pitnick *et al.*, 2006; Schillaci, 2006). In addition, comparative studies of birds and primates have shown that species with relatively large guts tend to have relatively small brains (Isler and Van Schaik, 2006). However, it is not known why this apparent trade-off occurs. Is it because of resource limits, space limits or because both are related to a third factor? Resource limits have been invoked to explain why artificial selection in chickens has resulted in larger guts, but smaller brains (Jackson and Diamond, 1996). However, the trade-off observed among species may be, at least in part, due to diet, which is related independently to both gut size and brain size. For example, herbivorous mammals tend to have larger guts, but smaller brains than mammals of an equivalent body size that feed on higher quality, but more dispersed foods (e.g. seeds, fruits and animal prey; Harvey and Krebs, 1990; Stevens and Hume, 1995; Langer, 2002).

The latter pattern deserves further discussion because it raises the intriguing possibility that organ allocation may be fundamentally linked to the ecological niche of an animal. Consider that animals have two major kinds of partially overlapping somatic organ systems: ectosomatic and endosomatic. This distinction was recognized by Romer (1972), who used the nearly synonymous terms ‘somatic

body' and 'visceral body', but I prefer my terms because all body structures are 'somatic', except for the reproductive organs. The ectosomatic organs and structures are required for moving through and interacting with the external environment, whereas the endosomatic organs and structures are required for maintaining the internal environment, including stable levels of nutrients, respiratory gases and other substances in the fluids bathing the tissues of the body. Specific brain centres (e.g. motor cortex and cerebellum), the somatic and sympathetic visceral nervous systems, externally directed sensory organs (eyes, ears, nose and skin receptors) and various locomotor structures are chiefly ectosomatic, whereas other specific brain centres (e.g. brain stem), the parasympathetic visceral nervous system, internal sensory receptors (e.g. taste buds and interoceptors) and the various alimentary and glandular organs are primarily endosomatic.

How may the relative development of the ecto- and endosomatic systems be affected by an animal's ecology? As a heuristic exercise, consider two closely related species of animals whose interaction with their respective environments differs in only one fundamental respect: the kind and location of the food that they eat. Other environmental factors are assumed to have essentially the same effects on both species. Given these ecological and phylogenetic controls, species A, which exploits easy to find and capture low-quality food should require a relatively less-developed ectosomatic system, but relatively more developed endosomatic system than species B, which exploits hard to find and capture high-quality food. This is because easily accessible food requires little movement, mental capacity and sensory ability, as compared to hard to find and capture food. However, since the easily accessible food is also of lower quality (i.e. physically and chemically protected, and thus relatively indigestible), it requires extensive internal processing as compared to the less-accessible food, which is of higher quality (i.e. highly nutritious and digestible). This dichotomy in food quality is to be expected, because natural selection should favour well-defended tissues in easy to find prey, whereas hard to find or capture prey are able to use inconspicuousness or evasiveness, rather than chemical tissue defences, to avoid predation (cf. Feeny, 1976).

Given the above scenario, it should not be surprising that animals that eat fibrous, relatively indigestible plants tend to have relatively small brains, eyes and ears, but more elaborate dentitions, jaw muscles and guts, than closely related animals that eat relatively digestible, but harder to find or capture seeds, fruit or animal prey. These differences have evolved repeatedly in the speciose rodent family, the Muridae. For example, compare the herbivorous, ground-dwelling voles (*Microtus*) and lemmings (*Lemmus* and *Dicrostonyx*), which have small eyes, ears and brains, and short legs and tails, but voluminous guts, highly elaborate dentitions, and broad heads containing large jaw muscles and skull attachments for masticating fibrous food, with the omnivorous, tree-climbing white-footed mice (*Peromyscus*) and wood mice (*Apodemus*), which have relatively large eyes, ears and brains, long legs and tails, but relatively small guts and narrow heads, and less-elaborate dentitions. I predict that such diet-related variation in ecto- versus endosomatic organ development not only may be common within a variety of groups of closely related animal species, but also may be relevant to the level of priority that an organ has in a given species, and to practical efforts to increase farm animal production.

2.2 Environmental effects on resource allocation priority rules

Variation of the resource allocation system among individuals of the same species not only reveals the malleability of this system, but also provides insight into its priority rules. These priority rules are best revealed by stressing organisms to see which components of the system maintain their integrity better than others. For example, maintenance (survival or longevity) is usually given precedence over growth and reproduction when animals are given limited food, or are stressed in other ways (e.g. Bronson, 1989; Boggs, 1994; Schneider and Wade, 2000; Glazier, 2002b). The importance of this priority system to animals is indicated by its apparent mediation through multiple neuroendocrine pathways that elicit stress-related reductions in growth and reproduction (Wade and Schneider, 1992; Rollo *et al.*, 1997). However, the resource allocation priority system may vary with age, gender, physiological state and environmental conditions, and among genetically different individuals and species (Bronson, 1989; Perrigo, 1990; Glazier and Calow, 1992; Wade and Schneider, 1992; Boggs, 1994; Kooijman, 2000). For example, food restriction revealed that young females of the crustacean *Daphnia magna* tend to put higher priority on growth relative to maintenance and reproduction than do older females (Glazier and Calow, 1992), as predicted in part by theory (Heino and Kaitala, 1999).

Ambient temperature can affect resource allocation patterns as well. Increasing temperature has been shown to increase relative allocation to reproduction or reproductive structures in fish (McManus and Travis, 1998). Perhaps enhanced allocation to reproduction is advantageous at high temperatures because they cause decreases in adult life expectancy, possibly as a result of enhanced rates of metabolism and ageing. However, by increasing metabolic rate, high temperatures may also increase maintenance costs in ectothermic animals, thus leaving fewer resources for reproduction (Bernardo and Reagan-Wallin, 2002). Alternatively, higher temperatures may permit longer daily activity periods for acquiring food, which results in increases in relative allocation to reproduction in the lizard *Sceloporus undulatus* (Niewiarowski, 2001). Temperature-caused changes in metabolic rate may additionally affect the relative investment that animals make in individual offspring (Sakai and Haradi, 2001).

Resource allocation may vary with body condition, reproductive state and levels of parasitic infection. Experimental manipulations have shown that barn swallows (*Hirundo rustica*) increase their energy expenditure for reproduction in response to increased energy reserves (Spencer and Bryant, 2001). In many (but not all) polygynous animals, where variation in male reproductive success exceeds that of females, females with good body condition or nutrition tend to produce more males than those in poor condition (Rosenfeld and Roberts, 2004), as predicted by sex allocation theory (Trivers and Willard, 1973; but see Hewison and Gaillard, 1999; Isaac *et al.*, 2005). The effects of maternal condition on offspring sex ratio may be mediated by stress hormones (e.g. corticosterone; Love *et al.*, 2005), direct effects of nutrients or other mechanisms (Rosenfeld and Roberts, 2004). In some ungulates, high-quality males sire more males than do low-quality males (Gomendio *et al.*, 2006; Røed *et al.*, 2007), though effects of paternal quality on sex ratio in birds have been mixed (Monaghan, 2004). Mate

quality may also affect the total investment in reproduction, as has been observed in a variety of animals (Sheldon, 2000). Presumably, high-quality mates make increased investment in offspring worthwhile in terms of evolutionary fitness, because of the high-quality traits that the offspring will inherit.

In mammals, the resource priority for reproduction increases from low in non-reproductive individuals to relatively high in pregnant and early lactating mammals to lower again in late lactating and post-lactation mammals (Hammond, 1947, 1952; Bronson, 1989; Perrigo, 1990; Wade and Schneider, 1992). Consequently, lactating ewes given restricted dietary protein place higher priority on milk production than immune defence (Houdijk *et al.*, 2003). 'Orchestrated changes for the priorities of a physiological state' have been called 'homeorhesis' (Collier *et al.*, Chapter 5, this volume).

Female damselflies (*Coenagrion puella*) infected with parasitic mites produce fewer, but larger offspring than do uninfected females (Rolff, 1999). A similar pattern is observed in *Daphnia* and guppies (*Poecilia reticulata*) given restricted food (Glazier, 1992; Bashey, 2006). Since both infection and food limitation are expected to decrease juvenile growth and survival, the production of larger young is as expected by theory (Sibly and Calow, 1986; see also Section 2.1). Seasonal and photoperiod effects on allocation priorities have also been observed. Seasonal shifts in resource allocation priorities have been observed in young fish (e.g. Biro *et al.*, 2005) where growth is prioritized in summer, whereas fat storage is prioritized in winter. This allocation pattern makes sense in terms of the greater expected food availability in summer versus winter (cf. Kooijman, 2000).

Phenotypically plastic responses of the resource allocation system to environmental change may vary among closely related species or populations with different life-history strategies. For example, two genetically distinct clones of *D. magna* differed in how much food limitation affected their relative allocation of energy to growth versus reproduction (Glazier and Calow, 1992). Perrigo (1990) observed that sensitivity of reproduction to food deprivation is greater in deer mice (*Peromyscus maniculatus*) than in house mice (*Mus musculus*), a difference that may be related to the greater seasonal predictability of food resources in the natural habitats of the deer mice. In addition, species with high adult mortality are expected to place a higher priority on reproduction over growth than those experiencing low adult mortality (Stearns, 1992; Heino and Kaitala, 1999). Semelparous species tend to prioritize reproduction when starved, in contrast to that observed in iteroparous species. For example, when starved, two of the three semelparous flatworm species increased the proportion of energy devoted to reproduction, whereas two iteroparous species decreased it (Woolhead, 1983). Similarly, relatively short-lived fish may increase resource allocation to reproduction in response to food scarcity, whereas long-lived fish decrease it (Wootton, 1990).

2.3 Environmental effects on resource allocation to body organs and structures

As noted for various life-history traits in Section 2.2, variation in the sizes of body organs among individuals and in response to environmental change can reveal the priorities of resource use for various anatomical structures and physiological functions. For example, in mammals, individual variability in organ mass is usually least

for the brain (≤10%, C.V.), intermediate for the heart, kidneys, liver and lungs, and greatest for the spleen and gonads (usually >20%, and sometimes >40%; Jackson, 1913; Brown *et al.*, 1926; Yablokov, 1974; Friedman *et al.*, 1995).

This high priority of the brain, and the relatively low priority of the spleen, gonads and fat tissues, is also supported by relative changes in the sizes of these organs in response to resource deprivation and other changes in body state. When birds and mammals are deprived of food or water, the brain shows the least change in size, whereas the spleen, gonads and fat tissues usually shrink greatly in size (Jackson, 1915; Kleiber, 1961; Ghosh, 1975). Similarly, biased protection of the brain, eyes and central nervous system, relative to that of other organs, is observed when a mammal is diseased (Brown *et al.*, 1926; Lee and Dubos, 1971), when its hormonal state is altered (e.g. by removal of the thyroid gland; Hammett, 1929), or when it is hibernating (Zhou *et al.*, 2001). However, specific organs that produce stress-related hormones, e.g. adrenal glands, may temporarily show relatively high priority, e.g. enlargement during disease in the rabbit (Brown *et al.*, 1926) and during dehydration in the gerbil (Ghosh, 1975).

In the gerbil *Tatera indica*, starvation results in tissue glycogen content decreasing relatively little in the brain, heart and kidneys, but relatively much in the liver and muscle (Purohit *et al.*, 1986). Maternal undernutrition in mammals results in similar 'sparing of the brain' in the fetus, but more marked effects on other fetas organs (e.g. Jackson and Stewart, 1920; Lee and Dubos, 1971; Desai *et al.*, 1996; Metcalfe and Monaghan, 2001). During nutritional or respiratory stress, blood flow of nutrients and oxygen to the brain appears to be maintained at the expense of other tissues (Jackson, 1987; Desai *et al.*, 1996). Food-restricted pregnant mammals show less per cent mass loss of their own tissues as compared to fetas tissues, a difference which may be explained by differential blood flow to somatic versus reproductive tissues (Rosso, 1981). In addition, food-restricted lactating rats consume more of their low-priority fat tissues than higher-priority visceral and muscle tissues (e.g. Glore and Layman, 1985). Seasonal changes in organ masses are consistent with the patterns reported above. In the best available data set, Pucek (1965) showed that winter depression in body mass in the common shrew (*Sorex araneus*) is accompanied by a small decrease in brain mass, intermediate decreases in the masses of the heart, kidneys and liver, and large decreases in the masses of the spleen and gonads.

Another approach is to assume that organs that appear early in development are of higher priority for the extended life of an organism than those that appear at a later stage of development. In vertebrates, the brain, central nervous system and other vital circulatory, respiratory and alimentary systems develop early, whereas the reproductive and body-storage systems develop relatively late (Swan, 1990). This pattern can be quantified by measuring the degree to which various organs at birth have reached adult size. When this is done in the guinea pig, it can be seen that the brain is most advanced, whereas the gonads are least developed (Gericke *et al.*, 2005). Similar patterns can be seen in the rat (Rieck *et al.*, 1996; Stewart and German, 1999), the pig (Friedman *et al.*, 1995) and various primates (Larson, 1985).

All of these approaches show that in mammals the brain is of highest priority, other essential somatic organs are of intermediate priority, and the least essential organs and tissues, such as the spleen, gonads and fat tissues, are of lowest priority.

These patterns parallel the higher priority that somatic maintenance generally has over reproduction, at least in iteroparous animals. Secondary sexual characters are not essential for survival, and thus should have relatively low priority in the resource allocation system as well. As expected, secondary sexual characters tend to be highly variable (Darwin, 1859, 1874; Hughes *et al.*, 2005) and very sensitive to body condition and environmental stressors (Rowe and Houle, 1996).

Various hypotheses have been offered to explain why certain body organs and systems have priority over others. Hammond (1947, 1952) suggested that organs with the highest metabolic activity have the highest 'pull' on resources. However, although the brain has high metabolic activity (Mink *et al.*, 1981) in line with its high priority, other organs with less priority may have even higher metabolic rates, either on a whole organ or mass-specific basis (Schmidt-Nielsen, 1984; Wang *et al.*, 2001). Desai *et al.* (1996) suggested that the relative sensitivity of developing organs to resource deprivation is simply a function of their rate of cell multiplication. However, this hypothesis is inadequate in two respects. First, although the rate of cell multiplication of high-priority organs (e.g. brain) is lower than that of low-priority organs during relatively late stages of development, this is not true during earlier stages. During the earliest stages of development, the brain and other high-priority organs grow rapidly by cell multiplication (e.g. Riska and Atchley, 1985), and thus undernutrition throughout pregnancy should stunt the growth of high-priority organs just as much as that of low-priority organs. Second, the cell multiplication hypothesis cannot explain why mature, fully developed organs show differential shrinkage in response to resource scarcity. This pattern would appear to be more a function of organ-specific rates of cell death than rates of cell multiplication. Perhaps organ priority is related to cell turnover rates. For example, the adult brain may be more protected during nutritional stress because its low rate of cell turnover would not allow it to recover lost tissue mass as readily as that of lower-priority organs (e.g. liver) with higher rates of cell turnover. However, there are exceptions to this pattern – e.g. muscle has lower priority than the brain (e.g. Hammond, 1947, 1952), despite having a similar low rate of cell turnover (Goss, 1966).

In any case, it seems more likely that the priority of an organ is most related to how important its function(s) is (are) to the welfare of the whole body. In addition, high-priority systems should be more highly and indispensably interrelated with other systems of the body than are low-priority systems. Clearly, a mammal can survive without gonads or with significantly reduced liver, spleen or fat tissue, but it cannot survive for long with even a slightly impaired brain. This is because the brain is important for regulating virtually all of the body's activities, and removal of any of its control centres would have serious, if not fatal, effects. Regulatory systems, such as that of the brain and central nervous system, should be privileged components of the body's resource budget, because altering the allocation of resources to them would affect the homeostasis of the whole body, and even the resource allocation system itself.

In short, high-priority systems do not necessarily require the greatest resource investment, but rather investment in them is expected to be most tightly regulated. This tight regulation should arise from relatively intense selection on them, because they yield the most fitness benefit per unit investment (following Sibly and Calow,

1986; Perrin, 1992). The relatively high variability of non-functional, rudimentary or vestigial structures, noted by Darwin (1859), Rensch (1959), Yablokov (1974) and others, is consistent with this interpretation. As Darwin (1859) argued, the variability of rudimentary organs 'seems to be owing to their uselessness, and therefore to natural selection having no power to check deviations in their structure' (but see Jeffery, 2005).

Resource allocation priorities may also apply to very specific structures within an organ or anatomical part. For example, various parts of the brain may show different priorities. As evidence, when brain size decreases during winter in voles and shrews, the telencephalon or cortex decreases the most, whereas the myelencephalon or brainstem decreases the least (Yaskin, 1984). In addition, decreases in brain size associated with domestication in the Norway rat (*Rattus norvegicus*) are associated with larger reductions in the sizes of the cortex and cerebellum than that of the medulla oblongata (Kruska, 1975). The size of the brainstem and its components also varies relatively little among bird species, as compared to that of the telencephalon and cerebellum (Boire and Baron, 1994). These patterns are not surprising because the brainstem contains many control centres responsible for the homeostasis of the body, whereas the cortex involves higher brain functions, such as cognition, that may have less immediate effects on survival (but see Ricklefs, 2004; Sol *et al.*, 2007). Note also that these differences cannot be explained by different rates of cell multiplication or turnover, which are low throughout the brain (see also above).

In addition, Badyaev (2005) has shown that traits that are highly integrated with other traits in a specific structure, such as in the wings of bumble bees (*Bombus empatiens*) and the mandibles of soricid shrews (*Sorex*), show less stress-induced variation than do less-integrated traits. These observations dovetail nicely with the notion just described that high-priority systems are more integrated with other systems of the body than are low-priority systems.

3. How Malleable Are Resource Allocation Systems?

The extensive variation in resource allocation systems described in Section 2 implies that these systems are very malleable, being highly responsive to both the internal state of an organism and its external environment. Species differences also imply that resource allocation systems can evolve readily by natural selection, a fact that has been profitably harnessed by animal breeders. However, some components of the resource allocation system may be more or less flexible than others. For example, a variety of approaches have shown that in birds and mammals, the brain and associated structures almost invariably have the highest priority for resources. However, the relative priority of other organs may vary, as shown by differences due to age, gender, genetic strain or species. This suggests that not only do some organs and systems in the body have higher priority than others, but also those high in the hierarchy are less likely to vary in their hierarchical status among genotypes, species or physiological states, compared to those low in the hierarchy. In other words, high-priority organs are not only more tightly regulated within any given organism in comparison with low-priority organs, but their priority status is

also less likely to become modified by selection or direct environmental effects than that of low-priority organs. The high malleability of some components of the resource allocation system is also seen by how rapidly some body organs expand or contract in response to various environmental challenges. For example, the alimentary tract and other visceral organs may increase substantially and reversibly in size during periods of high-energy demand (e.g. migration, reproduction and temperature stress; Piersma and Lindström, 1997; Starck, 1999).

Although some interesting insights have been gained, we still have much to learn about how much resource allocation systems can evolve and respond to environmental change, and about the proximate and ultimate mechanisms underlying these changes. In particular, intergenerational phenotypic and genotypic effects require further attention. The maternal environment can affect both the resource acquisition and allocation systems of offspring in numerous fundamental ways that are still little understood. For example, maternal stress or malnutrition may have profound effects on 'fetal programming', altering offspring-feeding rates, relative allocation of resources to various body organs, tissues and systems, and the neuroendocrine regulatory systems responsible for these changes (e.g. Lumma and Clutton-Brock, 2002; Worthman and Kuzara, 2005; Chadio *et al.*, 2007; De Blasio *et al.*, 2007). Compensatory growth by offspring stunted at birth or hatching may also exact various physiological and ecological costs with long-term, intergenerational fitness consequences (Metcalfe and Monaghan, 2001; Monaghan, 2004). In addition, differential expression of maternally or paternally inherited genes may affect resource acquisition by offspring in the womb, and later in life as well (Haig, 1993; Constância *et al.*, 2004).

Many other intriguing questions can be asked about the evolution of resource allocation systems. For example, should selection for hierarchical priority systems be stronger in variable, harsh versus constant, benign environments? One might expect that mechanisms for ensuring the protection of high-priority systems should be more developed in environments offering frequently stressful conditions than in more constant, benign environments. As expected, waterstriders (*Gerris*) inhabiting unpredictable environments defend somatic maintenance more strongly, by depressing reproductive investment in response to food scarcity, than do those from relatively constant environments, which lack food-dependent variation in reproductive investment (Kaitala, 1991). Another prediction is that wild animals should more strongly defend high-priority systems in response to environmental stress than should related domesticated animals. Perhaps this is why increased variability of the size of the high-priority brain and its parts has accompanied domestication in ducks (Ebinger, 1995) and the mink (Kruska, 1996). Similarly, gerbils appear to show less individual variability in brain mass and less loss of brain mass during resource deprivation than does the more domesticated laboratory rat. It would also be interesting to explore further whether the priority ranking of various organs and systems is related to specific lifestyles and environmental conditions.

In addition, what effect does the allocation priority system have on the evolvability of various traits? Glazier (2002b) has hypothesized that high-priority traits should exhibit little environmental variance, thus increasing the proportion of phenotypic variation that is genetic and thus heritable, relative to low-priority traits. In support, high-priority morphological and physiological traits related to body

maintenance tend to have higher heritabilities than low-priority life-history traits related to growth and reproduction (Glazier, 2002b; Roff, 2002). As expected, the high-priority brain tends to have high heritability that is often twice as high as that of overall body size (Riska and Atchley, 1985; Cheverud *et al.*, 1990; Miller and Penke, 2007). Offspring or propagule size, which has higher priority than that of offspring number as evidenced by its lower individual variability and sensitivity to maternal or environmental differences in a variety of organisms (e.g. Morris, 1987; Stearns, 1992; Boggs, 1994), generally has higher heritability as well (Roff, 1992; Christians, 2002; Brown and Shine, 2007).

Based on their high heritability one might suppose that high-priority morphological traits should respond to selection more readily than low-priority life-history traits. However, little is known about the evolutionary responsiveness of traits in natural populations. The survey of Kingsolver *et al.* (2001) showed that natural selection on morphological traits was stronger than selection on life-history traits, but no information was given on the responsiveness of these traits to selection. In any case, a complete understanding of the relative evolvability of traits requires an examination of their relative genetic variance, as well. In fact, the additive genetic variance and the mutational variabilities of low-priority, life-history traits average higher than those of high-priority, morphological traits (Houle, 1992; Houle *et al.*, 1996; Merilä and Sheldon, 1999). Indeed, one might predict that high-priority traits should have less additive genetic variance than low-priority traits because they are under more intense selection, following the models of Sibly and Calow (1986) and Perrin (1992). Based on this reasoning, high-priority traits may actually be less evolvable than low-priority traits, if both the genetic and environmental variance of these traits is relatively low. Perhaps this explains why morphological traits do not evolve faster, on average, than life-history traits (Kinnison and Hendry, 2001), despite their apparently being under stronger selection (Kingsolver *et al.*, 2001). It may also explain why the size of the high-priority brain seems to respond relatively little to artificial selection (Fuller, 1979; Ricklefs and Marks, 1984), and also usually appears to evolve more slowly than overall body size (Riska and Atchley, 1985; Kruska, 1987; Nealen and Ricklefs, 2001; but see Deaner and Nunn, 1999). Brain volume may have relatively low evolvability because, as observed in humans, it has a coefficient of additive genetic variance that is lower than that of any other organ or life-history trait (Miller and Penke, 2007). In contrast, low-priority secondary sexual characters exhibit high levels of phenotypic and genetic variance (Pomiankowski and Møller, 1995; Hughes *et al.*, 2005), and thus should have high evolvability. This hypothesis is supported by recent studies on birds showing that secondary sexual characters tend to evolve faster than non-sexual characters (Cuervo and Møller, 1999; Møller and Szép, 2005).

However, the above discussion is largely speculative. Required are more studies of the relative genetic variance, environmental variance, heritability and response to selection of various high- and low-priority traits, including various organs of the body. These studies would be especially insightful if they compared species with markedly different life histories and resource management systems. For example, if the above reasoning is correct, the genetic and environmental variance of reproductive traits, and thus their evolvability, should be lower in semelparous species than in related iteroparous species. Since reproductive traits appear to have relatively

high priority in semelparous species, their environmental variance should be relatively low because of tight physiological regulation (cf. Glazier, 2002b), and their additive genetic variance should be relatively low because of intense selection.

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3 Trade-offs

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It is commonly assumed that trade-offs (negative associations) between life-history traits are due to resource allocation. However, although competition between activities and structures for limited resources appears to underlie many negative associations between traits, trade-offs may or may not be seen for other reasons that are briefly reviewed here. Several recent reviews should be consulted for more details (e.g. Roff, 1992, 2002; Stearns, 1992; Rollo, 1995; Rose and Bradley, 1998; Sinervo and Svensson, 1998; Zera and Harshman, 2001; West-Eberhard, 2003; Harshman and Zera, 2007; Roff and Fairbairn, 2007).

1. Causes of Trade-offs

Trade-offs between traits may result not only from resource allocation, but also from ecological constraints, opposite independent effects of an environmental factor, morphological constraints, physiological limits, functional conflicts, multiple trait interactions, physiological regulation, genetic regulation and conflicts over gene expression. In many cases, these mechanisms are complementary rather than mutually exclusive, which I discuss in turn.

1.1 Resource allocation

Since all functions and structures require resources, in a general sense, all negative associations between the magnitudes of two traits involve resource allocation. I call this 'resource allocation in the broad sense'. Resource allocation in the broad sense that is the direct result of competition for limited resources is 'resource allocation in the narrow sense'. Resource allocation in the narrow sense is suggested by life-history trade-offs that are more strongly expressed under food limitation (e.g. Hammond, 1952; Roff, 1992; Stearns, 1992; Ebert, 1993; Sikes, 1995; French

 Resource Allocation Theory Applied to Farm Animal Production (ed. W.M. Rauw)

et al., 2007; Waelti and Reyer, 2007; but see Reznick, 1985; Glazier, 1992; Rollo, 1995). Even stronger evidence for resource allocation in the narrow sense comes from direct experimental manipulations. For example, artificially enlarged litters result in diminished rates of milk-energy intake and growth by mammal sucklings, thus causing a trade-off between offspring size and number (e.g. Hammond and Diamond, 1992; Rogowitz, 1996). Similarly, in various birds, enlarged clutches result in eggs and parents having lower nutrient reserves (Monaghan, 2004; Williams, 2005). Experimental manipulations suggest that muscle proteins are used for reproduction in, for example, zebra finches (*Taeniopygia guttata*) (Veasey *et al.*, 2000; but see Williams, 2005). Additional experimental manipulations of resource allocation to specific body parts have revealed compensatory changes in relative allocation to other body parts (e.g. Nijhout and Emlen, 1998). Genetically engineered mice exhibit profound changes in resource allocation in response to enhanced growth rates (Rollo, 1995). Indeed, commonly observed negative associations between growth and reproduction are difficult to explain other than as being the result of competition for limited resources (Stearns, 1992; Tsikliras *et al.*, 2007). Bone demineralization observed in various breeding birds and mammals (e.g. Williams, 2005; Hood *et al.*, 2006) is also hard to interpret other than as being diversion of resources away from the soma to reproduction. Zera and Zhao (2006) have even identified the specific metabolic pathways that are altered to fuel flight versus reproduction in wing-morphs of the cricket *Gryllus firmus*. Nevertheless, resource allocation in the broader sense may be affected by other factors as well.

1.2 Ecological constraints

Increased reproduction or growth may be correlated with decreased survival not only because of physiological resource costs, but also because of ecological costs, such as increased exposure to predation or other harmful environmental factors associated with increased foraging or parental care (Calow, 1979; Stearns, 1992; Glazier, 1999, 2002a; Roff, 2002). Both of these kinds of costs may even occur simultaneously. For example, egg-laying in the zebra finch not only diverts resources from muscle tissue, but also impairs flight, and thus possibly escape from predators (Veasey *et al.*, 2000). However, Reid (1987) has suggested that the trade-off between reproduction and survival in the glaucous-winged gull (*Larus glaucescens*) is probably due to physiological costs, and not ecological costs, because decreases in adult survival associated with reproduction occur mostly after the breeding season. In addition, negative associations between traits may arise because they are independently, but oppositely related to a common environmental factor. For example, sweating and skin whiteness are negatively related due to their independent relation to sunlight (West-Eberhard, 2003).

1.3 Morphological limits

The model of Sibly (1991) shows that body-space limits may cause trade-offs between body organs. Body space may also limit reproductive investment and

cause trade-offs between offspring size and number. For example, small mothers of the freshwater amphipod *Gammarus minus* show more pronounced trade-offs between egg size and number than larger mothers, apparently because of space limits associated with their small brood pouches (Glazier, 2000b). Body space limits may, in turn, be caused by resource limits on body growth, or by ecological constraints (e.g. size-selective predation). In particular, size-selective predation by fish has apparently caused the evolution of small body size in some populations of *G. minus* (Glazier, 1998), which, in turn, causes a trade-off between egg size and number.

Another morphological trait that may limit reproductive investment in mammals is mammary teat number (Gilbert, 1986; Beckman *et al.*, 2007). By limiting milk availability to sucklings, teat number may cause resource limits that result in trade-offs between litter size and offspring size and (or) survivorship. These trade-offs were verified in lactating mice with surgically varied teat number (Hammond *et al.*, 1996). However, the highly social naked mole rats (*Heterocephalus glaber*) have been able to successfully rear litters that are much larger (maximum = 28) than their teat number (maximum = 15), apparently because breeding females are fed and protected by other members of their colony, thus enabling them to allocate more time and energy to lactation (Sherman *et al.*, 1999).

1.4 Physiological limits

Physiological limits on food processing and resource use by specific tissues may affect the resource allocation in the broad sense (see Weiner, 1992; Bacigalupe and Bozinovic, 2002; Speakman and Król, 2005). Constraints on efficiencies of the assimilation and conversion of food into body tissue may also limit options for resource allocation (e.g. Glazier, 1990a; Naya and Bacigalupe, Chapter 4, this volume). In addition, excessive heat produced during lactation may limit reproductive investment in mammals. For example, lactating rats and mice suffer from heat load, which causes them to restrict contact with their pups, which then have reduced milk uptake and growth (Adels and Leon, 1986; Król and Speakman, 2003; but see Speakman and Król, 2005). Nevertheless, 'resource association' (see Glazier, Chapter 2, this volume) between lactation and thermogenesis permits lactating rats to prolong pup contact in the cold, which could be an advantage in their natural environments. Furthermore, the thermal benefits of sibling huddling may favour larger litter sizes in colder environments, as shown in European rabbits (*Oryctolagus cuniculus*) (Rödel *et al.*, 2008).

Trade-offs may also result from negative associations between rates and efficiencies of biochemical, physiological and behavioural processes (Smith, 1976; Sibly and Calow, 1986; Parker and Maynard Smith, 1990). For example, given constant food quality, the rate of food processing by the gut tends to be inversely related to the efficiency with which that food is digested and assimilated by body tissues (but see Rollo, 1986). McClure and Randolph (1980) have reported a trade-off between growth rate and efficiency in a comparison of the precocial cotton rat (*Sigmodon hispidus*) with the altricial wood rat (*Neotoma floridana*). However, within species and among closely related species, rates and efficiencies of tissue production tend to covary positively, because the cumulative, proportional energy costs of maintaining previously grown tissue are less when

production rates are rapid (Glazier, 1990a). Effects of reproductive stage on the efficiency of conversion of food into offspring may also affect the total level of reproductive investment. For example, in rodents, reproductive efficiency is higher during gestation than lactation, thus enabling more offspring production per unit investment during gestation (Glazier, 1990b). As a result, overall reproductive efficiency is a positive function of the time that the young spend *in utero* during their preweaning lives. However, despite the efficiency advantage of gestation, mammals have not abandoned lactation entirely apparently because of various costs of extending gestation time (Glazier, 1990b). Boggs (1992) presents a graphical model showing that the shape of the trade-off curve between two life-history traits (e.g. growth and reproduction) depends on both the rates and efficiencies of resource use by those traits.

1.5 Functional conflicts

Some functions cannot be performed simultaneously, thus leading to a trade-off between them. Examples include vocalizing versus swallowing (West-Eberhard, 2003), shivering versus locomotion in skeletal muscles (Chappell and Hammond, 2004) and cell division versus cilia production in cells (Buss, 1987). It is also possible that the operation of one function may impair the effectiveness of another. For example, Barnes and Partridge (2003) have suggested that trade-offs between reproduction and longevity may result from somatic damage caused by the reproductive process. However, the nature of this damage, and the mechanisms causing it, have yet to be elucidated, though it may involve oxidative stress (Salmon *et al.*, 2001), possibly related to enhanced metabolic rates (Nilsson, 2002; Alonso-Alvarez *et al.*, 2004). Possible effects of reproduction on longevity that occur via somatic damage may also involve resource allocation (in the narrow sense), if reproduction diverts resources away from mechanisms (e.g. antioxidant defences) that protect against somatic damage (Cichon, 1997; Harshman and Zera, 2007). Nevertheless, in an experimental study, Tatar and Carey (1995) showed that a trade-off between reproduction and survival in the beetle *Callosobruchus maculatus* was probably due to resource allocation (in the narrow sense), rather than to somatic damage directly caused by reproduction.

In general, time allocation of various behavioural activities can be considered the result of functional conflicts. For example, animals divide their time by exclusively engaging in foraging, mating, grooming, sleeping and other behaviours because none of these activities can easily or effectively be done simultaneously. Time allocated for foraging is of special significance because foraging not only consumes resources, but also provides resources that can then be allotted to various functions and structures (Ricklefs, 1991; Steiner and Pfeiffer, 2007). The relative priorities that animals give to behavioural activities in specific environments are of great theoretical and practical interest. For example, high-priority activities should be sustained more in response to time limitation or enhanced energy costs, than low-priority activities (e.g. Houston and Macfarland, 1980; Munksgaard *et al.*, 2005). However, these theoretical expectations have rarely been studied in wild animals. Risk of predation appears to influence the relative amounts of time that

mammals spend foraging versus being vigilant (e.g. Tchabovsky *et al.*, 2001). Diet can also affect the relative amount of time spent foraging.

1.6 Multiple trait interactions

Trade-offs between traits may occur not because of any direct interaction between them, but because they are both related to a third trait. The most obvious third trait is body size or metabolic rate, because a plethora of traits are related to them (as reviewed by Peters, 1983; Calder, 1984; Schmidt-Nielsen, 1984; McNab, 2002; Brown *et al.*, 2004; Chapter 9). For example, in interspecific comparisons, reproductive rate and longevity may be inversely correlated because they are independently, but oppositely related to body size and metabolic rate. Indeed, mammalian life histories largely follow a slow–fast continuum from large, long-lived species with slow metabolic, growth and reproductive rates to small, short-lived species with fast metabolic, growth and reproductive rates. However, the relative importance of physical, developmental and metabolic constraints versus adaptive optimization and resource allocation (in the narrow sense) in causing body-size scaling relationships is presently controversial (Promislow and Harvey, 1990; Rollo, 1995; Speakman *et al.*, 2002; Glazier, 2005; Prinzinger, 2005). In any case, several recent studies show the promise of using both allometric relationships and evolutionary optimization to understand life-history patterns (e.g. Charnov *et al.*, 2001; Sibly and Brown, 2007; Walker *et al.*, 2008).

Other trade-offs between traits may involve indirect effects mediated by additional traits or functions. For example, traits A and C may be negatively associated because trait A negatively affects trait B, which then negatively affects trait C. It is also possible that multiple traits may act as intermediary links in causing the negative association between two traits. For example, in birds a trade-off between current and future reproduction may occur because delayed reproduction in one breeding season may reduce the time for adequate moulting, thus decreasing effective thermal insulation, which, in turn, increases heating costs that may divert energy away from reproduction during the next breeding season (Nilsson and Svensson, 1996). A trade-off between reproduction and longevity may result because increased reproduction requires increased foraging, which may, in turn, cause increased somatic damage that reduces future survival (Yearsley *et al.*, 2005). Additionally, increased reproductive effort may suppress immune function, which may, in turn, decrease the probability of survival (Lochmiller and Deerenberg, 2000; Lee, 2006; Harshman and Zera, 2007).

1.7 Physiological regulation

Some negative associations between traits may simply be due to hormones or growth factors having opposite, pleiotropic effects on two traits that do not directly interact (Zera and Harshman, 2001; Williams, 2005; Flatt and Kawecki, 2007). However, a major function of the neuroendocrine system is to regulate the acquisition and allocation of resources and their rates of use by different tissues (Bronson,

1989; Wade and Schneider, 1992; Finch and Rose, 1995; Rollo *et al.*, 1999; Ricklefs and Wikelski, 2002), and thus explanations of trade-offs based on hormonal control largely complement those based on resource allocation (in the narrow sense). Resource allocation theory provides insights into the ultimate (evolutionary) causes of life-history trade-offs, whereas physiological analyses reveal important proximate (functional) causes for the expression of these trade-offs (Sinervo and Svensson, 1998). As an example, the insulin/IGF signalling pathway mediates the expression of key life-history trade-offs, such as that between reproduction and longevity (Leroi *et al.*, 2005; Partridge *et al.*, 2005; Van Straalen and Roelofs, 2006), and between offspring size and number (Gluckman *et al.*, 1992). Endocrine stress systems appear to control the priority rules of resource allocation. When stressed, birds and mammals enter into an 'emergency' physiological state that prioritizes maintenance (survival) over growth and reproduction (Wade and Schneider, 1992; Rollo *et al.*, 1997; Wingfield *et al.*, 1998), a response that is stronger in long-lived or iteroparous organisms than in short-lived or semelparous organisms (Ricklefs and Wikelski, 2002). By contrast, in semelparous *Octopus vulgaris*, hormones from the optic gland and ovaries prioritize reproduction over maintenance, resulting in release of amino acids from muscle to support rapid growth of the reproductive organs (O'Dor and Wells, 1978).

1.8 Genetic regulation

A classic explanation of the trade-off between reproduction and longevity is based on the antagonistic pleiotropy of genes. Because of the increasing probability of death with increasing age, selection may favour genes that promote early reproductive performance, even if they cause ageing later in life (Williams, 1957). In short, youthful fitness is favoured over elderly sickness, especially in short-lived organisms. Quantitative genetic studies of negative associations between early reproduction and adult ageing or survival in fruit flies (*Drosophila melanogaster*) (Rose, 1991) and red deer (*Cervus elaphus*) (Nussey *et al.*, 2008), and the pleiotropic effects shown by many hormones (Finch and Rose, 1995), suggest that age-dependent pleiotropic genes must exist, but very few of these have ever been identified (Leroi *et al.*, 2005).

Currently there is a debate over whether trade-offs between reproduction and longevity are caused by resource allocation to these two traits, as traditionally believed, or by gene-controlled molecular signals emanating from specific reproductive tissues that cause independent effects on fecundity and longevity (Leroi, 2001; Lessells and Colegrave, 2001; Barnes and Partridge, 2003; Partridge *et al.*, 2005; Barnes *et al.*, 2006; Van Straalen and Roelofs, 2006). A similar debate is occurring over whether the reduction of eyes in cave animals is the result of resource allocation or the pleiotropic effects of signalling genes (Fong *et al.*, 1995). This debate has been spurred by several observations calling into question the importance of energy conservation, including that eye reduction appears not to be related to environmental resource availability (Jeffery, 2005; Mejía-Ortíz and Hartnoll, 2005). Distinguishing between resource allocation in the broad and narrow sense, and between proximate and ultimate causes, may help clarify these debates. Any negative association between the magnitudes of two traits, however

caused, must involve resource allocation in the broad sense, but need not involve direct competition for resources (i.e. resource allocation in the narrow sense). Furthermore, the existence of proximate, molecular signalling mechanisms that act at the cellular level to cause trade-offs does not necessarily preclude the existence of ultimate, evolutionary mechanisms that generate adaptive optimization of resource allocation at the level of the organism (Lessells and Colegrave, 2001; Bochdanovits and De Jong, 2004). In any case, mapping out the network of cascading effects that genes have on life-history trade-offs will be very challenging because genes and their products may have pleiotropic effects in diverse ways, e.g. through differential effects at different ages and in different tissues, and through differential effects on other genes and their phenotypic expression (Leroi *et al.*, 2005).

1.9 Conflicts over gene expression

Stearns and Magwene (2003) have suggested characterizing trade-offs in terms of conflicts between functions over genome-wide patterns of gene expression. Such studies are not meant to offer an alternative to resource allocation in explaining trade-offs, but rather to increase our understanding of the interconnections between the genotype (genome) and the resource allocation phenotype (metabolome). This kind of work awaits formidable challenges with respect to statistically analysing and functionally interpreting huge amounts of gene expression data (Stearns and Magwene, 2003; Van Straalen and Roelofs, 2006; Roff, 2007). However, Bochdanovits and De Jong (2004) have already used a quantitative genomic approach to show that the expression of each of 34 genes in *D. melanogaster* causes opposite effects on two negatively associated life-history traits: larval survival and adult size. Furthermore, several of these genes affect energy metabolism and protein synthesis, thus suggesting that they act on the trade-off between larval survival and adult size via resource allocation.

2. Absent or Masked Trade-offs

Although various mechanisms may cause trade-offs between traits, trade-offs are frequently not observed (e.g. Reznick, 1985; Glazier, 1999; Reznick *et al.*, 2000). The absence of apparent trade-offs between life-history traits, such as reproduction and somatic maintenance, may be explained in several non-exclusive ways (Glazier, 1999):

1. Phenotypic differences among individuals may mask underlying genetic trade-offs between traits (Reznick, 1985; Glazier, 1999). For example, a model developed by Malausa *et al.* (2005) indicates that phenotypic plasticity in resource allocation may obscure negative genetic correlations between traits. Furthermore, experimental studies have shown that the magnitude and even sign of genetic correlations between traits may depend on environmental conditions (Sgrò and Hoffmann, 2004).

2. Since organisms are highly integrated multi-trait systems, trade-offs between every pair of traits should not be expected. For example, a trade-off between two

life-history traits may not be observed because both traits covary with one or more other traits (Roff, 1992, 2002; Rollo, 1995).

3. Enhanced resource input or reduced maintenance costs in favourable environments may alleviate conflicts over resources by different traits (e.g. Hirshfield and Tinkle, 1975; Tuomi *et al.*, 1983; Reznick *et al.*, 2000; but see Van Noordwijk and De Jong, 1986). This explanation is supported by several studies showing that trade-offs between life-history traits are stronger when food supply is low versus high. Allocation-related trade-offs should be expected only when the upper limits of resource uptake and metabolic performance have been reached (Weiner, 1992; Rollo, 1995).

4. Body reserves accumulated during times of plenty, and increased efficiencies of resource use, may allow animals to avoid trade-offs between specific life-history traits during times of food scarcity (Tuomi *et al.*, 1983; Rollo, 1995).

5. Since growth, reproduction and other energy-intensive activities are costly, natural selection may favour restraint (risk avoidance) so as to prevent costs (trade-offs) from being fully expressed (Rollo, 1986; Jönsson and Tuomi, 1994). Furthermore, natural selection should eliminate any individuals with non-optimal resource allocation strategies, thus severely limiting the range of phenotypes and genotypes over which trade-offs may be detected (Sibly and Calow, 1986; Roff and Fairbairn, 2007).

6. Organisms may adjust specific components of their energy budgets so as to prevent excessive energy costs that may negatively impact other functions. For example, various vertebrate animals have been observed to reduce activity or maintenance costs, thus freeing up more energy to support growth, reproduction or strenuous exercise, without substantially elevating total metabolic rates (Gittleman and Thompson, 1988; Wieser, 1989; Deerenberg *et al.*, 1998; Krockenberger, 2003; Vezina *et al.*, 2006). Furthermore, individuals may use different levels of compensation to offset varying levels of reproductive expenditure, thus accounting for why relationships between reproductive effort and field metabolic rates have been difficult to detect (Vezina *et al.*, 2006).

7. Resource association between two or more traits may also reduce energy costs that may have negative fitness effects. For example, koalas (*Phascolarctos cinereus*) do not exhibit increases in their field metabolic rate during the energy expensive period of lactation, apparently because they use heat generated by food processing and milk production for thermoregulation, a resource association that entails large compensatory energy savings (Krockenberger, 2003). As a result, the cost of lactation is masked, despite a nutrient-poor diet of *Eucalyptus* leaves.

8. Life-history trade-offs may be obscured by variation in resource acquisition that allows some individuals, populations or species to increase the energy expenditure of multiple functions simultaneously, thus resulting in positive correlations between traits. For example, positive correlations between somatic and reproductive investments among populations of the freshwater amphipod *G. minus* appear to be the result of variation in resource availability among the springs that they inhabit (Glazier, 2000a). Similarly, species of white-footed mice (*Peromyscus*) that inhabit resource-rich habitats have been able to increase energy expenditures for both maintenance and reproduction relative to those species from resource-poor habitats (Glazier, 1985a,b; Mueller and Diamond, 2001). Other interspecific comparisons

showing positive associations between metabolic rate and life-history traits in mammals (McNab, 2002; White and Seymour, 2004) may be the result of interspecific differences in resource availability, as well. Although higher metabolic rates may directly support higher rates of biosynthesis during reproduction (Glazier, 1985b; McNab, 2002), it is also possible that these two traits are independently related to food availability. Higher food availability permits higher feeding rates that require larger, more metabolically costly visceral organs (hence higher metabolic rates; e.g. Daan *et al.*, 1990; Książek *et al.*, 2004) and that also simultaneously support higher rates of offspring production. These associations are supported by experiments that have shown that mice (*Mus musculus*) selected for high rates of food intake have higher metabolic rates (Selman *et al.*, 2001) and produce larger litters (Brien *et al.*, 1984). However, these positive associations should not be expected when food availability is limited or varies little. This may explain why positive associations between metabolic rate and growth or reproductive rate have usually not been observed within species fed uniform diets in the laboratory (e.g. Johnston *et al.*, 2007). In support, higher metabolic rates, induced by implanted thyroxine pellets, were associated with higher growth rates in cotton rats (*S. hispidus*) fed food *ad libitum*, but no association was seen when food was restricted (Derting, 1989). *Ad libitum* rations probably permitted individual variation in food intake, whereas restricted rations did not.

The above hypothesis may explain the lack of a trade-off between fecundity and somatic protection, as seen in mice (*M. musculus*) that were fed diets at different caloric (fat) levels, and then fed a control diet *ad libitum* for >10 days before breeding (Johnston *et al.*, 2006). The mice that were conditioned on a restricted diet unexpectedly showed the highest levels of both fecundity and somatic protection (longevity assurance) because they apparently increased their food intake more when placed on the control diet than did the mice given other diets. The mice conditioned on the restricted diet enhanced their somatic protection, as predicted by resource allocation models (e.g. Shanley and Kirkwood, 2000), and then additionally took advantage of the later increased availability of food by markedly increasing their fecundity, as would be expected since *M. musculus* is known to be an opportunistic breeder (Perrigo, 1990).

3. General Conclusions

Organisms are informed resource users (see also Glazier, Chapter 2, this volume). They have evolved diverse resource management systems to cope with a variety of challenging environmental conditions. Because of limited and variable resources, organisms have evolved priority systems for allocating resources to various activities and structures in a hierarchical fashion. In environments where adult survival is good, natural selection has favoured iteroparous species that prioritize maintenance over reproduction. By contrast, in environments where adult survival is poor, natural selection has favoured semelparous species that prioritize reproduction over maintenance. A variety of iteroparous animals appear to place highest priority on the brain and central nervous system, and least priority on the gonads and fat tissues.

Resource allocation priority systems provide many insights into several basic features of organisms, including the relative variability, developmental timing, functional importance and evolvability of traits, and how each trait is related to other traits. High-priority traits are less variable (phenotypically and genetically), appear earlier in development, are functionally more important and seem to be less evolvable than low-priority traits. In addition, since all activities and structures use resources, trade-offs between traits always involve resource allocation, though the mechanisms causing these trade-offs may vary. However, trade-offs between traits may not occur because of resource association (sharing) among traits, variation in resource acquisition, or for other reasons that are reviewed in this chapter. In particular, if variation in resource acquisition exceeds that of variation in resource allocation, positive associations between energy-requiring traits may occur. This perspective appears to provide insight into why positive correlations between maintenance metabolism and growth, reproduction and other life-history traits are often observed among species with different resource availabilities, but not within species given uniform ration levels.

Despite the insights that the study of resource allocation priority systems can provide for understanding phenotypic variation, they have received little consideration in this respect (Glazier, 2002b). This is a glaring omission especially considering that we still do not have a coherent theory of phenotypic variation, even though variation is of central importance to developmental and evolutionary processes (Hallgrímsson and Hall, 2005). To achieve this goal, Hallgrímsson and Hall (2005) call for an integration of morphometrics, developmental biology and population genetics. However, a truly general theory of phenotypic variation should also draw on physiology, behaviour and ecology, and in particular patterns of resource acquisition, allocation and association. After all, the sizes and shapes of biological structures, and the rates and timing of biological processes all depend on spatial and temporal patterns of resource supply and allocation (cf. Sibly and Calow, 1986; Beilharz *et al.*, 1993; Rollo, 1995). In addition, patterns of resource acquisition and allocation may affect both the variance and covariance of organismal traits, and thus potentially their evolvability (Houle, 1991; De Jong and Van Noordwijk, 1992; Glazier, 2002b).

A focus on the regulation of resource use throughout the life history of organisms shows great potential for conceptually unifying all of the fields of biology. All organisms use genetic, neural, endocrine and (or) other forms of information to regulate the timing, rates and spatial distribution of resource use. These patterns of resource use, in turn, affect the resource regulatory systems themselves, which also require resources for their operation. Exploring this nexus of interactions brings us face to face with the most fundamental questions in biology, including: How are the genotype and phenotype related? How and why do organisms develop and evolve the way they do? How do organisms cope with environmental change? And so on.

Given that a finely adjusted interdependence between information use and resource use is essential for maximizing the evolutionary fitness of organisms, it is not surprising that regulatory systems, such as the brain and central nervous system, have the highest priority for resources. The brain evolved to help organisms cope with environmental vicissitudes (Allman, 1999), and given this critical role, the body

has in turn evolved to protect the brain from these vicissitudes. Thus, the regulatory systems of the body are also themselves most tightly regulated. A new focus on these regulatory systems as causes and results of resource allocation promises to expand the scope of life-history research, both theoretically and empirically, to include a variety of biological fields not traditionally included. Some of these fields that are already gaining attention include molecular biology, neurobiology, endocrinology and immunology. By including these fields, we will be in a better position to sort out the proximate and ultimate causes of the various resource investment strategies that we see in the living world. As a result, the statement by Stearns (1992) that 'life histories lie at the heart of biology' will ring all the more true.

This expanded view of life histories and phenotypic variation should have not only broad theoretical significance, but also practical significance for improving human health and farm animal production, both of which require a better understanding of how phenotypes are integrated and regulated. The animal and health sciences, too, can benefit from a resource-use perspective (cf. Hammond, 1947, 1952; Beilharz *et al.*, 1993; Rauw *et al.*, 1998; Worthman and Kuzara, 2005).

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4 Metabolic Constraints to Resource Allocation

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1. Introduction

One of the major goals of evolutionary physiology is to understand the intrinsic and the extrinsic factors that impose limitations on an animal's energy budget (McNab, 2002). Animals cannot expend energy at will and it is well known that a negative relationship exists between the rate of energy expenditure and the duration of activity that an organism is performing (Weiner, 1989; Peterson *et al.*, 1990; Speakman, 2000). In this sense, burst metabolic rates of activity or thermoregulation performed during short periods of time (i.e. minutes, hours) cannot be sustained indefinitely because organisms are not in energy balance during the exertion (Hammond and Diamond, 1997). In fact, an important part of the expenditure is fuelled by the body's reserves, which are depleted while activity is maintained. On the other hand, during longer activity periods (i.e. days or weeks), energy expenditure must be fuelled by concurrent energy intake, which is known as the sustained metabolic rate (SusMR). Specifically, SusMR is defined as a 'time-averaged energy budget that an animal maintains over times sufficiently long that body mass remains constant because time-averaged energy intake equals time-averaged energy expenditure' (Hammond and Diamond, 1997).

An important difference between burst and long-term expenditures concerns the level they can achieve. Rates of expenditures sustained over longer periods are limited to a lower level than rates of expenditure sustained over shorter periods. Specifically, SusMR is almost five times lower than burst expenditures (Bozinovic, 1992; Bundle *et al.*, 1999), and hardly exceeds seven times the resting levels (Peterson *et al.*, 1990). For small mammals in particular, asymptotic ceilings on SusMR could limit individual reproductive effort (since offspring number and quality depends on milk production; Knight *et al.*, 1986; Rogowitz and McClure, 1995; Rogowitz, 1996, 1998), activity (i.e. foraging and escape from predators), thermoregulatory capabilities and survival to long-term cold exposures (Konarzewski and Diamond, 1994; McDevitt and Speakman, 1994a), as well as other ecological processes, such as diet selection, geographical distribution and breeding ranges. This is because

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ceilings on sustainable energy expenditure represent the upper limit below which all energy-consuming activities performed by an individual must be engaged. Thus, given the important ecological and evolutionary consequences that sustained energy budgets have on many aspects of animal life, it is important to determine which factors are imposing ceilings on SusMR.

2. Early Ideas on Energy Budget Limitations

Poo *et al.* (1939) analysed the changes in weight and protein concentration of different internal organs during pregnancy and lactation in albino rats. As a final conclusion to this work the authors pointed out that:

> whether the decrease in serum, clot and carcass protein is a depletion effect arising because the gastrointestinal tract was mechanically incapable of carrying the quantity of food containing only 16% of protein that was required to meet the increased demand [i.e. a digestive central limitation] is a question that can be answered by observing the effect of foods with higher protein concentrations. If with increase in total protein consumption we no longer find any decrease in the amount of protein allocated to these tissues, the simple depletion hypothesis is validated. But if this decrease is found even when more than adequate quantities of protein are taken, we must look for some other mechanism [i.e. a central non-digestive limitation or a peripheral one].

However, after this work there were no further attempts to determine what imposed a limit to SusMR, and the dominant view until the 1980s was that digestive processes limited energy budgets. This view was based on some circumstantial evidence, such as the existence of a maximal food consumption capacity (see Karasov and McWilliams, 2005) and the experimental evidence of a 'digestive bottleneck' (see Jeschke *et al.*, 2002; Karasov and McWilliams, 2005).

However, during the 1960s and 1970s it was observed that digestive features (i.e. gut morphology and histology) of several species change on a seasonal basis, in parallel to alimentary and reproductive cycles (e.g. Davis, 1961; Myrcha, 1964, 1965; Juszczyk *et al.*, 1966; Gebczynska and Gebczynski, 1971; Ankey, 1977). In addition, at the same time experimental studies demonstrated that changes in environmental factors, such as temperature (Geuze, 1971a,b) or diet quality (Miller, 1975; Moss, 1972), produce notorious adjustments in gut attributes. After these works, and the development of optimal digestion models (Sibly, 1981; Penry and Jumars, 1986, 1987), a myriad of studies demonstrated that the digestive system is probably the most reactive system to change in environmental conditions (for a review see Piersma and Lindstrom, 1997; Starck, 1999; McWilliams and Karasov, 2001; Naya and Bozinovic, 2004; Naya *et al.*, 2007). All these studies on digestive flexibility indicate that if there is a digestive limit to SusMR, it is not a rigid, but a highly flexible one (Karasov and McWilliams, 2005).

3. Central, Peripheral or Optimal Design?

Drent and Daan (1980) reviewed the evidence on energetics of reproduction in birds, and reached the conclusion that a 'prudent parent' should not allocate

more than four times its basal level of energy expenditure to reproduction. At the same time, Kirkwood (1983) analysed maximum metabolizable energy intake in mammals and birds, and suggested the existence of a shared absolute ceiling for all the species evaluated. Finally, Peterson *et al.* (1990) reviewed the evidence of metabolic scope (i.e. the ratio between sustained and basal metabolic rate) for 37 species of vertebrates, and found that in all the cases the value was less than 7, and for most of the species it fell between 1.5 and 5. From these seminal papers to the present, there has been an increasing interest in understanding the effect of design constraints on energy budgets (e.g. Weiner, 1992; Speakman, 2000). Three hypotheses have been proposed to explain the physiological limits on energy budgets: (i) the 'central limitation hypothesis', where the shared central machinery limits SusMR; (ii) the 'peripheral limitation hypothesis', where the energy-consuming machinery limits SusMR; and (iii) the 'symmorphosis hypothesis', where the capacity of the central machinery closely matches that of the peripheral tissues.

3.1 The central limitation hypothesis

This hypothesis proposes that SusMR is limited by the central machinery involved in acquisition, processing and allocation of energy, resources and waste products. In this sense, metabolic limits are independent of the way energy is expended. That is, the same metabolic ceiling will be reached regardless of the mode of energy expenditure, and peripheral organs present always an excess capacity. Although there are different basic processes of central limitation (Speakman, 2000), historically, most authors have suggested that the capacity of energy assimilation is the principal limit for sustainable energy budgets. A way to evaluate the presence of metabolic ceilings, and at the same time to determine if they are centrally limited, comes from laboratory studies in which animals fed *ad libitum* are forced to reach their maximal SusMR under different modes of energy expenditure (e.g. lactation, thermoregulation and activity). If the central machinery is what limits SusMR, metabolic ceilings would reach the same value irrespective of the mode of energy expenditure (but see Bacigalupe and Bozinovic, 2002). However, it should be noted that this procedure does not allow the exclusion of a possible peripheral limitation (see below), because it could happen that by chance, different modes of energy expenditure have equal maximum values. Thus, a way to discriminate between both hypotheses is through a combination of peak energy demands. If central limitation really is the cause of the metabolic ceiling, one would expect a conflict in energy allocation when different high-energy-demanding activities are being performed simultaneously.

3.2 The peripheral limitation hypothesis

This hypothesis proposes that the central processing and transport organs may be able to supply energy and nutrients faster than the peripheral organs can convert and mobilize into work and heat. This implies that SusMR is peripherally limited,

i.e. at the site of energy use. Accordingly, the peripheral limitation hypothesis predicts different metabolic ceilings under different modes of energy expenditure. This is because limits are set by the proper limitations of tissues and organs where the energy is being used, while central organs present an excess capacity (Hammond and Diamond, 1997). Thus, like the central limitation hypothesis, a key way to empirically evaluate peripheral limitations on SusMR is from laboratory studies, in which animals fed *ad libitum* are pushed to their maximal SusMR under different modes of high-energy expenditure (e.g. lactation, thermoregulation and activity). In addition, in experiments that use a combination of peak energy demands, the peripheral limitation hypothesis predicts no conflict in energy allocation because central organs present an excess capacity.

3.3 The symmorphosis hypothesis

Perhaps organisms do not have excess capacities, and the capacity of central organs to supply energy has evolved to match expenditure capacity in peripheral tissues. This possibility, in which there is no limiting step on SusMR, rather an optimized design of organisms, is called symmorphosis (Taylor and Weibel, 1981). Basically, the principle of symmorphosis states that no extra structure is formed and maintained unless it is required to satisfy an organism's functional needs (Taylor and Weibel, 1981). Although this principle was first proposed to study the relationship between structure and function in the mammalian respiratory system, it has since been established as a general hypothesis of economic design (Weibel *et al.*, 1998; Weibel, 2000). Optimal design represents an almost perfect match between structure and function (Weibel *et al.*, 1991; Weibel, 1998). As a result, the structural trait becomes the factor that sets the limit of functional performance (Weibel, 1998, 2000). An important prediction of this principle is that if functional needs change, then structural components must change accordingly. This is because the building and maintenance of structures, above what is actually needed, is costly (DeWitt *et al.*, 1998). In the context of physiological limitations on SusMR, the symmorphosis principle predicts a match between central and peripheral organs and tissues. To test for this match, SusMR should be determined under different levels of demand (e.g. −10°C, 0°C and 10°C for SusMR during cold exposure). The next step is to evaluate the adjustment between the different SusMRs obtained, and the morphometric parameters of central and peripheral organs and tissues (e.g. the dry mass of these organs might be considered a good first approximation). Nevertheless, we must keep in mind that a better quantitative approach is necessary to test for symmorphosis (Weibel, 2000).

3.4 Sorting out the evidence

Many studies of mammalian energetics have confirmed that reproduction is the most demanding period in the life of a mammal female (e.g. Bronson, 1989). Moreover, energy expenditure in offspring during lactation is considered the most

demanding period of the reproductive cycle (Millar, 1979; Oftedal, 1984a,b; Sadleir, 1984; Thompson and Nicoll, 1986; Kenagy, 1987; McClure, 1987; Kenagy *et al.*, 1989). In small-sized species, the amount of nutrients and energy that can be supplied from body reserves obtained prior to lactation is restricted, and thus these organisms strongly depend on an increase in food ingestion at this time (Oftedal, 2000). For these reasons, lactation in small rodents has been widely used as a key stressor in the study of limits to energy expenditure (Speakman and Krol, 2005). However, it should be noted that during lactation, considerable amounts of energy are not metabolized by the mothers, but exported as milk (Kenagy *et al.*, 1989; Speakman and Krol, 2005). This determines that utilization of energy at the end of the system (SusMR) does not match sustained energy intake (SusEI), i.e. the variable that is assessed in most of the studies. Hence, in what follows, we will refer mainly to the current evidence on limitation to SusEI (see Speakman and Krol, 2005).

The fact that peak SusMR falls between two and seven times the basal metabolic rate (Drent and Daan, 1980; Kirkwood, 1983; Peterson *et al.*, 1990) was originally interpreted as evidence for the existence of a central limitation to energy budgets, i.e. regardless of the particular mode of energy expenditure, shared features of energy acquisition and utilization limit energy expenditure to a common value. This idea was reinforced by some experimental results obtained during the last decades of the last century. For example, energy assimilation in the Djungarian hamster (*Phodopus sungorus*) was virtually the same during the peak of cold acclimatization and during lactation (Weiner, 1987). Indeed, a study that combined lactation with locomotor activity (Perrigo, 1987) showed that food intake reached a plateau at high levels of activity, after which mothers cannibalized some of their offspring (*Mus musculus*) or extended lactation (*Peromyscus maniculatus*). Finally, an experiment in mice in which litter size was manipulated found that food intake increased with litter size from 5 to 14 pups, but after this value mothers were not able to further increase their food consumption, and consequently, were not able to rear more pups (Hammond and Diamond, 1992). All these results seem to support the hypothesis of a central limitation, probably at the digestive level. However, as we already mentioned, the alternative hypothesis that food intake was peripherally limited cannot be discarded based on these data (Bacigalupe and Bozinovic, 2002; Speakman and Krol, 2005).

Others studies conducted during the same years provided information that was more in accordance with the peripheral limitation idea. For example, in golden-mantled ground squirrels (*Spermophilus saturatus*) it was observed that, although milk production approached the limit for largest litter size (five pups), mothers were able to increase their energy intake an additional 10% if exposed to cold temperatures (Kenagy *et al.*, 1989). In addition, for Swiss-Webster mice it was demonstrated that energy assimilation rates were two times higher at the peak of lactation at room temperature (Hammond and Diamond, 1992, 1994) than those of virgin females at the peak of cold acclimatization (Konarzewski and Diamond, 1994), and that the effect on food intake of both factors combined was additive, i.e. mothers at 5°C increased their food consumption beyond the values considered the limit at the peak of lactation at room temperature (Hammond *et al.*, 1994). These results suggest that the capacity of the mammary

glands may determine the limit of food consumption during lactation. In order to test this, variation in the number of teats was created surgically while simultaneously varying the number of pups reared, under the rationale that: (i) if the capacity of the mammary gland was limited, then, when mammary tissue was reduced in size, the remaining tissue would be unable to compensate; and (ii) if the capacity of the tissue was centrally limited by the supply of energy, then it would respond to the absence of tissue by expanding its capacity (Hammond *et al.*, 1996). In this experiment it was found that mammary glands were unable to compensate milk production when their size was reduced, again supporting the hypothesis that the system capacity is limited at the site of energy use (i.e. peripherally, Hammond *et al.*, 1996). Finally, three additional lines of evidence also suggested a peripheral limit to energy expenditure. First, contrary to what was observed in Swiss-Webster mice, in the deer mouse (*P. maniculatus*), maximal SusEI during cold exposure exceeded that at the peak of lactation (Koteja, 1996). Second, by manipulating pup numbers and the environmental temperature, Rogowitz (1998) demonstrated that pup demands do not drive the capacity of the mother to deliver milk energy. Third, lactating females fed on low-energy food were able to compensate the deficit by increasing their food intake (Speakman *et al.*, 2001).

Nevertheless, in recent years, the emerging consensus on peripheral limitation on energy expenditure was undermined by a series of studies, conducted by Speakman and co-workers, who evaluated milk production in house mice at thermoneutrality (see Johnson and Speakman, 2001; Krol and Speakman, 2003a,b; Krol *et al.*, 2003). These authors evidenced that, although food intake at 30°C (i.e. within the thermoneutral zone) was lower than at 21°C and 8°C, as predicted by the hypothesis of peripheral limitation, milk production mirrored the pattern of food consumption. This result agrees, at a first glance, with the idea that food intake was driven by pup demands. However, this was not the case because pup growth followed the same trend as food consumption: growth was greater at 8°C than at 21°C, and greater at 21°C than at 30°C. In other words, when these new data are considered together with all the previous evidence, the results can not be explained by an extrinsic limitation due to pup demands, a central limitation by the digestive tract or a peripheral limitation by the mammary gland. As an alternative, Krol and Speakman (2003a,b) proposed the heat dissipation limit hypothesis, which states that the limit to SusEI is central and imposed by the capacity of the females to dissipate heat. This idea could explain why, at 21°C, lactation to support an increased litter size, or lactation plus an additional energy demand did not result in increased food intake or milk production (e.g. Koiter *et al.*, 1999; Johnson *et al.*, 2001). It also explains why at lower temperatures, lactating females are able to increase their food intake (e.g. Rogowitz, 1998; Hammond and Kristan, 2000; Johnson and Speakman, 2001), milk production and pup growth (Krol and Speakman, 2003a,b).

More recently, the same authors suggested that the capacity to dissipate heat may influence lactational performance, and proposed two additional hypotheses: the seasonal investment hypothesis and the saturated neural control hypothesis (Speakman and Krol, 2005). The first hypothesis proposes that the reproductive value of mice offspring born early in the reproductive season (i.e. under cold

temperatures) is higher than that of offspring born later in the season, and that mothers use ambient temperatures as a cue to determine their investment in offspring. The second hypothesis states that endocrine stimulation of food intake reaches a maximum during the latter half of the lactation period, and consequently, food consumption cannot be augmented whatever sort of manipulation is performed on animals; only ambient temperature acting via a different signalling route can do that. However, current data did not offer unequivocal support for any of these hypotheses (Speakman and Krol, 2005).

4. Concluding Remarks

It has been proposed that different patterns of energy expenditure among species (i.e. central versus peripheral, and within this latter category, differences in levels and modes of energy expenditure) could be related to each species' life-history strategy (Koteja and Weiner, 1993; Koteja, 1995, 1996; Hammond and Diamond, 1997). Accordingly, there is an implicit consideration that SusMR is adaptive. However, at this moment, it is difficult to confirm this assertion (but see Koteja *et al.*, 2000). Empirical data on physiological limitations on energy budgets is scant. Only a few studies have been explicitly designed to measure SusMR, and evidence shows that sustained energy expenditure does not exceed seven times the resting expenditure (Speakman, 2000). This fact raises two important questions: (i) Why is energy expenditure during long periods only slightly elevated above resting requirements compared to energy expenditure during short periods?; and (ii) Do organisms function at their physiological limits (Speakman, 2000)?

The answer to the first question has been associated with the potential decrease in fitness a mammal may experience if it expends more energy than it routinely does (Murie and Dobson, 1987; Wolf and Schmidt-Hempel, 1989; Stearns, 1992; Daan *et al.*, 1996; Finkel and Holbrook, 2000; Speakman, 2000). However, the evidence for this trade-off (i.e. energy expenditure versus fitness) is not conclusive (Tuomi *et al.*, 1983; Hare and Murie, 1992; Speakman, 2000). Regarding the second question, organisms could function at or near their physiological limits, but are prevented from doing so because of energy limitations imposed by the environment (e.g. Stenseth *et al.*, 1980; Speakman, 2000). At present, there is no sufficient evidence to offer definitive answers to these questions and neither are we able to conclusively identify which physiological factors may impose limits on SusMR. Hence, there is a need for insightful studies that aim to unravel the type of physiological limits on SusMR (i.e. central, peripheral or symmorphosis) and the steps at which these limits occur.

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5 Homeorhesis During Heat Stress

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1. Resource Allocation

1.1 Production

The last half of the 20th century witnessed unprecedented gains in agricultural productivity worldwide with US agricultural output increasing at an average annual rate of 1.76% from 1948 to 2002 (Fig. 5.1). Growth rates in crop output were 1.62%, while livestock output averaged 1.72%. Output growth can be further broken down into growth related to inputs and that related to total factor productivity. As shown in Fig. 5.1, the real cost of inputs has changed very little since 1948, while the gains in total factor productivity account for all of the growth in output. Reasons for these gains in productivity include improved understanding of nutrient requirements, improvements in ration formulation, improved reproductive practices such as artificial insemination, applying more accurate genetic selection methods, improved animal health and welfare practices and improved housing. New technologies and management tools such as use of recombinant DNA technologies, computerized animal identification systems and improved oestrus synchronization techniques are more recent contributors to improved productivity of domestic animals. All of the above improvements have resulted in improved resource allocation within animals. In other words, improved regulation of resource allocation has been and will continue to be a major opportunity for improved productivity of our domestic animal population (Bauman and Currie, 1980; Collier *et al.*, 2004).

1.2 Nutrient partitioning

Resource allocation is another way of describing nutrient partitioning, which is the physiological process by which the metabolizable nutient pool is divided among tissues. There is abundant evidence indicating that at different stages of the life cycle

 Resource Allocation Theory Applied to Farm Animal Production (ed. W.M. Rauw)

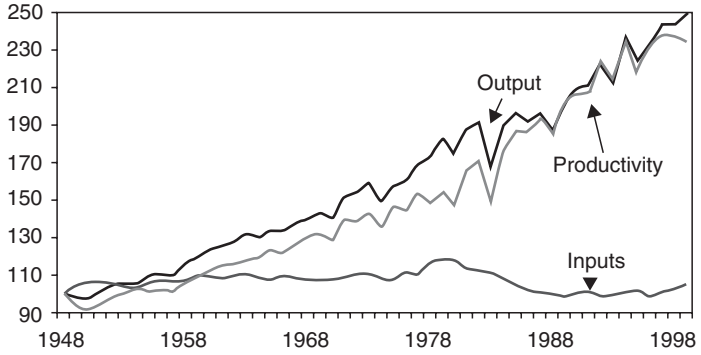

Fig. 5.1. Agricultural productivity in the USA from 1948 to 2002.

various metabolic pathways, such as lipolysis and lipogenesis in adipocytes, muscle and bone, accretion and mobilization of amino acids and calcium from muscle and bone, are up- or downregulated. The net effect of these activities is that nutrients are divided in various amounts to different tissues, biological functions and end products. This is not a homeostatic mechanism of altering nutritional regulation, but is a homeorhetic mechanism caused by changing tissue responses to homeostatic controls.

1.3 Homeorhesis

Homeorhesis is the 'orchestrated changes for priorities of a physiological state' (Bauman and Currie, 1980). The concept was crystallized during research studies involving metabolic adaptations during pregnancy and lactation (Bauman and Currie, 1980), but presently the general concept has been extended to include many other physiological states, nutritional and environmental conditions and even pathological states summarized in Collier *et al.* (2004). The key feature of homeorhetic controls is its chronic nature; several hours and days versus seconds and minutes for the majority of homeostatic events. The homeorhetic process involves hormonal regulation of multiple tissues and physiological systems that results in an overall coordinated response, which is mediated through altered responses to homeostatic signals. In other words, the response of cells to homeostatic signals is up- or downregulated (Bauman and Elliot, 1983; Vernon, 1989; Bell and Bauman, 1997). Interestingly, these same features were identified as features of acclimatization (Bligh, 1976).

1.4 Acclimatization

Acclimatization is a process that takes several weeks to occur and when this process is closely examined it is apparent that it occurs via homeorhetic and not homeostatic mechanisms. As described by Bligh (1976), there are three functional differences between acclimatory and homeostatic responses. 'First, the acclimatory response

takes much longer to occur (days or weeks versus seconds or minutes). Second, the acclimatory responses generally have a hormonal link in the pathway from the central nervous system to the effector cell. Third, the acclimatory effect usually alters the ability of an effector cell or organ to respond to environmental change' (Bligh, 1976; Collier *et al.*, 2004). These acclimatory responses are symptomatic of homeorhetic control mechanisms as described earlier and the overall effect is to coordinate metabolism to respond to environmental change. Thus, the seasonally adapted animal is different metabolically in winter than in summer. Acclimation is the physiological process by which animals reduce impact of stressors in their environment on their biological systems. It is a chronic process, which takes days to weeks to occur and therefore is homeorhetic rather than homeostatic. The end result of acclimation is to change target tissue responses to homeostatic signals. Often this process results in production losses as resource allocation is altered to adjust to the stress.

One example of this process is the acclimatory response of domestic animals to environmentally induced hyperthermia. Heat stress adversely impacts a variety of dairy and beef production parameters including milk yield, growth and reproduction, and therefore is a significant financial burden (~$900 million/year for dairy and >$300 million/year for beef in the USA; St Pierre *et al.*, 2003). Advances in management (i.e. cooling systems; Armstrong, 1994; VanBaale *et al.*, 2005) and nutritional strategies (West, 2003) have alleviated some of the negative impacts of thermal stress on cattle, but production continues to decrease during the summer. Accurately identifying heat-stressed cattle and understanding the biological mechanism(s) by which thermal stress reduces milk synthesis, growth and reproductive indices is critical for developing novel approaches (i.e. genetic, managerial and nutritional) to maintain production or minimize losses during stressful summer months.

2. Resource Allocation During Heat Stress

The biological mechanism by which heat stress impacts production and reproduction is partly explained by reduced feed intake, but also includes altered endocrine status, reduction in rumination and nutrient absorption, and increased maintenance requirements (Collier and Beede, 1985; Collier *et al.*, 2005) resulting in a net decrease in nutrient/energy availability for production. This decrease in energy results in a reduction in energy balance, and partially explains (reduced gut fill also contributes) why dairy cattle lose significant amounts of body weight when subjected to unabated heat stress.

2.1 Lactation

Reductions in energy intake during heat stress result in a majority of dairy cows entering into negative energy balance, regardless the stage of lactation (Moore *et al.*, 2005a). Essentially, because of reduced feed and energy intake, the heat-stressed cow enters a bioenergetic state, similar (but not to the same extent) to the negative energy balance observed in early lactation. The negative energy balance associated with the early post-partum period is coupled with increased risk of metabolic

disorders and health problems (Goff and Horst, 1997; Drackley, 1999), and decreased milk yield and reduced reproductive performance (Lucy *et al.*, 1992; Beam and Butler, 1999; Baumgard *et al.*, 2002, 2006). It is likely that many of the negative effects of heat stress on production, animal health and reproduction indices are mediated by the reduction in energy balance (similar to the way it is during the transition period, which is the period between 2 to 3 weeks prepartum until 2 to 3 weeks postpartum). However, it is not clear how much of the reduction in performance (yield, daily gain and reproduction) can be attributed or accounted for by the biological parameters effected by heat stress (i.e. reduced feed intake versus increased maintenance costs; Baumgard and Rhoads, 2007).

2.2 Growth

In general, heat stress-induced production losses for beef cattle are not as severe as those for the dairy industry. Reasons why growing cattle tolerate higher temperature–humidity index (THI) conditions and exhibit a greater heat strain threshold than lactating dairy cows is not entirely clear, but may involve the combination of various issues including: (i) reduced surface area to mass ratio; (ii) reduced rumen heat production (because of the mostly grain diet); and (iii) reduced overall metabolic heat production (on a body-weight basis). In addition, beef cattle will often experience compensatory gain after mild or short periods of heat stress (Mitlöhner *et al.*, 2001). The combination of these factors translate into heat-related reduced gain that is typically less than 10 kg, which amounts to ~7 extra days in the feed lot (St Pierre *et al.*, 2003). Furthermore, the impact of heat stress on reproductive indices is typically not as severe in beef cattle due to the seasonal nature of breeding programmes (often occurring during the spring in the USA).

3. Metabolic Adaptations to Reduced Feed Intake

A prerequisite to understanding the metabolic adaptations which occur with heat stress is an appreciation of the physiological and metabolic adaptations to thermal-neutral negative energy balance (i.e. underfeeding or during the transition period). Probably because of the reduced economic impact, there is much less known about the metabolic and physiological effects of hyperthermia in beef cattle as compared to dairy cows. Consequently, the changes in heat-related metabolism will be compared and contrasted primarily to the better-known changes in lactating dairy cows.

Cows in early lactation are classic examples of when nutrient intake is less than necessary to meet maintenance and milk production costs and animals typically enter negative energy balance (Drackley, 1999). Negative energy balance is associated with a variety of metabolic changes that are implemented to support the dominant physiological condition of lactation (Bauman and Currie, 1980). Marked alterations in both carbohydrate and lipid metabolism ensure partitioning of dietary-derived and tissue-originating nutrients towards the mammary gland, and not surprisingly many of these changes are mediated by endogenous somatotropin, which is naturally increased during periods of negative energy balance (Bauman and Currie, 1980).

One classic response is a reduction in circulating insulin coupled with a reduction in systemic insulin sensitivity. The reduction in insulin action allows for adipose lipolysis and mobilization of non-esterified fatty acids (Bauman and Currie, 1980). Increased circulating non-esterified fatty acids are typical in 'transitioning' cows and represent (along with non-esterified fatty acids-derived ketones) a significant source of energy (and are precursors for milk fat synthesis) for cows in negative energy balance. Post-absorptive carbohydrate metabolism is also altered by the reduced insulin action during negative energy balance with the net effect of reduced glucose uptake by systemic tissues (i.e. muscle and adipose). The reduced nutrient uptake coupled with the net release of nutrients (i.e. amino acids and non-esterified fatty acids) by systemic tissues are key homeorhetic (an acclimated response versus an acute/homeostatic response) mechanisms implemented by cows in negative energy balance to support lactation (Bauman and Currie, 1980). The thermal-neutral cow in negative energy balance is metabolically flex-ible, in that she can depend upon alternative fuels (non-esterified fatty acids and ketones) to spare glucose, which can be utilized by the mammary gland to copiously produce milk.

4. Heat Stress and Production Variables

4.1 Lactation

Heat stress reduces feed intake and both daily gain in beef cattle and milk yield in dairy cattle. The decline in nutrient intake has been identified as a major cause of reduced production (Fuquay, 1981; West, 2002, 2003). However, the exact contribution of declining feed intake to the overall reduced milk yield or average daily gain remains unknown. To evaluate this question in both dairy and beef cattle we designed experiments that utilized a group of thermal-neutral pair-fed animals to eliminate the confounding effects of nutrient intake. First we used lactating Holstein cows in mid-lactation that were either cyclically heat stressed (THI = ~80 for 16 h/day) for 9 days or remained in constant thermal-neutral conditions (THI = ~64 for 24 h/day), but pair-fed with heat-stressed cows to maintain similar nutrient intake (Rhoads *et al.*, 2007). Cows were housed at the University of Arizona's ARC facility and individually fed *ad libitum* a total mixed ration consisting primarily of lucerne hay and steam-flaked maize to meet or exceed nutrient requirements (NRC, 2001).

Heat-stressed cows had an average rectal temperature of 40.6°C (~105°F) during the afternoons (maximum THI) of the treatment implementation. Heat-stressed cows had an immediate reduction (~5 kg/day) in dry matter intake (DMI) with the decrease reaching nadir at ~day 4 and remaining stable thereafter (Fig. 5.2). As expected and by design, thermal-neutral pair-fed cows had a feed intake pattern similar to heat-stressed cows (Fig. 5.2). Heat stress reduced milk yield by ~14 kg/day with production steadily declining for the first 7 days and then reaching a plateau (Fig. 5.3). Thermal-neutral pair-fed cows also had a reduction in milk yield of approximately 6 kg/day, but milk production reached its nadir at day 2 and remained relatively stable thereafter (Fig. 5.3). This indicates that the reduction in DMI can only account for ~40–50% of the decrease in production when cows are heat-stressed and that ~50–60% can be explained by other heat-stress-induced changes.

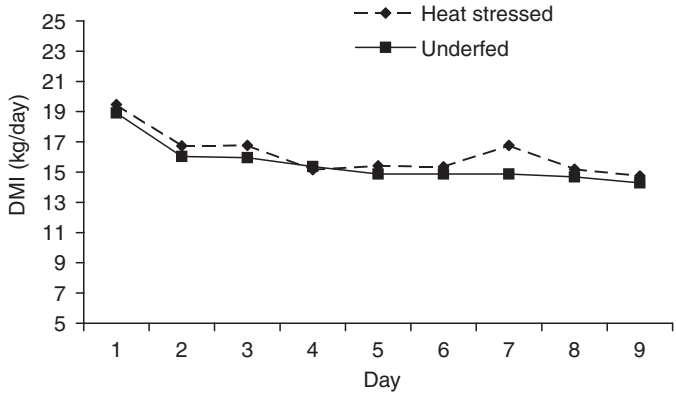

Fig. 5.2. Effects of heat stress and pair-feeding in thermal-neutral conditions on dry matter intake (DMI) in lactating Holstein cows (Rhoads *et al.,* 2007).

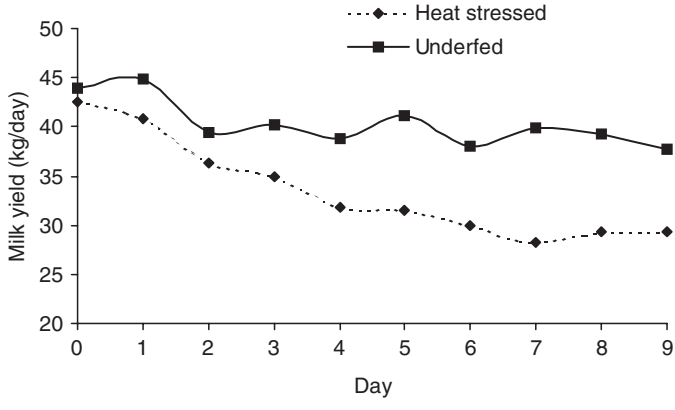

Fig. 5.3. Effects of heat stress and pair-feeding in thermal-neutral conditions on milk yield in lactating Holstein cows (Rhoads *et al.,* 2007).

We have repeated this experiment multiple times and the effects on DMI and milk yield are remarkably consistent (Wheelock *et al.*, 2006; Shwartz *et al.*, 2008).

4.2 Growth

To evaluate the differential effects of heat stress versus reduced nutrient intake in beef cattle we studied growing Holstein beef bulls (n = 12, 4–5 months of age, 136–182 kg body weight; O'Brien *et al.*, 2008). Bulls were either cyclically heat-stressed (29.4–40°C, 25–40% humidity and 12 h of light, conditions slightly warmer than during our dairy experiments) or were maintained in thermal-neutral conditions (18–20°C, 12 h of light), but pair-fed (86% concentrate, 14% protein, 2×/day) with heat-stressed bulls to maintain similar nutrient intake. Heat-stressed bulls had an average rectal temperature of ~40.6°C (105.1°F) during the afternoons (peak ambient THI). Heat-stress-reduced

DMI by ~12% and as expected (and by design) thermal-neutral pair-fed bulls had a feed intake pattern similar to heat-stressed cows (O'Brien *et al.*, 2008). Heat stress eliminated body weight gain and thermal-neutral pair-fed animals had a similar reduction in performance (O'Brien *et al.*, 2008).

4.3 Lactation versus growth

Despite being exposed to a slightly more extensive heat load, heat stress does not reduce DMI to the same extent in growing beef cattle as it does in lactating dairy cows (12% versus 30%). In addition, the reduction in feed intake accounts for only ~50% of the decrease in milk yield (Figs 5.2 and 5.3), but appears to explain most (if not all) of the reduction in growth. Gaining a better appreciation for the biological reasons for the aforementioned discrepancy between beef and dairy may theoretically provide insight on how to prevent or ameliorate the exaggerated decrease in milk synthesis during hyperthermia.

5. Heat and Maintenance Costs

Estimating the energy balance during heat stress, for both dairy and beef cattle, introduces problems independent of those that are inherent to normal energy balance estimations (Vicini *et al.*, 2002). Considerable evidence suggests that heat stress is associated with increased maintenance costs (7–25%; NRC, 2001); however, due to complexities involved in predicting upper critical temperatures, no universal equation is available to adjust for this increase in maintenance (Fox and Tylutki, 1998). Maintenance requirements are thought to increase, as there is presumably a large energetic cost of dissipating stored heat (McDowell *et al.* 1969). Not incorporating a heat-stress correction factor results in overestimating the energy balance and thus inaccurately predicting energy status (Baumgard and Rhoads, 2007).

In the beef study, the pair-fed thermal-neutral controls did not gain or lose body weight suggesting nutrient and energy intake satisfied maintenance requirements (O'Brien *et al.*, 2008). The heat-stressed bulls consumed similar quantities of the exact ration fed to the pair-fed thermal-neutral control animals and also had static body weight. This latter observation may indicate that, at least in growing bulls, heat stress does not increase overall maintenance requirements. If heat stress were to increase maintenance costs as reported (Fox and Tylutki, 1998; NRC, 2001), then the energy requirements of heat-stressed bulls should have exceeded the pair-fed thermal-neutral counterparts. In turn, the heat-stressed bulls would have been consuming inadequate energy/nutrients and should have (by definition) lost body weight. However, this was not the case and heat-stressed bulls did *not* lose body weight (O'Brien *et al.*, 2008), indicating that maintenance cost may not have been increased. Further research is needed to evaluate the effects of heat on maintenance requirements and to determine if physiological state (growth versus lactation) influences energy partitioning during thermal challenges.

6. Metabolic Adaptations to Heat Stress

6.1 Lactation

Due to the reductions in feed intake and increased maintenance costs, and despite the decrease in milk yield, heat-stressed cows enter into a state of negative energy balance (Moore *et al.*, 2005b). In a similar trial as to the one described above, heat-stressed cows entered into and remained in negative energy balance (~4–5 Mcal/day) for the entire duration of heat stress (Wheelock *et al.*, 2006). However, unlike a negative energy balance in thermal-neutral conditions, heat-stress-induced negative energy balance does not result in elevated plasma levels of non-esterified fatty acids (Wheelock *et al.*, 2006; Rhoads *et al.*, 2007). This was surprising as circulating non-esterified fatty acids are thought to closely reflect calculated energy balance (Bauman *et al.*, 1988). In addition, using an IV glucose tolerance test, we demonstrated that glucose disposal (rate of cellular glucose entry) is greater in heat-stressed compared to thermal-neutral pair-fed cows (Wheelock *et al.*, 2007). Furthermore, heat-stressed cows have a much greater insulin response to a glucose challenge when compared to underfed cows (Wheelock *et al.*, 2007).

Both the aforementioned changes in plasma non-esterified fatty acids and metabolic/hormonal adjustments in response to a glucose challenge can be explained by increased insulin effectiveness. Insulin is a potent antilipolytic signal (blocks fat break down) and the primary driver of cellular glucose entry. The apparent increased insulin action causes the heat-stressed cow to be metabolically inflexible, in that she does not have the option to oxidize fatty acids and ketones. As a consequence, the heat-stressed cow becomes increasingly dependent on glucose for her energetic needs and therefore less glucose is directed towards the mammary gland (Baumgard and Rhoads, 2007).

6.2 Growth

Although both the heat-stressed and pair-fed controls quit growing, neither mobilized adipose tissue (plasma levels of non-esterified fatty acids remained <100 μEq/l), which is agreement with a lack of body weight loss (O'Brien *et al.*, 2008). However, despite similar changes in production and post-absorptive lipid variables, there were heat-stress-induced changes in the post-absorptive carbohydrate metabolism. Similar to lactating dairy cows, heat-stressed growing bulls appear to have an increase in glucose disposal rate and have a much greater insulin response to a glucose challenge (O'Brien *et al.*, 2008).

6.3 Lactation versus growth

The changes in lipid and carbohydrate metabolism in heat-stressed dairy cattle may ultimately decrease the glucose availability needed for lactose synthesis. As a consequence, milk yield will decrease (milk synthesis is in large part dependent on lactose synthesis) and this may quantitatively equal the extra amount that peripheral or

extra-mammary tissues (primarily skeletal muscle) utilize. In beef cattle, the magnitude of heat-induced decrease in feed intake is not sufficient enough to signal adipose mobilization in pair-fed thermal-neutral controls. However, there remains the enhanced insulin response to supplemental glucose and an increase in exogenous glucose disposal. A possible reason why beef cattle appear to cope with heat stress better is that their production (tissue synthesis) does not rely on glucose to the extent that milk production does.

7. Theoretical Reasons for Altered Metabolism

Well-fed ruminants primarily oxidize (burn) acetate (a rumen produced volatile fatty acid, VFA) as their principal energy source. However, during negative energy balance, cattle also largely depend on non-esterified fatty acids for energy. Therefore, it appears that the post-absorptive metabolism of heat-stressed cattle markedly differs from that of thermal-neutral cattle, even though they are in a similar negative energetic state. The apparent switch in metabolism and the increase in insulin sensitivity is probably a mechanism by which cattle decrease metabolic heat production, as oxidizing glucose appears more efficient (Baldwin *et al.*, 1980). *In vivo* glucose oxidation yields 38 ATP (assuming that the ΔG (free energy) of ATP hydrolysis is −12.3 kcal/mol under cellular conditions; Berg *et al.*, 2007) or 472.3 kcal of energy (compared to 637.1 kcal in a bomb calorimeter) and *in vivo* fatty acid oxidation (i.e. stearic acid) generates 146 ATP or 1814 kcal of energy (compared to 2697 kcal in a bomb calorimeter). Despite having a much greater energy content, due to differences in the efficiencies of capturing ATP, oxidizing fatty acids generates more metabolic heat (~2 kcal/g or 13% on an energetic basis) compared to glucose. Therefore, during heat stress, preventing or blocking adipose mobilization/breakdown and increasing glucose 'burning' is presumably a strategy to minimize metabolic heat production (Baumgard and Rhoads, 2007).

For dairy cattle, the mammary gland requires glucose to synthesize milk lactose and lactose is the primary osmoregulator and thus determinant of milk volume. However, in an attempt to generate less metabolic heat, the body (primarily skeletal muscle) appears to utilize glucose at an increased rate. As a consequence, the mammary gland may not receive adequate amounts of glucose and thus mammary lactose production and subsequent milk yield is reduced. This may be the primary mechanism which accounts for the additional reductions in milk yield that cannot be explained by decreased feed intake (Figs 5.2 and 5.3).

Heat-stressed cattle require special attention with regard to heat abatement and other dietary considerations (i.e. concentrate/forage ratio, HCO_3^-, etc; Kadzere *et al.*, 2002; Baumgard *et al.*, 2007). In addition, they may also have an extra requirement for dietary or rumen-derived glucose precursors. Of the three main rumen-produced VFAs, propionate is the VFA primarily converted into glucose by the liver. One option to increase rumen propionate production is by feeding highly fermentable starches. However, this strategy may be risky as heat-stressed cattle are already susceptible to rumen acidosis (Kadzere *et al.*, 2002). Further research is needed to identify safe methods of increasing dietary or rumen-derived glucose precursors during heat-stress conditions.

Clearly, heat-stressed cattle implement a variety of post-absorptive changes in both carbohydrate and lipid metabolism (i.e. increased insulin action) that would not be predicted based upon their energetic state. The primary end result of this altered metabolic condition is that heat-stressed cattle have an extra need for glucose, theoretically due to its preferential oxidization in order to reduce metabolic heat. Therefore, any dietary component that increases propionate production (the primary precursor to hepatic glucose production), without reducing rumen pH, will probably increase production.

8. Heat Stress During Pregnancy Results in Placental Insufficiency

Resource allocation of the heat-stressed maternal and fetal systems during pregnancy results in intrauterine retardation of fetal growth. Smaller birth weights and increased mortality rates were observed in ewes lambing in the summer months in northern Australia (Shelton and Huston, 1968). In these semitropical regions, exposure to warmer climates was associated with smaller birth weights indicating that environmental heat stress slows the rate of fetal growth. This association was confirmed under experimental conditions conducted on pregnant ewes or dairy cows that were exposed to chronically elevated ambient temperatures from mid- to late gestation. The findings revealed that fetuses from heat-stressed dams were smaller than those delivered by pregnant females reared in moderate ambient temperatures (Collier *et al.*, 1982; Reynolds *et al.*, 1985; Bell *et al.*, 1987; Thureen *et al.*, 1992; Galan *et al.*, 1999; Regnault *et al.*, 2002b). Alexander (1978) proposed that heat-induced reductions in fetal growth rates in late pregnancy were caused by an earlier decline in placental growth and impaired placental development, which was reaffirmed by Bell *et al.* (1987).

In early studies, there was some ambiguity in the interpretation about whether the placental limitations precede the fetal growth restriction because some investigators viewed the placenta as an extracorporeal fetal organ. For example, restriction of fetal growth could lead to a smaller placenta; however, this does not appear to be the case in hyperthermia-induced fetal growth restriction. In sheep, the highest rate of placental growth occurs from 40 days of gestational age (dGA) to 75–80 dGA (term is 147 dGA) with maximal tissue accretion rates at approximately 55 dGA (Ehrhardt and Bell, 1995). After 80 dGA the dry matter content of the placenta remains constant, but structural modifications that enhance the placenta's ability to transport nutrients to the fetus continues until term (Wooding *et al.*, 1986). Vatnick *et al.* (1991) and Regnault *et al.* (2002b) have shown that reductions in placenta weights precede decreases in fetal weights. These early reductions in placental mass were due to fewer cell numbers, not smaller placental cells, because DNA content, protein content, and protein/DNA ratios were not different between the heat-stressed cotyledons compared to thermo-neutral control cotyledons (Vatnick *et al.*, 1991). Furthermore, imposing heat stress after the apex of placental growth at 64 dGA also resulted in smaller placental and fetal weights, indicating that exposure to environmental hyperthermia even during later stages of gestation impacts placental development (Bell *et al.*, 1989). Together, these data show that heat stress negatively influences placental formation and function, which leads to fetal intrauterine growth restriction during the final trimester when the fetus is rapidly growing.

A reduction in placental mass was not the only factor contributing to slower fetal growth rates. Evidence for decreased placental oxygen, glucose, and amino acid transport capacity per placenta mass was also apparent in heat-stressed fetuses (Bell *et al.*, 1987; Limesand *et al.*, 2004; Limesand *et al.*, 2007; Regnault *et al.*, 2002a; Thureen *et al.*, 1992; Wallace *et al.*, 2005). The rationale for impaired placental transport capacity stems from the findings that the uterine vein and umbilical arterial difference for oxygen and glucose were greater in fetuses previously exposed to chronic heat stress. Increased gradients between the mother and fetus are hallmarks for placental insufficiency, and might be partially compensated for by a reduction in umbilical blood flow. This physiological response increases the ratio of uterine to umbilical blood flow, which maintains equal clearance rates per tissue mass for ethanol, a blood flow-limited molecule (Bell *et al.*, 1987; de Vrijer *et al.*, 2004; Regnault *et al.*, 2003). These findings indicate that uterine blood flow is not disturbed by uneven perfusion or abnormal shunting bypassing the placenta, rather oxygen or nutrient clearance is impeded.

Abnormal placental vascular organization and angiogenesis have been examined as potential causes for insufficient placental diffusion (Regnault *et al.*, 2002a). Placental vasculature resistance has been shown to be increased for all umbilical artery resistance indices (Regnault *et al.*, 2002a, 2003). In the heat-stressed placenta, angiogenic growth factors (vascular endothelial growth factor, VEGF and placenta growth factor, PlGF) and their receptors have aberrant expression patterns during gestation in relation to transplacental oxygen diffusion (Regnault *et al.*, 2002b, 2003), thus providing evidence for placental diffusion deficiencies.

Attenuated umbilical (fetal) uptakes of glucose have also been documented in heat-stressed fetuses, showing that not only is the oxygen diffusion capacity reduced, but that facilitated glucose transporters (GLUT) were also decreased. Thureen *et al.* (1992) used glucose clamp procedures to assess placental glucose transport over a range of steady state maternal-fetal transplacental plasma glucose concentration gradients. The relationship for placental glucose uptake was lower than the normal thermo-neutral pregnancies and the absolute placental glucose transport is reduced by approximately 64% in the heat-stressed sheep fetuses (Thureen *et al.*, 1992). This difference in weight-specific placental glucose transport in the heat-stressed placenta corresponds with lower expression of placental GLUT-1 mRNA and GLUT-3 mRNA (S.W. Limesand, T.R.H. Regnault and W.W. Hay Jr, unpublished data), and GLUT-8 mRNA (Limesand *et al.*, 2004) in heat-stressed pregnancies at later stages of gestation (135 dGA). In the heat-stressed placenta, reductions in facilitated glucose transporters will lower the placenta's permeability for glucose (e.g. the capacity to move glucose from mother to fetus), and explain the greater difference between uterine and umbilical arterial glucose concentrations.

In addition, two essential amino acids, leucine and threonine, were reported to have markedly lower transport into the pregnant uterus and from the placenta into the fetal plasma compartment in heat-stressed fetuses compared to normal, thermoneutral fetuses (Anderson *et al.*, 1997; Ross *et al.*, 1996). Confirmation of inadequate amino acid transport was demonstrated for branch-chain amino acids using a nonmetabolizable neutral amino acid aminocyclopentane-1-carboxylic acid (ACP) to examine entry rates and movement into the fetal compartment. The ACP transport was lower in heat-stressed fetuses with severe growth restriction. Together, these studies show that active amino acid transport systems within the placenta are

also negatively influenced by heat stress during pregnancy, and decrease placental amino acid transport capacity to the growing fetus.

9. Placental Insufficiency Induces Developmental Adaptations in Fetal Metabolism

Testing the hypothesis for hyperthermia-induced placental insufficiency and fetal growth restriction has been the focus of several investigations since the late 1980s (Bell *et al.*, 1987; Regnault *et al.*, 2002a; Wallace *et al.*, 2005). We discussed earlier how heat stress affects the placenta's ability to modulate nutrient reallocation to the developing fetus. Surprisingly, very few investigations have been performed on heat-stressed fetuses to determine the impact of hyperthermia-induced placental insufficiency on the fetus and the developmental adaptations caused by intrauterine growth restriction in the fetus's organs and tissues, even though this is a widely accepted phenomenon in ruminant species.

Ultrasonographic measurements indicate that biometric parameters for determining fetal growth retardation, for example, abdominal circumference, begin to diverge from normal as early as 70 days gestational age (dGA; mid gestation) (Anderson *et al.*, 1997; Galan *et al.*, 1999), which is just prior to rapid fetal growth and after the apex of placental growth. Furthermore, these measurements parallel the decreases in fetal weights observed at 55 dGA (~15 days in treatment (dit)) when no reduction was apparent, at 90 dGA (~50 dit) when there is a tendency for smaller fetuses, and at 130–135 dGA (~80 dit) when a significant reduction of >50% was observed (Anderson *et al.*, 1997; Galan *et al.*, 1999; Regnault *et al.*, 2002b; Ross *et al.*, 1996; Thureen *et al.*, 1992). In all of these studies, when fetal and organ weights were measured, asymmetric fetal growth was found. An asymmetric fetal growth pattern is demonstrated by sparing of the fetal neuronal tissues at the expense of visceral organs; thus, the brain/liver ratio is increased during fetal growth restriction. This signifies that the fetus has altered their normal developmental programme to adapt to the low nutrient supply caused by the placental insufficiency.

The endocrine pancreas has been shown to be disrupted by fetal nutrient deprivation and might promote developmental adaptations. In the fetus, β-cells play a role in coordinating fetal growth by releasing the anabolic hormone, insulin, in response to higher glucose and amino acid concentrations (Aldoretta *et al.*, 1998; Limesand and Hay Jr, 2002). The placental insufficiency caused by heat stress results in fetal hypoglycaemia and hypoinsulinaemia. The low circulating plasma insulin concentrations are due to lower fetal insulin secretion because insulin does not cross the placenta. Therefore, we examined the fetal sheep pancreas and found that structural and functional adaptations take place in response to heat-stress-induced placental insufficiency. Furthermore, these adaptations in the endocrine pancreas might promote whole body changes in fetal insulin sensitivity and glucose homeostasis, creating lifelong nutrient allocation deficiencies.

The structural adaptations in the endocrine pancreas following hyperthermia-induced placental insufficiency appear to be specific to the pancreatic β-cells in comparison with other endocrine cell types within the pancreas. At 133 dGA (0.9 of gestation), heat-stressed fetal and pancreatic weights were 58% and 59% less than the thermoneutral controls, respectively, and pancreas weights correlated significantly with

fetal weights (Limesand *et al.*, 2005). These results indicate that the pancreatic mass declines proportionally with the fetal weight, but the endocrine cells only make up 4–6% of the pancreas. The insulin positive area of the pancreas was reduced 42% compared to control fetuses, which corresponds to a 76% reduction in β-cell mass (% insulin + area × pancreas weight). The reduction in β-cell mass was also confirmed by lower insulin mRNA content and insulin protein content in heat-stressed fetuses. No reductions in hormone mRNA contents were found for α-cells (glucagon), δ-cells (somatostatin), or F-cells (pancreatic polypeptide). Further investigations into how the β-cell mass was reduced revealed slower rates of fetal pancreatic β-cell replication rather than programmed cell death in heat-stressed fetuses (Limesand *et al.*, 2005). Reduction in β-cell mass and insulin availability during fetal growth might define a mechanism by which the fetus adapts its somatic growth rate to nutrient availability. If this pancreatic endocrine adaptation that appears necessary to preserve fetal life is not corrected after birth, it might contribute to inappropriate β-cell mass and insulin secretion, which will lead to glucose intolerance.

In addition to structural adaptations, we also showed that the pancreatic islet insulin secretion responsiveness and glucose metabolism was impaired in the heat-stressed fetuses (Limesand *et al.*, 2006). In the heat-stressed fetus, plasma insulin concentrations were 69% lower at baseline and 76% lower after glucose stimulated insulin secretion, and similar deficits were observed with arginine-stimulated insulin secretion (Limesand *et al.*, 2006). Isolated fetal sheep islets from normal control fetuses were shown to respond to glucose as they do *in vivo*. However, the islets isolated from heat-stressed fetuses had greater glucose-stimulated insulin release as a fraction of total insulin content, but the amount of insulin released per islet was significantly less due to their low islet insulin content (82%). Additionally, a deficiency in islet glucose metabolism was also found in the rate of islet glucose oxidation at maximal stimulatory glucose concentrations (11 mmol/l). Thus, pancreatic islets from heat-stressed fetuses have impaired insulin secretion due to reduced glucose-stimulated glucose oxidation rates, insulin biosynthesis and insulin content. This impaired glucose-stimulated insulin secretion occurs despite an increase in the fractional rate of insulin release that resulted in a greater proportion of releasable insulin most likely due to lower insulin stores.

In the sheep fetus with placental insufficiency and fetal growth restriction, a consistent and relatively large difference between the rate of net umbilical (fetal) glucose uptake from the placenta and their whole body rate of glucose utilization was found. This difference demonstrates fetal glucose production accounting for 41% of the glucose utilization rate. It has been shown many times that normal well-nourished fetal sheep do not exhibit measurable rates of glucose production (Hay Jr *et al.*, 1988, 1989). However, heat-stressed fetuses have increased plasma concentrations of catabolic hormones, glucagon and norepinephrine, but not cortisol (Limesand *et al.*, 2006). This in combination with the lower plasma insulin concentrations and relative hypoglycemia creates a hormonal milieu initiating hepatic glucose production via gluconeogenesis (Apatu and Barnes, 1991; Devaskar *et al.*, 1984; Teng *et al.*, 2002). We confirmed this by showing enhanced mRNA expression of hepatic gluconeogenic enzymes, phosphoenolpyruvate carboxykinase (PEPCK) and glucose-6-phosphatase, and no difference in glycogen content in the livers of the heat-stressed fetuses versus thermoneutral fetuses (Limesand *et al.*, 2007). Furthermore, the results indicate that the stimulation of these genes may be

mediated through the cAMP response element binding protein (CREB), which when phosphorylated transactivates PEPCK and G6Pase genes (Bady *et al.*, 2002; Hanson and Reshef, 1997; Thiel *et al.*, 2005). This higher rate of hepatic glucose production puts the heat-stressed fetuses at a disadvantage, requiring them to utilize other substrates for glucose production rather than fetal tissue accretion, likely explaining a potential mechanism for slower growth.

Even though the insulin secretion responsiveness and circulating concentrations are significantly reduced in the heat-stressed fetus with placental insufficiency, they appear to adapt to the hypoinsulinaemia by augmenting their sensitivity to insulin (Limesand *et al.*, 2007). Fetal glucose utilization rates are dependent on plasma insulin concentrations (DiGiacomo and Hay Jr, 1990; Fowden and Hay Jr, 1988). The fetal body weight-specific glucose utilization rates in the growth-restricted fetuses were not different from controls, even though their insulin concentrations were significantly lower (Limesand *et al.*, 2007). Together, these changes in fetal glucose metabolism demonstrate an increased avidity for glucose uptake and utilization by fetal tissues that helps maintain normal rates of fetal glucose metabolism per whole body weight. Moreover, our preliminary data indicate that this increased insulin sensitivity persists after birth, which identifies fetal programming events that can alter nutrient allocation in postnatal life.

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6 Residual Feed Intake

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1. Introduction

Feed intake and its utilization by the animal involve a complex of biological processes and pathways, and interactions with the environment (Fig. 6.1). That individuals of the same liveweight require rather widely different amounts of feed for the same level of production was acknowledged by Byerly (1941) in his preparation for experiments to determine whether or not individual differences in net efficiency of laying hens are inherited. The concept of residual feed intake (RFI) was one of a number of indices for calculating feed efficiency of growing cattle used by Koch *et al.* (1963) which recognized that differences in both body weight maintained and body weight gained affect feed requirements. They suggested that feed intake could be adjusted for body weight and body weight gain, effectively partitioning feed intake into two components: (i) the feed intake expected for the given level of production; and (ii) a residual portion. The residual portion of feed intake could be used to identify animals which deviate from their expected level of feed intake, and was moderately heritable, with efficient animals having lower (negative) RFI.

Because RFI is by definition phenotypically independent of the production traits used to calculate expected feed intake, it allows comparison between individuals differing in level of production during the measurement period. This independence of RFI from production has led some authors to suggest that RFI may represent inherent variation in basic metabolic processes. For example, genetic variation in maintenance energy requirement per kilogram of metabolic liveweight is closely associated with genetic variation in RFI in young Hereford bulls (Herd and Bishop, 2000). In laying hens, variation in RFI is mainly caused by variation in maintenance energy expenditure (Luiting *et al.*, 1991a). In a typical beef cattle herd the feed energy for maintenance represents 60–75% of the total energy requirements of individual breeding cows and the cost of maintaining cows is clearly an important factor in determining the efficiency and profitability of beef production systems (Archer *et al.*, 1999b).

 Resource Allocation Theory Applied to Farm Animal Production (ed. W.M. Rauw)

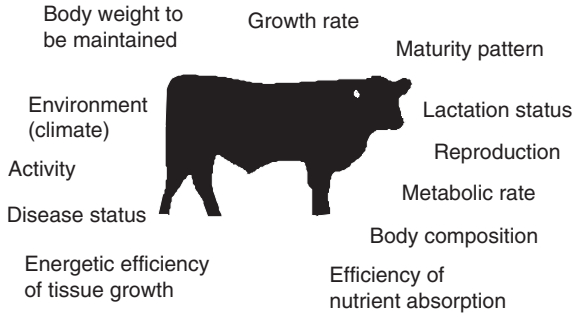

Fig. 6.1. Factors affecting feed utilization by the animal. (From Arthur *et al.,* 2004.)

Proponents of the RFI concept view it as a measure of efficiency that allocates feed eaten to that required for maintenance of liveweight and to components of production. In essence, RFI is a resource allocation theory, from which animal deviants can be identified, these being animals that require more or less feed than predicted. Study of the underpinning biological processes for which feed energy is required for an animal to grow and reproduce in terms of resource allocation can inform prediction of the consequence to the reduction in feed intake sought by breeding animals for lower RFI.

Over the past decade there has been greatly increased interest in breeding to improve beef cattle feed efficiency, which has been regularly reviewed (Archer *et al.*, 1999b; Herd *et al.*, 2003a; Arthur *et al.*, 2004; Arthur and Herd, 2005). In some reports RFI is also called net feed intake (NFI), referring to its derivation as actual feed intake net (or less) of expected feed intake for liveweight maintained and level of production over a test period. The term 'net feed efficiency' is also used for RFI and derives from it being a measure of feed efficiency net of size and level of production.

This chapter reviews RFI in the context of its potential use for farm animal genetic improvement aimed at reducing the feed cost of production. Evidence for phenotypic and genetic variation in RFI is presented. Causes of variation in the biological processes likely to contribute to differences in RFI are explored. The consequences of selection for low RFI are reviewed, with an emphasis on favourable, and potentially unfavourable, outcomes in terms of farm animal productivity and profitability.

2. RFI and Livestock Production

Providing feed to animals is a major cost input in almost any animal production system. For the pig and poultry industries, where cost of feed is easily quantified, the importance of the cost of feed has long been recognized and some improvements in feed efficiency, through both genetic and non-genetic means, have been made (Fairfull and Chambers, 1984; Luiting, 1991; De Vries and Kanis, 1992). Although the cost of providing feed to sheep and cattle in extensive grazing industries is more difficult to quantify, it is nevertheless a major cost of production, and improvement in the output of product per unit of feed used over the whole production system

would be of significant economic benefit. Non-genetic avenues for improvement of production system feed efficiency include many management choices such as housing, health treatments and feeding system. Genetic improvement can include choice of livestock breed, cross-breeding and selection within breeds.

The majority of national genetic improvement programmes for farm animals have traditionally emphasized within-breed selection for increased output, such as number of eggs in poultry, wool in Merino sheep, turnoff weight in meat sheep and beef cattle breeds, and milk from dairy cows. More recently, fertility and product quality and/or composition traits have also been included in breeding decisions. Direct selection to reduce the major cost of production, by selection for lower feed intake, has been problematical. Feed intake is strongly associated with level of production, so direct selection against feed intake has been viewed as undesirable. Feed intake is also expensive and laborious to measure on large numbers of individual animals despite recent advances in electronics and computerized feed intake recording.

Considerable individual animal variation in feed intake above and below that expected or predicted on the basis of size and growth rate have been reported in, for example, poultry (Luiting and Urff, 1991), pigs (Foster *et al.*, 1983; Hoque *et al.*, 2007; Gilbert *et al.*, 2007) and cattle (Archer *et al.*, 1999b). There is also evidence for a genetic basis to this variation in efficiency across these species, with estimates for the heritability of RFI that range from low to moderate presented in the reports just aforementioned and in the reviews by Archer *et al.* (1999b) and Pitchford (2004). In beef cattle, at least, there is evidence for differences between breeds in RFI under feedlot conditions (Schenkel *et al.*, 2004b; Moore *et al.*, 2005). Within beef cattle breeds, genetic variation in feed efficiency has been regularly reviewed and published (Archer *et al.*, 1999b; Herd *et al.*, 2003a; Arthur *et al.*, 2004; Robinson and Oddy, 2004; Arthur and Herd, 2005; Nkrumah *et al.*, 2007). Only in high-producing dairy cows do some studies report a zero or near-zero heritability, but these may reflect the method of feeding or small dataset examined, rather than a true lack of underlying genetic variation (Pitchford, 2004).

The opportunity to improve production system efficiency through exploitation of genetic variation in RFI is dependent not only on the existence of genetic variation in young animals, but also on the magnitude of the genetic correlations with other key production traits. In beef cattle these traits include growth and feed intake during finishing, and carcass and meat quality traits at slaughter, and in cow traits such as mature size, feed intake, milk production and lifetime reproductive performance.

In beef cattle, this opportunity to improve production system efficiency is demonstrated by research conducted over a 10-year period at the Agricultural Research Centre, Trangie, New South Wales, Australia. Young Angus bulls and heifers tested between postweaning between 8–12 months of age were used to estimate genetic and phenotypic parameters for feed intake, feed efficiency and other postweaning traits. The results presented in Table 6.1 show that there was genetic variation in the traits measured, they had a moderate heritability, genetic improvement in feed efficiency (RFI and feed conversion ratio, FCR) could be achieved through selection against RFI and the correlated responses in liveweight and growth rate were minimal.

Following the postweaning test, all heifers entered the cow herd. After the birth of their second calf, cows were not mated and approximately 10 weeks after

Table 6.1. Trangie Angus postweaning bull and heifer, and adult cow, efficiency test trait heritabilities (on the diagonal) and genetic correlations (above the diagonal) for feed intake (FI), test average daily gain (ADG), metabolic mid-test liveweight (MMWT), residual feed intake (RFI) and feed conversion ratio (FCR), and genetic correlations between postweaning traits (below diagonal) and adult cow traits (above diagonal; adapted from Arthur *et al*., 2001b; Archer *et al*., 2002.)

Trait	Mean	FI	ADG	MMWT	RFI	FCR
Postweaning test						
FI (kg/day)	9.65	***0.39***	0.54	0.65	0.69	0.31
ADG (kg/day)	1.26		***0.28***	0.53	−0.04	−0.62
MMWT (kg)	68.8			***0.40***	−0.06	−0.01
RFI (kg/day)	0.05				***0.39***	0.66
FCR (kg/kg)	7.79					***0.29***
Adult cow test						
FI (kg/day)	15.7	***0.28***	0.57	0.45	0.71	−0.57
ADG (kg/day)	1.19		***0.33***	0.37	0.02	−0.87
MMWT (kg)	110			***0.71***	−0.21	−0.12
RFI (kg/day)	−0.54				***0.23***	−0.21
FCR (kg/kg)	14.3					***0.26***
Genetic correlations between postweaning and adult cow test traits						
FI (kg/day)		0.94	0.67	0.69	0.69	−0.12
ADG (kg/day)		0.73	0.72	0.91	0.20	−0.30
MMWT (kg)		0.51	0.39	0.82	0.06	0.05
RFI (kg/day)		0.64	0.22	−0.22	0.98	−0.06
FCR (kg/kg)		0.15	−0.33	−0.54	0.75	0.20

the calf was weaned, the cows were re-tested for feed intake and growth in a similar manner to the postweaning test. Genetic parameters for the adult cow test traits are presented in Table 6.1. All traits were moderately to highly heritable. The results show that there is significant genetic variation in daily feed intake by the cows and the two measures of efficiency: RFI and FCR. The phenotypic and genetic relationships between traits measured during the postweaning and adult cow tests show that selection for lower postweaning RFI will lead to a reduction in the intake of the test ration by dry, non-pregnant cows, together with a slight increase in cow weight, thus improving the efficiency of the cow herd. There are few reports of the genetic correlations of feed intake and RFI from young growing animals to mature adults for comparison. For example, Nieuwhof *et al.* (1992) found a genetic correlation between RFI of dairy heifers measured postweaning with metabolizable energy intake during first lactation of 0.52 and with RFI of 0.58. Strong relationships present the opportunity to utilize selection to improve feed efficiency of growing animals and adult animals simultaneously.

In Australia, the major cattle breed societies have adopted RFI for the purpose of genetic improvement in feed efficiency. Breeding values describe the genetic merit of an animal used in a breeding scheme and are published in the Australian beef recording system 'Breedplan' as an estimated breeding value EBV. Trial EBVs for RFI were first published in 1999 for the Australian Angus breed, and for the Australian

Hereford and Poll Hereford breed in 2002. The Angus RFI EBVs were computed using 2128 animals with individual feed intake records. The EBVs generated ranged from −1.41 to +1.14 kg/day, compared to an average feed intake by the cattle of about 12–13 kg/day (Angus Society of Australia, 2002; D.J. Johnston, 2002, Armidale, personal communication). This implies that in this breed there existed genetic variation in feed intakes ranging from at least 10% below to 10% above that expected on the basis of an animals size and growth rate. This provides an opportunity to select low-RFI bulls for use in breeding programmes to reduce the feed cost of beef production. Fewer records existed for the Hereford and Poll Hereford breeds, with EBVs being computed using 579 animals with individual feed intake records. The EBVs generated ranged from −0.63 to +0.90 kg/day (Australian Hereford Society, 2002).

Comparisons of RFI may be less influenced by pre-test environmental affects than are growth-related traits. For example, Herd and Bishop (2000) showed that RFI over a performance test was not affected by differences in pre-test rearing treatments, in contrast to growth-related traits such as start-of-test weight and end-of-test weight, and in some years average daily gain and FCR. Age of dam is another non-genetic factor known to influence liveweight and growth of young cattle. Arthur *et al.* (2001c) showed that while age of dam affected average daily gain, feed intake, FCR and final weight, it did not affect RFI, in weanling tests on Charolais bulls.

While the utility of RFI for genetic improvement in feed efficiency is attracting the interest of animal breeders, it is worth noting that the concept of RFI can also be used in nutrition studies to detect differences in the efficiency of feed utilization not revealed by measurement of average daily feed intake, average daily gain or FCR, presumably because of the correlation between these traits. The report by Okine *et al.* (2001) provides an illustration of the use of RFI to detect differences in efficiency of utilization of energy in feeds.

2.1 Measurement of RFI

RFI measures whether an animal eats more or less feed than predicted by either accepted feeding standards, or by comparison to measured feed intakes of like-type animals (same breed, sex and age) eating the same feed. Accurate measurement of RFI is a prerequisite for genetic improvement and to understanding the biology of variation.

The basic requirements are that feed in excess of expected requirement is available to all animals so differences in appetite can be expressed, and the component traits (feed intake, liveweight, weight gain, etc.) are measured with sufficient accuracy. Sufficient accuracy is usually obtained by increasing the length of the RFI test period over which the traits are measured. Reduction in residual phenotypic variation resulting from environmental variation, while maximizing genetic variation, is desired. This approach has been used to determine minimum test periods for recording feed intake, liveweight and daily weight gain for the purpose of nutritional and genetic comparisons in beef cattle (Archer *et al.*, 1997; Archer and Bergh, 2000) and pigs (Arthur *et al.*, 2008). Interestingly, while measuring daily feed eaten is often viewed as the most difficult and expensive trait to record, it is actually measuring daily weight gain that has proved more problematical and

determined that test periods are longer than might otherwise be the case if feed intake alone was the only trait of interest. To reduce the length of test (and hence cost) required for daily weight gain, the merit of more frequent weighing of animals has been evaluated (Archer *et al.*, 1999a; Graham *et al.*, 1999; Kearney *et al.*, 2004), as well as approaches to modelling liveweight change over time (Robinson and Oddy, 2001; Schenkel *et al.*, 2002), and feed intake (Schenkel *et al.*, 2004a), but have yet to be routinely adopted for RFI tests of cattle.

National standards for RFI tests for beef cattle were developed in consultation with the major beef cattle breed societies in Australia (Exton, 2001), and may serve as a guide to the conduct of RFI tests in other livestock industries. The guidelines are available for download from http://www.dpi.nsw.gov.au/agriculture/livestock/beef/breeding/general/feed-efficiency (accessed 24 April 2008).

3. Biological Basis of RFI

The existence of genetic variation in RFI offers the potential that selection for low RFI (for higher efficiency) will produce progeny that eat less with no compromise in performance, and thereby provide a real opportunity to significantly reduce the feed cost of animal production. However, the biological basis of such variation is yet to be fully understood. In the late 19th century it was established that farm animals did indeed follow the physical laws of conservation of mass and energy. It follows that, apart from error in measurement of its component traits (feed intake, liveweight, weight gain, etc.), variation in RFI must be underpinned by measurable differences in biological processes.

Efficiency is usually defined as a ratio where both the denominator and numerator have the same units. However, the concept of efficiency is open to misinterpretation. In animal production circles it is used to describe variation in weight (product) of output with respect to feed input. Equal weights of output, for example equal weight gain, can consist of quite different amounts of energy due to variation in composition of the output, such as difference in lean and fat content of weight gain. Feed may deliver different amounts of energy depending on variation in composition, digestion and nutrient absorption and metabolism. Accordingly, it is almost impossible by simple measures of weight of output and feed intake to reliably achieve an understanding of sources of variation in 'efficiency'.

In broad terms there are likely to be at least five major processes by which variation in efficiency can arise (Herd *et al.*, 2004). These are associated with variation in: (i) intake of feed; (ii) digestion of feed (and the associated energy costs); (iii) metabolism (anabolism and catabolism associated with and including variation in body composition); (iv) activity; and (v) thermoregulation.

3.1 Feed intake

Variation in feed intake per se is associated with variation in 'maintenance' requirements of ruminants. As feed intake increases, the amount of energy expended to digest the feed increases also, in part because of a change in size of the digestive

organs. However, the amount of energy expended by the tissues themselves also increases per unit weight of the animal. This heat increment of feeding (HIF) has been known for considerable time (e.g. it was routinely measured by Kellner in the 1890s), and in ruminants is approximately 9% of metabolizable energy intake (Standing Committee on Agriculture, 2000). Webster *et al.* (1975) measured the amount of energy expended in the gut of the sheep as a consequence of eating and estimated that it could account for about 40% of the total HIF. They considered that the remainder was due to increased metabolism in peripheral tissues. Given that selection for RFI is associated with differences in intake, then those animals which eat less for the same performance could be expected to have less energy expended as HIF.

The rate of ingestion and duration of the meal have been reported as key factors in determining the energy cost of eating in cattle (Adam *et al.*, 1984). A study of feeding patterns of Angus steers bred for high or low RFI (Richardson, 2003) reported a trend ($P < 0.10$) for the high-RFI steers to have faster decline in average daily feeding session times over their feed intake test and to spend more time eating early in the test compared to low-RFI steers. Spectral analysis of feeding patterns for another group of these Angus steers found that the high-RFI steers had more variable temporal patterns of feed intake early in the RFI test period compared with low-RFI steers, which appeared to quickly settle into a regular feed intake cycle (Dobos and Herd, 2008). Robinson and Oddy (2004) reported genetic variation in three feeding behaviour traits of feedlot steers, that they had moderate heritabilities, and were positively correlated with RFI, such that higher RFI was associated with longer time feeding per day, more eating sessions per day and faster rate of eating (g/min). Feeding time and number of eating sessions (but not eating rate) also had positive genetic correlations with RFI, indicating that effects of some genes for these feeding behaviours were common with their effect on RFI. Relationships between RFI and feed intake behaviour have been described in pigs also.

3.2 Digestion

It is known that as level of feed intake relative to maintenance increases, the digestion of feed (as measured by total tract disappearance) tends to decrease (SCA, 2000). Over and above systematic variation due to amount of feed eaten, there is also genetic variation in total tract digestion of feed. In ewes from lines of sheep selected for high and low weaning weight, the magnitude of the difference was about 2% units of organic matter digestibility around a mean of 70% (Herd *et al.*, 1993). From these same sheep selection lines, 16-month-old rams from the high weaning weight line were found to have a higher digestibility by 4% units compared to rams from the low weaning weight line when fed near *ad libitum* (Oddy, 1993). Richardson *et al.* (1996) found that young bulls and heifers, phenotypically ranking low or high for RFI, tended to differ in their ability to digest dry matter by about 1% unit when tested on a pelleted ration with a calculated dry matter digestibility of 68%. This difference in dry matter digestibility accounted for 14% of the difference in intake between the two groups of cattle. Digestibility was correlated with RFI in cattle fed a high grain-content ration while housed in individual pens in an animal house. The magnitude of the correlation ($r = -0.44$)

indicated that differences in digestibility accounted for 19% of the phenotypic variation in RFI. The direction of the correlation indicated that lower RFI (higher efficiency) was associated with higher digestibility (Richardson and Herd, 2004). The difficulty in precisely measuring small differences in digestibility suggests that caution should be used in assigning variation in digestion as a major factor in explaining differences in RFI in beef cattle. Studies on monogastrics indicate that differences in digestibility are not important sources of variation in RFI (chickens: Luiting *et al.*, 1994; pigs: De Haer *et al.*, 1993; mice: Bunger *et al.*, 1998).

There is known to be variation in the supply of amino acids due in part to variation in efficiency of microbial protein production in the rumen (Kahn *et al.*, 2000) and appearance in the portal vein (Lush *et al.*, 1991). Between line differences of 9% in microbial protein production per unit of feed intake (as measured by urinary allantoin excretion), and of about 28% in appearance of amino acids in portal blood (direct measure) have been reported in sheep fed relative to liveweight at levels above maintenance (Kahn *et al.*, 2000; Lush *et al.*, 1991). In dairy cows, there is evidence that selection for high milk yield is accompanied by improvement in digestion and/or absorption of dietary energy and protein (Adams and Belyea, 1987).

Together these results suggest that differences in the processes of digestion and in substrate availability, at least in portal blood, do occur. They provide a possible mechanism to explain variation in 'efficiency' of feed utilization, without the need to invoke variation in nutrient utilization per se.

3.3 Body composition and metabolism

The deposition of the same weight of lean tissue and fat has different energy costs. There is more variation in the efficiency of depositing lean gain than fat gain. Theoretical partial efficiencies of nutrient use for fat gain are in the range of 70–95%, and for lean gain about 40–50%. However, there is more variation in efficiency of lean (protein) gain due to greater variation in protein turnover than in fat turnover. Moreover, protein turnover varies to a much greater extent between organs than does fat turnover. Accordingly, any variation in composition of gain, and in composition of the body, can influence the apparent efficiency of nutrient utilization. Notwithstanding any within-organ variation, there is considerable potential for variation in whole animal energy use, simply through differential organ growth. In the few cases where contribution of body composition to genetic variation in heat production or feed efficiency has been studied, it was found that variation in composition was small relative to variation in heat production (Herd *et al.*, 2004). Results for beef steers divergently selected for RFI (Richardson *et al.*, 2001) show that chemical composition was correlated with genetic variation in RFI, with steer progeny of low-RFI parents having less whole-body chemical fat and more whole-body chemical protein than progeny of high-RFI parents. The differences in energy retained in the body accounted for only 5% of the difference in feed intake, with the remainder (95%) due to heat production.

Tissues of the splanchnic bed include the gastrointestinal tract (GIT), liver, spleen, pancreas and mesenteric fat depots. Cumulatively, these organs, together

with the associated connective tissue and blood vessels, comprise approximately 15–20% of the total body mass in ruminants (Seal and Parker, 2000). Estimates of the total oxygen consumption attributed to the tissues of the whole splanchnic bed in ruminants range from 35% to 60% (Seal and Reynolds, 1993), and approximately 20% for the GIT alone (Cant *et al.*, 1996). Eisemann and Nienaber (1990) reported that portal-drained viscera consumed 25.4% and liver consumed 20.5% of whole-body oxygen uptake in steers. In a slaughter experiment using cattle divergently selected for RFI, Richardson *et al.* (2001) concluded that the weight of the highly active tissues of the GIT tract and internal organs were not related to genetic variation in RFI. While this evidence suggests there may be no difference in total weight of these tissues for low and high-RFI steers, oxygen consumption by the portal-drained viscera has been reported to be directly associated with feed intake in beef cattle (Huntington *et al.*, 1988). Given the strong correlation between feed intake and RFI, it is possible that there are associated decreases in oxygen consumption of these tissues following selection for improved (lower) RFI.

Differences in metabolites reported by Richardson *et al.* (2004) are in agreement with the body composition results described above. Leptin concentration, typically associated with increased fatness in cattle (Ji *et al.*, 1997; Chillard *et al.*, 1998; Minton *et al.*, 1998), was positively correlated with steer RFI, and in line with the greater fatness of the less-efficient steers. Urea, reported to be negatively related to protein content in bulls (Robinson *et al.*, 1992), negatively related to lean growth (Cameron, 1992; $r = -0.50$ in Clarke *et al.*, 1996) and positively related with backfat in sheep ($r = 0.24$; Clarke *et al.*, 1996), was positively related to genetic and phenotypic measures of RFI in steers (Richardson *et al.*, 2004). Creatinine, positively associated with muscle mass (Cameron, 1992; $r = 0.35$, Clarke *et al.*, 1996) and negatively associated with fat depth in sheep ($r = -0.44$, Clarke *et al.*, 1996), was negatively associated with steer RFI (Richardson *et al.*, 2004), providing indirect evidence of the greater muscle mass (and possibly also the lower fat content) of the more efficient steers.

The magnitude and direction of genetic correlations for measures of body composition with RFI provide evidence on the size of the effect of genes that affect body composition and RFI. Arthur *et al.* (2001b) found subcutaneous fat depth measured over the 12th/13th ribs and rump to have positive genetic correlations with RFI of $r = 0.17$ and 0.06 in beef weaner bulls and heifers, respectively. For yearling bulls from a number of beef breeds, Schenkel *et al.* (2004b) reported genetic correlations between RFI with fatness traits of similar magnitude to those reported by Arthur *et al.* (2001b), being $r = 0.16$ with scanned backfat thickness and $r = -0.02$ with scanned intramuscular fat percent. So in these young cattle, while these measures of body fat had statistically significant correlations with genetic variation in RFI, they explained less than 5% of the variation in RFI. In young feedlot steers, Nkrumah *et al.* (2007) reported slightly stronger genetic correlations for phenotypic RFI with backfat thickness and marbling fat score, both for scanned measurements on the live animal ($r = 0.35$ and 0.32, respectively) and measurements on the carcass ($r = 0.33$ and 0.28, respectively). In older feedlot steers and heifers, Robinson and Oddy (2004) report genetic correlations of 0.48 and 0.72, respectively, for 12th/13th rib and rump fat depths with RFI, and 0.22 for intramuscular fat percent: evidence for a much stronger association between the effect of genes controlling these measures of fatness and their effect on RFI.

In pigs, where attainment of moderate levels of fatness in the carcass is also required, a moderately strong (and antagonistic) genetic relationship for RFI with carcass backfat thickness ($r = 0.44$) has been reported (Gilbert *et al.*, 2007). The results suggest that the magnitude of the association between body composition and variation in RFI is influenced by age, sex and diet of the test animals.

Differences in body composition have not been reported as being major contributors to variation in RFI in other species. In chickens there are variable reports as to its contribution to the variation in RFI. Luiting (1990) summarized reported genetic and phenotypic correlations of body fat traits with RFI that ranged from −0.40 to 0.45. In a later paper, Luiting *et al.* (1991b) found that the low-RFI line contained 3.4% more fat than the high-RFI line. In mice, improved RFI was associated with a slight increase in fat postweaning and a decrease in fat at maturity (Archer *et al.*, 1998).

Variation in metabolism can impact on heat production. Many of these processes contribute to the maintenance energy requirement of an animal. There are demonstrated differences in efficiency of energy use for maintenance between animals (reviewed by Archer *et al.*, 1999b) and there is evidence that maintenance energy requirement per unit metabolic weight is closely associated with genetic variation in RFI (Herd and Bishop, 2000). Protein turnover in living animals is an energetically expensive process and variation in protein metabolism has been shown to accompany genetic selection for growth and other traits in domestic animals (reviewed by Oddy, 1999). There is genetic variation in energy utilization within a tissue. In sheep and cattle selected for and against growth rate, the amount of energy expended per unit mass of muscle varied between the selection lines by about 20%. A significant part of this variation was shown to be due to differences in the relative rates of protein degradation and protein synthesis within the muscle (Oddy *et al.*, 1995, 1998). Calpastatin, a specific inhibitor of the calcium-activated protease calpain system, and thus protein degradation, has been reported to differ for cattle selected for efficiency of feed use (McDonagh *et al.*, 2001). Richardson and Herd (2004) reported a higher level of total plasma protein and higher blood concentrations of urea and aspartate amino transferase (a marker of liver function indicative for higher levels of protein catabolism) in cattle with high RFI, compared to cattle with low RFI, which together provide evidence for higher protein turnover in high-RFI cattle. Further evidence for an association between protein turnover and RFI was reported by Tatham *et al.* (2000). They found a positive relationship between RFI and plasma creatinine/urea ratio indicative of higher turnover of creatine phosphate in the muscle of high-RFI (low efficiency) bulls.

Changes in efficiency of conversion of feed to gain and in the rate of protein degradation in response to selection for growth and leanness have been observed in many species ranging from chickens (Pym, 1990; Tomas *et al.*, 1991) to rainbow trout (McCarthy *et al.*, 1994). For example, chickens from lines selected for lean gain, or increased efficiency of conversion of feed to gain, had lower rates of fractional protein degradation than control line chickens (Pym, 1990). Moreover, Tomas *et al.* (1991) found that differences in fractional degradation rate were associated with differences in net efficiency of protein utilization. In this study decreased rates of degradation gave rise to improved efficiency of protein gain.

3.4 Activity

Variation in heat production (and thus energy available for maintenance and growth) also occurs as a result of differences in energy expenditure associated with activity. Studies on monogastric species reveal the potential importance of differences in activity to variation in RFI. For example, in pigs, total daily feeding time and number of visits to a feeding station may be positively correlated with RFI (Rauw *et al.*, 2006). Activity contributes to a substantial proportion of the variation in RFI in chickens (Braastad and Katle, 1989; Katle, 1991; Luiting *et al.*, 1991b). Luiting *et al.* (1991b) concluded that 80% of the genetic difference in RFI between lines of chickens divergently selected for RFI could be related to differences in physical activity. In lines of mice divergently selected for heat loss, Mousel (1998) showed that the high heat loss (low efficiency) mice were twice as active as the low heat loss (high efficiency) mice and that this difference in activity accounted for 10.5% of the difference in feed intake between the selection lines. In lines of mice divergently selected for food intake corrected for body weight, Bunger *et al.* (1998) found mice in the high food intake (low efficiency) line to be three times more active than mice in the low feed intake (high efficiency) line.

Differences in activity can also be associated with variation in RFI in cattle. Richardson *et al.* (1999) reported a phenotypic correlation of 0.32 for RFI with 'daily pedometer count' that would indicate that about 10% of the observed variation in RFI was explained by this measure of activity. Mechanisms associated with variation in activity include work involved in feeding, ruminating and locomotion at various speeds. Herd *et al.* (2004) calculated the energy cost of these activities for high- and low-RFI selection-line bulls and heifers under standard test conditions to account for approximately 5% of the increased feed energy intake by high-RFI (low efficiency) selection-line cattle.

3.5 Thermoregulation

The principal route for energy loss in ruminants is evaporative heat loss (through heat exchange in the lungs and nasal turbinates; Blaxter, 1962). To a large extent this is regulated by rate of respiration, yet the author is not aware of any study of the relationship between respiration rate and RFI. Postural change and other adaptations such as wetting, seeking shelter (and huddling) do not by themselves constitute a large proportion of variation in heat loss except in extreme situations.

Hens with lower RFI reportedly have smaller nude body areas through which they could lose energy and were also slightly better feathered and less active (Luiting *et al.*, 1994). Luiting *et al.* (1991b) had earlier suggested that each of these factors was likely to impinge on thermoregulation, and suggested that this may be a contributing factor to variation in RFI in chickens. However, the large difference in body size between these species suggests that the contribution of thermoregulation to variation in energy expenditure could differ markedly.

3.6 Integration of biological mechanisms

Experience with studies of animals from lines selected for other traits (e.g. growth rate and wool production) indicates that no single mechanism is likely to be primarily responsible for the associated change in phenotype (Oddy, 1999). For example, studies of replicated lines of mice selected for divergence in growth rate resulted in mice with similar divergence in the selected trait, but markedly different phenotype with respect to body composition, feed intake, metabolism and activity (Falconer, 1973). There are a limited number of cases in farm animals where a single gene mutation has occurred which led to marked phenotype differences, for example, the mutation in the myostatin gene that causes the double-muscled phenotype in cattle (Grobet *et al.*, 1997). In short, the expectation is that many mechanisms are associated with the RFI phenotype.

In recent years the advent of whole-genome association (WGA) studies, based on proprietary chips able to test for allelic variants in thousands of genes, have been used to find gene variants associated with variation in RFI and to identify possible biological pathways contributing to the observed variation. Barendse *et al.* (2007) conducted a WGA study for RFI measured on feedlot cattle from seven beef breeds in Australia. They found 161 single nucleotide polymorphisms (SNPs), representing 141 genetic regions of the bovine genome, providing evidence for association with RFI for multiple genes in a wide diversity of metabolic pathways that include all the processes described above. Sherman *et al.* (2008) report six SNPs that have an effect on RFI in feedlot cattle in Canada. Not all these RFI SNPs showed association with feed intake and FCR, showing that these SNPs may be affecting the underlying biological mechanisms of feed efficiency beyond feed intake control and weight gain efficiency (Sherman *et al.*, 2008).

That many physiological mechanisms contribute to variation in RFI was shown in experiments on Angus steer progeny following a single generation of divergent selection for RFI (Richardson and Herd, 2004). Difference in energy retained in protein and fat accounted for only 5% of the difference in RFI following divergent selection. Differences in digestion contributed conservatively 10% and feeding patterns 2% to the variation in RFI. The HIF contributed 9% and activity contributed 10%. Indirect measures of protein turnover suggest that protein turnover, tissue metabolism and stress response contributed to at least 37% of the variation in RFI. Approximately 27% of the difference in RFI was due to variation in other processes such as ion transport, not yet measured.

4. Implications for Livestock Improvement

4.1 Production response to selection

Feed efficiency

The existence of phenotypic and genetic variation in RFI in a number of livestock breeds offers the opportunity to improve enterprise productivity and profitability. The direct and correlated responses in the component traits that go into the calculation of RFI is well demonstrated by the comprehensive study of divergent RFI

selection lines in Angus cattle established at the Trangie Agricultural Research Centre in Australia. Given the many biological processes that appear to underpin variation in RFI, it can be anticipated that a number of correlated changes in other important production and fitness traits may result from selection for RFI. Indeed, it is indirect improvement in many of these traits that is sought through implementing a breeding program that includes selection for lower RFI. Responses in the Trangie divergent RFI cattle selection lines are presented as an example of the range of correlated changes that have been observed. Possible consequences to fitness traits are discussed in more detail in Section 4.2.

Direct and correlated responses in postweaning feed efficiency and growth traits resulting from 5 years of divergent selection for RFI produced low-RFI selection-line progeny that had significantly lower RFI (−0.54 kg/day), ate less feed (9.4 kg/day) and had better feed conversion (6.6) than progeny in the high-RFI line (0.71 kg/day, 10.6 kg/day and 7.8, respectively; $P < 0.05$), but did not differ in average daily gain (1.44 versus 1.40 kg/day) over the test period or in final liveweight (384 versus 381 kg; Arthur *et al.*, 2001a). Superior growth and feed efficiency on pasture and in the feedlot was recorded for steers from the low-RFI line compared to those from the high-RFI line. After just a single generation of divergent selection, some trait means for the selection lines could not be shown to differ statistically, but for several other traits, significant regression coefficients with RFI EBVs provided evidence for change accompanying genetic differences in RFI, such as for average daily gain (Herd *et al.*, 2002, 2003b).

When tested as adult cows, those forming the parental generation of the low-RFI selection line ate less feed (15.6 kg/day) and had significantly lower RFI (−0.41 kg/day) than those forming the high-RFI line (16.3 and 0.26 kg/day, respectively; $P < 0.05$), but did not differ in liveweight or in average daily gain over the test period (Arthur *et al.*, 2005). The direction and magnitude of the genetic correlations for postweaning RFI with adult cow traits (see Table 6.l) would indicate that subsequent generations of adult cows in the selection lines should differ in feed intake, RFI, FCR, but not in liveweight, when re-tested on a high-energy ration. The significant, but low, genetic correlation (0.20) with daily gain during the adult cow test reported in Table 6.l was not apparent in the parental generation of cows. Cows subsequently born in the low-RFI selection line have shown slightly superior growth on pasture, to become heavier at maturity than cows in the high-RFI line (Arthur *et al.*, 2005).

In summary, selection for low RFI has been accompanied by a reduction in intake without a compromise in growth, in young bulls and heifers postweaning, in steers in the feedlot and in adult cows fed medium- to high-energy-content rations, and by superior growth and better feed efficiency in steers and cows at pasture: outcomes that would generally be considered favourable for beef cattle enterprises.

Carcass and meat attributes

Evidence exists that in beef cattle there is a genetic relationship between RFI and subcutaneous fat depth, with more efficient (lower RFI) animals being leaner than less-efficient (high-RFI) animals. Correlated changes in body composition and differences in a mechanism of protein turnover have been observed between the

Trangie RFI cattle selection lines. These changes have, or may have in the future, an impact on aspects of carcass and meat quality. Ultrasound measurement before slaughter showed that low-RFI line steers had less subcutaneous fat over their ribs (10.2 mm) and rump (13.1 mm) than high-RFI line steers (11.6 and 14.8, respectively; $P < 0.05$). The low-RFI line steers had less fat depth at the rump on the hot carcass (14.9 mm) and there was a small difference in dressing percentage (52.1) than high-RFI line steers (16.5 mm and 52.9, respectively; $P < 0.05$; Richardson *et al.*, 2001). Statistically significant regressions for the three fat traits and dressing percentage with RFI EBV provided additional evidence of genetic association. Using total tissue dissections of bodies of steers selected for RFI, Richardson *et al.* (2001) reported significant selection-line differences in per cent carcass fat (9.9 versus 11.3) but not in percent retail beef yield. These results demonstrate a realized response in fatness after just a single generation of divergent selection. The regression coefficient for retail beef yield percentage with RFI EBV is favourable but low. Breeding for low RFI will need to be balanced with selection on fat traits to ensure compliance with market specifications for fatness.

After a single generation of divergent selection on postweaning RFI, meat samples taken from the *M. longissimus dorsi* of feedlot-finished steers showed no difference between selection lines in shear force and compression values after 1 and 14 days of ageing, nor in initial concentrations in muscle of m- and μ-calpain (enzymes associated with initiation of muscle fibre breakdown; McDonagh *et al.*, 2001). However, muscle from low-RFI line steers contained a slightly higher concentration of calpastatin (an inhibitor of the action of the calpain enzymes) and lower level of myofibre fragmentation, than muscle from high-RFI line steers. These results provide evidence that ongoing selection for low RFI (high efficiency) could negatively affect meat tenderness, and this association needs to be monitored. Small differences in myofibre fragmentation are consistent with differences in protein degradation and turnover proposed as one of the mechanisms contributing to variation in RFI (Richardson and Herd, 2004).

Maternal efficiency and productivity

Results show that in beef cattle, selection for lower RFI can lead to improvement in cow feed efficiency. Pasture intakes were similar for lactating cows that had previously been ranked as above or below average for RFI measured when they were younger, but the low-RFI cows were 7% heavier, had similar subcutaneous fat levels and reared calves of similar weight to high-RFI cows (Herd *et al.*, 1998). The advantage in efficiency of the low-RFI cows, when expressed as a ratio of calf weight to cow feed intake, was 15% although only statistically significant at $P = 0.07$, presumably due to a small number of animals in the experiment. The results in Table 6.1 discussed previously show that selection for lower postweaning RFI will lead to a reduction in the intake of a pelleted ration by dry, non-pregnant cows, together with a slight increase in cow weight, thus improving the efficiency of the cow herd. These favourable relationships present the opportunity to utilize selection to improve feed efficiency of growing animals and adult cows simultaneously, based on measurements taken postweaning before selection decisions are made.

Maternal productivity of Angus cows divergently selected for RFI were studied across three mating seasons. The cows were the result of 1–2.5 generations of

selection and differed in RFI EBV by 0.8 kg/day. No significant selection-line differences in weight (measured four times a year) were observed, although the low-RFI cows were heavier over the course of the study (Arthur *et al.*, 2005). In general, the cows lost subcutaneous fat (measured two times a year) during the period when they were nursing calves, and gained fat thereafter, with the high-RFI cows having significantly greater rib-fat depths late in lactation at joining time. There were no significant selection-line differences in pregnancy, calving and weaning rates, milk yield and weight of calf weaned per cow exposed to bull. The study indicated that after an average of 1.5 generations of divergent selection for RFI there are no significant selection-line differences for these important maternal productivity traits. The low-RFI line cows tended to calve a little later than the high-RFI cows, presumably because they fell pregnant later in the previous mating season. Such an association with RFI would be unfavourable in herds with a restricted breeding period. It could offset the benefit in cow feed efficiency and will require further research to determine the nature of the association.

4.2 RFI and fitness

In recent times, two main challenges are emerging in the area of genetic improvement in feed intake and efficiency. One challenge is to address the serious problem in high-producing animals, such as in lactating dairy cows and sows, where the high demand for energy following parturition frequently results in negative energy balance and the onset of metabolic diseases. For these animals, the current approach to genetic improvement seems to lean towards the development of strategies to increase feed intake capacity of the animal (Veerkamp and Koenen, 1999). The second challenge applies more to beef cattle and poultry where the energy demands for production are not so high and negative energy balance immediately after parturition is less of an issue. In these species there is evidence of genetic variation in feed efficiency, which can be exploited directly to improve efficiency and profitability of production (Archer *et al.*, 1999b).

A review by Rauw (1998) proposed that genetic selection, so effective in increasing production levels of livestock, has concomitantly increased the occurrence of behavioural, physiological and immunological problems in livestock. Basic to the computation of RFI is calculation of the expected feed intake by an animal based on a measure of its liveweight and an allowance for level or quantity of product output. Rarely is composition of the output considered, nor are differences in locomotion, disease status, immunocompetence or other metabolic processes that use energy. It follows that if there is no allowance made for the energy requirements of these processes, the reduction in feed intake sought by selection for low RFI may compromise an animal's capacity to sustain these functions.

Perhaps paradoxically, for beef cattle at least, it has been hypothesized that susceptibility to stress is a key driver for many of the biological differences observed following divergent selection for RFI. This hypothesis by Richardson and Herd (2004) is supported by measurement of a number of parameters that indicate high-RFI (low efficiency) steers to be more susceptible to stress than low-RFI (high efficiency) steers, and as a consequence metabolize more feed energy than predicted on the basis of

weight and weight gain alone. The postulated greater susceptibility to stress by high-RFI steers refers to a greater likelihood of these animals being affected by particular stressors, and/or having less-effective mechanisms to cope with and adapt to these stressors. Genetic variation in susceptibility to stress has been reported in pigs (Zhuchaev *et al.*, 1996). High rates of cortisol production were reported in pigs and were associated with the stress of frustration or lack of control or predictability of their environment (Dantzer, 1981). It has also been suggested that many of the differences in activity of chickens divergently selected for RFI are a type of frustration behaviour, connected with long-term stress of the animals (Luiting *et al.*, 1994).

The increasing awareness of animal welfare issues, tighter specifications for carcass and meat quality attributes and narrow profit margins in animal production are justification for fuller knowledge of the biological consequences of selection for RFI (Rauw *et al.*, 1998). The problem arises because energy balance during the different stages of the animal's life has usually been assessed as independent events, rather than as part of a whole. There is a need for a better understanding of the energy requirements for maintenance and production (including reproduction) and the variation in the efficiency of energy utilization for these processes throughout the animal's life. In practical terms, there is a need for a better understanding of the genetic and phenotypic relationships between feed intake and the components of production at different phases of the animal's productive life, in order to be able to effectively utilize feed intake to optimally improve whole production system efficiency. The challenge then will be to develop breeding programs that avoids negative energy balance during periods of peak energy demand, while exploiting the variation in the efficiency of feed energy utilization.

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7 Allocation of Resources to Maintenance

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1. Describing Maintenance Requirements

This chapter deals with aspects of the maintenance energy (ME) requirements in growing pigs. This is a risky topic to write about: almost everyone seems to have some intuitive feeling for it, but the scientific literature is full of imprecise and contradictory descriptions and definitions of 'maintenance', and of its requirements. I do not attempt to clarify that particular issue. On the contrary, I assume some common understanding of the matter (rightly or wrongly), and look in more detail at some components of the aggregate. This section considers the various ways the aggregate has been approached, in order to put those components in their proper perspective.

There are at least three operational definitions of when an animal can be regarded to be 'at maintenance'. Close and Fowler (1982): (i) state that 'the concept of maintenance […] relates to an animal in energy equilibrium, neither losing nor gaining energy'; (ii) stress that this would mean that the sum of the energetic equivalents of body protein and lipid deposition (the energy retention) is zero, but not necessarily that *both* these deposition rates would be zero; and (iii) continue with references to studies that have shown that immature animals have the tendency to deposit protein and catabolise lipid when fed at that particular level. The US National Research Council (NRC, 1996) writes in its nutritional recommendations for beef cattle: '[E]nergy maintenance does not necessarily equate to maintenance of body fat, body protein, or body weight.' This is the most common approach.

Others have restricted 'the concept of maintenance' to the true steady-state situation where 'strictly there should be no translocation of material within the animal' (Armsby and Moulton, 1925), so that both protein and lipid deposition rates are zero (e.g. Kielanowski, 1965; Emmans, 1994).

Because it is much more difficult to monitor the body energy balance than to monitor body weight, animals are often assumed to be fed 'at maintenance' when their body weight does not change (long-term trials such as by Taylor and Murray

(1991); short-term trials such as by Kolstad and Vangen (1996) and Ball *et al.* (1998a)) although this may be accompanied by considerable changes in their body energy balance.

Similar to many other authors, NRC (1988), when dealing with maintenance requirements of pigs, seems to have chosen not to commit itself to any of the above options, and to leave the choice to the reader. They immediately focus on the *requirements*: 'Maintenance energy requirements include needs for all body functions and moderate activity. Many factors influence these requirements, including environmental temperature, activity level, group size, stress […] and body composition.' This is in contrast to, for example, Stephens' (1991) description, which would not allow for any heat increment of feeding or activity in its specification of maintenance: 'The term *maintenance requirement* as it is used in nutrition and metabolism literature is essentially conceptual, and represents that portion of heat production which is not attributable to productive processes such as growth, gestation and lactation, or to other identifiable energy costs such as the heat increment of feeding or activity.' In fact, this specification comes close to the fasting heat production (FHP).

Such specifications of maintenance requirements are important in animal science because applied feeding levels are often related to the presupposed maintenance requirement, e.g. 'animals were fed at 2 or 3 times maintenance'. The base level is commonly adopted from recommendations such as the UK Agricultural Research Council (ARC, 1981) and NRC (1988).

A more precise and quantitative description of maintenance energy requirements (ME_{maint}, in kJ/day) was presented by Emmans (1994). When ignoring methane production in monogastrics, his equation builds upon FHP (the energy expended when maintenance requirements are met by metabolism of body tissue) as follows:

$$ME_{maint} = FHP + [w_d \times FOM + w_u \times (UN - FUN)] \quad (7.1)$$

FOM, the faecal organic matter produced from the diet, and UN, the nitrogen excreted in the urine (both in g/day), relate to the steady state with zero protein and zero lipid deposition. FUN represents UN at fasting. The constants w_d and w_u (estimated at 3.8 and 29.2 kJ/g, respectively) translate mass into energy. The heat increment of feeding 'at maintenance' corresponds to the term in square brackets in Equation 7.1, but any heat increment associated with protein or lipid deposition is explicitly excluded from ME_{maint} as both are zero in this approach; as mentioned above, this does not hold for the majority of maintenance studies. Walker and Young (1993) made the same explicit distinction between 'energy used for vital processes' (analogous to Emmans' ME_{maint}) and 'extra energy costs associated with the productive state', and refer to the aggregate as *support costs*: 'the machinery costs necessary to support the animal in a productive state, which have been shown to vary with growth rate'.

The main picture that emerges from all this is one of confusion. There is neither general agreement about what ME_{maint} actually represents nor about its components, and most descriptions are of a qualitative nature. This is not likely to change in the foreseeable future. For our current purposes, the main issue is which metabolic processes should be included in the aggregate.

There is no disagreement about 'physiological service functions' (Gill and Oldham, 1993) such as circulation, coordination, respiration and excretion. The levels of these in the absence of production are commonly included in the 'basal metabolic rate' together with cell maintenance functions such as the active transport of ions through cell membranes (further referred to as 'membrane transport') and turnover of the established body protein mass. It is also common to make some allowance for 'basic activity', which in monogastrics includes little more than just standing upright rather than lying down, but in ruminants sometimes allows for grazing activity.

But actions such as thermoregulation, immune response and coping with other stressors are often excluded from the specification of the aggregate, although the literature is full of references to the apparent maintenance requirements of animals that were not kept in thermoneutral, pathogen-free and welfare-friendly conditions. A similar situation applies to all physical activities beyond the basic level, especially in young animals, and to physiological service and cell maintenance functions 'above maintenance'. Much of the disagreement about the proper definition of maintenance processes seems to stem from the difficulty of separating out the costs involved with the above-mentioned functions from the measured heat production.

Taken together, these views would seem to allow for the quantitative description of the maintenance requirement of a mature animal in metabolic 'steady state' that does not have to cope with any kind of stress on its system and that is engaged in only a basic level of physical activity (cf. Van Es, 1972; Webster, 1988; McCracken, 1992). Naturally, this steady state would require the absence of dynamic processes such as growth, reproduction, lactation or physical work.

Maintenance costs of mature animals have received much scientific attention in the extensive meat production sectors (in the western world mainly sheep and beef cattle). Because of the low prolificacy of these species, a relatively large proportion of the total nutrient input into such production systems is required for 'maternal overhead', i.e. for maintaining the parental generation rather than for bringing the progeny generation to its required slaughter point. The classical study of this issue is by Dickerson (1978), who made use of mid-1970s US performance trait levels to parameterize his bio-economic model (Dickerson, 1982), and calculated that maintenance plus replacement of the parental generation of sheep and beef cattle requires 50–58% of the total feed energy input per kilograms of edible meat protein produced from the slaughter progeny generation. Webster (1989; his Table 1) gives (undocumented) corresponding values of 52–70%. The maintenance costs of the progeny itself play a much less important role in such production systems (17–23% from those same calculations). Hence, the latter issue has been the subject of serious scientific study only since the late 1990s (e.g. Ball *et al.*, 1998a,b, and references provided there).

By contrast, in the intensive meat production systems based on broiler chickens, turkeys or pigs, the maternal overhead requires a much smaller proportion of the total feed energy input (6.5–20% according to Dickerson (1978); 4–20% according to Webster (1989)). The 20% figures are for pigs; its current value would be much lower due to increased reproductive rates since that time (see also Large, 1976). The maintenance costs of the slaughter progeny have a considerable impact on the overall energetic system efficiency (31–60% from those same calculations,

which seem unrealistically high values nowadays). Hence this issue merits, and has attracted, more scientific attention than in the extensive production systems.

We have to consider, then, the maintenance requirements of a growing immature animal that, by definition, is not in metabolic steady state. This makes it again difficult to decide what should be included in our conceptual maintenance processes. Young *et al.* (1989) measured oxygen consumption in fetal, neonatal, growing and adult sheep, and found significantly elevated metabolic rates per $kg^{0.81}$ metabolic body weight during the stage of highest relative growth rate (28–74 days of age). These authors conclude that the elevated metabolic intensity associated with production processes makes the scaling of metabolic rate with a common body weight exponent inappropriate. Hence, when maintenance requirements are expressed as a function of metabolic body weight with a fixed exponent (e.g. α kJ/$kg^{0.75}$/day), they (or rather, α) would become inflated during rapid growth.

This seems to build upon one of Stephens' (1991) surmises: '[I]n immature animals that are in positive energy balance, the physiological processes which make up maintenance requirement are running at elevated levels (Milligan and Summers, 1986)'. In order for this to make literal sense, maintenance would have to be partly defined as the direct result of production processes, which goes far beyond more consistent definitions such as Emmans' in Equation 7.1. But although Stephens' 'maintenance' is clearly confounded with production-related metabolic processes, it represents one of the dominant views on the issue in animal science (Cleveland *et al.*, 1983; Tess *et al.*, 1984; Summers *et al.*, 1986; Baldwin and Hanigan, 1990).

Part of the associated confusion may be eliminated by using the terms *metabolic intensity* (Turner and Taylor, 1983) or *support functions* (Walker and Young, 1993) instead. For example, the former authors' statement 'an animal at equilibrium is in a 'tuned-down' physiological state. The food used in such a state can hardly be equated quantitatively to the food used for basic vital functions in a productive animal' conveys roughly the same information as Stephens' above, but it is unambiguous because the term 'maintenance' is avoided.

The elevated physiological processes referred to by Stephens are mainly the functions related to increased nutrient intake (foraging and feed intake activity; digestion and its associated enzyme production and wear and tear on digestive tissues; and excretion) summarized in increased heat increment of feeding, and intensified cellular functions such as membrane transport and protein turnover (cf. Milligan and Summers, 1986). Obviously, metabolic intensity would be more strongly elevated by the process of growth when that growth is more intense, due to two possible factors: (i) the rate of growth, e.g. in kilograms per day; and (ii) its composition, in terms of the ratio of protein to lipid deposition.

2. Explaining Maintenance Requirements

Both growth rate and growth composition have been the subject of substantial genetic change through artificial selection in commercial pig and poultry populations, especially since 1970; other chapters in this book give examples. Although this genetic change has dramatically increased the gross production efficiency of

pig and poultry meat, the growth-related elevation of metabolic intensity in young growing pigs, turkeys and broiler chickens makes the individual animal more and more expensive to maintain (or more appropriately: to support) on a daily basis. A large part of this apparent trend is caused by the widely established habit to express maintenance requirements per $kg^{0.75}$ metabolic body weight: modern lean genotypes contain more protein per $kg^{0.75}$, and it is mainly the proteinaceous tissues that generate the maintenance-related metabolic functions.

For example, Campbell and Taverner (1988) and Rao and McCracken (1992), who measured maintenance requirements of growing males of 'high lean growth' pig genotypes by extrapolation of energy intake at various feeding levels towards zero energy retention, report estimates of 600–610 $kJ/kg^{0.75}$ metabolic body weight per day (further denoted as $kJ/kg^{0.75}$/day), and note that these are much higher than the levels between 420 and 460 $kJ/kg^{0.75}$/day recommended for growing pigs by ARC (1981) and NRC (1988). Those recommendations were compiled from much earlier sources, which form a mixture of: (i) studies similar to Campbell's and Rao's, extrapolating energy intake to zero energy retention; and (ii) factorial analyses according to Equation 7.2 in Section 3.

Similarly, Kolstad and Vangen (1996) estimated maintenance requirements of Norwegian Landrace and Duroc pigs, noticing that these breeds differ considerably in body composition (3.3 mm lower backfat depth in Landrace), growth rate (145 g/day higher in Landrace) and feed conversion ratio (0.22 units lower in Landrace). These pigs were kept at 58 kg body weight on a 1× maintenance regime for 6 weeks, measuring body composition by computer tomography in order to adjust for protein and lipid metabolism. These measurements had to be readjusted because the pigs had positive protein deposition rates but the authors had used the NE content of body protein, rather than the ME cost of protein deposition, for their adjustment; the revised results are in Fig. 7.1, showing the much higher maintenance requirements of the Landrace.

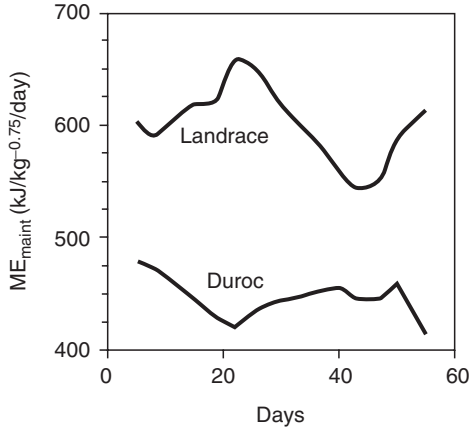

Fig. 7.1. Maintenance energy (ME) requirements estimated on Norwegian Landrace and Duroc pigs. (Data from Kolstad and Vangen, 1996, re-analysed to allow for a 53 kJ/g ME cost of protein deposition.)

Following the above reasoning, part of these differences would disappear when maintenance requirements would be expressed per unit of body protein mass, as stressed by Whittemore (1983), Webster (1983, 1988) and Emmans and Fisher (1986), among others. Indeed, Kolstad and Vangen (1996) report (wrongly adjusted, see above) estimates of the average ME_{maint} of the Landrace and Duroc pigs of Fig. 7.1 at 677 and 618 kJ/day/kg$^{0.75}$ metabolic body weight (i.e. a 9.5% difference) and at 386 and 357 kJ/day/kg non-fat soft body tissue (i.e. an 8.1% difference).

But the metabolic intensity of the 'lean' tissue varies considerably between tissue pools as well. This issue was reviewed by Archer *et al.* (1999) who focused mainly on ruminants, but a convenient example in growing pigs is from Pekas and Wray (1991). These authors subjected pigs to indirect calorimetry to measure FHP, and related the results to the mass of several tissues by cluster analysis. Strong relations were found between FHP and the mass of the gastrointestinal tract (particularly the small intestine), the liver, pancreas and kidneys. Likewise, the maintenance requirements of immature pigs were related to muscle mass and viscera mass by Van Milgen *et al.* (1998) and Van Milgen and Noblet (1999), who report a contribution (per unit of tissue mass) of the viscera to FHP (in fasted pigs) and to the maintenance requirements (in pigs fed *ad libitum*) three to four times as high as the contribution of muscle. As Webster (1988) noticed, this raises the question as 'to what extent differences in maintenance requirements [can] be attributed to differing proportions of the different organs and tissues of the body, each having different metabolic rates'.

Knap (1996, 2000) studied this issue through simulation. His results were combined with data from Beisel (1985), Baracos *et al.* (1987), Kluger (1989) and Demas *et al.* (1997) to give a tentative partitioning of the mean level of ME_{maint} in growing pigs into its component processes. In Fig. 7.2, the four parts on the left of the major plot (service functions, protein turnover, membrane transport and basal activity) are meant to represent thermoneutral, welfare-friendly and healthy individual housing conditions.

The fifth part of the major plot contains functions that may come in addition to that first group of four, when the environment becomes less optimal. Its possible

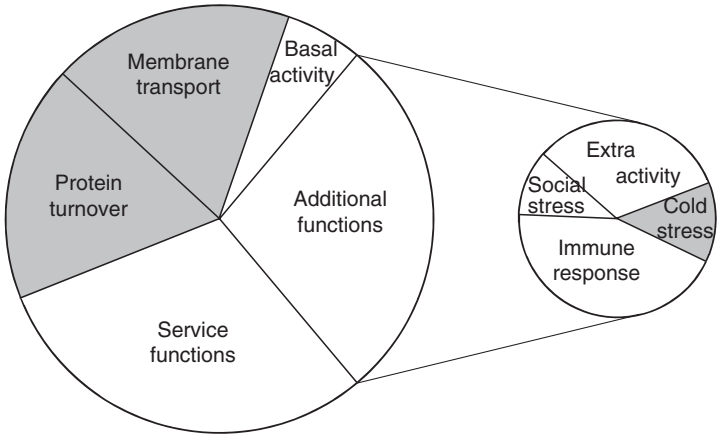

Fig. 7.2. Tentative partitioning of maintenance energy requirements (ME_{maint}) in growing pigs.

subdivision is in the minor plot, which has the same area as the 'additional functions' part of the major plot. Thermoregulation in Fig. 7.2 applies to continuously cold conditions; hot thermoregulation is more difficult to visualize because it reduces maintenance requirements rather than adding to them, but the magnitude of its impact is of the same order as of cold thermoregulation. Immune response in Fig. 7.2 applies to conditions of continuous (chronic) subclinical infection. In real life, pigs are rarely cold or subclinically ill continuously; so the 'additional' fractions in Fig. 7.2 must be seen as upper limits. Those same simulation results allow for the partitioning of the *between-animal variation* in ME_{maint} into the same components; see Section 5.

The notion has been developed in animal ecology that 'investment into production [is] traded off against investment into maintenance' (Wieser, 1994), specifically in 'conditions of ecological stress [where] an environmental change [...] disturbs the balance between maintenance and production'. The author continues with examples of reduced maintenance functions (protein turnover and membrane transport) with increased production levels in a wide variety of animal species, and concludes that high levels of maintenance functions naturally lead to high levels of metabolism and the associated energy requirements but at the same time provide a 'greater [...] range over which the activity of cells can be controlled' and a 'greater flexibility and richer behavioural repertoire'. From that point of view, it is more appropriate to use the term 'maintenance *processes*' than 'maintenance *requirements*', the latter term emphasizing the implied costs rather than the functionality of the processes involved. Insight in this matter would benefit from 'maintenance' being regarded as a set of fitness-related functions rather than merely as a cause of nutritional inefficiency. The allocation of sufficient resources to these fitness-related functions is crucial for homeostasis, and it is clear now that this allocation is at least partly genetically regulated (see Van der Waaij, 2004; Friggens and Van der Waaij, Chapter 18, this volume). Hence, the consideration of maintenance requirements as a full-fledged component of livestock breeding objectives becomes more and more relevant.

3. Measuring Maintenance Requirements

Although in Section 2 we cited studies that 'measured maintenance requirements of growing pigs', this measurement is by no means straightforward, mainly because the statistical partitioning of many physiological service functions, and of the metabolic costs of protein synthesis and membrane transport, into maintenance- and growth-related processes is not feasible. It is notoriously difficult to obtain estimates of the ME requirements (ME_{maint}) that are unconfounded with the energetic efficiencies of protein and lipid deposition (k_P and k_L) when ME_{maint} is not specified consistently such as in Equation 7.1.

The straightforward approach towards measuring a process that requires a zero energy retention in order to be consistently defined, would seem to be to subject an immature animal to a feeding level that keeps it in that state, and measure its metabolic intensity as a direct estimate of ME_{maint}. Such data have been reported for immature pigs by Jentsch *et al.* (1989) and Hoffmann *et al.* (1993), and by Vangen (1980) and Kolstad and Vangen (1996; see Fig. 7.1). But it has been

argued that the resulting measurements would reflect the animal's metabolic intensity at maturity (which it has not attained yet) rather than the elevated intensity in its undisturbed growing state. Stephens (1991) used evidence from an experiment by Taylor *et al.* (1981) on immature cattle to conclude that 'immature animals not in positive energy balance are likely to make metabolic adjustments which render estimates of maintenance requirements suspect' and 'maintenance requirements per unit body weight at artificially imposed equilibria [are] identical to those at maturity; [...] a sufficiently long equilibration period [allows] the animal's metabolism to settle at the same base level as it would ultimately reach if development were allowed to proceed normally'.

Van Milgen and Noblet's (1999) analysis of deposition data measured in growing pigs fed *ad libitum* suggests that if these pigs would be fed 'at maintenance', they would deposit some body protein and catabolize body lipid (much like the pigs of Fig. 7.1, treated that way by Kolstad and Vangen (1996)). These authors are uncomfortable with that result (for reasons not relevant here), and give three reasons why it may be an anomaly: erroneous data, an inadequate statistical model, or 'probably most important, the concept of maintenance [involving zero energy retention] may not be appropriate for growing animals'. The latter notion (which is supported by Close and Fowler (1982) and Walker and Young (1993), among others) would again imply that the result of extrapolation of observations on growing animals towards their state of energy equilibrium (or vice versa) should not be treated as a meaningful physiological characteristic. As Moe (1992) put it, 'it is possible to extrapolate [...] to zero growth rates to identify a maintenance component. If this hypothetical maintenance component is accepted as a mathematical entity rather than a physiological one, many conceptual problems can be avoided'. Webster (1988) characterized maintenance in growing animals as 'an operational description'.

Interestingly, Dawson and Steen (1998) estimated ME_{maint} in growing immature sheep and beef cattle, and found the results to be much higher than the corresponding ARC (1980) and AFRC (1990) recommendations. They attribute the difference not to genetic changes in growth intensity (as in the above-mentioned pig studies of Campbell and Taverner (1988), Rao and McCracken (1992) and Kolstad and Vangen (1996)) but to changes in measurement conditions: the earlier estimates derive from trials that attempted to keep the animals in steady state and it 'would be expected that heat production by the visceral organs would be lower than in fully fed animals' because 'higher maintenance requirements associated with higher rates of gain appear to be due to the increased mass of metabolically active organs such as the liver, intestines, heart and kidneys'. Walker and Young (1993) and Van Milgen *et al.* (2000) notice the same trend in growing pigs kept on various feeding levels.

An alternative, and widely used, approach to measuring ME_{maint} is by extrapolation of observations on animals in positive energy balance, applying Kielanowski's (1965) 'factorial analysis' to regress ME intake on protein and lipid deposition:

$$ME_{intake} = ME_{maint} + \frac{23.8}{k_P} \times P_{dep} + \frac{39.6}{k_L} \times L_{dep} \quad (7.2)$$

where P_{dep} and L_{dep} denote protein and lipid deposition in kilograms/day, respectively; k_P and k_L denote the energetic efficiencies of these deposition processes; the

constants 23.8 and 39.6 kJ/g are the net combustion energy contents of protein and lipid. The estimate for ME_{maint} follows as the intercept of the regression analysis, usually from extrapolation.

It has been noticed that this multiple linear regression approach has the disadvantages of intercorrelated independent variables (e.g. Kielanowski, 1976; Close and Fowler, 1982; Tess *et al.*, 1984; Walker and Young, 1993; Noblet *et al.*, 1999) and larger measurement errors on the independent variables than on the dependent one (Emmans and Kyriazakis, 1997). This causes the associated parameter estimates to be confounded and biased, respectively, which makes it statistically hazardous to interpret them independently from each other. In accordance with this, Tess *et al.* (1984) reviewed literature estimates of k_P and k_L for growing pigs (all obtained with models like Equation 7.2), which they found to range from 0.36 to 0.76 and from 0.58 to unity, respectively, and which they found to depend strongly on ME_{maint}, which was either estimated as a parameter in the same analyses or assumed fixed. The common practice of relating ME_{maint} to metabolic body weight with a fixed rather than a simultaneously estimated exponent is likely to cause even more interdependence of the estimates (Noblet *et al.*, 1999).

Hence, the factorial analysis estimates its three parameters (k_P, k_L and ME_{maint}) rather inappropriately. Van Milgen and Noblet (1999) present an alternative statistical model with two simultaneous non-linear equations that relate protein and lipid deposition, respectively, to ME intake above maintenance. This produces estimates for the same three parameters mentioned above plus the fraction of ME intake above ME_{maint} that is designated for protein (as opposed to lipid) deposition, and the 'change in energy gained as protein (and lost as lipid) relative to the change in BW for animals fed at zero energy retention'. The statistical improvement of this approach over the factorial one is the simultaneous solution of the protein- and lipid-related processes, which allows for taking into account the relation between these so that the estimates are less confounded and therefore more reliable. The price to pay is the necessity to estimate two extra parameters.

In another attempt to avoid the above-mentioned confounding between parameter estimates, Emmans (1994) proposed an alternative (and this time internally consistent) arrangement of the ME-requiring body functions. In contrast to Van Milgen and Noblet (1999), whose main contribution is in the statistical processing of the data, this approach involves a change of model. It redefines ME_{maint} according to Equation 7.1, and allows explicitly for the heat increment of feed intake 'above maintenance'. In this extended model, k_P and k_L are true and unconfounded constants for a given diet composition.

4. Variation in Maintenance Requirements

Much of the increase in gross efficiency of pig meat production mentioned in Section 1 was brought about by a genetic change towards increased leanness. This effect will reach a plateau when the economically optimum levels of pig carcass leanness are achieved, which will not take long in the western world. When a further increase of efficiency is desired, it will then have to come from a reduction of overhead costs, either by a further increase of growth rate (reduction of the time

to slaughter) or by a reduction of the overall maintenance requirements per unit of metabolic body weight per day. As discussed in Section 1, this in itself is likely to reduce the metabolic scope and make the system more sensitive to environmental instability. A prerequisite for control of such, more environmentally sensitive, production systems would be to study the maintenance-related processes and their metabolic costs and benefits in more detail. To quote NRC (1996): 'Successful management of beef cattle, whether for survival and production in poor nutritive environments or for maximal production, depends on knowledge of and understanding their maintenance requirements.'

In order to achieve that, we would have to consider the physiological service functions and processes like body protein turnover, cell membrane transport, thermoregulation, immune response, coping with other stressors (most notably, social ones) and physical activity. Because there is a considerable between-animal variation (largely of a genetic nature) in body composition, the maintenance functions related to body composition must be expected to show such variation as well, certainly as long as maintenance requirements are expressed in relation to metabolic body weight. This would hold for protein turnover and membrane transport, for thermoregulation, and possibly also for some immune response functions (Demas, 2004). The other functions, not obviously related to body composition, have been found to vary between individuals too. This holds true for many immune response functions (see Knap and Bishop, 2000; Van Eerden, 2007), for physical activity (Dunnington *et al.*, 1977; Heckl-Ensslin *et al.*, 1991; Van Milgen and Noblet, 2003) and for response to social factors (Jonsson, 1985; Hohenboken, 1986; Koolhaas and Van Oortmerssen, 1998; Muir, 2005; Chen *et al.*, 2007; Bergsma *et al.*, 2008; Turner *et al.*, 2008). It follows that we must expect ME_{maint} to vary between animals within a genotype, and that this variation is partly of a genetic nature.

This can be illustrated with data from Thorbek (1975), the classical source of information on maintenance requirements of individual growing pigs. Her experimental results are summarized in Fig. 7.3, which shows the estimated ME_{maint} of 48 scale-fed growing pigs in relation to body weight. Each pig was subjected to indirect calorimetry eight times between 23 and 80 kg BW, and k_P and k_L were estimated (at 0.48 and 0.77) on the data while assuming a constant value for ME_{maint} at a given body weight. Estimates for ME_{maint} were then obtained as $ME_{maint} = ME_{intake} - \frac{23.8}{k_P} \times P_{dep} - \frac{39.6}{k_L} \times L_{dep}$ for each of the 48 × 8 records (using the above estimates for k_P and k_L, but with $k_L = 1$ in case of lipid catabolism). Reanalysis of these data produced an estimate for the between-animal variance component of ME_{maint} at 0.24.

There is ample documentation now of variation in the maintenance requirements of mature cattle, poultry and mice, and of growing cattle, mice and pigs, and even more for variation in residual feed intake (RFI) in those same species. RFI (Koch *et al.*, 1963) provides an approximation of ME_{maint} (Luiting, 1991): compare the expression $MEI = b_P \times P_{dep} + b_L \times L_{dep} + ME_{maint}$ with the expression $MEI = b_{GR} \times GR + b_{comp} \times BodyComp + RFI$ (where GR is growth rate, BodyComp denotes some measurement of body composition, commonly backfat depth or lean content, and RFI is expressed on an energy basis). The variation of RFI then approximates the variation of ME_{maint}, with a bias that depends on the equivalence of $(b_P \times P_{dep} + b_L \times L_{dep})$ with $(b_{GR} \times GR + b_{comp} \times BodyComp)$.

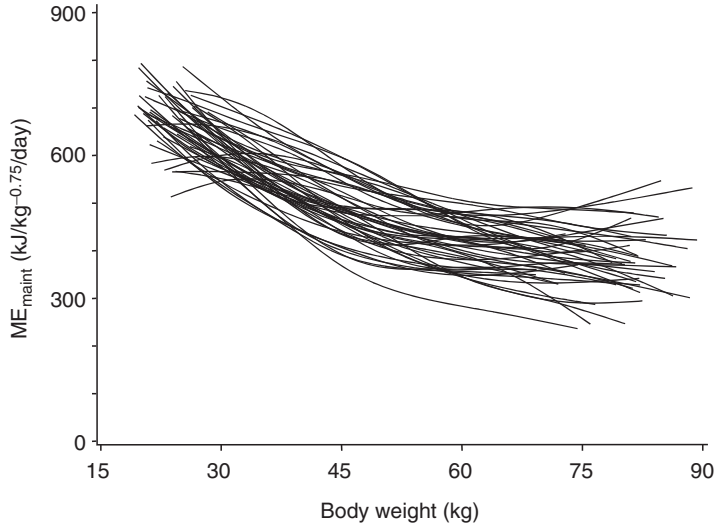

Fig. 7.3. Estimated maintenance energy requirements (ME_{maint}) of 48 growing pigs in relation to body weight. Spline interpolation curves through data from Thorbek (1975).

Expressing the between-animal standard deviation of RFI as a fraction of the ME_{maint} population mean then provides an (over-)estimate of the coefficient of variation of ME_{maint}. Estimates of such coefficients of variation (CV) of ME_{maint} derived from RFI are summarized in Fig. 7.4, together with CV of directly measured ME_{maint} in various species and the published heritability estimates for these traits.

It follows from Fig. 7.4 that: (i) ME_{maint} in immature growing mammals shows a phenotypic CV within populations of about 0.1; (ii) the CV of RFI has the tendency to overestimate this parameter, as expected; and (iii) given the heritability estimates, about 30% of the variance of ME_{maint} in these animals is of a genetic nature.

5. Partitioning Maintenance Requirements

Foster *et al.* (1983), De Haer *et al.* (1993), Mrode and Kennedy (1993), De Vries *et al.* (1994), Von Felde *et al.* (1996), Labroue *et al.* (1999), Johnson *et al.* (1999), Nguyen *et al.* (2005) and Cai *et al.* (2008) studied RFI in growing pigs, as the residual term of a regression of feed intake on body weight plus production traits, as mentioned in connection to Fig. 7.4. The R^2 values reported by these authors range from 0.06 to 0.71, dependent on the design of their regression model and on experimental (housing and feeding) conditions. The average figure of $R^2 = 0.4$ indicates that only about 40% of the variance in *ad libitum* feed intake of growing pigs is typically due to variation in the underlying production traits (growth rate, backfat depth and lean content). This value is much smaller than what is commonly found in productive mature animals such as laying hens or lactating cows

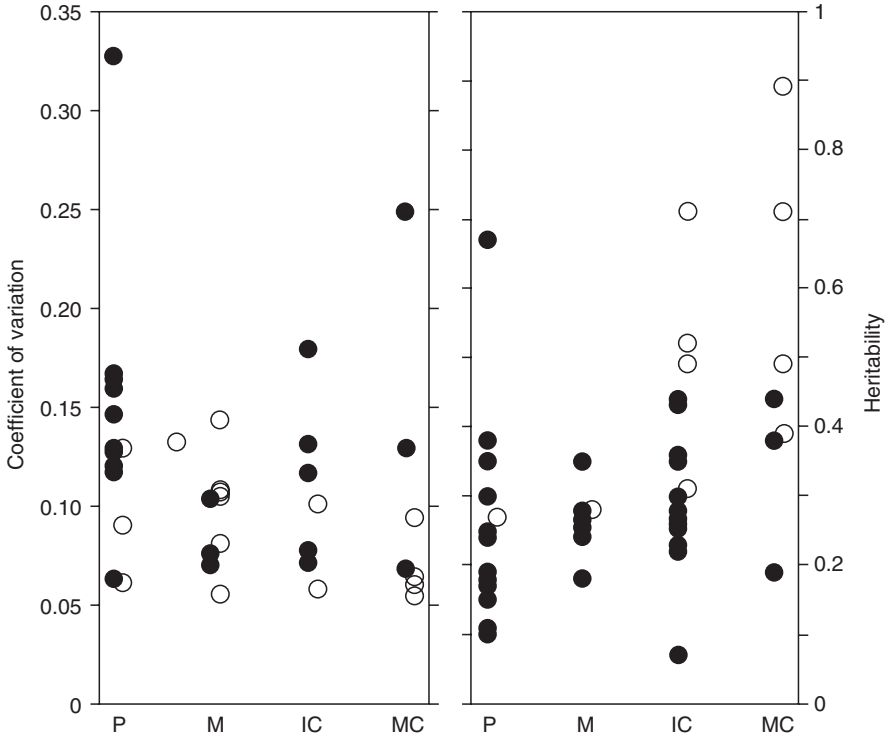

Fig. 7.4. Estimates of phenotypic coefficients of variation (CV) (left) and heritabilities (right) of ME_{maint} (○) and residual feed intake (RFI) (●) in immature pigs (P), mice (M), and immature (IC) and mature (MC) cattle. (Data from 48 literature sources.)

(see Luiting, 1999). In growing immature animals, the variation in RFI comprises (apart from measurement error) variations in ME_{maint} and growth efficiency. The latter element is largely due to variation in body growth composition because of the different ME requirements of protein and lipid deposition. Any associated variation that ends up into estimated RFI is a reflection of the less-than-perfect representation of protein and lipid deposition by the underlying production traits. The former element can be dealt with as follows.

We can now come back to Webster's (1988) issue 'to what extent differences in maintenance requirements [can] be attributed to differing proportions of the different organs and tissues of the body, each having different metabolic rates' mentioned in Section 2. Knap (1996, 2000) simulated protein turnover and thermoregulation in growing pigs, and concluded that the variance of ME_{maint} includes only a small portion that depends on body (growth) composition. Assuming that the membrane transport-related variance has at least the same magnitude as the protein turnover-related variance, this portion was quantified as 110–150 out of a total variance of 2540 $(kJ/kg^{0.75}/day)^2$ (set to 130 in Fig. 7.6). The resulting partitioning is in Fig. 7.5. Only to facilitate the comparison of the corresponding portions, both plots in this figure have been drawn the same size as their counterparts in Fig. 7.2.

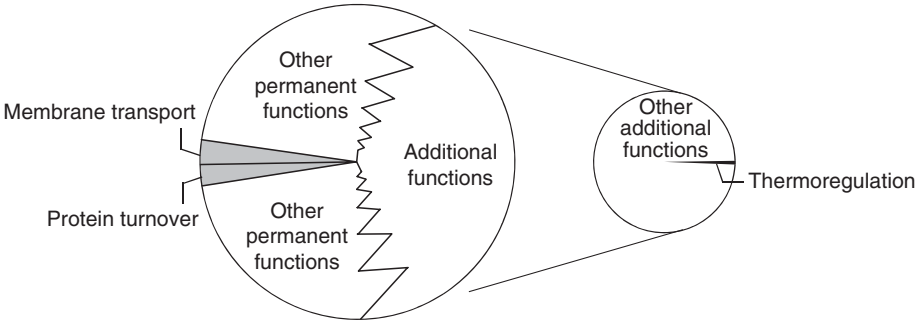

Fig. 7.5. Tentative partitioning of the within-population variance of maintenance energy requirements (ME_{maint}) in growing pigs. The jagged lines indicate uncertainty about the division among these components.

The contribution of protein turnover to the variation of ME_{maint} in Fig. 7.5 is caused by variation in body (growth) composition only. Specifically, between-animal variation in the turnover rate for any particular tissue is assumed to be absent; hence this portion may be underestimated. The contribution of membrane transport has been set, arbitrarily, just somewhat larger than the protein turnover fraction (as in Fig. 7.2), and may therefore be underestimated as well. The 'other permanent functions' in the major plot of Fig. 7.5 comprise the service functions plus basal activity, as in Fig. 7.2. The magnitude of this variance, as compared to the variance due to the additional functions that are triggered by suboptimal environmental conditions, is very unclear. Hence the jagged division lines in Fig. 7.5; the portion of the 'additional functions' may well have to be larger than suggested here. Physical activity is probably the most important single source of variation in the ME_{maint} of healthy thermoneutral pigs (De Haer *et al.*, 1993). The minor plot in Fig. 7.5 shows that the contribution of thermoregulatory metabolism (cold or hot) to the variance of ME_{maint} is very small.

This leads to a partitioning of the variance in *ad libitum* ME intake of growing pigs as shown in Fig. 7.6. The proportion of the variance of RFI (feed intake adjusted for growth and body composition) that is still associated with body composition and with the composition of body growth is (3 + 8)/60 = 18%. This is much more than what is commonly found in productive mature animals (see Luiting, 1991, 1999) that do not show much body growth and often vary less in body composition than growing animals do. This consequence of a less-than-perfect representation of body (growth) composition in the regression model is one of the reasons why R^2 values for the regression of feed intake on production traits (e.g. the value of 40 in Fig. 7.6) are usually much higher in mature animals. With a perfect representation, the RFI regression model would have been based on ($b_P \times P_{dep} + b_L \times L_{dep}$) and body protein and lipid mass, and the anomalous 3 + 8 = 11 units would have been included in the 'production' term in Fig. 7.6. The values 40 and 60 in Fig. 7.6 would become 51 and 49, respectively and, apart from measurement errors, RFI variation would be fully equivalent to ME_{maint} variation, the estimate of which would then be truly independent of body (growth) composition.

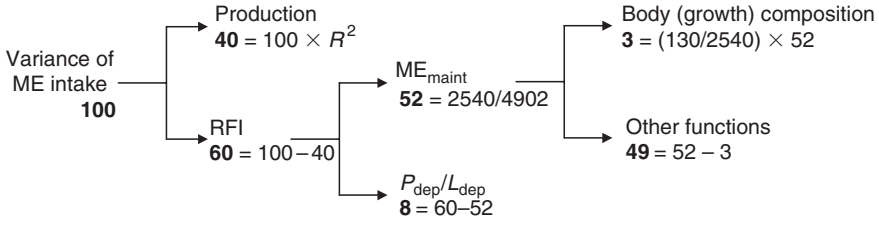

Fig. 7.6. Tentative partitioning of the within-population variance of *ad libitum* maintenance energy (ME) intake (set at 100) of growing pigs. The values shown for 'Production' and 'residual feed intake (RFI)' are averages of literature values. These differ considerably among sources, dependent on the design of the regression model to estimate RFI and on environmental conditions. The values 4902, 2540 and 130 (kJ/$kg^{-0.75}$/day)^2 are simulation results from Knap (2000). RFI: residual feed (ME) intake; production: processes represented by growth rate plus backfat depth or body lean content; P_{dep}/L_{dep}: composition of body growth (protein versus lipid deposition).

It follows that about half of the variance in *ad libitum* feed intake of growing pigs must be due to variation in maintenance-related processes that are independent of body (growth) composition. As mentioned above, an important component of these 'other functions' in immature pigs is physical activity. De Haer *et al.* (1993) report that 44% of the variance in RFI of their group-housed pigs was due to variation in feed intake activity alone; in Fig. 7.6 this would be a value of 26 and the corresponding value for total activity would be somewhat higher, perhaps up to 40. Again, the corresponding value for mature animals must be expected to be lower.

6. Modelling Maintenance Requirements

Black *et al.* (1995) referred to the various maintenance-related processes as discussed in Section 5 when they wrote: 'ideally, these components of maintenance should be represented also within a comprehensive model of animal growth'. Similarly, 'in order better to address this variation [in ME_{maint}], several groups have developed mechanistic models which attempt to capture cause and effect relationships underlying maintenance energy expenditure as they vary across physiological states, environmental conditions, breed and other factors. A number of physiological/metabolic functions which contribute to variance in apparent maintenance requirements have been identified; these functions have been characterized, at least partially, using mechanistic models' (Baldwin and Hanigan, 1990). These authors claim that many physiological service functions, and also the metabolic costs of protein synthesis and membrane transport, 'manifest themselves as components of both maintenance energy expenditures and costs of production [...] As a result, mechanistic models are increasingly deviating from [the] use of the classical concept of depicting costs of maintenance and production separately'. This coincides with the surmise of Van Milgen and Noblet (1999) quoted in Section 5, and with the reasoning on support costs by Walker and Young (1993). It also illustrates the

potential value of the use of mechanistic simulation models (Thornley and France, 1984), rather than empirical statistical models, for studying the above-mentioned processes.

Of course, the increasing complexity of simulation models when mechanistic routines are added to them has its disadvantages as well: simulation models require data to be parameterized. At the same time, it must be recognized that existing data becomes less representative over time; Turnpenny *et al.* (2000) write: 'The breeds referred to in the [literature] are no longer used, and the breeds used today partition energy […] differently. Increased growth rates of [pigs] result in higher metabolic heat production and […] future work should concentrate on collecting comprehensive up-to-date heat loss data from animals rather than on further theoretical modelling'. It should be obvious that any model *extension* will increase the information requirement for a proper model parameterization even further. It may be sufficient for research models to derive their data from the literature, but as soon as such a model is applied to a specific animal population (which turns it into an *application model*) it may be wiser to remove such routines from the program, and accept an incomplete predictive functionality until that population has been properly characterized for the relevant traits; Knap *et al.* (2003) and Doeschl-Wilson *et al.* (2007) provide suggestions for such characterization. Failure of model developers to exert such discipline may easily lead model end-users to the type of disappointments that led Conceição (1997) to propose that 'the power of modelling techniques in the understanding of biological systems will be undermined by premature attempts to use models to predict the behaviour of the systems'. Of course for model developers, such 'premature' attempts are precisely the way to find out if their models are yet good enough.

It follows from Fig. 7.6 that the variation in ME_{maint} of growing immature animals is at least as important a cause of variation in their *ad libitum* feed intake as variation in their growth-related processes (the emphasis on variation is important). But pig growth modellers have mostly concentrated on the description of the growth potential, as is illustrated by the succession of papers that follow and/or attempt to improve upon the linear-plateau concept of Whittemore and Fawcett (1974), as reviewed by Luiting and Knap (2006). By contrast, the whole aggregate of maintenance functions is often condensed into a single function of metabolic body weight or body protein mass. Given this imbalance of developmental activities, future dynamic modelling should focus on a more comprehensive description of maintenance processes rather than on an even more detailed description of the growth potential. Knap (2000) found that very different sets of potential growth rules produced surprisingly similar simulation results, up to and including the within-genotype variation of protein and lipid deposition and implied maintenance requirements.

For modelling purposes, it is useful to distinguish between three partially overlapping groups of maintenance-related processes: (i) the physiological service functions; (ii) processes triggered by environmental factors (thermoregulation, immune response and reactions on social stressors); and (iii) processes related to body composition (protein turnover, membrane transport, thermoregulation and possibly some immune response functions). Group (iii) is of the most immediate interest in a pig breeding context, because it is body composition that is influenced most by pig breeding activities. But given the 'advanced' stage of the current production

genotypes in some meat-producing species, group (ii) is of rapidly increasing interest to animal breeders, because some of these traits seem to be primarily responsible for the environmental sensitivity of highly productive genotypes that leads to genotype by environmental interactions (see also Knap, Chapter 17, this volume). Of course, in the animal breeding context, the interest is as much directed to the between-animal variation of these processes as to their mean levels.

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8 Allocation of Resources to Growth

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1. Introduction

1.1 Choice of approach

For principles of resource allocation to apply to farm animal growth, it is necessary to assume that an animal can master the full hierarchy of information (data), knowledge (understanding the data) and wisdom (rational use of information and knowledge). Such assumptions may not be safe. The presumption that animals allocate nutrient resources to growth according to some rationale, presumes in turn that:

1. The animal recognizes the separate elements of its nutrient resource base, and knows that these may be partitioned in different ways;
2. There is information flow in relation to the resources that the animal can interpret;
3. The animal is in control of its circumstances; and
4. The animal prioritizes in a logical and beneficial way.

The experimental programmes necessary to explore these presumptions are as immense in scale as they are unlikely in funding. None the less, without some *understanding* of nutrient resource allocation, there can be no lasting progress in enhancing the efficiency of farm animal production.

1.2 Empiricism and deduction

The purpose of investigatory experimentation is to interrogate data streams from which patterns may be elucidated, and insights gained. The particular must be capable of being widened to the general. Were these things not so, then investigation would not progress beyond observation; and any interpretation that might be possible would have no function outside of the time, place and circumstance within which the data stream first arose.

 Resource Allocation Theory Applied to Farm Animal Production (ed. W.M. Rauw)

The empirical measurement of outputs of lean and fatty tissue growth, resultant from measured input of protein and energy, will yield information for allocations of nutrients to growth. With the help of proper statistical analysis, it may further be determined what the probability might be of the same outputs resulting from the same inputs under the same conditions. Little is learned about other input levels and combinations, or other conditions. If animals exercise choices over both the balance and level of energy and protein input, and if they make further choices over the allocation of nutrient resources to the level and balance of lean and fat growth, the empirical information is of limited use – limited, in fact, to the past, whereas interest lies in the modulation of the future.

In accepting the notion that resource allocation theory applies to animal growth, there must also be acceptance that empiricism will be a poor servant to help understanding, and only the deductive approach will take the science forward. This is not to say that empirical observation has no value at all, just that the value is limited.

Does this mean that all farm animal science can only be useful if it has achieved understanding? That no experiment can be launched without a hypothesis? That 'base' empiricism must always give quarter to 'higher-minded' deduction? To take this position is to condemn as of little value the major part of farm animal research effort. But not to take it, and to acquiesce to the idea that understanding is not a requirement for technological advance, is to accept farm animal research as no more than a never-ending cyclical procession of empirical experiments that answer only today's question, and have no bearing on the morrow. This conundrum is not a matter of degree, it is a matter of type. Thus some activities can and must adopt technological solutions on the basis of empirical observations alone; others equally must not. Resource allocation falls into that category of study where empiricism has little to offer, and deduction is prerequisite to progress.

Deduction requires understanding; not complete understanding, but sufficient. Understanding requires not just information (the data stream), but also knowledge, and sometimes wisdom. The information can be obtained. Knowledge and wisdom are scarcer commodities. The dilemma is in the choice between: (i) frank empiricism, with the putting aside of deduction and hypothesis making, which, although limited in application, cannot be 'wrong'; or (ii) deduction based on partial understanding and faulted hypotheses, which, although much broader in utility, can never be 'right'. The danger in the application of deduction is that deduced outcomes may be influenced by human preconceptions. And these can be wrong.

1.3 The specific and the general

When faced with a level of understanding of a system that is inadequate for the management of that system, there is a natural tendency to de-complex and to reduce. The component parts can then be observed and patterns of logic sought and described. Then, the specific can be scaled up to the general, and the general can be used in systems management. But these steps have pitfalls for those who try too hard. De-complexing the complex can result merely in naivety. Reductionism,

by addressing the particular, may cause any greater understanding of the interacting whole to be lost. The seeking of logic is a subjective activity, influenced by the social mores and environment in which the logician resides. This the more so since, in the applied sciences, the rules of contemporary scientific publication and peer review looks for 'in-step' progression rather than 'out-of-step' disaffection with current presumptions.

While it is evident that systems management requires general forward-planning rules, rather than specific retrospective ones, the transfer from the one to the other may be uneasy. The specific and the general may not be closely linked. The transfer of an observation of the specific into an understanding of the general is not merely a matter of assuming that observation *is* understanding. Nor is it true that the specific, if closely enough observed and verified, is *synonymous* with the general.

1.4 The demands of deduction and generalization

The hypothesis proposed is that animals *can* allocate their nutrient resources, and that for efficient feeding of animals the allocation rules must be understood. The methodology for the exploration of this hypothesis is suggested as deduction (rather than empiricism), and it is further suggested that only if open to generalization are specific understandings likely to be useful. The challenges met by the resource-allocating animal must, necessarily, be reflected in the challenges met by the deductive modeller. Categorized generally, these are:

1. Data recognition, capture and analysis to yield information about working functions, relationships and efficiencies (such as the nutrient costs of protein retention, lipid retention and bodily activity and the utility of dietary amino acids);
2. The need to make decisions and choices, and to determine priorities relating to allocation of nutrients to: (i) the different components of body growth; and (ii) alternative usages to growth (such as maintenance, storage and reproduction);
3. Definition of the limits to both inputs and responses that define the envelope within which allocation rules have freedom of operation (such as mature size, the physiological minimum for body lipid and the maximum attainable rate of protein retention).

2. Product

Resources allocate to processes and products. These need to be defined in terms of both level and priority of resource demand. The important products are retention in the body of protein mass, lipid mass, water mass and mineral mass; the important processes are maintenance of body functions when there is no productive activity, and driving the body functions associated with productive activity. With a view to allocating resources to these products and processes, the animal must have a view of the size and nature of the envelope within which the allocation activity will be restricted and the decision structure for allocations within the envelope.

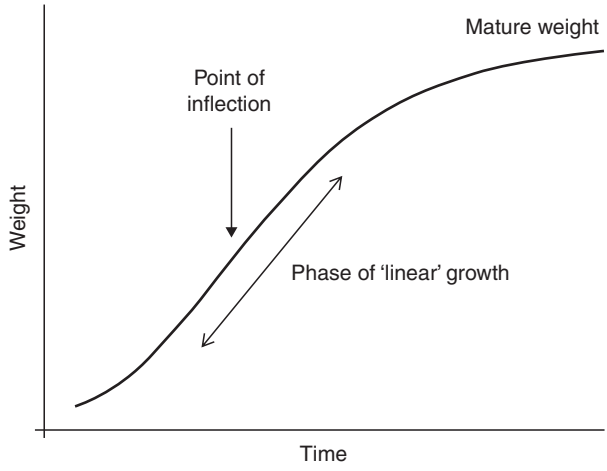

Fig. 8.1. The Gompertz function describing animal growth in terms of weight and time.

2.1 Retention

It is the deposition and retention of protein, lipid, water and minerals that causes the animal to grow. Growth may be described as the relationship between the weight of the animal and time. An appropriate function for the relationship between weight (W) and time (t) would be that of Gompertz:

$$W = A\ e^{-e^{-B(t-t^*)}} \tag{8.1}$$

where A is the weight (W) at maturity, B is the growth coefficient and t^* is the time at the point of inflection. This function produces a simple, but asymmetrical, sigmoid curve (see Fig. 8.1).

2.2 Mature size

Resource allocation thus requires information on the final size (A) that the animal seeks. Mature mass and size, the asymptote to the growth curve, is characteristic of species, of breed within species, and of strain within breed. Adult pigs are smaller than cattle, Galloway cattle smaller than Friesian cattle and British Friesian smaller than the Holstein. Indeed, final size is a defining characteristic. Its correct definition is fundamental to the management of nutrient allocation. Unfortunately, there is little to be had of this essential information. Few studies have had the patience (or finance) to delineate growth to the point of maturity of the body mass, and at that point to separate the component parts into mature protein mass, mature water mass, mature lipid mass and mature mineral mass. It is possible that only one of these (mature protein mass) has any constancy and meaning.

The reader may baulk at the proposition that the final size of livestock is an inadequately known parameter. But consider:

1. Domestic breeding populations are rarely grown or kept in uninhibited circumstances.
2. Their mature mass and its composition are of little commercial interest.
3. Female growth to maturity is interrupted by reproduction, which is a higher imperative.
4. The animals whose growth is of interest and utility are usually not of the same genotype as the breeding populations which begot them.
5. Animals grown for meat production are slaughtered at weights greatly below their mature size (usually at 50% of mature size or less), thus rendering their continuing growth to maturity and subsequent body analysis a seriously costly exercise.

2.3 Time

Resource allocation is a matter of both absolute amount and rate of usage. If A is the ultimate value of the y-axis, then the rate at which it is achieved (the slope of the Gompertz function) is dependent upon the extent of the x-axis; the time it takes to reach maturity. Animals reaching A at similar times, but having differing values for A, will have differing growth rates. Knowledge of t, the time at maturity, is therefore fundamental to the optimum provision of nutrients for growth (see Fig. 8.2).

An animal knowledgeably allocating nutrients to growth needs not only a conception of the final size to which it may aspire, but also the time it wishes to take to get there: A and t fix growth rate. As little is known of t as of A, and for similar reasons. Importantly, most studies of growth rate terminate at the point of commercial slaughter, which is far earlier than the age at maturity. In passing, there is general agreement that t should be measured in days. There is little logic in this. Small animals with short lives might resent being expected to operate on the same timescale as larger animals with long lives.

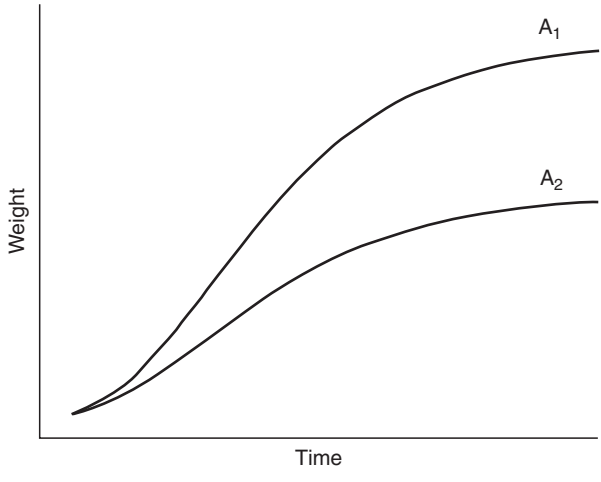

Fig. 8.2. Animals with different mature weight, but reaching that weight in similar times, will have differing rates of growth.

2.4 Curvature

If it is accepted that:

1. In the initial phase of growth, the daily accumulation of body mass will be less than in the middle stages, even if only on the basis of the contemporary body mass to which the gain needs to be added (1 kg of daily gain on the body of a 50 kg calf is a rather more daunting proposition than the same kilograms on the body of a 500 kg steer); and
2. In the final phase of growth, the daily accumulation of body mass will be less than in the middle stages, even if only on the basis of the logicality of no biological system instantaneously switching from full on to full off;

then the relationship between weight and time must show a sigmoid curvature with accelerating and decelerating phases. There is convenience in suggesting that this sigmoid is balanced, with the point of inflection (maximum growth rate) in the middle. This also means that daily gain (dW/dt) may be described as a simple quadric function. However, acceleration in early growth and deceleration in later growth do not have a common causation, so there is little justification for a response curve with symmetry below and above the point of inflection. None the less, there is no particular reason why the Gompertz function should be the 'best' descriptor; others have been forwarded. Crucial to its acceptability is the choice of t at which the description begins (and ends). Is zero t conception, birth or weaning?

The Gompertz function suggests that maximum growth occurs at $1/e$ (0.37) of A, and the rate achieved at this point will be $(A.B)/e$. High values for A are therefore associated with faster-growing genotypes. Gain may be described as:

$$dW/dt = BW\ln(A/W) \tag{8.2}$$

This function is also shown in Fig. 8.3. As suggested earlier, the concept of 'A' may only be safe for protein mass. In particular, there is little observed evidence of a mature lipid mass; lipid being accumulated in time of plenty, and called upon in time of need. Thus, better might be:

$$dW_p/dt = BW_p\ln(A_p/W_p) \tag{8.3}$$

where W_p is the protein mass at any given time (t), and where A_p is the protein mass at maturity. In effect, dW_p/dt is the maximum potential rate of protein retention at any given point in growth, and A_p is the mature protein mass. If the former is designated as the upper limit for protein retention ($P_{retention}max$) and the latter as $P_{total}max$ (for mature body protein mass) and W_p is referred to as P_{total} (for current body protein mass), then:

$$P_{retention}max = Pt\ B\ln(P_{total}max/P_{total}) \tag{8.4}$$

At this point, the animal's desired rate of growth is calculable, and thereby the nutrient resources for its attainment; albeit on fragile knowledge of mature mass and time.

Helpfully, the Gompertz curve tends to be rather flat-topped, and therefore a description of 'linear' growth can be accepted over a large proportion of post-weaning animal growth to the point of slaughter for meat (see also Fig. 8.1).

Fig. 8.3. Gain (change in weight/change in time) in relation to live weight.

As has been established, there is neither safety nor logic in using the same argumentation for lipid. There may be no merit in seeking algorithms to describe lipid growth, which may have only extrinsic drivers (such as availability of food). Lipid deposition may best not be considered as one single function, but three:

1. There appears to be 'physiological' requirement for a small, minimum level of body lipid sufficient to allow effective body functioning. This can be expressed as a function of body protein:

$$(L_{\text{total}} : P_{\text{total}})_{\text{min}} \tag{8.5}$$

2. Then there is lipid retention occurring during growth, which may be related to the rate of protein retention. There may be some preferred ratio of lipid to protein in the body of a normally growing animal. The proposition here is that in deciding upon the ratio of lipid to protein retention in the gain, the animal is aware of the lipid to protein ratio extant in its body, and that the animal aspires to adjust the composition of its growth to achieve that ratio. This preferred ratio is likely to be species-, breed-, strain-, genotype- and sex-specific. It may change slightly as the animal ages/grows bigger.

$$(L_{\text{total}} : P_{\text{total}})_{\text{preferred}} = (L_{\text{total}} : P_{\text{total}})_{\text{perferred}} + \text{b}(P_{\text{total}}) \tag{8.6}$$

Otherwise, there seems to be a reasonable expectation of an allometric relationship between lipid and protein masses:

$$L_{\text{total}} = aP_{\text{total}}^{\text{b}} \tag{8.7}$$

3. And at last, lipid is associated with energy storage when nutrient supply is in excess of immediate need. This latter may occur, depending on food supply, both during

growth, and at maturity. An animal cannot fatten when food is limiting, so the rates of gain in protein and lipid bear a constant relationship (that of the preferred ratio, and the animal will not 'fatten'). Where an animal prioritizes nutrient usage towards protein growth in order to maximize its rate of growth to mature size, then fattening during growth will only occur if there are nutrients in excess of those needed for protein deposition. At maturity, however, when protein growth slows towards a rate of zero, fattening may proceed at any level of feed supply above that needed for body maintenance.

2.5 Water and minerals

Little useful can be forwarded with regard to the definition of an envelope for mineral retention. That there is a minimum is self-evident, else the bones would not support the muscle, and be prone to breakage. But what level of support, and what degree of bone strength is appropriate, and over what timescale (point of slaughter or natural lifespan)? Mineralization of bone can also be a storage function; in which event, like lipid, the concept of an 'end point' is inappropriate. The association between bone and muscle tempts the presentation of bone growth as a function of muscle mass growth, thus, in terms of ash (Ash) and protein (P):

$$\text{Ash}_{\text{total}} = nP_{\text{total}}^{\text{b}} \tag{8.8}$$

Water is the most significant part of the estimation of live weight growth from the allocation of nutrients to lipid and protein retention. The water associated with lipid in the formation of fatty tissue can vary from 0.1 to 0.2. This is influenced by level of body fat, rate of lipid retention, age of the animal and sex. Further complications arise when an animal chooses to utilize some of its stored fatty tissue to generate needed energy not available from the dietary intake. It appears that the catabolized lipid may be replaced with water. Thus the animal loses tissue, but not weight!

More important is the association of water with protein in the growth of the lean mass. Small errors in the estimate of this parameter can result in large errors in the estimation of growth from the retention of protein. It is a prerequisite that the valuation of body water content is accurate. Water comprises some two thirds to three quarters of the weight of the growing animal's body. Faster and more youthful gains appear associated with more water than slower and older ones. Slow-growing and older animals have drier lean tissue. If related to protein mass, it may be proposed that:

$$\text{Water}_{\text{total}} = aP_{\text{total}}^{\text{b}} \tag{8.9}$$

'a' is likely to be a variable number reflecting the general water content of the body, and 'b' is likely to be a variable number (less than one) reflecting a decrease in water content as body mass increases. In the accurate 'multiplying up' from retention of protein and lipid to live animal growth, it is greatly to be regretted that the importance of these parameters being allocated their correct values is matched only by our ignorance of them.

2.6 Product prioritization

Three assumptions dominate:

1. There is a limit to the potential rate of growth of the priority body tissue.
2. The animal, in prioritizing, seeks to maximize the rate of growth of that tissue; that is, to reach its desired mature tissue mass at the earliest possible opportunity.
3. The tissue concerned is lean mass.

This being so, dietary protein and energy will be partitioned to body protein retention until the limit of the envelope is reached, the dietary supply of amino acids is used up, or the dietary supply of energy needed to fuel protein synthesis is used up. There is no reason to propose any other than linearity in this response to nutrient supply, the rule being not that of 'diminishing response', but rather that of 'first limiting resource'. On the level of the individual animal this will show as a 'broken stick' function. Only on the level of the population, where there is variation in the point of the 'break', will the function appear curvilinear. There may however be a possibility that the efficiency of digestion of amino acids, and the efficiency of their arrangement into animal body protein may be greater in animals 'challenged' by lower dietary protein supply.

Modulation to the three dominant assumptions occurs:

1. To divert dietary protein and energy to the functions of 'maintenance', in the support of life functions;
2. To divert energy to the formation of a minimum level of physiological lipid; and
3. To match each unit of retained protein with the preferred ratio of lipid in 'normal' body growth.

After these allocations are satisfied, excess dietary protein moieties may be denatured to provide further energy, and excess dietary energy may be retained in the form of storage fatty tissue. In passing, there should be recognition of a possibility of removal of excess energy as heat, but this would be unusual in growing domestic animals, and would require some upper limit either to daily retention of lipid or to total body lipid.

2.7 Likelihood of moderate insufficiency of nutritional resource

Domestic animals may find themselves with less than the desired level of nutrient intake as a result of season and husbandry. Animals such as pigs and poultry can be provided with sufficient food while they grow to fully satisfy their lean tissue growth ambitions. Fattening during growth is therefore likely unless intake is restricted below appetite. For ruminants with access to forage, the greater likelihood is that nutrient intake is inadequate to satisfy lean growth potential. Thus, the composition of the gain will remain at $(L{:}P)_{\text{preferred}}$, and the animal will not fatten. For large genotypes with high values for A, and a high lean growth potential, it is necessary for a more concentrated diet to be offered if the animal is to fatten during growth. That fattening during growth does occur becomes a prerequisite for larger types; otherwise slaughter weight would become excessive. Alternative

husbandry strategies would be: (i) to use slower growing animals, which would mature at smaller size, and then fatten consequent upon $P_{retention}$ reducing as maturity is approached; or (ii) to slaughter animals in a leaner state.

2.8 Negative growth

During growth, dietary nutrient supply may be of sufficiently low level, or of sufficiently poor nutrient balance that growth cannot proceed, or indeed may become negative. As the major demand for the maintenance of life functions is energy, negative growth is usually associated with the catabolism of body lipid, primarily that part of body lipid which is excess to the preferred ratio with protein (and therefore seen as 'storage depot fat'). The growing animal holding fatty tissue reserves may also use those reserves to fuel protein deposition. Thus, in the presence of sufficient dietary protein, but insufficient dietary energy, $P_{retention}$ may be maximized, lean tissue growth proceed apace and body lipid stores diminish. At lower feed intakes, this situation is quite likely, and is a perfectly satisfactory purpose for lipid storage in the first place ($P_{retention}$ being assumed the dominant priority). At levels of nutrient supply considered only adequate for maintenance, the young animal, while showing no change in live weight, may well be losing lipid and making measurable protein (and water) gains.

It appears that associated with lipid breakdown there may also be some inevitable breakdown of protein. The rate of body protein loss associated with body lipid loss seems greater as: (i) the rate of lipid catabolism increases; and (ii) the total available lipid diminishes. This presupposes that some part of retained protein can act in a storage capacity. *In extremis*, the unavailability of body lipid to fuel maintenance will result in the use of body protein for that purpose (gluconeogenesis). But for purposes of normal growth, negative growth may be associated primarily with use of fatty tissue stores as a result of temporary failure in nutrient supply, or ill health.

2.9 Compensatory growth

The 'comfort zone' for an animal allocating its dietary nutrients to growth has been suggested as a rate of protein retention at, or near the potential for the genotype concerned, together with the preferred rate of lipid retention:

$$P_{retention} = P_{retention}\max \tag{8.10}$$

$$L_{retention} = P_{retention}\,(L_{total} : P_{total})_{preferred} \tag{8.11}$$

If the first (achievement of potential rate of protein retention) does not pertain, the animal may perceive that it has lagged behind its expected weight-for-age (for this, of course, the animal must be aware of both its weight and its age). The most likely cause of such a lag is inadequate supply of diet nutrients or disease. Upon the return of adequacy of nutrient supply, or health, the animal may expect to return to linear

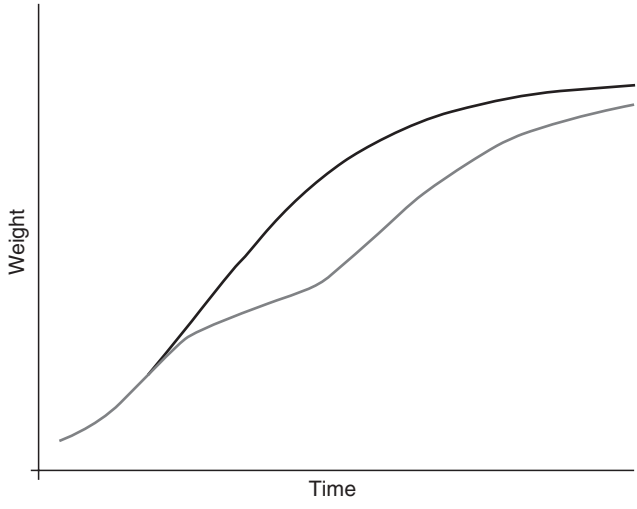

Fig. 8.4. After a period of suboptimal growth, growth rate may return to that previously attained.

growth with a similar slope, but at delayed time (see Fig. 8.4). To do otherwise would require either: (i) that the system had been permanently damaged – in which case there is a new, lower, value for $P_{\text{retention}}$max; or (ii) that the system had been invigorated by the experience – in which case there is a new, higher, value for $P_{\text{retention}}$max and the previous genetically determined potential may now be exceeded (this latter phenomenon would of course *appear* to be observed if the previous growth performance had not been at or near the limit). This 'invigoration' might also need to be accompanied by enhanced appetite and/or enhanced efficiency of nutrient use if the new (higher) targets are to be met. (In passing, the reader may note from observation of Fig. 8.4 that it is the case that animals having suffered a period of reduced growth will *appear* to 'catch up' with their less-disadvantaged peers as the latter slow down their growth towards mature size, but the former are still in the linear phase).

If the second part of the requirement for 'comfortable growth' (achievement of the preferred ratio of body lipid to body protein) does not pertain, then the animal may perceive that it has more fat than it needs (and has spare energy in the system), or less fat than it would prefer (for this, of course, the animal must be aware of both its lipid mass and its protein mass). The likely cause of superfluous fat growth would be a diet imbalanced against protein supply, while the likely cause of inadequate fat growth would be a diet imbalanced against energy supply. If the animal has 'available energy' additional to that in the diet, then, upon a return to a sufficiency of dietary protein, it may reduce its rate of lipid retention, use available lipid stores, increase its rate of protein retention towards the maximum potential, and return to $(L_{\text{total}} : P_{\text{total}})_{\text{preferred}}$. If the animal has inadequate body lipid, then upon return to a sufficiency of dietary energy, it may increase its rate of lipid growth above the preferred ratio in order to return the total body ratio to 'normal'. In both of these cases the

animal has made compensatory gains to adjust for previous imbalances and return to its body composition 'comfort zone'. Particularly in the case of protein shortage by imbalance, followed by protein sufficiency, enhanced rates of gain may be observed (but there is no necessity for any presumption that the envelope of $P_{\text{retention}}$max be breached).

3. Competitive Products

As considered earlier, the growing animal has resource allocation decisions to make in relation to the chosen components of its growth. For the female there come resource allocation decisions on a grander scale; for the female reaches reproductive capacity while still growing. In the domestic species (possibly as a result of purposeful selection for the early breeding potential) the ability to produce the next generation occurs at a point around or less than half way to maturity. Two considerations arise:

1. Can the animal support a continuing full rate of growth simultaneously with pregnancy and lactation?
2. If nutrient resource is not adequate for the continuation of full growth ($P_{\text{retention}}$max), what are the natures of the resultant allocations?

3.1 Pregnancy

Upon falling pregnant, appetite will rise, and nutrient selection choice may change. The animal then has already opted for a strategy, which attempts to maintain the rate of previous growth. At the same time, the sigmoid growth curve for lean tissue growth will flex downwards, a reduced rate of lean growth indicating that the attempt at compensation has not fully succeeded. It is also instructive to note that the composition of the maternal gain changes at this point in favour of lipid deposition; that is $(L_{\text{total}} : P_{\text{total}})_{\text{preferred}}$ increases. It may be argued that the imperative has changed. The allocation of nutrient resource is no longer prioritized to growth of the present generation, but to the sustenance of the next. The gravid female is preparing for lactation in the certain knowledge that dietary intake post-partum cannot provide sufficient nutrient flow to support the required rate of milk synthesis; the prospective mother is laying down energy stores as fat. None the less, maternal body growth, although slowed to about half the previous rate, does not stop.

In the face of an insufficiency of nutrient supply, the partition decisions become acute. In the face of *moderate* insufficiency, all productive functions will reduce, but the relative prioritization for nutrient resources appears to be first fetus, then body stores and maternal growth. The fetus will be smaller, but in relative terms there will be proportionately less body lipid and protein retention. In the face of *severe* nutrient insufficiency, a tipping point is reached at which the pregnancy may be abandoned; but by this time damage and stunting of the maternal body will have occurred (Target A_{p} has been reset).

3.2 Lactation

After parturition, a leap in appetite occurs, driven by a nutrient demand that far outstretches any previous requirement for satisfying the animal's ambition to reach mature protein mass within a given time frame. Milk yield rises with the increasing demand of the offspring. In these circumstances it is difficult to sustain any rational argument for prioritization of even the lowest level of maternal body growth. Normally, the appetite increase is insufficient to meet demand. The maternal investment in the offspring has already been considerable. Lactation synthesis overrides all other considerations, and the maternal body goes into negative growth. First, lipid is lost, then lipid and labile protein, and finally body protein itself.

The greater the rate and extent of body tissue loss in lactation (the greater the lactation yield in relation to the nutrient supply) the more likely will be a failure in the return of the reproductive cycle, rebreeding needing to await adequate nutrition and replenishment of essential lost body tissues. It may be surmised that tissue loss and suspension of maternal growth is 'normal'. However, when there is abundance of food it does appear possible for weight loss to be avoided in lactation. The length of the lactation period also impinges on this discussion. Most mammals are lactationally anoestral to some or other extent, but as the hormone balances alter through the course of lactation, this effect weakens. In dairy cattle, the reproductive cycle can return soon after peak lactation, and the end of lactation is accelerated by the presence of the next offspring in the uterus. In the pig, lactation is usually in noticeable decline (or has been terminated by weaning) before the reproductive cycle re-establishes.

There is little doubt that the primiparous mammal, because it is breeding while growing, is a quite different beast to the multiparous. Much of this is a result of a change after the first parturition in the priorities for nutrient use, and a likely resetting of life objectives. For the female, therefore, the die is thrown at first impregnation, and lands at parturition. The main part of the required final size needs to be attained by the end of the first pregnancy, for growth thereafter will be slow and interrupted by negative phases. This may be a reason for the prime imperative of earlier life – maximization of the potential for lean tissue growth (attainment of $P_{\text{retention}}$max, and early realization of A_{p}).

Not only does the primiparous female make a bad model for the multiparous, but once bred, the decision drivers seem rigorously set *against* maternal growth and *for* regular breeding. Breeding populations of domestic animals do not provide good data streams for the study of growth to maturity.

4. Process

4.1 Maintenance of body functions when there is no productive activity

A resting non-productive animal expends nutrients to maintain its life processes. This is a fixed cost which must be paid before any other use for ingested nutrients is considered. When comparing species of differing size it is apparent that smaller

animals have a higher demand for maintenance energy ($E_{maintenance}$) than larger animals. The regression seems to fit $W^{0.75}$.

$$E_{maintenance} = aW^{0.75} \tag{8.12}$$

The three-quarter power has been taken forward to provide a common base for the expression of maintenance requirements for nutrients within species, breeds and strains; although the rationale for this is obscure. If there is some view that heat lost at rest may be associated with the relationship between mass and surface area, 0.66 might be expected. More reasonable would be to ask from whence the major cost comes. As the likely answer is protein turnover, maintenance might be better related to the protein body mass. The rate of turnover of that mass decreases with animal size and degree of maturity, thus for the growing animal there is logic in:

$$E_{maintenance} = a^1 P_{total}^{0.75} \tag{8.13}$$

As the first step in nutrient allocation is to meet maintenance requirement, considerable effort has gone into its empirical measurement by the classical methodology of measuring heat output in a resting state. Once determined, the value for '*a*' is presumed constant, and at any value for W (or P_{total}) the flow of energy to $E_{maintenance}$ is fixed as a base requirement. Only subject to the subtraction of $E_{maintenance}$ from the ingested energy flow can the resourcing of growth begin. There is little logic in this, as the actively growing animal is not resting. None the less, the regression of energy intake on energy gain will show, by back extrapolation, a residual positive constant, representing energy expended when energy gain was zero (Fig. 8.5). While demonstrating that there is some fixed cost in the system with a priority demand for nutrients, even when there is active growth, it may bear little relationship to energy usage measured at rest.

If the costs of protein turnover lie at the heart of $E_{maintenance}$, they also lie at the heart of $P_{retention}$. Thus, as the animal progressively increases its rate of protein

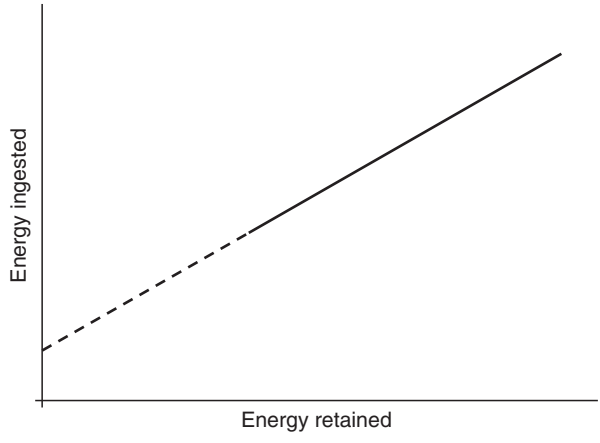

Fig. 8.5. The regression of energy intake on energy retained will show a constant value representative of energy expended when there is no growth activity (maintenance).

retention, the 'maintenance cost' is progressively shared and swallowed up. It is unsurprising therefore that when empirical determinations of maintenance are lower than expected, the simultaneous empirical determination of heat output consequent upon protein retention is higher than expected, and vice versa.

The actively growing animal is unlikely to perceive maintenance and production costs as separate entities. Therefore, while it may be rational for maintenance to be considered a fixed cost for the animal that is not actively growing, it may not be rational to consider this activity as a priority for resource allocation in the actively growing animal, where protein turnover is accounted within the demands of retention.

At zero protein retention there will be losses of protein moieties from the body. This level of loss may be termed 'protein maintenance'. Similar argumentation should apply as for energy.

4.2 Driving the body functions associated with productive activity

Protein retention has two associated energy costs: that of the energy residing in the retained body protein, and the energy cost of getting the absorbed amino acids structured and accreted. The energy cost of protein retention appears closely related to protein turnover, the rate of turnover being a number of multiples greater than the rate of accretion. As suggested above, the major heat output from the growing animal will emanate from the anabolic processes associated with protein retention. The cost per kilogram of accreted protein will increase with increasing mass of protein being turned over and the rate of the turnover. These are probably inversely related, but for any given animal the cost of protein retention appears to become greater as its mass increases. Overall these possibilities, the efficiency of use of energy for protein retention appears to vary rather considerably from some 0.4 to some 0.6.

Aspects of turnover are less relevant for the energy costs of lipid retention, and energy efficiency is relatively stable at around 0.75.

The outcome of this discussion on process is that in terms of the animal's knowing allocation of nutrient resources to functions, the following is likely:

1. At rather slow rates of growth, the animal is making partition decisions which prioritize to satisfaction of energy and protein requirements for maintenance before movement of nutrient resources to growth ($P_{\text{retention}}$ and $L_{\text{retention}}$).
2. At rather faster rates of growth, the animal is making partition decisions which elide *from* prioritization to satisfaction of energy and protein requirements for maintenance *towards* prioritization of nutrient resources to growth as adumbrated earlier.
3. At target growth rates where the potential for $P_{\text{retention}}$ is approached, the animal may substantially disregard any interest in prioritizing to the maintenance functions.
4. The demand upon nutrients per unit of lean tissue growth may increase with increasing animal size.
5. The costs of maintenance at lower rates of gain would suggest that there is intrinsic efficiency and logic in the animal driving to maximize its rate of protein growth per unit of time.

5. Competitive Processes

5.1 Activity

Foraging involves expenditure of the very nutrients sought. Activity is often considered as part of the 'maintenance requirement', and as such will be prioritized above growth. However, at high rates of activity the expenditure may become uneconomical.

5.2 Disease

Ill health causes a reduction in food intake, simultaneously with an increase in the demand for nutrients to support either the defence response to overt disease, or the immune response to prevent overt disease. Animals in environments with high levels of endemic disease have substantially increased 'maintenance' requirements. Disease will also increase the rate of nitrogen loss from the body, reducing the available protein resource. The animal has no option, when faced with disease, but to prioritize the flow of nutrients towards disease response and control.

5.3 Cold

Body core temperature must be maintained. In the cold, energy will be diverted away from growth creation and into heat creation (cold thermogenesis). However, the heat output from essential body functions is able to double in cold environments to keep the animal warm. Thus, cold will only impact upon animals whose rate of heat loss resultant from driving the anabolic processes of growth are insufficient in themselves to keep the body warm. In effect, metabolically busy bodies (those growing faster) will not be cold at the same ambient temperature as metabolically less busy animals. It is the latter that will need to respond to cold environments by burning dietary energy to keep warm. This exacerbates the problem: a reduction in growth reduces the rate of waste heat loss, which in turn causes the animal to need to use dietary (and body storage) energy to keep warm. Slow-growing animals need warmer environments than faster-growing ones.

6. Context

6.1 Product quality

Over recent decades selection in our domestic meat-producing animals has been in favour of increasing lean tissue growth rate ($P_{\text{retention}}\text{max}$). With this there has been an increase in mature lean mass. Meat breeds of pigs and poultry are substantially larger at maturity than half a century ago, and similarly (but to a lesser extent) for cattle. Selection for efficiency may also have reduced $(L_{\text{total}} : P_{\text{total}})_{\text{preferred}}$. The more rapid path to the latter is to use entire males for meat production in place of the castrate.

The outcome from this selection has been fast, lean and efficient meat production. In the context of a need for human food protein at a low price, this outcome is laudable. But the context is changing. In many parts of the world animal protein is no longer in short supply, and the cost of food is no longer a primary consideration. The quality of the eating experience is becoming dominant. The selection pressures of the previous century have not favoured eating quality, and the perceived wisdom is that modern livestock do not deliver this attribute well. The growth rate may be slowed by dietary manipulation. However, a fast-growing (high P_{total}max) large genotype will respond to this to produce a different product to that which would have resulted from the same (absolute) nutrient supply delivered to a slower-growing (low P_{total}max) genotype. Further, slower growing animals require to be slaughtered at lower weights, and as they will be older for any given weight, the use of the entire male becomes questionable.

6.2 Passive allocation

Often in the course of an animal's growth to maturity, the envelope for lean tissue growth potential is never approached. The rate of nutrient flow into the animal may be adequate only for limited growth, and the animal well content to attain $(L_{total}:P_{total})_{preferred}$. This allocation rule may therefore be more important than the attainment of $P_{retention}$max. Meanwhile, the preferred ratio of lipid to protein in the body is rather variable. Not only does it differ widely between species and breeds (often being a defining characteristic of a species and breed), but also within strain, and there are substantial sex differences. Furthermore, this 'preference' may be overridden with manageable consequences for the animal concerned. In cases of dietary imbalance in favour of protein, the body lipid resource will be used to optimize the use of available amino acids for growth. In cases of dietary imbalance in favour of energy, the body lipid stores will be supplemented.

The mammalian neonate has little lipid in its body. The priority for early growth (regardless of the previous discussion about the nature of the protein growth curve) is evidently lipid deposition. Indeed, the rate of lipid retention is never greater in the life of a mammal than when it is young, and well outstrips protein retention. The composition of mammalian milk, being energy rich, enforces this. Efficiency is not compromised; the youngster simply uses all the nutrients presented to it in an optimal way.

Throughout the period of growth to maturity, in most of the circumstances likely to prevail, it may be argued that the animal may not so much make active resource allocation decisions, as simply adapt passively to the nutrient resources that are available. Are animals more often to be found in environments where adaptability and flexibility are the operational attributes that prevail? It may be concluded that animal growth response to nutrient supply will utilize the strategy of optimization of use of the nutrient supply as presented within the larger framework of a mindful allocation of nutrient resources according to preferences and priorities. That is to say, animals may not always be proactive resource allocators, but often reactive resource optimizers.

9 Genetic Size-scaling

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1. Introduction

Despite some striking differences in appearance, all sizes and shapes of mammal have virtually the same biochemistry and physiology. The question is how to make use of this fact to benefit animal production theory.

Most traits of relevance to animal production are quantitative and their relationship to body size is surprisingly uniform over the whole mammalian range. This uniformity can be encapsulated in two formal genetic size-scaling rules, and these can then be used to furnish a quantitative description of the growth and development of a typical mammal (Taylor, 1980a,b). Genetic size-scaling is thus a body of quantitative theory that stems from the underlying physiological and biochemical similarity of mammalian growth. Genetic size-scaling depends on each genotype having its own genetic size parameter that operates at every stage of growth from embryo to adult. This size parameter can be estimated in various ways. At present, the simplest acceptable estimate is adult body weight standardized to a constant lipid content.

2. Defining the Parameters

2.1 Standard adult body weight, *A*

Body weight, changing continuously during growth, eventually, with adequate feeding, becomes more or less stationary at a level intimately related to the genotype of the individual. For this reason adult body weight plays a dominant role in studies of mammalian growth. The main non-genetic variable influencing adult body weight in healthy, moderately active mammals in a thermoneutral environment is nutrition. Provided they have not been severely undernourished during growth, and thereby permanently stunted, mammals of the same genotype, even with quite

 Resource Allocation Theory Applied to Farm Animal Production (ed. W.M. Rauw)

different growth and food intake curves in early life, will eventually grow to the same adult body weight when given the same adequate rate of food intake.

Equilibrium adult body weight changes as the level of constant food intake changes, varying from the low extreme of an emaciated adult, all skin and bone, to an extremely fat adult. Body fat represents reserves of energy stored internally. How much reserve should an animal have? It all depends on the circumstances. Let us therefore agree upon a conventional standard for body fat reserves where lipid constitutes a proportion of 0.15 of adult body weight. The associated adult body weight, A, can then be adopted as a quantified expression of an animal's genetic size-scaling parameter. If it were considered more appropriate within some particular context, a different lipid proportion could be adopted for standardizing adult body weights. The disadvantage, however, would be that such results could not be used in comparative mammalian studies unless all species were standardized to this same lipid proportion.

Jenkins and Ferrell (1997) found that, within each of nine breeds of cattle, equilibrium (empty) body weight, W_E, was linearly related to its lipid proportion, l_E, by:

$$W_E = a + b\,(l_E - l_A) \tag{9.1}$$

where l_A is the lipid proportion adopted for defining standard adult body weight, A, and where the regression constants a and b differed to some extent from breed to breed. If $l_E = l_A$ in (9.1), then $W_E = A$, so that a is seen to be an estimate of A, available only if a regression line has been calculated for that breed or sire group. Otherwise, given an approximate value for b, an estimate is $A = W_E - b\,(l_E - l_A)$. As it was more appropriate for their data, Jenkins and Ferrell (1997) set l_A to 0.25 and estimated $A_{0.25}$ for breeds and also for individual animals, using the appropriate b-value for each of the nine breeds. A most useful result was that the slope b was proportional to A, with $b = b^*A$, where b had an overall average value for all breeds of $2.5A$. Given l_E and W_E, standard adult body weight, A, with 0.15 chemical fat, can be estimated as:

$$A = W_E\,/[1 + 2.5(l_E - 0.15)] \tag{9.1a}$$

For dissected carcass fat and equilibrium body weight, a similar (unpublished) value of b^* was obtained from an experiment with identical twin Ayrshire cattle described by Taylor and Young (1968). The value of b^* for other species would have to be established, but equation (9.7a), which is the size-scaled form of Equation 9.1, suggests that other mammalian species might have a similar value.

When a standard lipid proportion other than $l_A = 0.15$ is used, the difference between two equilibrium weights with different lipid proportions remains unchanged at $2.5A(l_1 - l_2)$. For example, for $l_1 = 0.35$ and $l_2 = 0.15$, the difference is always $0.5A$. Their ratio, however, is different for every l_A. The advantage in taking $l_A = 0.15$ is that it yields realistic ratios. Equation 9.1 gives $W_E = 0.67$ for $l_E = 0.02$ only when $l_A = 0.15$, in line with a normal animal, neither fat nor lean, being able to lose about one third of its body weight, before the lower limit of 0.02 essential lipid is reached. Moreover, an animal fattened from $l_A = 0.15$ up to $l_E = 0.35$ shows a realistic increase in body weight of 50%, from A to $1.5A$.

The emphasis in this section is on defining A in terms of equilibrium body weight, rather than on how to estimate it, which is discussed in Section 6. Since

the general size of a species is roughly in keeping with its body weight, it is not unreasonable to refer to A as a general genetic body-size parameter.

2.2 Body size and biological time

Genetic size-scaling for time variables had its genesis when Clark (1927) published his extraordinary collection of data on heart pulse rate. He gave an interspecies regression coefficient of −0.27 for both mammals and birds, which implied that pulse rate varied with $A^{-0.27}$. Since then, the length of most biological periods has been found to increase systematically with increasing adult body size of species. The relationship is well documented by Gunther and Guerra (1955), whose average coefficient for many traits was 0.27. Lindstedt and Calder (1981) extended the list of traits and gave interspecies regression coefficients ranging from 0.18 to 0.39. Peters (1983) further extended the number of traits and gave coefficients ranging from 0.08 to 0.49. The average coefficient was close to 0.27 in both cases. Bonner (1965), in a fascinating study of life cycles, used the enormously extended size range from viruses to sequoia, to show that generation interval was related to body mass with a coefficient of 0.27.

These genetic relationships between biological time variables and body size are thus well authenticated and well established in the literature. What is not clear is whether all coefficients are uniformly 0.27 or whether (as was widely believed) there are genuine variations about 0.27 for different traits and in different subgroups of species. For the same trait, different authors have reported coefficients which, based on their quoted standard errors, were significantly different, but these were almost certainly due to non-random sampling of species. Taylor (1980a) believed that the majority of reported regression coefficients were potentially consistent with a single common value of 0.27. Even if they are not, there is a huge advantage in postulating that they are, and seeking an (adaptive) explanation for any genuine proven deviation. For example, the almost constant gestation length of breeds within a species is presumably necessary for interbreeding, and has several interesting consequences. If the value of 0.27 were to be universally adopted, it would provide a uniform scale for genetic comparisons of time variables.

2.3 Metabolic age, θ

In order to make full use of the relationship between biological time variables and standard adult body weight, Taylor (1965) proposed accepting 0.27 as a universal value, thus enabling ordinary age to be replaced by a metabolic age scale. This metabolic age scale is derived from Brody's (1945) physiological age scale, as discussed in Section 3.2. To distinguish it from Brody's age scale, the word 'metabolic' was chosen in acknowledgement of Kleiber's (1961) phrase 'metabolic turnover time' or simply 'metabolic time', which he used when discussing the time interval between successive administrations of a drug. Brody's physiological age is based on two parameters, whereas metabolic age is based on only one, namely the standard adult body-size parameter A. Metabolic age, θ, can be defined as:

$$\theta = \lambda t A^{-0.27} \tag{9.2}$$

Table 9.1. Expected temporal lifeplan of a mammal.

Metabolic age (metabolic days)	Developmental event
0	Egg enters the uterus (3.5 days after ovulation)
4	Egg implants
7	First somites appear
10	Is 0.00001 mature in body weight
14	End of metamorphosis
15	Fetal eyelids close
40	Is 0.04 mature in body weight
50	Is born 0.05 mature in body weight
90	Is weaned
100	Is 0.4 mature in body weight
110	Becomes sexually mature
115	Is 0.5 mature in body weight
150	Starts first lactation
200	Ends first lactation, having yielded 16A MJ of milk energy
270	Tibial epiphyses fuse
440	Is 0.98 mature in body weight
500	Is fully mature in body weight
700	End of prime of life
1000	First onset of cancer ($P > 10^{-3}$)
2000	Dies (maximum life span, after 5×10^8 pulse beats)

where t is the age in days from conception, A is the standard adult body weight parameter, *always measured in kilograms*, and λ is a dimensional unit that allows metabolic age to be expressed in metabolic days (which can readily be converted to metabolic seconds, minutes, hours, weeks or years as seems appropriate). It should be stressed that the metabolic age scale is always based on the standard adult body weight parameter A, and never on any immature body weight. For embryonic stages of development, a slightly modified and more precise definition of metabolic age should be used (see Taylor, 1965).

When all time variables associated with mammalian growth and metabolism are transformed to this metabolic age scale, there emerges a pattern of developmental events common to all species. This temporal life plan for a typical mammal is illustrated in Table 9.1, with a selection of events taken from Taylor (1986). The metabolic age estimated for several of the events may not be very accurate. The numerous deviations of species from these expectations illustrate the genetic diversity among mammals.

2.4 Degree of maturity, *u*

One further variable has still to be defined. Any immature body weight, W, can be expressed as a proportion of its standard adult weight, A, to give a degree of maturity, u, defined as:

$$u = W/A \tag{9.3}$$

so that:

$$W = Au \tag{9.4}$$

which allows any weight, W, to be partitioned into two components: its current degree of maturity, u, and its unchanging genetic body-size parameter, A.

When different genotypes are compared at the same degree of maturity, all immature body weights are, by definition, directly proportional to their size parameter A. The same is true when normally growing mammalian species are compared at the same metabolic age, since (as we shall see shortly) they will all have reached closely similar degrees of maturity.

Direct proportionality to parameter A has also been shown to apply to amounts such as energy stored, energy inputs and outputs, and volumes, provided these have been cumulated over corresponding metabolic age intervals.

2.5 Genetic size-scaling rules

The two genetic size-scaling rules proposed by Taylor (1980a) for applying to bioenergetic and growth variables can be simply stated as follows:

1. Treat all age and biological time variables for the ith genotype as proportional to $A_i^{0.27}$, where A_i is the body-size parameter (in kilograms) of the ith genotype.
2. At ages standardized as in Rule 1, treat all cumulate growth and bioenergetic variables for the ith genotype as proportional to A_i.

These two rules provide a simple but general procedure for introducing information on genotypic differences in size and time into equations, experimental designs and quantitative calculations.

'Cumulate variables', or simply amounts, are normally weights measured in kilograms, energy measured in joules or volume measured in litres. The conditions under which the scaling rules can legitimately be applied to these variables are discussed later.

It is immediately clear from the scaling rules that any amount per unit time will be proportional to $A^{0.73}$. During the second third of the 20th century, it was established that growth rate, caloric intake and metabolic rate of mammalian species were dependent on the 0.73 power of their adult body weight. It began with Kleiber (1932), who found that the basal heat production of mature mammals was proportional to their adult body weight to the power 0.74. A few months later, Brody and Proctor's (1932) famous mouse-to-horse (later mouse-to-elephant) relationship yielded an exponent of 0.73, a value adopted as an international standard in 1935 (Brody, 1945, page 373), and amply confirmed (Kayser and Heusner, 1964; Peters, 1983). In pre-computer days, the data-based value of 0.73 was commonly modified to 0.75 for ease of calculation. Normal adult heat production (which is about twice basal) has an expected value of $7A^{0.73}$ Watts. Humans, for example, have $A^{0.73}$ roughly equal to 20, so the heat an adult produces (midway between basal and normal) is roughly equivalent to a 100-Watt electric bulb.

There has been, and to some extent there still is, confusion in the scaling used for metabolic rate. Often no distinction was made between comparing traits in genetically different animals or species all at the same stage of development, and comparing traits at different stages of growth in the same animal, breed or species. A common misconception led to the indiscriminate and unjustified scaling by $W^{0.73}$, widely referred to as metabolic body weight, and often used to scale growth rate, metabolic rate or caloric intake at any or every stage of growth. Despite this, Brody (1945) has shown that metabolic rate (measured as basal heat production) increased during normal growth with $W^{0.58}$ (not $W^{0.73}$). Immature body weights cannot legitimately be used for scaling. All genetic size-scaling uses A_i (or some equivalent) as the one-and-only size-scaling parameter over the whole period of growth.

3. Genetically Size-scaled Growth and Food Intake Curves

We now have all the ingredients needed to enable us to look at some examples of genetic size-scaling. These ingredients are the genetic body-size parameter, A, metabolic age, θ, degree of maturity, u, and the two genetic size-scaling rules.

3.1 Equilibrium maintenance requirement, e_m

A brief outline of the first equation to be size-scaled is as follows. For the same genotype, Taylor and Young (1968) found what they considered a key result, namely, that *equilibrium* body weight, W_E, associated with a proportion of body lipid, l_E, was directly proportional to the constant level of feed intake, f_E, of a standard pelleted diet. For f_E in MJ of metabolizable energy (ME) per day, they gave:

$$W_E = e_m f_E \tag{9.5}$$

where e_m is equilibrium maintenance efficiency in kilograms of body weight maintained per MJ of ME consumed per day. From their experiment with identical twin Ayrshire cattle with $A = 500$ kg, they found:

$$e_m A^{-0.27} = 1.7 \tag{9.6}$$

The standard daily adult food intake, f_{Ai}, for genotype i, is the daily food intake actually required (rather than that estimated from A_i) to maintain standard adult body weight, A_i, in equilibrium. The ith genotype's maintenance efficiency, e_{mi}, is the ratio of A_i to f_{Ai}.

Equation 9.5 can now be used as a simple but important first example of genetic size-scaling. The same result as that found by dividing an unscaled variable by A or $A^{0.27}$ can be obtained by replacing the unscaled variable by the product of the size-scaled variable and A or $A^{0.27}$ as appropriate. For example, age t would be replaced by $\theta A^{0.27}$. To size-scale Equation 9.5, we therefore replace the constant level of food intake per day, f_E, which is an amount divided by a time, by $f_E{*}A^{0.73}$, where $f_E{*}$ is size-scaled food intake, that is, intake per kilogram of A per metabolic day. Then replace e_m, which is the ratio of an amount to a rate, by $e_m{*}A^{0.27}$, where

e_m* is the size-scaled efficiency of equilibrium maintenance. Finally, writing $u_E A$ for W_E and dividing both sides by A, gives the size-scaled equation:

$$u_E = e_m * f_E * \quad (9.7)$$

where e_m* is a mammalian constant that applies to any equilibrium body weight, mature or immature, and to any species. The value of the equilibrium constant e_m* has already been well established in mature animals. When degree of maturity $u_E = 1$, then f_E* = f_A*, i.e. the size-scaled food intake in MJ per metabolic day required to maintain standard adult body weight, A, so that e_m* = $(f_A*)^{-1}$. Brody's (1945) value for maintenance requirement f_A*, in MJ of ME, was 0.60 (per unit metabolic weight per day or rather per unit weight per metabolic day) for adult animals in a normal (non-fasting) state. The efficiency of maintenance, e_m*, is therefore $(f_A*)^{-1} = (0.6)^{-1} = 1.67$ to be compared with the value from Equation 9.6 of 1.70 (kg weight per MJ of ME per metabolic day), obtained from immature equilibrium weights. A value for e_m* of 1.7 would appear to be applicable to all equilibrium weights, both *immature and mature*, including over-mature. Since equilibrium body weights can vary considerably in lipid proportion, equilibrium maintenance efficiency, e_m*, would appear to be independent of body composition.

Establishing an immature equilibrium body weight on a constant feeding level is, however, not something that can be done quickly. The metabolic fire in a normally growing animal takes a long time to die down, and any attempt to hold an animal at its current weight by feeding the appropriate equilibrium amount will almost certainly result in an initial loss in weight, due in part to the reduction in the weight of the digestive organs.

As another simple example, Equation 9.1, which dealt with the lipid proportion, l_E, associated with an equilibrium weight, W_E, can be size-scaled by replacing W_E with $u_E A$, replacing constant a, being a weight, with $a*A$, and likewise b with $b*A$, while l_E and l_A, being proportions, remain unchanged. Dividing throughout by A, gives the size-scaled equation as:

$$u_E = 1 + b*(l_E - l_A) \quad (9.7a)$$

since setting $l_E = l_A$ makes $u_E = 1$, and therefore $a* = 1$ also.

3.2 Postnatal growth

In his extraordinary and comprehensive book *Bioenergetics and Growth*, Brody (1945) analysed data on the growth in body weight of nine domestic mammalian species, ranging in size from mice to horses. For each species, he represented the postnatal growth curve (for $u > 0.3$) by a negative exponential asymptoting towards an adult body weight, A, namely:

$$W = A[1-\exp\{-k(t-t_0)\}] \quad (9.8)$$

where W is body weight, k is the exponent for rate of maturing towards asymptote A, and t is age from birth, with t_0 the extrapolated starting age when $W = 0$. He called $k(t - t_0)$ the physiological age of the species, and plotted the sequence of

immature body weights for each species, as a percentage of its adult weight, against its physiological age, and obtained a common curve for all nine species.

Taylor (1965) found a strong relationship (r = 0.94) between Brody's parameters k and A, namely, $k = cA^{-0.27}$, where c is a constant. Since k has the dimension of (time)$^{-1}$, this relationship was in line with the many previously published results for time intervals. He suggested that the most effective way to use this relationship was in terms of metabolic age θ. To size-scale Brody's Equation 9.8, write Au for W, and replace k with $cA^{-0.27}$ so that physiological age, $k(t - t_0)$, equals $cA^{-0.27}(t - t_0)$ = $c(\theta - \theta_0)$. Brody's equation yields the genetically size-scaled maturing curve in terms of degree of maturity u and metabolic age θ:

$$u = 1 - \exp\{-c(\theta - \theta_0)\} \tag{9.9}$$

where c and θ_0 are mammalian constants, estimated from Brody's data as c = 0.01 and θ_0 = 50. This genetically size-scaled equation for $u > 0.3$ provides a common expected mammalian postnatal maturing curve for all species. To obtain the expected curve for any genotype with a body-size parameter A, replace u in Equation 9.9 with WA^{-1}, and θ with $tA^{-0.27}$, and set c equal to 0.01 and θ_0 equal to 50. This gives the expected (predicted) one-parameter description of postnatal growth, for $u > 0.3$, as:

$$W = A[1 - \exp\{-0.01\ (tA^{-0.27} - 50)\}] \tag{9.10}$$

The estimate of 0.01 for c will be dependent on how these domestic species were fed. Obtaining accurate estimates for mammalian constants is a recurring problem. Not only are large and comprehensive sets of data needed, but comparable systems of feeding also need to be used (see Section 3.5). Consistency among the estimates for different but related traits is also required. Unfortunately, the accuracy of predicted values or curves depends on how well the mammalian constants have been estimated.

While for each normally growing species metabolic age leads to a growth curve that can be represented by one genetic size-scaling parameter, a further important advantage is that the deviation of any one species from the common expected curve for all species can now be measured. This makes it possible to assess species' deviations freed from the effect of body size. In the case of Brody's growth curves, genetic size-scaling showed that, after eliminating the inherent differences associated with body size, rabbits appeared to have an inherent capacity to mature more rapidly than any of the other species, with guinea pigs the slowest maturing. This difference, being in line with a similar difference in early growth, is probably genetic, although a difference in nutrition cannot be ruled out.

3.3 Cumulate food intake, F_t, and net efficiency of growth, e_g*

Spillman (1924) described body weight W_t at age t for normally growing species in terms of their food intake F_t, cumulated from birth up to age t, by the equation:

$$W_t = A[1 - \exp\{-\alpha(F_t - F_0)\}] \tag{9.11}$$

Size-scaling by replacing W_tA^{-1} with u, also F_tA^{-1} with F_t^* and F_0A^{-1} with F_0^*, gives:

$$u_t = 1-\exp\{-\alpha^*(F_t^*-F_0^*)\} \tag{9.12}$$

where α^* replaces αA to match the change from F to F^*, and where the subscript specifying age at observation can be either t or θ at choice. Values found for α^* averaged about 0.04 (Parks, 1982), although some uncertainty attaches to the conversion of units of measurement for food intake. For rapidly fattening ruminants, a value of 0.025 may be more appropriate. Differentiating (9.12) gives:

$$\frac{\mathrm{d}u}{\mathrm{d}F^*} = \frac{\mathrm{d}u}{\mathrm{d}\theta}\Big/\frac{\mathrm{d}F^*}{\mathrm{d}\theta} = e_g^*(u) \tag{9.12a}$$

the gross efficiency of growth measured as the amount of growth per unit of food energy at degree of maturity u, namely:

$$e_g^*(u) = \alpha^*(1-u) \tag{9.13}$$

Gross efficiency declines as the maintenance requirement increases from zero at $u = 0$, so that $e_g^*(0) = \alpha^*$ is an estimate of the (net) efficiency of growth, which will be referred to simply as e_g^*. Note that $e_g^* = e_g$, both being the ratio of growth and feeding rates, and therefore not affected by size-scaling. The growth efficiency will usually be written as e_g^* to make it clear that it is size-scaled. Since Equation 9.13 does not necessarily give a good fit to data, this method of estimating e_g^* may not be reliable. The observed decline was exponential rather than linear in the ABRO multibreed experiment described by Taylor *et al.* (1986), where extrapolation yielded a tentative extreme estimate of 0.04 for e_g^*, but any value between this and 0.02 was acceptable.

3.4 Daily food intake, *f*

An equation describing the feeding rate of animals growing normally on an adequate diet is basic information in any study of growth and food efficiency. Loosely defined as the '*ad libitum*' feeding curve, it defines the upper limit set by appetite, and so depends on the quality of the diet. Published data (from a variety of sources) on feeding rates for eight mammalian species yielded the following sized-scaled equation for food intake in MJ of ME per metabolic day as a function of degree of maturity for normally growing animals:

$$f^* = 0.81[1.02-\exp(-4u^{1.4})] \tag{9.14}$$

which gave an excellent fit to the mean curve, although a poor fit to several of the individual curves, possibly because of different experimental conditions and uncertain food energy values. With so much variety in the observed curves, taking their mean may well have distorted the shape of any common underlying feeding rate curve.

Nevertheless, this equation can be used to obtain another estimate of the growth efficiency, e_g^*. Normally, growing animals reach their maximum growth rate when about one-third mature, and this part of the growth curve provides a reasonably good size-scaled estimate for growth rate of about 0.007 degrees of maturity per metabolic day. Equation 9.14, with $u = 0.33$, gives feeding rate

f^* = 0.48 MJ of ME per kg of A per metabolic day. Equation 9.20 gives a corresponding value for total heat production h^* = 0.32, partitioned by Equation 9.7 into $u(e_m^*)^{-1}$ = 0.20 for equilibrium maintenance and the remaining 0.12 due to the work of growth. Growth energy stored is thus $f^* - h^*$ = 0.16. The total for growth becomes 0.12 + 0.16 = 0.28, giving an estimate for e_g^* of 0.007/0.28 = 0.025.

The growth and food intake equations of Parks (1975a), among others, were given in their size-scaled form by Taylor (1980b).

3.5 Combining growth rate, equilibrium maintenance and feeding rate

Studying the growth of animals without simultaneously involving food intake is likely to give results that are quite limited in scope. The chemical composition of both diet and growth obviously need to be included also in any comprehensive description of animal growth. In an excellent paper, Emmans (1997) presented just such theory, based on his concept of 'effective energy', and incorporating genetic size-scaling. For present purposes – that of illustrating the use of genetic size-scaling – it will be sufficient to consider the following simple but basic equation, used by Taylor and Young (1966), Blaxter (1968), Montiero (1975) and Parks (1975b) among others:

$$e_g^{-1}\frac{dW}{dt} + e_m^{-1}\cdot W = \frac{dF}{dt} = f(W,t) \tag{9.15}$$

which, when size-scaled, becomes:

$$(e_g^*)^{-1}\frac{du}{d\theta} + (e_m^*)^{-1}u = \frac{dF^*}{d\theta} = f^*(u,\theta) \tag{9.16}$$

Rewriting Equation 9.16 with $r^* = e_g^*/e_m^*$, the ratio of size-scaled efficiencies, gives:

$$\frac{du}{d\theta} + r^*u = e_g^* f^*(u,\theta) \tag{9.17}$$

When food intake is given as a function of θ only, with $f^*(u,\theta) = f^*(\theta)$ in Equation 9.17, the general solution of linear differential Equation 9.17 is:

$$u = \exp(-r^*\theta)\,[c + e_g^* \int f^*(\theta)\exp(r^*\theta)d\theta] \tag{9.18}$$

where c is some constant, usually negative.

The reciprocals of the growth and maintenance efficiencies, e_g^* and e_m^*, convert the growth rate from kilograms per day into the food energy needed to produce the growth, and convert the kilograms of weight maintained into the food energy per day needed for equilibrium maintenance, so that the sum of these two terms equals the rate of energy intake. Note that higher values of e_g^* and e_m^* imply increased efficiency.

3.6 Relation of size-scaled normal growth rate to its energy cost

The use of $(e_m^*)^{-1}u$ as a maintenance term has already been justified in Section 3.1. The growth-rate terms in Equations 9.15–9.17 can, to some extent, be justified as follows. One of Brody's (1945) three equations for heat production, h, during normal growth was, in size-scaled form:

$$h^* = 0.6u^{0.58} \tag{9.19}$$

If the equilibrium maintenance requirement, given in (9.7) as $0.60u$, is removed from Equation 9.19, then the energy cost or 'work' of growth, h_g^*, becomes:

$$h_g^* = 0.6(u^{0.58} - u) \tag{9.20}$$

Figure 9.1 compares the size-scaled growth-rate curve derived from eight mammalian species by Taylor (1985) with the curve for the energy cost of growth predicted by Equation 9.20, multiplied by 0.06 so that the maxima of both curves are matched. In the same way, the size-scaled curve for the energy cost of growth, derived from another of Brody's (1945) equations, predicted a curve identical in shape to the Gompertz growth-rate curve, namely, $-0.02u\ln u$, also shown in Fig. 9.1, but it did not fit the data quite so well, although it might be more appropriate for today's highly selected pigs, rabbits and poultry.

This close matching of growth rate and work of growth makes it a reasonable, although possibly an approximate, procedure to have both cost and amount of growth energy combined in the first term of Equations 9.15, 9.16 and 9.17. The growth efficiency, e_g^*, in Equation 9.17 will remain approximately constant as long as the composition of the body weight gain does not vary to any great extent. Emmans (1994) found that the amounts of (effective) energy required to lay down 1 g of protein and 1 g of lipid were remarkably similar, being 60 kJ for protein (of which only 24 kJ were stored), and 56 kJ for lipid (of which 40 kJ were stored). Furthermore, he found that the result applied to different species and different stages of maturity. If we suppose that 1 g of protein is normally associated with

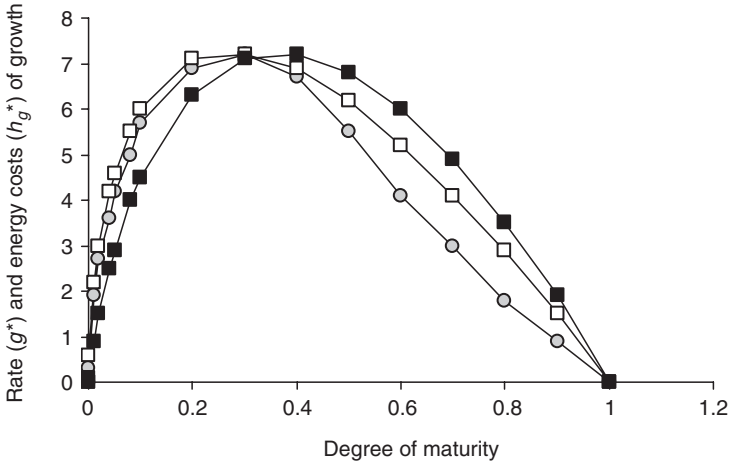

Fig. 9.1. Growth rate in degree of maturity per metabolic day ($g^* = du/d\theta$) and the energy cost of growth (h_g^*) in relation to degree of maturity (u); for g^* (–○–), the mean value from data on eight mammalian species; for h_{g1}^* (–□–) = $0.036(u^{0.58} - u)$ (which is 0.06 times Equation 9.20); and for h_{g2}^* (–■–) = $-0.02u\ln u$ (which is identical to the Gompertz maturing rate curve). The equations for h_{g1}^* and h_{g2}^* were converted to maturing rate equivalents by a factor equivalent to $e_g^* = 0.06$. The y-axis has been multiplied by 10^3.

about 3 g of water and ash, the energy cost of 1 g of lipid-free weight gain would be 15 kJ. The energy cost of 1 g weight gain with a lipid proportion l, would then be $(15 + 41l)$ kJ, and the efficiency of growth in mass, e_g*, being the reciprocal of this, would vary from a maximum of about 0.07 g/kJ for animals depositing very little lipid, down to about 0.02 for animals depositing large amounts of lipid. For intermediate levels of lipid, we might expect e_g* to be about 0.04 or 0.05. Montiero (1975) found that for Friesian cattle, the decline in his measure of efficiency of growth with age was slight, but was more apparent in the Jersey breed, which deposited relatively more fat.

Finally, with Equation 9.17 to some extent justified, we return to note that the feeding rate, f*, can be chosen in advance as a function of u and/or θ, and the differential equation can then be solved to give the resulting expected growth curve.

4. Growth Curves Derived from Given Feeding Rates

Size-scaled growth equations are now derived from five different types of feeding rates, including rates that are zero, constant, proportional to a power of body weight and cyclical.

4.1 Feeding rate proportional to body weight

Suppose feeding rate was set at $f = aW$ or in size-scaled form $f^* = a^*u$. Equation 9.17 then becomes:

$$\frac{du}{d\theta} + r^*u = e_g^*a^*u \tag{9.21}$$

which, on integration, gives:

$$u = \exp[(e_g^*a^* - r^*)(\theta - \theta_0)] \tag{9.22}$$

Feeding in proportion to body weight is thus expected to produce exponential growth, except when the exponent is set at zero by feeding an equilibrium amount given by $a^* = (e_m^*)^{-1}$, growth being positive when a^* is greater than this, and negative when less. Exponential growth, by keeping cumulate maintenance to a minimum, would probably be highly efficient. In practice, a limit to appetite will nearly always be reached, which will prevent this type of intake curve from being followed, except over limited periods. For early growth, St C.S. Taylor and G.B. Young (1967, unpublished) examined this feeding regime using twin Ayrshire cattle. The growth curves resulting from two levels of feeding in proportion to body weight were both rough approximations to those expected, but more linear than exponential, compatible with the animals fattening quite rapidly and growth efficiency, e_g*, possibly declining.

4.2 Feeding rate constant

As another simple example, suppose the feeding rate is kept at a constant level with $f^* = f^*_E$. The resulting growth curve obtained from Equation 9.17 is:

$$u = u_E\,[1-\exp\{r^*(\theta - \theta_0)\}] \tag{9.23}$$

where the equilibrium degree of maturity $u_E = e_m{}^* f^*_E$. For a constant level of feeding at or below the adult maintenance level, the growth curve thus has an asymptotic approach to the equilibrium degree of maturity. The full transition from $u = 0.3$ to 1 on the same constant feeding level is likely to be beyond the bounds of an animal's appetite, except perhaps with a highly nutritious diet. At full maturity, $u_E = 1$ and Equation 9.23 then becomes Equation 9.9, namely, Brody's postnatal growth curve for $u > 0.3$. But with one interesting difference. The exponent c in Equation 9.9, which has been replaced by r^* in Equation 9.23, can therefore be interpreted as the ratio of the efficiencies of growth and equilibrium maintenance. The estimate of r^* from Brody's growth curves was 0.01, but this has to be reconciled with some higher values. Blasco *et al.* (2003), for example, when fitting Gompertz growth curves to selected and control lines of rabbits, obtained values of r^* (their $k/A^{0.27}$) all close to 0.02 (range 0.019–0.021). However, Blasco *et al.*'s rabbits are likely to have had a much higher food intake than did Brody's domestic species in the 1930s. Adopting a value of 1.7 for $e_m{}^*$ leads to values varying from 0.017 to 0.034 for $e_g{}^*$, with a mid-point of 0.025, corresponding to a value of 0.015 for r^*.

If, at any point, the constant feeding rate were set to zero, Equation 9.17 then predicts an exponential decline in body weight, with the same exponent r^* for all mammalian species.

4.3 Feeding rate that results in a Gompertz growth curve

Equations 9.22 and 9.23 suggest that a sigmoid curve, with a point of inflexion at $u = 0.3$, would result from feeding in proportion to body weight, W, from $u = 0$ to 0.3, followed by a constant feeding level from $u = 0.3$ up to 1. Suppose we were to feed animals in proportion to W, but with a progressive exponential brake on appetite, to give a feeding rate specified by:

$$f = aW[1 + \exp\{-\beta(t - t_0)\}] \tag{9.24}$$

that is, in proportion to the product of their body weight and a quantity declining, at an exponential rate, from a large value when u is small, to 2 at the point of inflexion when $u = 0.37$, and finally to 1 at $u = 1$. After size-scaling and introducing Equation 9.24 into Equation 9.17, this becomes:

$$\frac{du}{d\theta} + r^*u = e_g{}^* a^* u\ [1 + \exp\{-\beta^*(\theta - \theta_0)\}] \tag{9.25}$$

If we confine our attention to continuous growth to a mature equilibrium at $u = 1$, when $du/d\theta$ is zero and θ is infinite, we get $e_g{}^* a^* = r^*$, and consequently $a^* = r^*(e_g{}^*)^{-1} = (e_m{}^*)^{-1}$. Then, dividing throughout by u and integrating, and also setting the origin at the point of inflexion with $\theta_I = \theta_0 = 0$, and $u_I = \exp(-1) = 0.37$, results in:

$$\ln u = -r^*(\beta^*)^{-1}\exp(-\beta^*\theta) \tag{9.26}$$

In order to get the usual Gompertz curve, the interesting condition that $r^*(\beta^*)^{-1} = 1$ must also hold, so that $\beta^* = r^*$, the ratio of efficiencies, the same as was found for Brody's growth curve in Equation 9.23. This gives:

$$u = \exp\{-\exp(-r^*\theta)\} \tag{9.27}$$

Since r^* is a mammalian constant, with a known value, and since $a^* = (e_m^*)^{-1}$ is also known, we can actually specify the size-scaled feeding rate curve as:

$$f^* = a^*u\{1 + \exp(-r^*\theta)\} \tag{9.28}$$

with approximate values of 0.015 for r^* and 0.60 for a^*. As u tends to zero, so also does f^*, since $u\exp(-r^*\theta) = -u\ln u$ from Equation 9.26 tends to zero as θ becomes large and negative. It may be simpler to have feeding rate determined, not by u and θ as in Equation 9.28, but by u only. Using Equations 9.28 and 9.27, we get:

$$f^* = a^*u(1-\ln u) \tag{9.29}$$

This is useful for applying at any point on the growth curve, but unfortunately the size-scaling parameter, A, needs to be known for the animal in question. For long-term planning in advance, the feeding rate is often best given in terms of age. Again combining Equations 9.28 and 9.27, we get:

$$f^* = a^*[\exp\{-\exp(-r^*\theta)\}]\{1 + \exp(-r^*\theta)\} \tag{9.30}$$

but we still need to know the body-size parameter, A.

4.4 Feeding rate proportional to a power of body weight

We next look for the growth curve produced by a feeding rate $f = aW^{1-m}$, and so have, in size-scaled form:

$$f^* = a^*u^{1-m} \tag{9.31}$$

which, combined with Equation 9.17, gives:

$$\frac{du}{d\theta} + r^*u = e_g^*a^*u^{1-m} \tag{9.32}$$

For $m = 0$, which is feeding in proportion to body weight, growth would theoretically remain exponential as in Equation 9.22. For $m = 1$, we get Brody's growth curve as in Equation 9.9. For $0 < m < 1$, multiplying throughout by mu^{m-1} gives:

$$\frac{d(u^m)}{d\theta} + mr^*u^m = me_g^*a^* \tag{9.33}$$

The general solution, for any value of m, is the growth curve of Richard's (1959). Realistic feeding rate curves, however, all lie in the range $0 < m < 1$. For this range of m, integration gives:

$$u^m = e_m^*a^*\{1 - \exp(-mr^*\theta)\} \tag{9.34}$$

with an origin at $u = 0$ when $\theta = 0$. The resulting growth curves are sigmoid with their point of inflexion, given by $u_I = (1 - m)^{1/m}$, which moves up from $u_I = 0$ for $m = 1$ until it approaches its limiting Gompertz value of $u_I = 0.37$ as m approaches zero. Note that when m changes the curvature, it compensates appropriately by also changing the exponent. Note also that all parameters are already either known, or appropriately fixed for a given context.

This power class of feeding rates always results in an exponential approach of u^m to an equilibrium asymptote at $u^m = e_m{}^*a^*$. If a^* is chosen so that $e_m{}^*a^* < 1$, it will be an immature equilibrium. If $e_m{}^*a^* = 1$ in Equation 9.34, then the feeding rate has the standard adult value f_A, and an animal will grow along what can be described as its *standard growth curve for a given value of m*, and reach its standard adult body weight, A, at $u = 1$. If $e_m{}^*a^* > 1$, animals will fatten more during growth, and a heavier and fatter adult equilibrium will be reached, with the upper limit set by appetite at $u = 1.5$ or possibly more. These sigmoid growth curves would also seem the most appropriate for an animal moving from one immature equilibrium to a higher one, slowly at first, and gradually faster as its digestive organs become adapted to a higher food intake.

4.5 Cyclically fluctuating feeding rates

In 1969, to test Equation 9.17, St C.S. Taylor and G.B. Young set up an experiment at ABRO in which identical twin cattle were fed from 12 to 108 weeks of age on a standard all-pelleted diet with a metabolisable energy of 10 MJ/kg. Food intake, dF/dt, followed three linearly increasing food intake regimes, the highest and lowest acting as boundaries for two sinusoidally fluctuating regimes with one twin cycling up and down while its co-twin cycled down and up, with crossovers occurring every 24 weeks (Fig. 9.2A).

Since the experimental feeding rates were specified as mathematical functions (fairly closely matched in practice), they could be entered in Equation 9.17 and integrated to give the predicted growth curves for each of the five feeding paths (Fig. 9.2B). The growth curves were predicted to crossover 11, 9 and 10 weeks later than did the food intake curves. The observed crossovers were delayed by 9, 5 and 10 weeks. The final predicted and observed crossovers occurred at 94 weeks, with both close to 230 kg. The observed growth curves were in some aspects amazingly close to those predicted, while in other aspects they were only rough approximations (Fig. 9.2C). The data were analysed by Parks (1975b) when he came to ABRO.

When A was taken as 500 kg, the estimates of the parameter values were $e_g{}^* = 0.028$, which was quite acceptable, and $e^*_m = 1.23$, which was about 30% lower than the well-established value of 1.7. When the efficiency parameters were allowed to change with age, a much better fit was obtained, but then predictions became quite unacceptable. A better fit might also have been obtained with e_m set at 1.7 and $e_g{}^*$ allowed to decrease with age, but this was not tested. Taylor and Murray (1987), when analysing the results of a time-controlled feeding experiment with six breeds of cattle, found that when $e_m{}^{-1}$ was kept constant at 0.11 (corresponding to $(e_m{}^*)^{-1} = 0.6$ for $A^{0.27} = 5.5$), then $e_g{}^*$ declined on average from 0.032 for the period '3–9 months of age' to 0.024 for the period '18–24 months of age'. They also found that a value of 0.028 for $e_g{}^*$ gave a reasonable approximation over the whole 2-year period. While Equation 9.17 might give only approximations, yet it never yielded nonsense. Parks (1982) continued to test Equation 9.17 by examining many other sets of data, finding approximate agreement in general, with occasional serious discrepancies, as when the value of parameter $e_g{}^*$ increased with the level of dietary

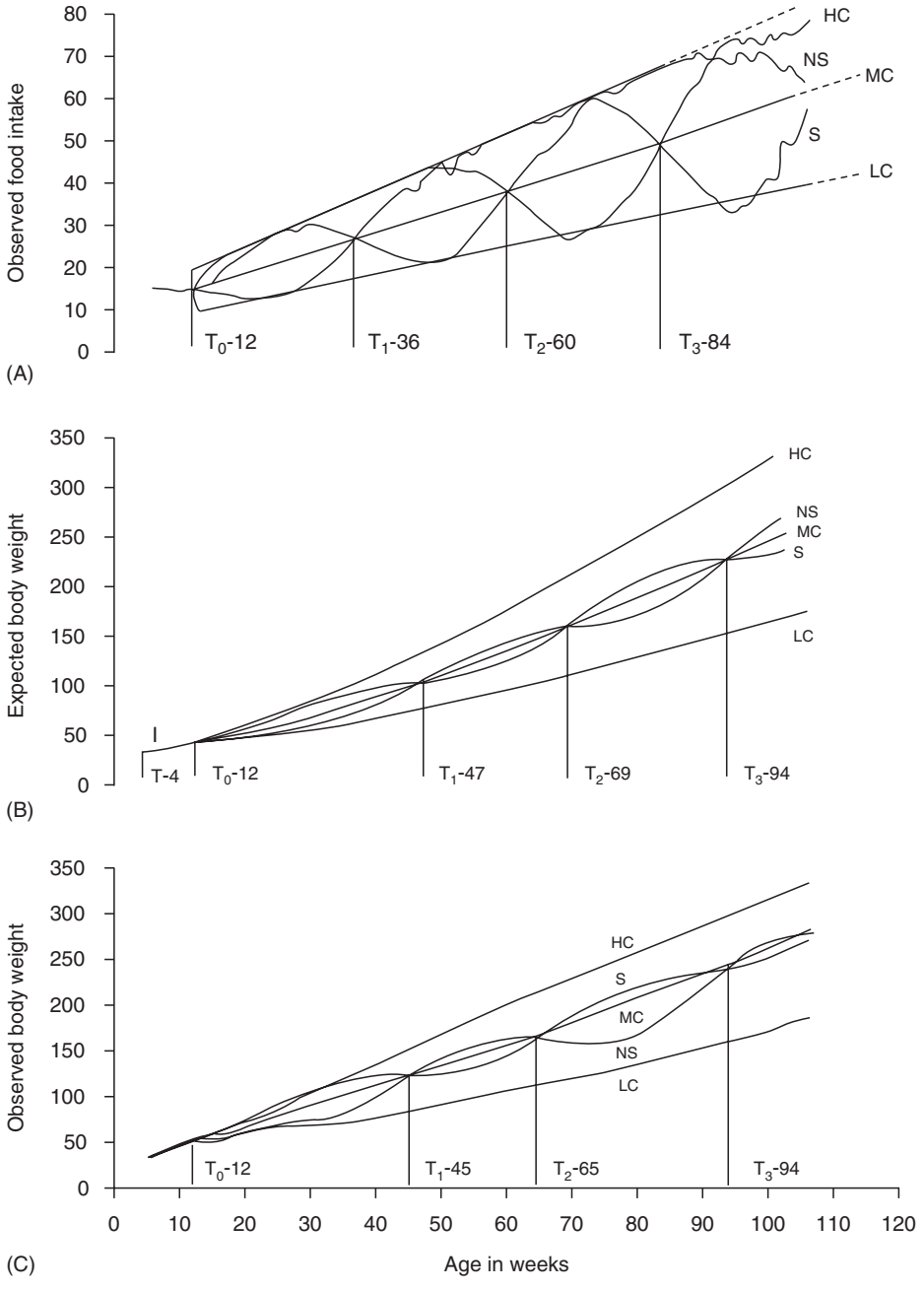

Fig. 9.2. Observed food intakes in MJ of ME per day (A), predicted body weights in kilograms (B) and observed body weights in kilograms (C); also ages at crossovers, for Ayrshire twin cattle on five feeding levels from 12 to 108 weeks of age. (After Parks, 1975b.) HC = high constant acceleration of feeding rate; MC = medium constant acceleration; LC = low constant acceleration; S = sine wave acceleration; NS = negative sine wave acceleration.

protein. A modified value of e_g* may therefore be required for certain foodstuffs. Nevertheless, better size-scaled equations, such as might be developed from those of Emmans' (1997) and based on his 'effective energy', should eventually replace Equation 9.17. For the present, the conclusion seems to be that, for any species, breed, sire group or individual, Equation 9.17 can be used to provide an approximate expected growth curve for a specified food intake function.

4.6 Food intake curves derived from given growth curves

The converse problem is, given a growth curve $u(\theta)$, to deduce what the associated food intake curve $f(u,\theta)$ might have been. It can readily be solved (since differentiation is simpler than integration) by differentiating $u(\theta)$ to give $\mathrm{d}u/\mathrm{d}\theta$, which can be substituted along with u itself into Equation 9.17. This gives the equation for food intake as $f^*(u,\theta)$. Since $u(\theta)$ is given, $f^*(u,\theta)$ can always be transformed into $f^*(\theta)$, the required food intake curve. As always, A is needed.

5. Allometry

Huxley (1932) first used the word 'allometry' as the measurement, during growth, of one body part, y, in relation to the whole or to another part, x. He also derived and popularized the power equation:

$$y = ax^b \tag{9.35}$$

where a and b are constants to be estimated. If y_A and x_A are the adult values of y and x, then $y/y_A = (x/x_A)^b$. Extending degree of maturity to individual measurements allows the size-scaled power equation to be written as:

$$u_y = u_x^b \tag{9.36}$$

The size-scaled constant $b^* = b$, and is thus unchanged by scaling, so that the power equation has the remarkable property of always yielding b values that are independent of genetic size, and immediately comparable across genotypes. This great advantage of the power equation (along with some others) is offset by two deficiencies. The first is lack of additivity of parts and the second is the inability to describe the relative growth of a trait that has immature values greater than its adult value.

These two serious deficiencies are remedied in the quadratic allometry equation of Butterfield *et al.* (1983), but at the cost of losing the considerable advantages of the power equation. If y_i is the ith part of some total, x, then Butterfield's equation is:

$$y_i = a_i x + b_i x^2 \tag{9.37}$$

where a_i and b_i are the constant coefficients of the quadratic. These individual equations can be summed over any or all of the parts. Omitting, for simplicity, the subscript in Equation 9.37, and size-scaling as for the power equation, and also using the condition that $u_y = 1$ when $u = 1$, gives:

$$u_y = q^*u + (1-q^*)\, u^2 \qquad (9.38)$$

This quadratic allometry equation is the size-scaled form of Equation 9.37, with the allometry constant q^* being independent of genetic size. It has the important property, when $q^* > 2$, of being able to describe a trait which has (in the range $0.5 < u < 1.0$) an immature maximum that is greater than its adult value, such as liver and intestinal weights (Butterfield *et al.*, 1983). Comparing coefficients in Equations 9.37 and 9.38 and writing p_A for the adult ratio, y_A/x_A, gives $q^* = a/p_A$ and $(1 - q^*) = x_A b/p_A$, so that $a + x_A b = p_A$. The size-scaled allometric constant q^* can therefore be estimated as $q^* = a/(a + x_A b)$, but only if x_A is known; or from a alone as $q^* = a/p_A$ if the adult ratio p_A is known. For most pairs of traits, p_A is likely to increase or decrease with genetic size. Both power and quadratic allometry equations have their advantages, to be made use of as appropriate.

The main aim of this chapter, however, is not to evaluate growth equations, but to illustrate the use of genetic size-scaling for combining data from genetically different material, for giving expected equations, curves, ages and values for any species, breed or genotype of given size, and finally for evaluating genetic deviations, both for biological interest and for use in animal production.

6. Some Final Comments

6.1 Conditions for valid comparisons

In general, traits should be compared at the same metabolic age or degree of maturity. Otherwise comparisons are doing no more than indirectly measuring obvious differences in body size. Thus, a large beef breed might appear to be producing beef more efficiently than a small breed if compared at the same immature body weight, but this advantage is simply an artefact of the large breed being at a younger metabolic age and less mature. For example, Cundiff *et al.* (1986) found that the inter-breed correlation between food efficiency and adult body weight, for the cross-bred steer progeny of 15 sire breeds with the same dam breed, was 0.85 when all steers were slaughtered at a constant body weight, but −0.05 at constant marbling.

Animal production experiments that have involved genetic size-scaling are, among others, those reported by Meyer (1995) in cattle, Butterfield (1988), Butterfield *et al.* (1983), Oberbauer *et al.* (1994) and Thonney *et al.* (1987) in sheep, and by Blasco *et al.* (2003) in rabbits.

6.2 Dimensional considerations

The body-size parameter, A, is measured in kilograms and so is strictly a mass, and not a weight which should strictly be measured in Newtons: but this equivalence is acceptable since gravity is effectively constant. Likewise, volume in litres is proportional to mass provided density is constant. Again, in the same way, energy is directly proportional to mass, provided caloric density or calorific value is constant. Constant mass and energy density are therefore theoretically necessary

conditions for the perfect accuracy of the size-scaling rules. However, these conditions of constancy need not apply restrictively. Deviations in mass or energy density about a regression on genetic size are quite acceptable and can provide important information – but only if they are not correlated with the body-size parameter, *A*. Fortunately, both regressions are effectively zero across the mammalian range of genetic body size.

6.3 Genetic selection

One of the main problems when attempting to improve animal production by breeding is deciding exactly which traits should be subjected to how much selection pressure. When selecting for growth rate, which is highly correlated with the body-size parameter *A*, most of the selection pressure will operate on *A*, and the outcome will be mainly larger animals that mature more slowly. A less-obvious example would be improving the important trait of maintenance efficiency by selecting for increased e_m values in Equation 9.1, using sire groups to measure the ratio of equilibrium weight, W_E, to the feeding rate, f_E, which maintains that weight. Equations 9.2 and 9.7, however, show that much of the genetic variation will be due to genetic variation in $A^{0.27}$, so that once again much of the effort will have been wasted on producing larger animals. To improve inherent maintenance efficiency that is independent of body size, one would have to select for $(e_m^*)_i = (e_m)_i A_i^{-0.27}$ for the *i*th genotype. Here again we come up against the major stumbling block of having to have a good estimate for A_i. If the considerable advantages of genetic size-scaling are to become readily accessible, it is important that clearly defined and reliable estimates are obtained, not only for A, but also for the mean values of the various parameters involved in mammalian growth, physiology and metabolism. Without these, the genetic deviations from expectation, which are needed for selection, cannot be evaluated.

6.4 Estimating body-size parameter *A* and efficiencies e_g^* and e_m^*

Undoubtedly the main difficulty in using genetic size-scaling lies in obtaining reliable parameter estimates. Good estimates of the parameter *A* require a greater effort than is usually considered acceptable, yet the improvement in genetic selection for growth and efficiency could more than compensate for the extra effort.

6.5 Estimation for species, breeds or sire progeny groups

Choose a growth curve appropriate to the context. Take (or evaluate if necessary) its associated feeding rate equation. Use it to feed two (or more) sire progeny groups so that they follow the chosen growth curve all the way to equilibrium body weights set at 20% or so above and below a guessed value of *A* (or use a series of levels). Record all food intakes, especially where there has been some food refusal. Interpolate, or calculate the regression of equilibrium weight on lipid level. The

weight corresponding to a lipid level of 15% is the required estimate of the sire's body-size parameter, A. The associated interpolated feeding rate will be the sire's standard adult feeding rate f_A. The ratio $A^{0.73}/f_A = e_m*$ will estimate the efficiency of maintenance for that sire. To estimate the efficiency of growth, fit the chosen growth curve to the data for each sire progeny group. The size-scaled maturing index, $r*$, has the same expected value for all sire groups, and the average value of $r*$ equals e_g*/e_m*, which yields an estimate of e_g*, the sire's growth efficiency parameter.

6.6 Estimation for individuals

One possible way to get a reasonable estimate of an individual's size-scaling parameter for A, would be to construct an estimation function based on a large number of traits and their variance–covariance matrix. The traits would include body weights and especially body measurements, both mature and immature, adult weight estimated by fitting a growth curve, measures of body composition, various time durations, in particular a maturing index estimated from a fitted curve, age at sexual maturity, lactation length, physiological measures such as minute volume, and so on. Of course, care must be taken not to prejudice the investigation of any trait by including it, too heavily weighted, in the estimation function. The rationale for this is the same as that for diagnosing the zygosity of twin cattle: one or two blood groups or one or two body measurements are of little value, yet in moderate numbers, both provide high accuracy, but only if all the information is used, and not just a linear regression or first principal component (Taylor and Murray, 1991). Such an estimate could be further reinforced by estimates of A that were available from parents and sibs and, since these might have observed adult body weights and measurements included in their estimates of A, they would be especially useful if the individual were itself immature.

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10 Allocation of Resources to Reproduction

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1. Introduction

For the progenitors of our domestic livestock, and for most modern animals under extensive management systems, the nutritional environment fluctuates widely throughout the year. During evolution, these animals developed physiological strategies for making the most efficient use of widely fluctuating energy supplies, with a special emphasis on coping with the nutritional demands of producing and rearing offspring. These strategies are still an integral part of the genetic make-up of most domestic species, even if management under domestication has reduced the potential impact of the environmental extremes. Our farm animals thus have a wide variety of intimate and complex connections between their metabolic and reproductive control systems, such that metabolic factors exert effects that range from enhancing reproductive output, when conditions turn favourable, to complete blockade of reproduction, when circumstances become so dire that the lives of parents and offspring are threatened.

In the context of resource allocation theory, the metabolic requirements for reproduction and the processes that control the allocation of resources are clearly relevant. However, it is essential to go well beyond the model of 'source, reserve and sink' because it implies passive rather than controlled processes. The concept of the fetus as a sink for resources is simply inadequate when both the fetus and the mother are making 'decisions'[1] in gene-determined processes at brain level that implement the finding, storing and transferring of energy for themselves and for each other. The same must apply to lactation, often the deepest 'sink' of them all. These maternal and fetal 'decisions' cover a range of timescales, from moment-by-moment perturbations

[1] We hope that we can be excused the teleology, admittedly a dangerous practice, but sometimes the arguments can be expressed more simply if we adopt the view that the animal 'considers' its options.

 Resource Allocation Theory Applied to Farm Animal Production (ed. W.M. Rauw)

to epigenetically programmed future performance in both mother (metabolic memory) and offspring (fetal programming).

The same criticism could perhaps be levelled at resource allocation theory. Concepts such as lifetime reproductive output, and the balance between length of reproductive life and rate of reproduction, are rarely an issue for farm animals; their life is usually cut short before their lifetime performance can be measured! It is also difficult to reconcile such concepts with the propensity for animals to make relatively instantaneous, but profoundly important, reproductive decisions, such as the production of two eggs rather than one only a few days before mating. As we shall see below, this decision can have little to do with any prediction of future abundance of energy or total lifetime output.

This chapter will begin with a brief overview of the physiological links between the metabolic and reproductive control systems, but we will not go into the details because authoritative reviews are already available (e.g. Robinson *et al.*, 2006; Blache *et al.*, 2007). Most of the chapter will, instead, focus on three major topics: (i) a quantification of energy demands and costs associated with the various stages of the reproductive process in males and females; (ii) the consequences of not satisfying those demands; and (iii) the value of 'nutritional pharmacology', in which we can take advantage of windows of opportunity in the reproductive process to provide acute, timely supplements that will improve reproductive outcomes at minimal cost. In covering these topics, we will deal mostly with the sheep, the animal with which we have the most direct experience, with occasional diversions to other species, because there is no information from sheep studies, or because there are species differences that need to be highlighted. In general, we will try to deal with broad principles that can be applied to all species.

2. Physiological Links Between the Metabolic and Reproductive Control Systems

Change in metabolic status, defined as change in the availability of nutrients and energy to the tissues, is a powerful regulator of reproductive function in both sexes. Studies in male and female ruminants, particularly the ram and the post-partum cow, have begun to reveal the nature and organization of the regulatory processes involved. This information has led to our current perspective of the relationship between reproduction and metabolism, in which there are four interdependent 'dimensions' (Blache *et al.*, 2007). In brief:

1. *Genetics*: The effects of metabolic status and dietary manipulation on the reproductive axis in males and females differ among genotypes in both sheep and cattle. Examples for post-partum anoestrus in cows and gonadotrophin secretion in rams are detailed by Blache *et al.* (2002, 2007).

2. *Structures*: The many organs involved are all localized points of regulation and integration. Within the reproductive system, these include the brain, the gonads and, to a lesser extent, the pituitary gland. Within the metabolic system, major roles for the liver have long been suspected, but the precise nature of the inputs is not clear. The relatively recent confirmation that fat depots are endocrine tissues

suggests that our list of regulatory organs is far from complete – for example, we now need to investigate the products of muscle (Chagas *et al.*, 2007).

3. *Communication networks*: The brain is the ultimate regulator of all bodily processes so the reproductive and metabolic centres of the brain are the destiny for all lines of information about metabolic status. This information is somehow integrated and conveyed to a common pathway that leads to the reproductive axis, the gonadotrophin releasing hormone (GnRH) neurons. The metabolic signals received by the brain centres can be hormones, nutrients or metabolites (glucose, insulin, leptin, ghrelin and kisspeptin), or they can be neural, such as inputs from the digestive system (review: Blache *et al.*, 2007).

4. *Time*: Metabolic history influences the response to changes in energy availability. For example, in dairy cows, the luteinizing hormone (LH) response to improved nutrition during post-partum anoestrus is affected by pre-calving feeding, regardless of body condition (Chagas *et al.*, 2007). In mature rams that are in high body condition, there is no increase in LH pulse frequency in response to an increase in intake, in contrast with animals in low body condition. Moreover, the LH response to an increase in intake fades within few weeks even if the nutritional input is maintained and the animals are still gaining weight (Blache *et al.*, 2007). Thus, some sort of 'metabolic memory' seems to be involved, perhaps modifying interactions among the various signalling systems according to metabolic history (Blache *et al.*, 2006).

This four-dimensional view of the relationship of reproductive function with metabolic status, as proposed by Blache *et al.* (2007), is a platform upon which we can build long- and short-term management practices to control reproduction in farmed ruminants. We need to add to it a thorough understanding of the energy requirements for each stage of the reproductive process, and that is the next topic in this chapter.

3. Quantified Energy Demands and Costs Associated with Reproduction

The best way to quantify the energy demands of reproduction is to use net energy (NE) so that variations in digestibility and metabolic efficiency are removed (Fig. 10.1).

In the interests of simplicity and economy of space, the values for energy requirements are rounded and, clearly, ignore other aspects of nutrition such as the requirements for protein, essential amino acids and fatty acids, vitamins and minerals (e.g. zinc with its quite specific roles in reproductive processes; Martin *et al.*, 1994). Thus, the first assumption that we have made, in the pursuit of brevity, is that these factors are not limiting. The other assumptions are:

1. For all homeothermic species, fasting heat (MJ/day) = $0.3BW^{0.75}$ (BW = body weight), and maintenance is fasting heat plus 20%.

2. Body mass is 50 kg for a female Merino sheep, 80 kg for a male sheep and 700 kg for a Holstein cow.

3. For the estimation of energy demands associated with milk production, a constant milk composition throughout lactation, no loss in body mass during lactation, energy densities (kJ/g) of 17.6 for lactose, 23.8 for protein and 39.3 for fat are assumed. Lactation in sheep is assumed to last 105 days with three phases (days 0–34, days 35–69 and days 70–105). Peak milk yield is assumed to be 1.0 L/day with a

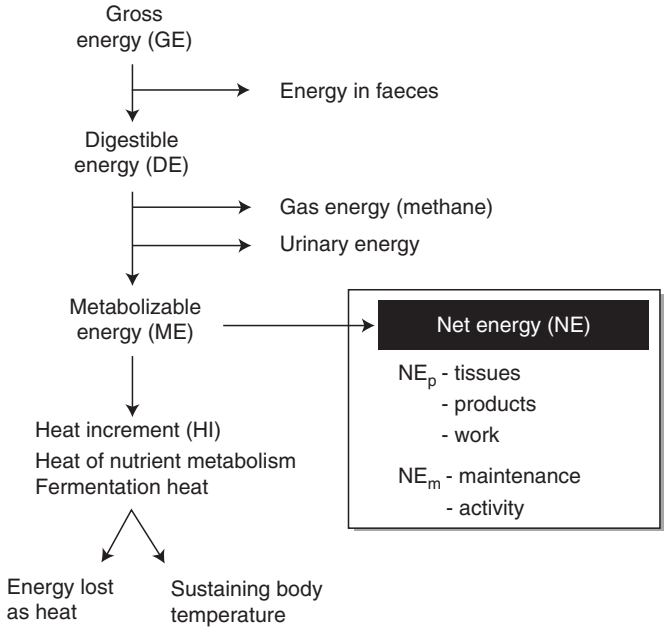

Fig. 10.1. The biological partitioning of food energy. Net energy is available for processes such as reproduction and lactation.

composition of 55 g lactose, 55 g protein and 80 g/l fat. The daily NE outputs are assumed to be 580 (phase 1), 470 (phase 2) and 350 kJ/Wm (phase 3).

4. The Gompertz equation (SCA, 1990) describes energy gains during pregnancy in sheep from 63 days after conception on: $\ln E\,(\mathrm{MJ}) = \mathrm{A} - \mathrm{B}\exp(-Ct)$, where, for the fetus $A = 4.695$, $B = 21.437$ and $C = 0.01728$, and for gravid uterus $A = 7.649$, $B = 11.465$ and $C = 0.00643$. For example, at 150 days after conception, the energy in the fetus is 1.13 MJ and the energy in the uterus is 1.18 MJ, so the total energy in the products of conception is 2.3 MJ. These values can be adjusted in direct proportion for twin fetuses by reducing the individual mass to 80% and then doubling the value: 3.68 MJ. Note: these equations do not take into account udder development in the latter stages of pregnancy.

4. Satisfying the Demands

Clearly, under very controlled conditions, the normal requirements for reproduction can be met, at least in theory, by supplying a complete, synthetic diet. This is typically the case for lactating Holsteins in North America, where normal industry practice is to keep the animals indoors and feed them a 'total mixed ration'. However, even under these conditions, internal drivers of appetite and metabolic balance, or perhaps genetic aberrations in the physiological systems that control appetite and metabolic balance, can prevent the animal from consuming its requirements, leading to unacceptably high levels of infertility (review: Chagas *et al.*, 2007).

In contrast, most of the world's domesticated herbivores are either free-ranging or extensively managed and live in a nutritional environment that varies from season to season. The feed energy that is available to the animals ultimately reflects the capacity of plants to grow and survive under seasonal patterns of temperature or soil moisture. In such environments, many forage plants survive by adopting an annual habit, growing rapidly during the favourable season, seeding copiously and then senescing before the harsh season. For the animals, the difference between periods of abundance and periods of deficiency can be enormous because, for much of the year, only poor-quality plant material is available to grazing animals and they need to select from stems, leaves, roots and seeds, and cope with a diet that is vastly different from the relatively brief period of lush, green pasture that they enjoy during the favourable months. Coping means at least surviving but, in the long run, it also means reproducing and providing for offspring. For domestic animals, it also means the economic delivery of animal products.

This problem is difficult enough in a climate that is seasonal but predictable from year to year, as is the case for most temperate regions. In Mediterranean, subtropical or semi-arid regions, however, the timing of the annual wet and dry periods is not entirely predictable and the nutritional value of pasture and forage can change over relatively short periods of time in response to seasonal rainfall events. The physiological systems in the animal need to be able to predict the annual cycle, but also respond to short-term fluctuations.

The annual cycle in reproduction is controlled by photoperiod and, in temperate regions, this leads to a harmonious relationship with the annual cycle in feed supply (Lincoln and Short, 1980). In contrast, in Mediterranean regions, for example, there is a shift in the phase relationship of the annual cycles and this leads to an incompatibility between the supply of nutrients and the seasons of reproduction. Thus, ewes that mate in late autumn, in response to photoperiodic drivers, would lactate over late summer and experience the greatest drain on their resources when the food supply was rapidly diminishing. Similarly, in subtropical conditions, where most of the rain falls in summer and autumn, the annual pattern of food supply differs from temperate regions primarily in spring when conditions are very dry. Under photoperiodic control, sheep and goats would kid on very poor pasture.

How do the animals cope with this problem? First and foremost, species that are adapted to non-temperate environments have usually developed an opportunistic reproductive strategy. Photoperiod still exerts a long-term, perhaps less-dominant, role in the control of the timing of reproductive events, but other factors, such as food supply and social conditions, become critical in the fine-tuning of the strategy (Bronson, 1985; Martin *et al.*, 2002). Since the late 1980s, we have been investigating these interactions and attempting to understand the physiological processes involved for sheep and goats. We will not detail this work here because it is not directly relevant and has been covered in many other review articles (e.g. Blache *et al.*, 2003, 2007).

Despite their opportunism, animals in these situations can struggle. To get a feeling for the difficulty, we will quantify the energetics for three scenarios with different relationships among the annual cycle of forage energy availability and the timing of reproductive events (Fig. 10.2). In Scenario 3, the energy available matches almost perfectly with the energy demands, whereas other regions, such as

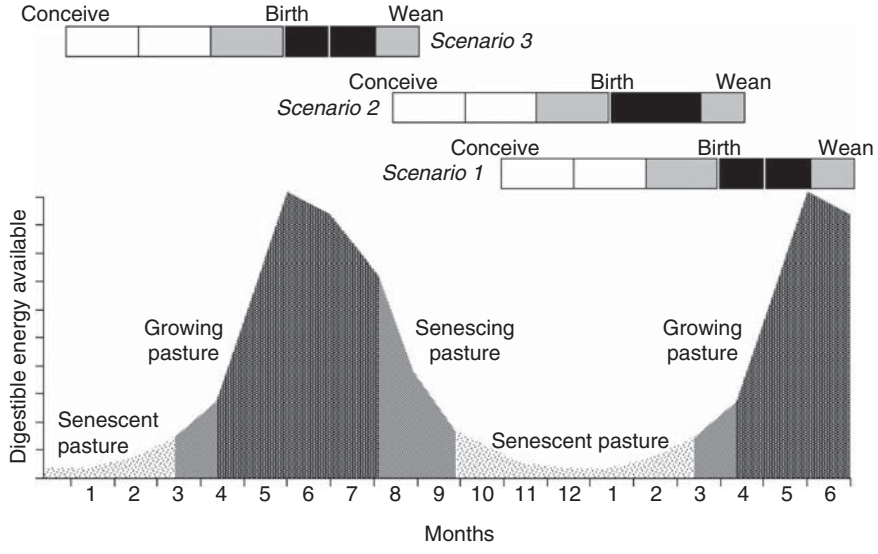

Fig. 10.2. The annual cycle of pasture availability and its relationship with three potential reproductive scenarios. Scenario 1 reflects the relationship for a temperate genotype trying to breed in a subtropical region. Scenario 2 reflects the relationship for a temperate genotype trying to breed in a 'Mediterranean' region. Scenario 3 is typical of animals adapted to temperate regions, with major reproductive events typical of a strict 'short-day breeder'.

the Mediterranean (Scenario 2) and subtropical (Scenario 1) are more problematic and the lack of coincidence of energy availability and demand can be a major risk factor in reproductive performance.

In contrast to the quantification of energy demands that we used above, it is not feasible to present the energy available in pasture as net energy because the efficiency of energy use varies with its destination in the animal and, during reproduction, we have major changes in the proportion of the NE that is used for maintenance, pregnancy and lactation. For example, the metabolizable energy (ME) of a high-quality pasture (14 MJ of ME/kg) will be used with an efficiency of 77% for maintenance and 60% for lactation. A low-quality pasture (7 MJ of ME/kg) will be used with an efficiency of 68% for maintenance (not much below the high-quality pasture), but only 30% for lactation. However, we can get a feeling for the demand–supply relationship if we make certain assumptions:

1. The digestibility of young, green pasture is 80%.
2. ME = 0.81 DE (i.e. the combined energy loss in urine and CH_4 is 19%).
3. ME is used with varying efficiencies depending on its use by the animal (SCA, 1990):

$$K_{\text{maintenance}} = 0.35\ Qm + 0.503$$

$$K_{\text{growth}} = 0.7Qm$$

$$K_{\text{fattening}} = 0.78\ \text{Qm} + 0.006$$

$$K_{\text{pregnancy}} = 0.12\ \text{Qm}$$

$$K_{\text{lactation}} = 0.35\ \text{Qm} + 0.42$$

where Qm is the MJ of ME as a proportion of the GE of the diet.

Importantly, applying these factors will accentuate the pasture curves shown in Fig. 10.2, heightening the peaks and depressing the troughs. Typically, the amounts of pasture available are 6000 kg/ha for Mediterranean pasture in spring, and 7200 kg/ha perennial pasture in the subtropics. Voluntary food intake of high-quality pasture will be 1500 g/day during pregnancy and 2500 g/day during lactation. On the basis of this and the other figures above, we can now address the three scenarios in Fig. 10.2, using the Merino sheep as our paradigm.

4.1 Scenario 1: Lactation coincides with the growing phase of the pasture

This is typical for Merino sheep mating in late summer, under photoperiod control, in Mediterranean regions. Birth coincides with the first appearance of new pasture, so the ewe has to cope with late pregnancy at a time when feed is in short supply and also of very poor quality (digestibility is 50% or less). The animals exacerbate the problem by attempting to select the better-quality parts of plants. This substantially increases their maintenance requirements because they walk much longer distances and spend much more time grazing and ruminating, activities that all require energy expenditure. It is not uncommon for sheep to graze for up to 10 h/day searching for the more digestible herbage. When sheep are grazing high-quality, green material, little selection is involved so the energy cost of searching, harvesting, ingesting and ruminating equates to about 20% of fasting metabolism. Therefore, maintenance requirements equate to fasting heat output plus about 20%. However, when food is scarce and of poor quality (months 10–13 in Fig. 10.2), maintenance requirements can increase by 60% (e.g. from 6.7 to 10.7 MJ NE/day). In addition, this poor-quality feed is converted to NE with only half the efficiency of high-quality green feed. Hence, the ME required for maintenance increases from 8.7 to 13.8 MJ. This translates to a DE requirement of 15.8 and a GE requirement of 31.6 MJ assuming material is 50% digestible. Thus, just to maintain herself, the ewe must consume 1.75 kg of the senescent pasture. She will struggle to consume and process more than about 1 kg/day of such material and would be in serious energy deficit in late pregnancy. This problem is exacerbated by two factors: first, the developing fetus(es) takes up progressively more space in the abdominal cavity, reducing rumen volume; second, poor-quality forage requires longer in the rumen for digestion. This combination of factors severely reduces the energy ewes can absorb. When high-quality green feed becomes available (from month 17 on), the ewe can compensate for low rumen volume by speeding up the rate of passage through the digestive system.

There are other ramifications for ewes spending their pregnancy in dry, harsh periods of the season, many overlooked because we tend to focus on the energy devoted to the products of conception, the feto-placental unit(s) (1.1 MJ NE) and

uterus (1.2 MJ NE). For example, there are energy costs associated with the development of the udder and the formation of colostrum (see below). The fetus itself is metabolically very active with an absolute requirement for glucose. In ruminants, high-fibre diets of low digestibility are ketogenic, producing abundant acetate but low amounts of the gluconeogenic precursor, propionate. This is why ewes grazing poor-quality diets in late pregnancy are prone to pregnancy toxaemia, a major risk if they are carrying more than one fetus.

During lactation, the ewe will be able to minimize the length of time for which she is in negative energy balance and also reduce the depth of her negative energy balance. Maximum milk production is generally reached by the end of the third week of lactation. For example, a Merino ewe weighing 50 kg and suckling a single lamb would excrete a maximum of about 5.7 MJ of NE/day in milk. When, the NE requirement for maintenance is added (6.7 MJ/day), the ewe requires a total of 12.4 MJ of NE daily. Actively growing grass is highly digestible (85%) and supplies all the nutrients required for lactation, so it has high ME concentrations and the ME is efficiently converted into NE. For milk plus maintenance, the ewe requires a total of 18.2 MJ of ME and, assuming a conversion of DE to ME of 0.81 and a digestibility of 0.85, the total GE of forage dry matter that is required is 22.3 MJ. Assuming 18.0 MJ of GE/kg forage, the ewe would need to harvest and process about 1.2 kg of dry matter (about 5 kg of green material) per day to remain in energy balance. This is well within her capacity.

Suckling twin lambs substantially increases the energy required. Twins are born 20% lighter than singles and they grow at about 80% of the rate of singles, but their combined demand would increase milk output to 9.1 MJ of NE daily, equivalent to 15.2 MJ ME from pasture. This translates to 1.9 kg dry matter (7.7 kg of green pasture), an amount that probably exceeds a Merino's voluntary food intake at this stage of lactation. In fact, the situation could be worse because the consumption and processing of this amount of pasture would increase the energy cost of grazing (a component of maintenance). So, a ewe with twins would almost certainly be in negative energy balance by the end of the third week in lactation. Most mammals do not reach their post-natal maximum voluntary food intake until the end of the first third of lactation (week 5 for a ewe). By this time, the ewe should be harvesting at her maximum and, since milk energy output is already declining, she is probably beginning to consume more energy than she requires.

4.2 Scenario 2: Milk production begins when energy availability is at its lowest

This scenario pushes the ewe into the deepest level of energy deficit because food mass and quality are both limiting, and maintenance costs are high, as described above. In addition, poor-quality forage is converted into milk energy with a very low efficiency (30%), so the ewe must eat much more dry matter than in Scenario 1. The NE required for milk is the same (5.7 MJ/day), but the ME required to generate that milk increases to 19.0 MJ. The cost of maintenance is also high, perhaps as much as 13.8 MJ ME. Hence total ME required would be 32.8 MJ. This could translate to a requirement of 40.5 MJ for DE and 81 MJ for GE. The ewe would need to consume about 4.5 kg forage/day which is well beyond her capacity.

About the only advantage of this scenario is that the young animals, if they survive, will be weaned on to good pasture.

4.3 Scenario 3: Late pregnancy and early lactation coincides with maximum energy availability

This timing probably offers the ewe the best chance of giving her offspring the best start in life. Fetal development will not be compromised and there are no limits placed on early lactation. Provided pasture is actively growing, digestibility will be high (80% or above) and its protein concentration will meet all requirements. Voluntary food intake will be such that all energy requirements can be met during pregnancy and during lactation except for early lactation. However, if the ewe is well fed during the later stages of pregnancy, she will have sufficient energy reserves of her own to fuel early lactation without limiting milk output. In turn, the lamb will not be compromised during its fetal development and will be born at a normal weight and have the necessary vigour to stimulate the udder to produce maximum milk. Early growth of the lamb will, therefore, be at a maximum.

However, when pasture senescence begins, the digestibility (i.e. quality) will fall rapidly (months 7–9). Although there is plenty of herbage for the ewe to fuel lactation, the low quality begins to limit the growth of the lamb. For the first 3 weeks of life, the lamb obtains almost all of its energy from milk and there are no problems. At about 3 weeks of age, the lamb begins to pick at green feed and the process of rumen development begins. By 8 weeks of age the lamb is fully functional as a ruminant and, provided its mother is still lactating well, would be obtaining perhaps half its energy from milk and half from pasture. However, despite its fully functional rumen, the lamb's voluntary intake of food other than milk is limited by the digestibility of the material it consumes. Succulent pasture is handled well by the 8-week-old lamb, but senesced pasture with a digestibility of 55% is handled poorly and will begin to limit growth.

5. The Consequences of Not Satisfying the Demands

The nutritional requirements over the course of the reproductive cycle are driven by the need to produce gametes, express sexual behaviour and sustain the embryonic, fetal and post-natal development of the young. Inability to meet the needs from metabolic inputs (intake and reserves) will affect all stages of the reproductive process in both sexes, although the degree of impact will differ. An important point is that undernutrition and overnutrition can have different outcomes on the reproductive system, for both males and females, according to their timing. One marked example is the different impact of dietary treatments for growing and mature animals, a clear demonstration that resource allocation to the reproductive axis depends on the needs for other functions (in this case, growth). In addition, for both sexes, inadequate metabolic supplies can have short-term and lifelong consequences, within the same generation and from one generation to the next. For example, the level of

nutrition during pregnancy can have a dramatic effect on the reproductive capacity of the next, and possibly to all future generations (Ashworth *et al.*, 2008).

We will begin with the short-term outcomes, focusing on the sheep. At the most basic level, the ewe has two 'decisions' to make: whether to ovulate or not (effectively, whether to reproduce or not), and whether to have one or more ovulations and thus bear one or more lambs. Both decisions are potentially life-threatening, for both herself and her offspring, because she must survive the extreme metabolic drains of pregnancy and lactation and, at the same time, equip her offspring with sufficient bodily reserves to survive any challenges thrown up by the environment during development to maturity.

So, how does the ewe make these decisions? The drive to reproduce is extremely powerful and any animal, male or female, has to be in terrible condition before the reproductive centres of the brain decide to completely shut down the reproductive axis. Even fairly thin ewes can raise one lamb. Thus, the 'decision to ovulate' is almost obligatory. Having made that decision, however, the decision to have more than one lamb is much more complicated. The risk is far greater, involving glucose imbalance in late pregnancy, calcium homeostasis during lactation and a massive loss of personal body reserves because of the need to double milk excretion. Generally, this risk is only accepted when the ewe is in top condition at mating. A guarantee of one healthy lamb, carrying her genes into the next generation, is a better option than the risk associated with multiple births.

Considering the different implications of these decisions, it is not surprising that different physiological mechanisms are involved. The decision to ovulate or not (i.e. reproduce or not) is made by the brain centres and is implemented through the GnRH cells; this system is only inhibited by extreme undernutrition. By contrast, the decision to have multiple ovulations has little to do with GnRH cells, and nutritional and metabolic factors play a major role, mostly acting within the ovary. In both cases, quantitative energy balance has little direct influence – instead, the reproductive control systems seem to respond to the regulatory signals that control, or are affected by, energy balance.

5.1 Onset of puberty

Puberty primarily concerns the decision to reproduce. Entry into reproductive life is a decision that commits the pre-pubertal ewe to enormous expenditure of energy and nutrients 5–8 months later. In this context, resource allocation theory would seem to be well supported. This is illustrated by the tension between the drive to begin reproductive life as early as possible and the need to be conservative and wait for the appropriate accumulation of resources so that reproductive effort is better rewarded over a long period. The value of the conservative strategy is evident in the desire of producers to delay puberty in environments where early pregnancy places the animal at risk, as happens in cattle grazing open ranges in the tropics. In this case, heifers that enter puberty early, in response to a flush of feed in the wet season, and conceive before being fully grown will find it difficult to survive the following dry season when the problems of feed shortage are compounded by the drains of pregnancy and lactation (Mackintosh and Pratchett, 1988).

We have previously analysed in detail the relationships between nutrition, age, growth, body mass, mature body size and puberty (Lindsay *et al.*, 1993), so we will not deal with those issues here. The most critical development since has been the advent of leptin, finally providing us with a regulatory process for lipid homeostasis and a potential link between body reserves and the reproductive centres of the brain. Effectively, leptin seems to allow the animal to 'weigh' its adipose mass physiologically and thus allow the brain to decide when to commence reproductive activity. However, the control of puberty is not as simple as the leptin enthusiasts would like to believe. Indeed, as we discover more about leptin and the way that its secretion and action are controlled, we are learning that, as with other nutrition–reproduction relationships, the situation is too complex for it to be dependent on a single hormone.

5.2 Sexual behaviour and sperm production

Studies of the effects of castration on Soay ram lambs have clearly demonstrated that survival rate (or longevity) is mainly due to differences in metabolic status, caused by variation in energy intake and energy expenditure through the expression of sexual behaviour and associated behaviours such as standing and aggressive behaviour (Jewell, 1997). Nevertheless, there is some controversy on the effect of undernutrition on the expression of sexual behaviour of male sheep, mainly due to the methods used to assess libido and the frequency of observations (Parker and Thwaites, 1972). If feed restriction is sufficiently long and severe, such that over 30% of body mass is lost, the expression of normal sexual behaviour is compromised (Parker and Thwaites, 1972). However, sexual behaviour requires fairly intensive motor activity (Banks, 1964) so a decline in libido could be caused by a general weakness in the rams rather than energy restriction (Tilbrook and Cameron, 1990). In fact, during joining, motor activity is greater in rams than in ewes, because of the very active part taken by the rams during courtship (Banks, 1964). Interestingly, it is also greater in rams with smaller testes than in rams with larger testes (Raadsma and Edey, 1985), suggesting that the cost of reproduction is greater in animals that produce less spermatozoa.

It is unclear if the effect of nutrition on the sexual behaviour of rams is due to energy or protein. Rams fed a high-protein diet have a greater sex drive than rams fed a low-protein diet (Salamon, 1964), but an increase in undegraded dietary protein has no effect (Fernandez *et al.*, 2004). Also, overfed males show reduced sexual activity possibly because they are overweight and have difficulty in expressing courtship or mounting (Okolski, 1975). Overall, libido is more sensitive to undernutrition than sperm production, but increases in the plane of nutrition stimulate sperm production before affecting libido.

Testicular size is more sensitive to undernutrition than body mass (Thwaites, 1995a) and is strongly correlated with both the level of nutrition (Martin and Walkden-Brown, 1995) and the level of exercise and, not surprisingly, these two effects are addictive (Thwaites, 1995b). In commercial practice, Dorper rams kept in intensive systems have better reproductive performance than those kept in extensive systems (Fourie *et al.*, 2004). The quality of the spermatozoa produced, measured as sperm count and sperm motility, is also decreased by undernutrition

for a period of time greater than the 7-week duration of spermatogenesis (Parker and Thwaites, 1972; Robinson *et al.*, 2006). It seems that this effect is caused by a lack of feed energy because manipulation of the amount of protein that escapes degradation in the rumen, and thus reaches the small intestine, does not affect semen characteristics or sexual behaviour, despite loss of testicular mass and sperm production when less undegraded dietary protein was available (Fernandez *et al.*, 2004).

5.3 Female sexual behaviour and gamete production

In adult ewes, severe undernutrition and thus extremely poor body condition leads to irregular oestrous cycles or acyclicity, preventing the expression of sexual behaviour (Allen and Lamming, 1961; Cahill *et al.*, 1984). On the other hand, mild decreases in nutritional input are unable to prevent ovarian cycles and behavioural oestrus in ewes, unless this is combined with a very high energy demand for another function, such as lactation (Hunter and van Aarde, 1973). Similarly, female sexual behaviour does not seem to be affected by the level of nutrition, probably because of the strong physiological relationship that links sexual behaviour to ovulation (Blache and Martin, 1995). In small rodents, energy-deficient diets can affect the expression of female sexual behaviour; probably the small amount of reserves carried by these animals makes them susceptible to severe changes in energy balance (Gill and Rissman, 1997). This may apply to sheep under extreme dietary deprivation.

When nutrition is adequate to favour the qualitative decision to reproduce, the animal can address the quantitative decision – the number of ovulations and thus embryos, fetuses and offspring that will be supported. This nutrition–reproduction relationship is dose-responsive, in contrast with the decision to ovulate (reproduce), which is based on a threshold.

Ovulation rate, defined as the number of ovulations per ovulating female (thus ignoring anovulatory females), is related to nutrition in several ways, reflected in the variety of variables that are significantly correlated with it (review: Scaramuzzi *et al.*, 2006). First, we have the long-term response to energy balance, known as the 'static effect' of nutrition, based on the relationship between ovulation rate and live weight, with the live weight being constant. In addition to this, short-term increases in dietary intake can also stimulate the number of ovulations. In the second type of relationship, the 'dynamic effect', ovulation rate is increased while body weight is rising. When the response is very rapid, well before any detectable change in live weight, it falls into the realm of nutritional pharmacology (see below) and is known as the 'acute effect'. There is still considerable debate as to whether these three classes of response are really different or are simply variations in expression of the same processes (Scaramuzzi *et al.*, 2006). The 'static' and 'acute' effects of nutrition alter ovarian folliculogenesis (Viñoles *et al.*, 2002, 2005), apparently via direct actions on the ovary by leptin, IGF-I, insulin and glucose, factors that increase the responsiveness of follicular tissues to follicle-stimulating hormone (FSH) (review: Scaramuzzi *et al.*, 2006). Importantly, all of these factors are involved in energy homeostasis.

5.4 Trans-generational effects of the metabolic status of the mother

The energy requirement for the ovarian cycle and the first two thirds of pregnancy is small in the context of total daily energy expenditure for reproduction (Blache *et al.*, 2007), but the metabolic status of the mother at the start of pregnancy appears to have profound effects on the processes that lead to the provision of a suitable uterine environment for embryonic and feto-placental development. In other words, with respect to the early conceptus, we are dealing with regulatory processes rather than quantitative energetics. Undernutrition, and perhaps overnutrition, has several consequences over a range of time frames, beginning with early oocyte development, moving to implantation (around day 14 in small ruminants), and finally to organogenesis. Some of the effects are relatively subtle, such as the 'programming' of future performance that is often difficult to detect and link back to the conditions of pregnancy. Other effects are dire, effectively ending in the death of the conceptus.

Early conceptus: This area is quite speculative, but recent studies of the metabolism of cumulus–oocyte complexes and early embryos suggest that we need to pay more attention to the effects of maternal nutrition in the period covering the last few days before ovulation and the first few days after conception (review: Thompson, 2006). The ovum and the conceptus both rely on a histotrophic source of nutrition for energy production and the synthesis of macromolecules. It seems that the balance of supply and demand for energy and other anabolic substrates during oocyte maturation and very early stages of embryo development can programme subsequent developmental potential, and this may include the fetal growth trajectory. For example, glucose is an essential nutrient for oocyte maturation, so too little glucose can reduce meiotic competence. Glucose appears to act through the hexosamine biosynthesis pathway, a 'fuel-sensing' pathway that seems to interact with systems for cell signalling and transcriptional regulation. Another example is the role of hypoxia during embryo development – ruminant embryos lack a key hypoxia responsive element during compaction, blastulation and subsequent development in the uterine cavity so, in contrast to rodents, hypoxia is important for normal development in sheep and goats. These issues might explain some of the effects of undernutrition during the 'Bloomfield period' (see below). Most of the studies cited by Thompson were done with oocytes and embryos *in vitro*, so need to be extended to the whole-animal situation.

Peri-implantational effects: A potential point of impact for nutritional input is the process of maternal recognition of pregnancy, around 14 days after conception in sheep, during which there is a precisely synchronized chemical dialogue between the embryo and the mother that determines the fate of the embryo (Spencer *et al.*, 2004). Embryo loss during the first few weeks after conception varies from 7% to 46% in sheep (Restall *et al.*, 1976), and both underfeeding and overfeeding are listed among the causative factors (Abecia *et al.*, 2006). Certainly, severe undernutrition is dangerous (e.g. Blockey *et al.*, 1974; Rhind *et al.*, 1989), but even relatively mild undernutrition delays the development of the embryo, mainly because it leads to a combination of hormonal imbalance and reduced sensitivity of the endometrium to progesterone, compromising the embryo-mother signalling system that is necessary for the establishment and maintenance of pregnancy (Abecia *et al.*, 2006).

Paradoxically, there is also evidence that overfeeding in the first few weeks after conception can cause problems. This is apparently due to an increase in the

clearance of progesterone (Parr, 1992; Parr *et al.*, 1993). In these studies, extreme dietary regimes were compared (e.g. 25% versus 200% of maintenance requirements) and we are still not certain how important this issue is under a more normal range of circumstances.

Post-implantational effects: High-quality ultrasound has allowed major advances in describing the problem, and we now know that, in sheep, 3–4% of conceptuses are lost every 20 days from day 25 postconception (Dixon *et al.*, 2007). As with early embryo mortality, maternal progesterone concentrations are a major factor (Dixon *et al.*, 2007). Nutritional constraint may contribute to these losses but we have too little robust information for quantification.

Feto-placental function: Maternal nutrition affects placental growth and this can lead to low birth weight with the attendant post-natal implications. This has been reviewed in detail by Lindsay *et al.* (1993), so we will not deal with it here. Since Lindsay's review, a new dimension to feto-placental development in sheep has been added by Bloomfield *et al.* (2003b). They found that undernutrition from 60 days before until 30 days after conception, during which there was only a moderate and temporary 15% loss of maternal body mass, increases the number of premature births, with some lambs born up to 20 days before term. This 'Bloomfield Effect', perhaps one of the most dramatic illustrations of the concept of 'metabolic memory', is caused by accelerated maturation of the fetal hypothalamic–pituitary–adrenal axis, effectively advancing the fetal trigger that initiates parturition (Bloomfield *et al.*, 2004).

Fetal programming: Inadequate nutrition during pregnancy has detrimental effects on both male and female offspring, a phenomenon called fetal programming (Symonds *et al.*, 2007). Fetal programming has been observed in the progeny of overfed young females and underfed mature females, with the offspring experiencing growth retardation in both cases (Wallace *et al.*, 2005). Undernutrition of the mother decreases the number of Sertoli cells in the testes of male lambs at birth (Bielli *et al.*, 2002). Sertoli cell numbers determine the capacity for sperm production in later life, but the long-term consequences of maternal dietary restriction during gestation on future fertility have not been studied.

Gonadal activity in the female lamb is also affected by the nutrition of her mother during gestation (Rhind *et al.*, 2001; Rhind, 2004). Restricting the level of intake to 70% of maintenance requirements, from 71 to 110 days, or from 101 to 126 days of gestation, reduces the developmental competency of oocytes harvested from 9-week-old lambs, compared to oocytes from lambs born to ewes fed 150% maintenance (Kelly *et al.*, 2005). Moreover, feeding only 50% of the maintenance requirement during the first 95 days of gestation reduces ovulation rate in the progeny at 20 months of age by 20% (Rae *et al.*, 2002). A long-term study suggests that these effects are maintained over life, because the percentage of multiple births was higher in ewes born to mothers that were supplemented during gestation than to mothers that were not supplemented (Gunn *et al.*, 1995).

There are only a few examples of these trans-generational effects of nutrition on the reproductive capacity of sheep because this field of research is fairly new and the experiments are protracted. For other aspects of the future productivity of sheep, evidence is accumulating. Factors related to energy homeostasis, such as glucose tolerance and the function of the somatotrophic and adrenal endocrine

axes, are associated with birth weight or with maternal nutrition during gestation (Bloomfield *et al.*, 2003a). These axes have connections with reproductive function, so this is clearly an area worthy of investigation. Moreover, the molecular mechanisms, such as DNA methylation, are now being pursued (Wu *et al.*, 2006).

5.5 Survival of the young

Undernutrition or overnutrition of ewes during gestation leads to low body weight and an associated decrease in the rate of survival of the lambs, because survival rate is related to birth weight, although the relationship is parabolic because both high and low birth weights are detrimental. Adequate birth weight gives the lamb the vigour it needs to stimulate and establish the vital bond with its mother (Nowak and Poindron, 2006). Maternal vigour is also important because undernourished ewes are less active after parturition, especially if the parturition is lengthy, and weakened ewes do not nurture their lambs quickly and strongly enough, decreasing the chances of survival of the newborn (Nowak *et al.*, 2008).

The critical nature of the contribution of maternal nutrition to a successful bond is illustrated by the impact of dietary intake during the final week of gestation on the production of colostrum and milk. Banchero *et al.* (2002) calculated the energy needs of newborn lambs and found that many of them did not receive sufficient energy for their needs from the colostrum. In particular, this problem was most pronounced in ewes bearing twins. An increase of intake to 130% of maintenance doubles the quantity of colostrum and improves its viscosity during the critical first 12 h after parturition (Banchero *et al.*, 2004). On the other hand, reducing nutrient availability by 50% during the last 3 weeks of gestation has a negative effect on milk yield for up to 3 weeks after parturition, even when intake is unrestricted during lactation (Tygesen *et al.*, 2008).

Interestingly, resource allocation theory is strongly supported by a major aspect of post-natal reproductive performance: post-partum anoestrus. In general, reproductive capacity is reduced by attempts to accelerate lamb production by shortening the interval between lambings (Cognié *et al.*, 1975; Hulet, 1978; Hogue, 1987). At a superficial level, it could be argued that the Holstein dairy cow has been successfully selected to have a very short post-partum anovulatory period. However, in reality, the outcome has been very poor fertility probably because the modern Holstein cow has an aberration in her somatotrophic axis, disrupting energy allocation (Chagas *et al.*, 2007). Thus, post-partum anoestrus and puberty, two major aspects of lifetime reproductive output, lend weight to resource allocation theory and, interestingly, neither of them are particularly susceptible to manipulation by nutritional pharmacology.

6. Windows of Opportunity: 'Focus Feeding' and 'Nutritional Pharmacology'

For all animal enterprises, feed is the primary limiting resource. For intensive industries, the benefit–cost relationships should be transparent, but the same

principles apply to grazing animals – and there is constant pressure to ensure that greatest benefit is gained from an annual cycle of forage production and to reduce the need for supplement. Careful planning is required to relate the amount, composition, duration and timing of nutritional inputs into the reproductive process. Obviously, this will vary between animal enterprises and between environments. Moreover, the list of potential options is lengthening as we gain a greater understanding of the nutritional and metabolic interactions with the reproductive control systems, as described above. For any or all of these options, we could use conserved or manufactured feed or, in extensive management systems, we could shift the entire reproductive process so that the critical periods are aligned with peaks and troughs in the availability of pasture and forage, as we have demonstrated with respect to the relationship between the pasture cycle and milk production. Effectively, this is the concept of *focus feeding* – cost-effective nutritional inputs into critical points in the reproductive process where the management of metabolic factors will improve productivity (Martin *et al.*, 2004).

An important issue in considering this situation is the somewhat extreme nature of some of the supplements that we are proposing. For example, consider the effect of feeding a ewe with 500 g high-protein supplement daily for only the final 3–6 days of the late luteal phase (e.g. Williams *et al.*, 2001). This short, heavy supplement will double her fecundity, stimulating her ovaries to produce two ovulations instead of one, with all the attendant risks that are incurred 5 months later. It is difficult to imagine that responding in this way to a sudden supply of rich feed, discovered daily for the only few days that coincide with one specific part of the reproductive cycle, is a normal biological response developed during evolution. It seems unlikely that a 3- to 6-day supplement will have the long-lasting effects on body reserves that are necessary to support a double pregnancy and a milk supply for twins. It therefore seems to us that this type of acute, rich supplement is somehow 'deceiving' the reproductive control systems so that they respond in the same way as they would to a sustained period of good nutrition leading to heavy body reserves. This is akin to the unexpected responses that can be induced by very high doses, often known as 'pharmacological doses', of exogenous hormones, and has led us to coin the term *nutritional pharmacology*. For focus feeding to reach its full potential, we may need to develop a range of options for nutritional pharmacology in which metabolic sensors are simply swamped with high concentrations of substrates, metabolites and hormones. The animal will be induced to follow a high-risk path in its reproductive strategy, thereafter relying unwittingly on human managers to cope with the consequences.

With ruminants, in particular, nutritional pharmacology has a high risk of acidosis if high-energy supplements are used. Thus, some aspects of focus feeding may require careful selection or development of supplements with a low content of soluble carbohydrate.

7. Critical Control Points: Targets for Focus Feeding

The success of focus feeding depends on the timing of the dietary stimulus (Fig. 10.3), on the quality and quantity of the supplement, and on factors that are more

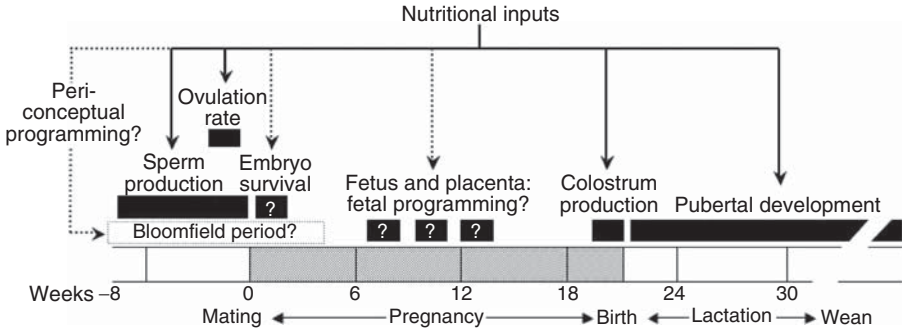

Fig. 10.3. Summary of targets for focus feeding that are known to improve reproductive output for sheep. Many of these also apply to goats. Approximate timing and duration are indicated. The broken arrows indicate inputs for which practical benefits are still under investigation. Quantity and quality of dietary treatments will vary with each point of the reproductive process, as well as among species, genotypes and environments. In the 'Bloomfield period', mild undernutrition leads to premature births. (Redrawn after Martin *et al.*, 2004.)

difficult to control, such as the metabolic history of the animals (particularly 'metabolic memory'). Focus feeding is already being used to boost sperm production, increase ovulation rate ('flushing') and improve offspring survival, so we can consider these applications to be already incorporated into industry practice.

7.1 Sperm production and sexual behaviour

Feeding supplements to rams and bucks for 8 weeks before mating ensures that their testicular size and sperm production are maximal (review: Martin and Walkden-Brown, 1995). These responses are reliable in genotypes that are not dominated by photoperiod, but also can be used in more photoperiod-sensitive breeds, such as the Corriedale and Suffolk (Pérez-Clariget *et al.*, 1998; Blache *et al.*, 2002; Hötzel *et al.*, 2003).

7.2 Ovulation rate

With respect to focus feeding and nutritional pharmacology, we are not considering the 'static effect' of live weight or even the 'dynamic effect' of a change in live weight. The phenomenon of interest is the 'acute effect' in which very short and well-timed periods of high nutrition (during the final 3–4 days of the luteal phase) increase twin ovulations by 20–30% (Oldham and Lindsay, 1984; Scaramuzzi *et al.*, 2006).

In Western Australia, we typically feed a Merino ewe with 500 g lupin grain daily for the final 3–6 days of the late luteal phase (e.g. Williams *et al.*, 2001). If the ewe has a condition score of 2.5 (on a scale of 5), this short, heavy supplement is likely to induce twin ovulations. This is only feasible because lupin grain is a highly

digestible source of protein with very little soluble carbohydrate, so acidosis is avoided without any need for a period of adaptation as would be required with, for example, wheat grain. For the same reason, lupin grain has been successful as an experimental treatment for studying metabolic pathways in male sheep (review: Blache *et al.*, 2002). However, we need to find alternatives to lupin grain because, first, it is not readily available in many parts of the world and, second, it is not always an effective nutritional pharmacological agent (e.g. for promoting colostrum production (see below)).

On the other hand, the nutritional pharmacological effect on ovulation rate can be achieved equally with relatively brief periods of feeding with high-quality pasture and forage (Viñoles *et al.*, 2008), and also with oral doses of a glucogenic substrate (Rodríguez Iglesias *et al.*, 1996; Williams *et al.*, 2001). The response to glucose suggests that the sudden influx of nutrients from supplements acts, at least in part, through pathways involving energy substrates, even when there is little soluble carbohydrate in the supplement.

7.3 Embryo mortality

Nutritional effects on the embryo are obviously critical because nutritional pharmacology treatments used to increase ovulation rate may need to be stopped precisely relative to the time of ovulation so that they do not intrude into the period of early oocyte or embryo development.

7.4 Colostrum production and offspring survival

Increases in litter size are economically and ethically justified only if all the offspring survive until sale or reproductive maturity. It is therefore essential for the lamb to have access to adequate amounts of high-quality colostrum during the first hours after birth. The colostrum provides energy and immunoglobulins and, in addition, will facilitate the establishment of the ewe–lamb bond (Dwyer *et al.*, 2003; Nowak and Poindron, 2006). This is particularly important for ewes bearing twins (Banchero *et al.*, 2002). Problems can be avoided by feeding 30% above maintenance requirements for the final 10 days before parturition (review: Banchero *et al.*, 2006). Generally, the supplement guarantees an adequate supply of colostrum so that this factor does not contribute to lamb mortality. The same applies to goats (Goodwin and Norton, 2004).

The nature of the nutritional pharmacology for colostrum is crucial and it differs from treatments that can be used to improve ovulation rate: starchy feedstuffs such as maize and barley are most effective, whereas lupin grain is less so (Banchero *et al.*, 2004a,b). This is because a major component of the effect seems to be provisioning of extra starch for digestion in the small intestine, providing a ready source of glucose for the synthesis of lactose at a time when the capacity of the pre-parturient ewe to ingest roughages is limited by the space in her rumen (Banchero *et al.*, 2006). Space is, of course, even more limiting in twin-bearing ewes. The supplement thus increases the colostrum concentration of lactose and

it also hastens the clearance of progesterone (Banchero *et al.*, 2006), two factors that accelerate the onset of lactogenesis (Hartmann *et al.*, 1973).

In addition to consideration of the quality of the supplement, there is a clear need for precise timing because, at the end of pregnancy, overnutrition can increase birth mass and cause distocia. Feeding strategies, therefore, have to be fine-tuned for single- and twin-bearing females, and for different genotypes. The necessary degree of precision is only possible if the number of fetuses is known (ultrasonography) and if there is a good synchronization of conceptions in the flock or herd.

7.5 The next steps for focus feeding

Focus feeding for sperm output, ovulation rate and colostrum production can be incorporated into industry practice immediately. However, for the Bloomfield period, embryo survival and fetal programming, the picture is not yet sufficiently clear. At this stage, it seems quite arbitrary to separate these three issues because, in reality, they are all part of the continuum of development from the oocyte to zygote to embryo to fetus-placenta. Some of the effects of nutrition may be directly on the developing tissues, whereas other might involve 'programming', perhaps through epigenetic processes.

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11 Allocation of Resources to Immune Responses

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In evolutionary biology, there has been considerable interest in recent years in the role of disease resistance as a life history trait (Owens and Wilson, 1999; Zuk and Stoehr, 2002). Life history theory investigates the extent to which the use of resources to resist disease is traded off against other components of fitness such as growth and reproduction (Schmid-Hempel, 2003). Consequently, much research has focused on attempting to quantify these costs both in wild populations and in domestic animals. To approach this question, we need first to consider the nature of the immune system, how it is regulated and how the host responds to challenge by infectious organisms. The review then considers the interplay between immune challenges and production traits in farm animals for evidence of trade-offs.

1. The Immune System as an Arm of the Host Sensory System

The word 'immune' is derived from the Latin 'immunis', which means exempt from duties or taxes. Attempts to define the immune system usually employ descriptions of its operation and outcomes. In these terms the immune system is comprised of those cells and proteins, and the tissues that support them, which react with molecular structures that pose a threat to the integrity of the host. From the commonly assumed goal of protection against molecular threats arises the description of immunity as a defence system. Even so central a concept of immunity as this has been questioned recently with the proposition that the primary driver of adaptive immunity in vertebrates may have been the beneficial consequences of commensal microorganisms nurtured by the immune system rather than the limitation of disease caused by pathogens (McFall-Ngai, 2007). It is not surprising, therefore, that there is ambiguity over what constitutes immune defence (Greenspan, 2007) and what are appropriate measures of immune activation (Zinkernagel, 2007).

Kelley *et al.* (2005) have estimated that in humans there are in excess of 1560 genes representing 7% of the genome that contribute to immune defence. These include

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genes involved in barrier functions of epithelia, innate and adaptive immune responses and in ontological development of the immune system. A growing number of germ line encoded receptor families, including toll-like receptors (TRLs), triggering receptors expressed on myeloid cells (TREM), sigelc molecules, C-type lectin receptors, Nod-like receptors and other molecules recognize the presence of pathogens (Sansonetti, 2006). When stimulated, these receptors provide signals for elevated expression of immune defence genes and mobilization of leucocytes to sites of tissue defence as part of the innate immune system. In addition to these proteins and receptors for recognizing foreign molecules, somatic mutation and selection of receptors for antigen on B and T cells can lead to 'learned' adaptive or acquired immunity.

When considered as a defence organ, the immune system is the arm of the host's sensory capabilities that has specialized in sensing noxious threats from foreign molecular structures that are typically associated with pathogenic organisms. Two important consequences flow from this view:

1. Like other sensory systems, the immune system requires afferent inputs for normal development and maintenance. This aspect is important when considering the maintenance costs of the immune system.
2. Like other sensory systems, the immune system is integrated into the coordinated host defence reaction to excessive environmental stimulation that manifests as stress. How does the host regulate its physiological responses to environmental and endogenous stimuli?

2. Models of Physiological Regulation

Davies (2003) noted that in information theory, information has meaning in a context or environment. Genetic information has meaning through the ability of transcribed RNAs and their translation products to utilize resources from the environment for the fulfilment of life history traits of the host. One hundred and fifty years ago, Claude Bernard recognized the importance of a constant internal environment to the maintenance of life. Cannon (1929) introduced the term homeostasis to describe the regulation of the internal environment of the animal by feedback mechanisms seeking a set point for physiological variables, and emphasized the importance of the autonomic nervous system in this process. Subsequently, Hans Selye (1936) described the general adaptation syndrome as the common response pattern of animals to diverse noxious stimuli including tissue trauma, exposure to environmental extremes, excessive muscular exercise and intoxications. Selye described several phases in the general adaptation syndrome that occur during sustained exposure to extremely noxious stimuli. An initial alarm reaction is followed by a stage of resistance, which has a degree of specificity for the eliciting agent, which in turn is followed by a stage of exhaustion (Selye, 1946). During the stage of resistance there is adaptation to the specific agent eliciting the stress response 'at the expense of resistance to other agents'.

The immensely detailed knowledge of specific pathways involved in response to particular stimuli that has been gained subsequent to Selye's work has tended to erode awareness of commonalities of the generalized adaptation syndrome. For instance, in common with the early phases of innate immunity, tissue trauma and

some psychological traumas can lead to induction of the acute phase response in which there is production of pro-inflammatory cytokines, decreased utilization of glucose by many non-immune tissues, resorption of amino acids from skeletal muscle and increased anabolic activity in the liver (Klasing, 1988). Thus, while the acute phase response can be one important cost of immune defence, it is not a cost that is unique to immune defence.

Studies with extremely noxious stimuli underlay research on the general adaptation syndrome. With the use of milder stressors it was apparent that repeated exposure to the stressor could often lead to more enduring adaptation that is typically specific to the stressor and that often employs behavioural strategies to avoid the physiological responses of the general adaptation syndrome. Moberg (2000) was a strong proponent of this type of model of the response to stressors. In economic terms, the animal is using energetically inexpensive, learnt neurological pathways for processing input stimuli to entraining behaviours that reduce exposure to or impact of the stressor, rather than initiating expensive systemic physiological responses to control the (potential) damage the stressor would otherwise invoke through activation of the general adaptation syndrome. A strong analogy exists here with innate and adaptive immunity. On exposure to a novel foreign molecular structure, general innate immune defence pathways are activated. During this non-specific general response, lymphocytes are also trained to recognize the structure of specific foreign molecules and on repeated exposure, a specific adaptive anamnestic immune response that remembers the foreign molecules is initiated and at least some of the costs of the more general innate immune response are bypassed.

Adaptation through learned responses as a strategy for reducing the threat from stressors has been extended to be the central concept of regulation of physiological processes by allostasis (Sterling, 2004). This theory developed by Sterling and Eyer (1988) holds that learned adaptation in the central nervous system leads to predictive regulation of physiological process. Variables are not regulated by feedback to maintain constant set points, but they are dynamically regulated through neural mechanisms to provide 'coordinated variation that optimizes performance at least costs'. Thus, physiological variables such as blood glucose concentration or blood pressure are not driven towards a constant set point by the homeostatic mechanisms proposed by Cannon but are modulated dynamically over short (minutes) and long (days to weeks) timescales to meet anticipated loads by central control mechanisms.

Allostasis has emerged as a powerful model of physiological processes that can account for diseases of physiological dysregulation such as type II diabetes and intrinsic hypertension in the absence of molecular lesions. There appears to have been little overt application of allostasis theory to models of regulation of immune function. None the less, immune responses are strongly influenced by neuropeptides, neurotransmitters and the neuroendocrine system, and immune responses can be influenced through learnt cognitive processes. For instance, both humans and animals can undergo behavioural (Pavlovian) conditioning to exhibit either immunosuppression or immunostimulation on re-exposure to the conditioned stimulus (1993; Husband, 1995). Thus, the immune system shares at least some attributes with allostasis in other physiological systems.

Moberg's model of responses to stressors emphasizes the multiplicative way in which stressors can impact on the animal, and that the capacity of the animal to

cope with additional stressors diminishes as the load of stressors increases. To describe this phase of limited capacity to cope with additional stressors, Moberg coined the term pre-pathological state, which under sustained exposure to a stressor can lead to pathology (Moberg, 2000). Like other modalities of the stress response, self-inflicted pathology is an important feature of excessive immune stimulation.

Finally, in this consideration of models of regulation of physiological processes is the concept of prioritization of nutrient use by tissues in growing and reproducing animals experiencing parasite infections (Bauman and Currie, 1980; Coop and Kyriazakis, 1999). This model provides an empirical ranking of the priorities for nutrient use during the acquisition phase and the expression phase of immunity in parasitized sheep of different growth status and reproductive status. An important issue that the nutrient partitioning model must accommodate is the need for all metazoan cells to receive external signals for them to modify their uptake of nutrients from extracellular fluid. Lymphocytes, muscle cells, reproductive tissues and so on lack the autonomy to control their own rate of accessing nutrients (Fox *et al.*, 2005). Rather, growth signals such as interleukins, somatotrophins and insulin regulate nutrient uptake by these cells and a mechanism of external regulation of target tissues, which was termed homeorhesis by Bauman and Currie (1980; Chapter 5), is required for any model of nutrient prioritization.

From this survey we can see that there is a unified defence strategy to protect the integrity of the host from perturbation that employs behavioural, physiological and immunological responses. Each of these defence and control elements has innate responses that provide defence of low specificity and learned responses that exhibit higher specificity for particular eliciting agents. This schema can be formalized as a hypothesis that generalized responses to noxious stimuli are likely to be more costly than specialized responses. If specialized responses are less costly, experimental approaches to measuring the cost of adaptive responses need therefore to consider whether the stressor under examination is a primary or a repeat stimulus. The benefit to the individual of reduced future cost on re-exposure also needs to be considered. As some adaptive behaviours are likely to be socially transmitted, reduced future costs can occur for conspecifics in an analogous manner to the reduced future cost to flockmates from reduced exposure to pathogens that accrue through the epidemiological effects of immunity in the individual. This has been demonstrated, for instance, for resistance to gastrointestinal nematodes in sheep. Sheep with acquired resistance to gastrointestinal nematodes pass fewer eggs, and through reduced pasture contamination with infective larvae expose their flockmates to lower levels of larval challenge (Bisset *et al.*, 1997; Eady *et al.*, 2003).

There are several implications of these models of regulation of physiological responses for understanding the costs of immune activation:

1. There is bidirectional integration of the immune system into the systemic physiology of the host.
2. The immune system interacts with other body systems in the nutrient economy of the host.
3. Response patterns of the immune system to stimulation are influenced by age, prior antigen exposure, duration of antigen exposure, phenotypic quality of the host, nutritional status, day length and concomitant infection status.

4. Immune responses are context-specific. That is, the strength and type of immune response are influenced by the nature of the pathogen and by other environmental conditions as well by the intrinsic host factors noted above.
5. Inappropriate or dysregulated activation of the immune system can incur costs through damage to host tissues.

It follows that few general estimates of the cost of immune activation can be calculated. Many measures of the costs of immune activation have been made in specific examples and some of these will now be considered. From the perspective of resource allocation theory there are two fundamental questions about the costs of immunity:

1. Are there costs to the individual of maintaining and using an immune system? These can be considered as phenotypic or physiological costs.
2. Is there an evolutionary (genetic) cost? Can natural or artificial selection for a trait limit the capacity of offspring to resist disease, or conversely, when selection pressure is on disease resistance, can there be negative genetic covariance with other fitness traits? A number of reports in the literature indicate the existence of evolutionary costs in laboratory models of selection for disease resistance (Schmid-Hempel, 2003). Examples in farm animals are considered below.

3. Physiological Costs of Immune Activation

A functional immune system is necessary for life in a field environment that exposes animals to commensal and pathogenic organisms. In such an environment there is continual stimulation of the immune system. While a class of antibodies, termed natural antibodies, can be produced by the B-1 subset of B lymphocytes in mice raised in a germ-free environment on an antigen-free diet, the repertoire of antibodies in an animal is enlarged by exposure to commensal bacteria that populate the normal gut (Cebra, 1999). Natural antibodies are low-affinity antibodies of broad antigen specificity and provide a level of a priori antibody-dependent immunity to the host (Baumgarth *et al.*, 2005). Natural antibodies occur in other species such as chickens (Matson *et al.*, 2005). Commensal bacteria and pathogens expose the host to antigens. The specificity of antibodies generated against these targets, sometimes called immune antibodies, is not absolute and thus the host gains some potential cross-protection against pathogens it has not been exposed to by the generation of antibodies against commensal microbiota.

Comparisons between animals raised in germ-free or conventional environments have been used to estimate the cost of maintaining the immune system. At one level this is a meaningless estimate, perhaps analogous to estimating the amount of metabolic energy used by the central nervous system that could be saved by the induction of general anaesthesia. Lochmiller and Deerenberg (2000) tabulate results from a number of experimental studies where the metabolizable energy requirement of conventionally reared animals was up to 44% greater than germ-free animals. Not all of these differences are necessarily attributable to activity of the immune system in conventional animals as gut flora may modify digestion and absorption of nutrients. In other studies cited by these authors lesser differences were recorded.

While it is not practical to turn off the immune system in farm animals by providing them with a microbiota and antigen-free environment, the antigenic load can be reduced by management practices. Quarantine practices to reduce exposure to specific pathogens reduces the risk of disease, while hygiene practices, housing and use of probiotics and antibiotics in feed modify gut microflora and reduce blood concentrations of pro-inflammatory cytokines in chickens, pigs and laboratory animals in the absence of clinical disease (Klasing, 1988; Roura *et al.*, 1992; Fossum, 1998; Lipperheide *et al.*, 2000). Bacterial products such as endotoxins induce pro-inflammatory cytokines, which modify the nutrient economy in the host by reducing protein accretion in muscle, skin and milk, increase protein resorption from muscle and skin and increase anabolic activity in the liver and gut tissue (Klasing, 1988). At higher concentrations these cytokines induce fever and are linked to pathways that induce anorexia and sickness behaviour characterized by reduced exploratory and foraging activity, and increased rest and sleep. Metabolic rate is increased by between 5% and 13% per degree Celsius of fever (Baracos *et al.*, 1987). Together, these changes induced by pro-inflammatory cytokines are part of the acute phase response, which, despite its name, can occur over chronic timescales when it has also been described as chronic immunological stress (Klasing and Barnes, 1988). More detailed accounts of the biochemical pathways modified during activation of the acute phase response are available (Klasing, 1988; Ebersole and Cappelli, 2000; Colditz, 2003).

Kyriazakis *et al.* (1998) have proposed that anorexia during infection aids survival of the host by promoting an effective immune response and by increased diet selectivity. During anorexia and the acute phase response there is a controlled reduction in serum iron and zinc concentrations. These changes reduce bacterial growth and reduce the risk of septicaemia. Intra-abomasal infusion of glucose increases egg production and worm burdens in lambs infected with the intestinal nematode *Trichostrongylus colubriformis* (Bown *et al.*, 1991). In addition, mice infected with the bacterial pathogen *Listeria monocytogenes* have higher mortality when force-fed to normal energy intake (Murray and Murray, 1979). These observations on pathogen survival provided Karyazakis *et al.* with compelling evidence of the host benefits of anorexia.

However, during the acute phase response there is increased gluconeogenesis and hyperglycaemia, but reduced glucose utilization by many tissues, and increased reliance on glutamine as a cellular energy source, particularly by leukocytes and gut tissues. Hyperglycaemia during the acute phase response can increase infection rates in human trauma and surgery patients (Burton *et al.*, 2007). Unlike most somatic cells, leukocytes lack glutamine synthetase and have a high requirement for this amino acid as an energy source and substrate for purine and pyrimidine synthesis (Pond and Newsholme, 1999). The priority given to glutamine production during the acute phase response and the regulatory role of pro-inflammatory cytokines in directing this change have led to the concept of a neuroendocrine-immune gradient that controls substrate availability and utilization (Elsasser, 1993; Husband, 1995). Environments with a high load of environmental and immunological stressors divert nutrients away from accretion in muscle, skin and milk towards production of acute phase reactants by the liver and towards host defence.

The **first cost** of immune activation then is the cost of elevated metabolic rates during fever and in activated leukocytes. Oxygen consumption, glucose

utilization and glutamine utilization increase twofold to threefold during lymphocyte activation (Cheung and Morris, 1984; Pond and Newsholme, 1999). The **second cost** of immune activation results from reduced nutrient availability through anorexia and the **third cost** arises from altered priorities for nutrient utilization that reduce the capacity for non-immune tissues to use nutrients for maintenance or growth. These costs are typically but not exclusively associated with innate immune responses.

There is ample evidence to support the concept of control of nutrient utilization by a gradient of neuroendocrine-immune regulatory factors; however, quantitative estimates of the cost of a level of infection or degree of antigenic load have proved to be highly variable. Such quantitative predictions are sought by animal scientists for more economical management of farm animals and by evolutionary biologists to resolve questions in life history theory. While data are available on the metabolic cost of fever, lymphocyte proliferation kinetics and glucose, and glutamine and energy requirements of leukocytes on a per cell basis during immune responses, prediction of the magnitude and duration of immune activation and the associated costs that will accompany vaccination or an experimental or field infection has proven difficult. Two general strategies have been employed to address these phenotypic costs (Zuk and Stoehr, 2002):

1. Manipulate host biology and measure the impact on immune competence.
2. Challenge the host immunologically and measure the impact on other traits.
Two important issues accompany such studies: the measurement of immune competence and the nature of the immunological challenge.

3.1 Measurement of immune responses

As noted above there are many modalities through which the immune system responds to foreign molecules, and thus operationally the immune system is comprised of many traits rather than representing a single unified entity. Furthermore, homeostatic or perhaps allostatic mechanisms regulate the immune system, and measures of specific components of an immune response, such as specific antibody or antigen reactive T cells, may not be indicative of the impact of immune stimulation on the total pool size and turnover kinetics of immune tissues. While there may be an increase in antigen-specific antibody and antigen reactive cells in response to vaccination, there is usually neither hypergammaglobulinaemia nor lymphocytosis in response to vaccination.

Antibodies are produced by plasma cells, the majority of which reside in bone marrow as long-lived cells continually producing antibody. During a new immune response, newly formed plasma cells, specific to antigens eliciting the response, migrate to bone marrow and in a random process displace a proportion of the resident population which, once outside the protective niche of bone marrow, die (Radbruch *et al.*, 2006). Thus the repertoire of circulating antibodies is constantly updated to reflect the recent history of antigen exposure of the host without expanding the pool of antibody producing cells or circulating immunoglobulin. This model applies predominantly to immunoglobulin (Ig) G. Within the circulation, IgG is protected from degradation by the neonatal receptor for the crystalizable fragment

portion of the immunoglobulin molecule (FcRn) on endothelial cells and some leukocytes to extend its half-life far past that of most circulating proteins (Qiao *et al.*, 2006). Thus, in the absence of an excessive immunological stimulus, there is an intrinsic regulation of the size of the IgG pool and its turnover kinetics. Regulation of size of the IgA plasma cell pool, which is important in mucosal defence, has not been elucidated.

While the alarm reaction to diverse stressors can have dramatic impacts on the numbers of leukocytes that can be assayed in peripheral blood due to redistribution of cells into the circulating pool, expansion of leukocyte numbers through increased proliferation in primary and secondary lymphoid organs is typical of unresolved infections rather than primary immune responses (Cole *et al.*, 1997). The argument here is that there may be a relatively constant rate of activation of cells by environmental organisms contributing to many ongoing primary and anamnestic immune responses and that most new primary responses do not alter the sum of these events. Responses to pathogens may be an exception to this pattern.

It follows then that the **fourth cost** of immune activation arises from the change in size and rate of turnover of cell and protein pools of the immune system. Therefore, to measure this cost we need to be able to estimate the pool size and turnover kinetics for the relevant immune defence constituents. The major pools to measure might be the major acute phase proteins, as markers of activation of innate immunity, and total immunoglobulins and the major leukocyte classes as markers of adaptive (acquired) immune responses. In this context, specific antibodies and antigen reactive cells may be a distraction.

3.2 Immunological challenges to invoke immune responses

Sterile non-replicating antigens such as sheep red blood cells or bacterial endotoxins are often used in studies to measure the impact of immune activation on life history and production traits (Zuk and Stoehr, 2002). These stimuli avoid the risk of pathology induced by pathogens, but suffer from the difficulty of comparative quantification across species because of potential differences in recognition mechanisms and response pathways. Furthermore, bacterial endotoxin is a very common contaminant of laboratory grade reagents including sterile phosphate-buffered saline and is likely to have been present in some antigen preparations without knowledge of the authors unless its absence was confirmed.

Immune defence is not a unitary trait and studies employing immunological challenges need to identify, which immunological traits are under enquiry, remembering of course that unmeasured components of the immune responses may be more important to the outcome of the immune response than measured components (Matzinger, 2007). Viney *et al.* (2005) emphasize that immune responses are context-specific and, together with Zinkernagel (2007), have emphasized that functional outcomes of pathogen challenges, such as viraemia, host pathology or mortality, may be more meaningful measures of immune competence than immunological measures such as antibody titres or the strength of antigen-specific proliferation of lymphocytes *in vitro*.

3.3 The cost of excessive or inappropriate immune activation

Pathology as a consequence of excessive stimulation is a feature of both the general adaptation syndrome and the allostatic model of physiological regulation. It is perhaps not surprising then that a **fifth cost** of immune defence that can be identified is damage inflicted on the host by activity of the immune system. Immunopathology is a prominent feature of unresolved infections and may be one of the driving forces for antagonism between the T helper cell modalities Th1, Th2, Th3 and Th17 as a means of reducing the risk of self-harm during chronic immune activation (Graham *et al.*, 2005).

As a driver of evolutionary change, the risk of immunopathology needs to be traded off against any benefits to fitness of the host that might accrue from activity of the immune system. Behnke *et al.* (1992) captured this dynamic in their proposal that the escalating costs of mounting an even more effective immune response to eliminate the diminishing pathology of a residual nematode parasite population under partial control by a non-sterilizing immune response, accounts for the failure of vertebrate hosts to have evolved sterilizing immunity against nematodes. Immune defence is imperfect in predicting which foreign entities pose a threat of pathology and hence which infectious agents it needs to mount an immune response against. Furthermore, many pathogens gain a benefit from deliberately activating ineffective host immune responses and hence they outwit the immune system.

The potential for the cost of mounting an immune response to exceed the benefits it delivers has been elegantly illustrated for infection of sheep with the gastrointestinal nematode parasites *T. colubriformis* and *Telodorsagia circumcincta* by Greer *et al.* (2005a,b). During primary infection in helminth naive lambs, both parasites induce anorexia and reduced growth rates. These deleterious effects can be significantly ameliorated by treatment with immunosuppressive doses of the glucocorticoid methylprednisolone acetate, despite the drug treatment resulting in higher worm burdens and higher worm egg counts (WECs) in faeces of immunosuppressed lambs. Interestingly, in adult ewes with acquired immunity, infection with *T. colubriformis* did not lead to a reduction in voluntary feed intake, but did lead to a reduction in the gross efficiency of use of metabolizable energy that was not modified by immunosuppression. It was found that immunosuppression did not significantly reduce the loss of plasma protein into the gut that occurs during *Trichostrongylus* infection, thus the costs of acquiring an immune response in helminth naive lambs is presumably not due to immunopathology in the gut. Immunosuppression did however protect lambs against the hypoalbuminaemia caused by infection (Vaughan *et al.*, 2006).

This change, together with increased amino acid incorporation into liver and gut tissues (Steel *et al.*, 1982) and retarded skeletal growth (Sykes and Greer, 2003) during *Trichostrongylus* infection are indicative of activation of the acute phase response. Further evidence for activation of the acute phase response during helminth infections in sheep was reviewed recently (Colditz, 2003). Activation of innate immune responses by gastrointestinal nematodes is well recognized (de Veer *et al.*, 2007). Greer *et al.*'s conclusion that the acquisition phase of immunity to gastrointestinal nematodes in sheep is more expensive than later expression of immunity against the same parasites is therefore in accord with the hypothesis proposed above that generalized responses to noxious stimuli are more costly than specialized responses and is

also in accord with Coop and Kyriazakis' (1999) model of nutrient prioritization during parasite infection.

It is noteworthy that despite the acquisition of specific adaptive immunity, during re-exposure of the host to many pathogens, there may still be activation of innate immune pathways by pathogen-derived molecules before the infection is cleared. Thus, specific adaptive immunity may not be able to circumvent all the costs of innate immune responses. The extent to which it can is a research issue worthy of investigation.

3.4 Interactions of immune responses with production traits in farm animals

Are these five potential costs of immune activation evident through trade-offs with production traits in farm animals? Let us continue with the example of gastrointestinal parasitism caused by nematodes in sheep. Many detailed studies have been undertaken on the nutritional costs of parasitism and its impact on production, and a range of estimates are reported in the literature. Liu *et al.* (2005) estimated, from regression equations calculated on data obtained in a nutritional study in 10- and 18-month-old castrated male Merino sheep infected with 3300 *T. colubriformis* and 3300 *Teladorsagia circumcincta* larvae three times per week for 12–18 weeks, that infection increased the requirement for metabolizable energy by around 28% and metabolizable crude protein by around 18% in a 30 kg animal. In a 60 kg animal, this additional requirement associated with infection reduced to around 7% for both metabolizable energy and metabolizable crude protein. The authors noted that the young sheep are likely to have had some prior exposure to parasites before the experiment and were unlikely to be experiencing a true primary exposure to the parasites. Houdijk *et al.* (2001a) estimated, from predicted changes in the mass of the immune system and parasite induced plasma loss into the gut (pathology), that immunity to *T. colubriformis* increased the requirement for metabolizable protein by 5% in the periparturient ewe. Such studies illustrate an increased requirement for energy and metabolizable protein during the immune activation and pathology that accompany parasite infection.

Numerous pen studies report an impact of parasite infections on wool growth in sheep. Barger *et al.* (1973) observed an 18% reduction in wool growth during infection of sheep with *T. colubriformis* in comparison with uninfected controls. Wool has a high content of the sulphur amino acid cysteine as do immunoglobulins, gastrointestinal mucins and glutathione. Supplementation with cysteine increased wool growth in both infected and uninfected groups by 33%, but was unable to restore wool growth in the infected group to the level seen in the control group. This discrepancy illustrates the cost of altered capacity of tissues to utilize nutrients under the neuroendocrine-immune gradient that parasite infection creates.

A breakdown in acquired immunity to infectious disease around the time of parturition occurs in many mammals (Houdijk *et al.*, 2001b). There is little evidence of a hormonal basis for this reduction in immunity to gastrointestinal parasites in sheep, where re-prioritization of metabolizable protein supply to reproductive tissues, especially the lactating mammary gland, is considered to account for this change (Coop and Kyriazakis, 1999; Sykes and Greer, 2003). The degree of

reduction of immunity in the periparturient period is very sensitive to the supply of dietary metabolizable protein (Houdijk *et al.*, 2005, 2006). Thus, dietary supplementation with metabolizable protein can minimize the loss of immunity seen at this time. In non-lactating, non-pregnant sheep, both resistance and resilience (high production in the face of ongoing heavy parasite burdens) are also sensitive to availability of dietary metabolizable protein (reviewed by Walkden-Brown and Eady, 2003). Elevated activity of the hypothalamus–pituitary–adrenal axis with impending parturition, and the metabolic demands of lactation appear to reduce the priority of the immune system for access to nutrients at this time.

The studies discussed above have largely examined the impact of gastrointestinal parasite infections on production traits in sheep in controlled pen studies. An alternative approach to the question is to examine phenotypic correlations between worm burden, as assessed by WECs in faeces, and production traits in field studies. Several studies with records on thousands to more than 10,000 Merino sheep report phenotypic correlations between WEC and wool and body growth traits that are low or close to zero and generally not significantly different from zero (Albers *et al.*, 1987; Eady *et al.*, 1998; Khusro *et al.*, 2004). The failure of these field studies to identify a combined impact of immune responses to parasites and the pathology of parasitism on production traits may lie in the short period of time over which parasite resistance is measured in comparison with the time needed for production traits to manifest themselves. Management of worm burdens as part of routine animal husbandry during the longer intervals over which production responses accrue may have minimized the impact on the measured production traits (Eady *et al.*, 1998).

3.5 Environmental influences on immune responsiveness

Availability of micronutrients and macronutrients, exposure to environmental stressors and the presence of concomitant infections can have substantial impacts on immune competence. Measuring immune competence in this scenario poses a different challenge to experimentalists from measuring the costs of immune activation. While environmental influences may have differential impacts on the capacity of the various modalities of the immune system to respond to immune stimulation, measurement of the strength of antigen-specific responses (antibody titres, lymphocyte proliferation and cytokine production) can be informative. None the less, the impact of environmental influences on pool sizes and kinetics should not be ignored. The more important challenge to the experimentalist, however, may be choosing the spectrum of activities of the immune system to assay that provide an informative representation of the diversity of host defence modalities.

When nutrients in the diet are not limiting, food intake may be able to be increased to meet increased demands of immune stimulation if the stimulus does not induce anorexia. Deficiency of zinc and energy can bias immune responses away from Th1, towards Th2 (Long and Nanthakumar, 2004). In contrast, deficiency in retinoic acid, an important determinant of IgA B cell migration to mucosal tissues (Mora *et al.*, 2006), can lead to suppression of Th2 responses and bias towards Th1 responses. Most trace element deficiencies reduce immune responses in ruminants (McClure, 2003).

4. Genetic Costs of Immune Activation

While the phenotypic associations discussed above consider the physiological interplay between economic traits of the individual such as growth and host defence responses when these are activated, this section considers the consequences for offspring when there is artificial selection for immune responsiveness, disease resistance or production traits. Does selection for immune responsiveness or disease resistance impose a cost by limiting the potential of offspring to exhibit other traits of economic value? These potential genetic costs are revealed as negative genetic correlations and can be considered to be the **sixth cost** of immunity.

Genetic correlations of production traits, with WEC as a measure of resistance to gastrointestinal nematode infections in sheep grazing at pasture, have been the subject of numerous studies. A high WEC indicates a heavy infection and hence poor resistance to parasitism. In six well-characterized resource flocks of Merinos, Eady *et al.* (1998) derived a pooled estimate of genetic correlations with WEC of 0.15 for greasy fleece weight, 0.10 for clean fleece weight, −0.06 for fibre diameter and −0.21 for body weight. In 16,669 yearling Merinos on commercial properties, Khusro (2004) estimated similar values: 0.07 ± 0.07 for greasy fleece weight, 0.11 ± 0.08 for yearling clean fleece weight, −0.05 ± 0.07 for fibre diameter and −0.14 ± 0.07 for yearling body weight. From their data, Eady *et al.* (1998) estimated that genetic selection for production would lead to an increase in WEC of around 1% per annum in a flock with an average generation interval of 3 years and a selection intensity of 1.35.

Detailed reviews of the relationship between genetic change in production traits and disease resistance in farm animals are provided by Knap and Bishop (2000) and Rauw *et al.* (1998). These reviews illustrate the occurrence of trade-offs between disease resistance traits and production traits. However, when traits exhibit additive genetic variation within a population, provided that negatively correlated traits are appropriately balanced in a breeding objective, positive progress in the traits can be made despite the unfavourable genetic correlations. The examples cited by Rauw *et al.* illustrate the consequences of failure to include selection for disease resistance traits in many contemporary breeding programmes. This leads to the question of whether a composite trait of immune competence can be included in such programmes or whether separate measures of resistance to specific diseases are needed. Wilkie and Mallard (1999) have addressed this question in Yorkshire pigs by selecting on an index for high or low immune responsiveness that included antibody responses to hen egg lysozyme and cell-mediated immunity. The high responder line had faster growth rate and stronger immune responses to a number of viral and bacterial pathogens; however, resistance to parasites was not investigated. The studies provide tentative support for the value of selecting for a composite trait of general immune responsiveness.

Importantly, when genetic parameters for WEC and production traits such as those reported by Eady *et al.* (1998) have been estimated in different breeds of sheep in different environments, the strength and favourability of genetic correlations with WEC have been found to differ substantially (Douch *et al.*, 1995; Bishop *et al.*, 1996; Bouix *et al.*, 1998; Eady *et al.*, 1998; Morris *et al.*, 2000; Coltman *et al.*, 2001). This variability led Bishop and Stear (1999) to conclude that there may be no general relationship between worm resistance and productivity. The differences could be due to differences in allele frequencies between populations of sheep, differences in the composition of the

parasitic challenge and differences between the environments in which the measures were made. Resistance to internal parasites is a highly polymorphic trait and selection for resistance in the various populations may have been for defence mechanisms that differ in their costs and in their efficacy in controlling parasites.

In the current research push for gene markers for resistance to disease it will be valuable to include studies on the relative costs of the immune mechanisms the various markers are linked to. When genes associated with autoimmune disease in laboratory mice are moved into different genetic backgrounds there can be a strong influence on the expression of the disease phenotype covering the spectrum from full-blown disease to normality. Similarly, genes associated with resistance to disease may entrain differing costs in farm animals of different genetic backgrounds. Animal breeding programmes might therefore be able to exploit genetic variation in the costs of immune activation associated with disease resistance.

5. Conclusions

The immune system, as one of the body's sensory organs for controlling interactions with the environment, is integrated into the physiological regulatory mechanisms that maintain the integrity of the host in the face of diverse environmental threats. The immune system shares responsibility with the neuroendocrine system in regulating nutrient utilization by somatic tissues during times of threat. Experimental evidence indicates that immune responses are influenced by the nature of the pathogen and by genotype, age, gender, passive immunity, prior exposure to the pathogen, capacity to recall the antigen, concurrent infections, physiological status, micronutrient status, macronutrient status, day length (Nelson, 2004) and presence of concurrent stressors.

The dynamic interplay of immune defence with production traits in farm animals reflects its participation in the nutrient economy of the host. The immune system shares with other physiological pathways dynamic, predictive regulation through neutral control processes that are indicative of allostatis. Regulation and learning in adaptive immune responses may also have some autonomy from allostatis. Behavioural, physiological and immune defences against environmental stimuli employ a common strategy of using expensive innate and less costly learned pathways to protect the integrity of the host. Six costs of the immune system are identified. These are:

1. Increased metabolic activity, systemically during fever, and locally during activation of immune system cells;
2. Reduced nutrient availability due to anorexia and sickness behaviour;
3. Altered priorities for nutrient utilization due to changes in the neuroendocrine-immune gradient during immune activation that reduce the capacity of many non-immune tissues to utilize nutrients;
4. Change in the size and rate of turnover of cell and protein pools of the immune system;
5. Damage to host tissues caused by inappropriate or excessive activity of the immune system (immunopathology); and
6. Genetic costs, which are changes in the capacity of offspring to express production (and life history) traits following selective breeding for disease resistance.

Immune responses are context-specific and the costs will vary with the pathogen, the environment and the genotype of the host. Benefits of immune defence can accrue not only to the individual, but to flockmates through epidemiological consequences of immunity and to offspring through impacts on fitness.

Standardization of environments in the intensive animal industries can reduce antigen loads on the immune system and thereby reduce the activation status of the immune system and its drain on production. However, continual immune stimulation by gut flora is necessary for health of the animal in all but germ-free environments.

For defence against many pathogens, there is substantial additive genetic variation in host immune responses, and their outcome of disease resistance. The example of genetic resistance to internal parasites in sheep illustrates that resistance can be positively genetically correlated with some production traits for some genotypes in some environments and negatively genetically correlated in other cases. Thus, failure to select for resistance to internal parasites in a production-based breeding objective can lead to increased susceptibility through negative genetic correlations with production traits in some genotypes in some environments. This suggests that breeding objectives should include disease resistance when such negative genetic correlations exist.

Genetic diversity is likely to exist in the costs of immune defence and it may be possible to select for immune defence that imposes fewer costs on production traits. It may be found that immune responses linked to gene markers of disease resistance incur different costs in animals of differing genetic background. Immune defence is a complex trait and the appropriate components of immunity required for disease resistance that should be included in the breeding objective may need to be balanced against the disease risks in the production environment. For instance, housed pigs that have adequate control through management practices of external and internal parasites may be able to forgo a strong genetic capacity to mount anti-parasite Th2 immune defence although consequences of the altered T helper cell bias on pregnancy would need to be investigated (Piccinni, 2006).

The context-specific nature of immune activation suggests that quantitative estimates of the costs of immune activation cannot readily be generalized. While immunity may reduce the tax imposed on the host by pathogens it does not occur without cost. Evidence in farm animals suggests that immune defence against disease can be an important life history trait.

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12 Selection for High Production in Pigs

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1. Developments in Pig Performance

The production performance of pigs, both in terms of growth and carcass traits and in terms of reproductive output, has been considerably increased through selective breeding, particularly since the 1960s. Figure 12.1 illustrates this by showing the developments over time of mature body lipid-to-protein mass and maximum protein deposition rate, and of litter size. As expected, the growth and carcass traits show a steady development over time since the late 1960s; genetic improvement of litter size, with its low heritability, became only feasible with the implementation of routine Best Linear Unbiased Prediction (BLUP) evaluation, which occurred in the early 1990s in the various breeding organizations behind this data.

This chapter describes some of the side effects of this genetic improvement, which are mainly due to inadequate breeding goals, not taking robustness traits into account sufficiently. Knap (Chapter 17, this volume) describes how that can be (and is, in practice) remedied. These two chapters should be read in conjunction with each other.

2. Compromised Fitness, Environmental Sensitivity

In highly productive pig genotypes, fitness may become compromised directly, in the form of gradually impaired development of supportive tissues, and of the reproductive endocrine system. Fitness may also become compromised indirectly, when production-related processes come to demand so many resources from the organism that functions such as immune response and coping with other stressors become resource-limited in adverse environmental conditions. This leads to loss of adaptive capacity to cope with intensive conditions (i.e. reduced robustness). This latter scenario assumes that the metabolic drive of the production-related processes is strong enough to dominate resource allocation at the expense of fitness-related traits, but

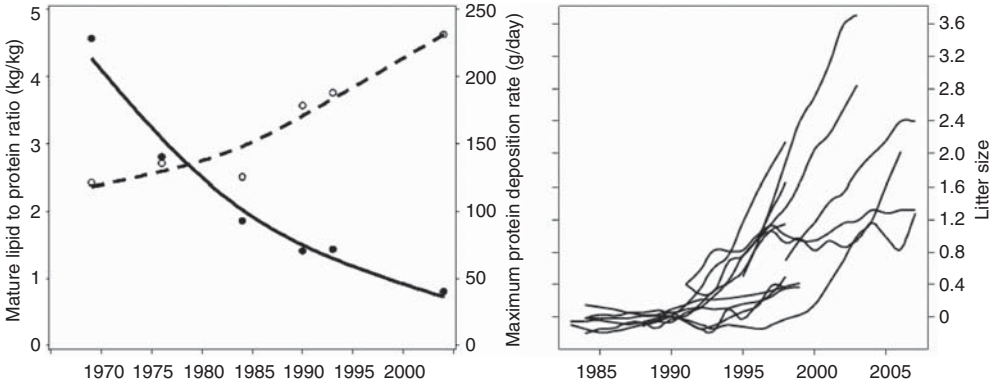

Fig. 12.1. Left: time trends of the ratio of mature body lipid-to-protein mass (solid line), and of the maximum rate of protein deposition (broken line), as estimated from Gompertz curves fitted for growing pigs of six sire lines. (Adapted from Doeschl-Wilson *et al.*, 2007.) Right: genetic trends of litter size at farrowing in 14 pig dam lines. (Data from eight Internet sources.)

it may also work the other way around: in highly productive pig genotypes, the allocation of sufficient resources towards the production-related processes may become constrained by: (i) the environment providing insufficient resources to begin with (e.g. inadequate nutrition and hot climate); or (ii) the environment making additional demands for resources (e.g. cold climate, subclinical disease and social stress). This leads to loss of adaptive capacity to cope with limiting conditions (environmental sensitivity). From the point of view of the production traits involved, it will be perceived as a genotype × environment interaction.

3. Resource Allocation

The production performance of high lean growth, feed-efficient genotypes is likely to lead to fitness constraints when the production environment is not under control and is not tailored towards the genotype's needs. Selection experiments in several livestock species confirm this. Luiting *et al.* (1997) and Luiting (1999) reviewed the relevant literature and showed that intensive selection for the above combination of traits in growing animals increases the gross production efficiency (by increasing the protein/lipid ratio of deposited tissue) and reduces maintenance requirements.

It is common in animal production to consider 'maintenance requirements' as an energy sink that should be reduced as much as possible to make resources available for production processes, and the work on residual feed intake in growing pigs (see Knap, Chapter 7, this volume) and other species (e.g. Chapter 6) shows very explicitly that this can actually be achieved through selection. Reduction of overhead costs increases the output/input ratio and this is a straightforward way to increase gross production efficiency. But many maintenance processes reflect vital functions that can only be allowed to diminish when environmental conditions do not place any extraneous load on the system (see also Chapter 7). Genotypes with the attributes that we perceive as reduced maintenance requirements may have lost their flexibility

to deal with suboptimum conditions, or in other words, may have lost fitness. Hence, the above surmise that the more efficient genotypes are often less 'robust'.

Fitness constraints play more or less important, but very different, roles in growing pigs, in young (peri-pubertal) breeding pigs, and in reproductive sows; boars are ignored here because of the very limited data available on male fertility (but see Brandt and Grandjot, 1998). We have to consider disorders in supportive tissues such as bones and the cardiovascular system, in muscle physiology, in fertility traits and in immunocompetence. From the point of view of the pig breeder, the situation is often confusing because what is needed here is quantitative information on the genetic variation of fitness-related traits within genotypes. Because many fitness traits are difficult to measure and to quantify on large numbers of animals, such information is commonly scarce and confounded. As a result, the issue has often been ignored.

4. Supportive Tissues

4.1 Bones

Leg disorders are the main cause of involuntary culling in young breeding pigs, and an important cause of early culling in sows (Stalder *et al.*, 2004) – leg weakness is unfavourably correlated with sow longevity (Grindflek and Sehested, 1996; Jörgensen, 1996; Serenius and Stalder, 2006). This is by no means a recent development: it was reported already by Smith and Smith (1965), and Bereskin (1979) produced a review arguing that the shift to confinement housing with hard-surface and abrasive floor structure had caused osteochondrosis, arthritis and varying degrees of lameness, affecting bone structure and configuration. Published genetic correlations suggest unfavourable relationships of leg weakness with growth rate and body composition, but the general picture is far from clear; correlated responses in selection experiments confirm this (see also Chapter 17).

Dämmrich and Unshelm (1975) raised the concept that muscle mass is outgrowing bone mass in fast-growing livestock (see Chapter 17 for more details). Likewise, Fernández (1995) noticed that maximum bone ash growth in pigs is consistently achieved at higher dietary mineral levels than those required for optimum body weight growth and feed conversion, and suggested that selection for lean growth has shifted physiological regulatory systems to different levels for muscle growth versus bone growth and development. This would result in bone development inadequacy in growing pigs that cannot be prevented solely by increasing mineral intake, but would require an increased focus on leg soundness in the breeding goal as well.

The five earliest entries of Fig. 12.1 represent sire lines recorded from 1969 to 1993 (compiled by Knap, 2000). These data sources provide information on body ash mass as well, and Fig. 12.2 shows the time trends of the ratios of mature body lipid and ash mass to mature body protein mass, and of the shape of the body lipid and ash growth curves relative to the shape of the protein growth curve. Whereas the mature body lipid/protein mass ratio shows a strong decline over time (the direct effect of genetic improvement), the mature body ash/protein mass ratio shows no discernible pattern, and the ash and protein growth curves (Gompertz) are very much equal in shape as judged by their points of inflection. These findings

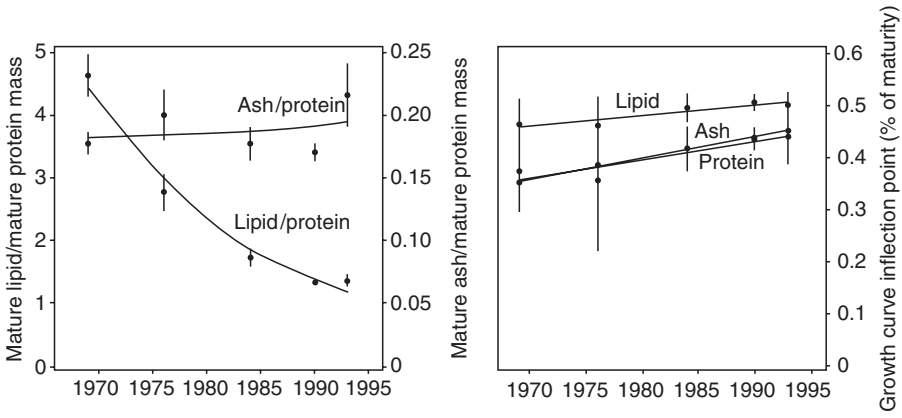

Fig. 12.2. Left: time trends of ratios of mature body lipid mass and mature body ash mass to mature body protein mass, as estimated from Gompertz curves fitted for growing pigs of five sire lines. Right: time trends of the points of inflection of the same Gompertz curves for body protein, lipid and ash mass. See Knap (2000) for details, and see also Fig. 12.1.

do not support the notion that muscle mass in modern pig genotypes has outgrown bone mass. It looks as if the *amount* of bone (ash) is not a prime candidate factor to cause fitness constraints in pigs.

Several studies (Van der Wal *et al.*, 1980; Lundeheim, 1987; Goedegebuure *et al.*, 1988; Jørgensen and Tang Sørensen, 1994; Stern *et al.*, 1995) have shown that the general 'leg weakness' problem is only partly explained by specific disorders such as osteochondrosis. The obvious conclusion is that the leg weakness issue is a complicated one that cannot be fully attributed to underdevelopment of bone (ash) mass, nor to clear-cut syndromes such as chondrosis-like lesions. The remainder may be related to impaired synchronization of maturation of bone growth plates, cartilage, ligaments and tendons with maturation of muscle mass; it will be very difficult to produce proper quantitative evidence for this.

Meantime, leg soundness traits show genetic variation, selection for leg soundness has been shown to work in pigs, and when incorporated in a sensible breeding objective it is perfectly possible to achieve genetic improvement of production traits and leg soundness at the same time (see Fig. 17.2). In practice, the main challenge for pig breeders is to implement a sufficiently sophisticated data collection and selection system. Because of the generally low heritabilities of these traits, there is little scope for real genetic improvement without making use of selection criteria that combine information from relatives (BLUP), but the issue suffers strongly from old-fashioned ideas about phenotypic selection and independent culling: proper genetic evaluation methods have hardly been applied in practice before the late 1990s.

4.2 The cardiovascular system

The cardiovascular system plays a key role in allocating resources (oxygen, substrates and metabolites) to the tissues that need them. Heart size in domestic pigs

is considerably smaller than in the wild boar (Schürmann, 1984; Dämmrich, 1987; Yang and Lin, 1997); Brambilla *et al.* (2002) mention heart weight values of 0.21% and 0.38% of body weight, respectively. This has obviously resulted from domestication and selective breeding; an early source is Engelhardt (1966). Robert *et al.* (1987) suggest a role of decreased levels of physical activity resulting from selection for feed efficiency. Myocardial hypertrophy was mentioned by Dämmrich (1987) as a consequence of the 'small heart' being 'overstrained' in genotypes with a strongly developed 'muscular periphery'. A large proportion of this (functionally small) cardiovascular system's volume capacity serves to supply the muscles in lean fast-growing pigs, and when the system is placed under stress (such as during weaning, re-grouping or transport to slaughter) the required redirection of blood supply to other tissues may be impaired. This is one of the causes of disorders in the gastrointestinal tract (oedema disease and intestinal acidosis, caused by leakage of the gut epithelium, caused by insufficient blood supply) in pigs as well as in humans. Limited heart capacity is therefore one of the potential constraints for the proper functioning of the gastrointestinal system and other organ groups in fast-growing pigs (Nabuurs, 1998; Nabuurs *et al.*, 2001).

Emmans and Kyriazakis (2000) made the logical comment that if one body component (such as muscle) increases due to selection, then other body components must necessarily be reduced. The general idea has always been that this 'other body component' is mainly adipose tissue, but any organ group that is not under direct control may be affected, particularly now adipose tissue is reaching its lower limits in high lean growth pig genotypes. A reduction of blood volume and heart weight in Pietrain (5.36 l, 295 g) as compared to Dutch Yorkshire pigs (6.56 l, 350 g) was already reported by Sybesma and Hart (1965), who related this breed difference to a difference in leanness (66.3% versus 60.4% lean) although their comparison of Pietrain with Dutch Landrace (5.88 l, 298 g, 60.6% lean) was much less convincing.

By contrast, Yang and Lin (1997) report that 'the size of the heart of domestic pigs varies in size proportionally with the changes of metabolism seen in terms of growth', based on their own experimental results and data from Davey and Bereskin (1978) and Cliplef and McKay (1993). They suggest that 'the bigger relative heart size caused by recent intensive selection for leanness should be [considered] from a different perspective. [...] Heavy selection pressure under the current intensive system of pig production may have exceeded innate physiological limits; this unfitness leading the animal to exhibit pathophysiological changes, perhaps including hypertrophic cardiomyopathy.'

Ballerini *et al.* (2003) studied wild boar, the traditional Cinta Senese domestic pig breed (CS), and a cross of CS with modern Large White. The latter showed considerably higher serum concentrations of reactive oxygen metabolites. Brambilla *et al.* (2002) and Brambilla and Cantafora (2004) suggest that cardiovascular inadequacy and oxidative stress in high lean growth genotypes is related to an increased prevalence of metabolic disorders such as Mulberry Heart Disease in weaner pigs (cardio-angiopathy due to lipid peroxidation) and Porcine Stress Syndrome (abnormal accumulation of lactic acid in muscle tissue). Of course, the latter syndrome is largely a consequence of the recessive allele of the *RYR* (Halothane) gene, which is well-documented to increase in frequency with selection for strong muscling.

This issue is under good genetic control since halothane testing started in the 1970s, and particularly since the HAL-1843 DNA test (Innovations Foundation, Toronto, Canada) became available in the 1990s (Fujii *et al.*, 1991), although there are indications of other mutations causing the same symptoms (Allison *et al.*, 2006).

Clearly, the evidence on this issue is conflicting and there is a need for more (and much better structured) research. The approach of Geers *et al.* (1990) to study electrocardiogram parameters rather than heart size would allow for the collection of data with a meaningful genetic structure, and given the complexity of the issue it seems that this should be the first priority.

4.3 Muscle physiology

To a large extent, post-natal growth is a matter of skeletal muscle differentiation, based on the differential expression of proteins involved in oxidative metabolism, oxidative stress and protein turnover (Hamelin *et al.*, 2007). Oxidative muscle fibres react more slowly when stimulated and fatigue slowly, while glycolytic fibres react more immediately but fatigue sooner (Rahelic and Puac, 1981). Oxidative versus glycolytic fibre proportions are influenced by intensity of physical activity (Bee *et al.*, 2004). Henckel (1992) suggested that an increase in glycolytic fibre proportions is due to selection for high lean growth rate, because these fibres have higher growth potential than the other types. This has been confirmed by comparisons of wild boar with domestic pigs (Rahelic and Puac, 1981; Essén-Gustavsson and Lindholm, 1984; Weiler *et al.*, 1995; also in terms of muscle fatigue resistance: Szentkuti and Sallai, 1988) and by comparisons of domestic pig strains with different selection histories (Karlsson *et al.*, 1993; Brocks *et al.*, 2000; Oksbjerg *et al.*, 2000). Because the ability of a muscle fibre to sustain physical stress is determined by its oxidative capacity, an increase in glycolytic capacity with selection may increase the animal's sensitivity to environmental stressors.

5. Reproduction

The genetic relationships of some sow fertility traits with growth and carcass traits has become properly documented since the statistical analysis of large field databases has become computationally feasible. Litter size is the most widely recorded fertility trait, and there is now a reasonable body of published estimates of its genetic correlations with growth rate and backfat depth. Published values for both correlations centre around a value close to zero, but unfavourable relations have been found just as well as favourable ones. See Fig. 12.3 where the mean values are −0.02 and +0.06, respectively. Since the early 1980s, this pattern has been confirmed by selection experiments.

Ignoring the fact that many of the apparently (un)favourable correlation estimates will not differ significantly from zero, it seems that the relationship of litter size with growth-related traits is largely population-specific. Resource allocation theory would then predict that the unfavourable relations occur particularly in the leaner, faster-growing genotypes. This is supported by the data presented in

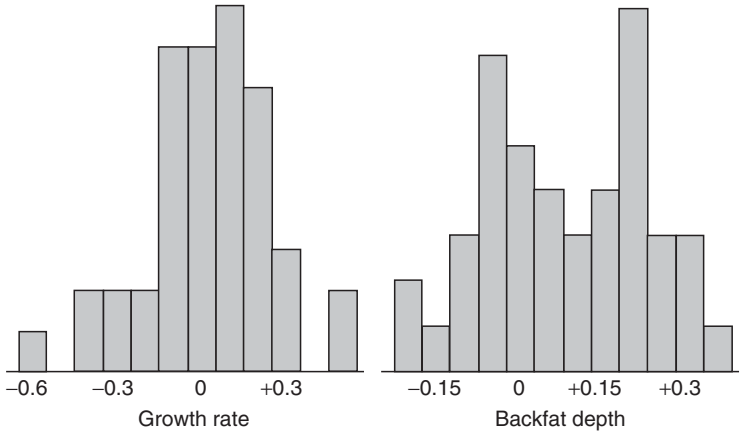

Fig. 12.3. Frequency distributions of published genetic correlation estimates of litter size at birth with growth rate (left) and with backfat depth (right). (Data from 19 literature sources.)

Fig. 12.4. The genetic lines in this lean growth selection experiment (Cameron, 1994) provide a much wider genetic range of growth and carcass traits than what is usually encountered within a commercial pig population. Hence, these data allow for a much more detailed view of the above-mentioned genetic relationship. Figure 12.4 shows that the genetic relationship of litter birth weight with backfat depth depends on the actual fat depth level. The linear correlation estimate on these data is effectively zero overall, but within a narrower range of the *x* variable the correlation would be favourable (in the fatter genotypes), unfavourable (in the leaner genotypes) or truly zero.

The relationships of puberty- and oestrus-related traits (age or weight at first mating or farrowing, weaning-to-mating intervals) with growth and carcass traits show a similar, as yet inconclusive, pattern as for litter size (e.g. Hutchens *et al.*, 1981; Johansson and Kennedy, 1983; Mabry *et al.*, 1985; Rydhmer *et al.*, 1992, 1994; Bidanel *et al.*, 1996; Tholen *et al.*, 1996; Tölle *et al.*, 1998; Cameron *et al.*, 1999; Torres Filho *et al.*, 2002; Holm *et al.*, 2004).

Sows reach their mature size around their seventh parity (Simmins *et al.*, 1994). This means that young sows are challenged simultaneously with the drive to grow and with the successive requirements of pregnancy, lactation and attaining oestrus. Because dam-line breeding goals have included selection for reduced fatness until very recently, the above-mentioned challenges have to be met with reduced amounts of body fat at times of conception, farrowing and weaning.

Noblet *et al.* (1990) calculated the metabolizable energy (ME) requirements of pregnant and lactating sows as 6–10 and 15–20 Mcal/day, respectively. Hence, lactation is by far the most energy-demanding element of the reproductive process. From that point of view, a counterpart of Fig. 12.3 focusing on lactation performance rather than litter size would be more to the point; but such information is very scarce as yet. When feed intake is insufficient to meet the energy requirements, body reserves of lipid and protein are catabolized, particularly in first parity (Whittemore, 1996; O'Dowd *et al.*, 1997; Grandinson *et al.*, 2005). Bronson and

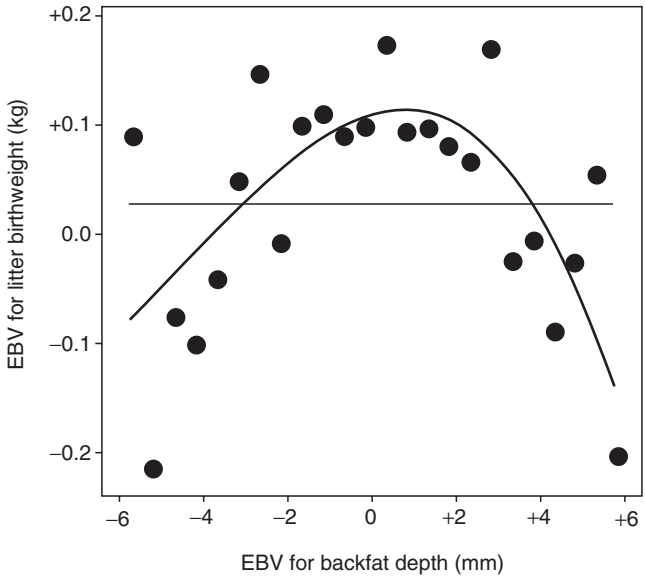

Fig. 12.4. Estimated breeding values for litter birth weight in relation to those for backfat depth in pigs of nine selection lines. The data points represent means of ½-mm backfat depth subclasses. R^2 = 0.0002 and 0.48 for the linear and cubic regression lines, respectively. (Data from Kerr and Cameron, 1996; N. Cameron, 1999, personal communication.)

Manning (1991) reviewed the literature on the relation between body composition and female fertility, and concluded: '[T]here is no doubt that ovulation can be regulated somehow in relation to whole-body energy balance, and certainly the amount of energy stored in adipose tissue is an important component of energy balance, but there is no evidential basis on which to accord body fat a direct causal role in regulating ovulation in either the pre-pubertal or adult female [mammal].' Such evidence has been produced more recently; Butler (2005) mentions an inhibitory effect on the reproductive axis, affecting luteinizing hormone (LH) and follicle-stimulating hormone (FSH) levels, and Kauffold *et al.* (2008) and Quesnel *et al.* (2007) report effects on follicular development during lactation and after weaning, respectively. A conceptual framework describing the relationship between body lipid reserves and the reproductive cycle was more recently presented by Friggens (2003). Reproductive failure due to a reduced ability to ovulate, delayed returns to oestrus, low litter size and poor milk production (Eissen *et al.*, 2003) are the predominant reasons for early culling of young sows (Stalder *et al.*, 2004; Butler, 2005; Thaker and Bilkei, 2005).

While reproductive output is genetically increasing and body reserves remain limited, nutrient intake of lactating (and pregnant) sows should then increase, and sow feed intake capacity becomes a critical factor. Eissen *et al.* (2000) show that lactating sows compensate for the higher energy requirements of increased litter size (milk production) by increasing their feed intake, but this compensation was inadequate in their sows. They conclude that the ongoing genetic trends of

decreasing fat reserves at farrowing and increasing energy requirements during lactation (as in Fig. 12.1) require voluntary feed intake of lactating sows to be included as a breeding goal trait. We come back to this in Section 7.

6. Immunocompetence

Amadori (2006) refers to the various cardiovascular and metabolic disorders mentioned in Sections 4.2 and 4.3, and concludes that 'undoubtedly, the widespread appearance of devastating viral diseases in the 90s (Porcine Reproductive and Respiratory Syndrome (PRRS), post-weaning multisystemic wasting syndrome) is related to the above phenotypes of swine breeds. This concept can be adequately grasped having in mind that PRRS virus can increase the susceptibility of pigs to bacterial endotoxin, and that Porcine Coronavirus 2 (related to PMWS) can down-regulate the homeostatic IFN-α response of pigs. The resulting cascade of inflammatory cytokine responses and/or its effects could thus be more serious in pigs with the above negative traits.' A worrying example of a multidimensional cause-and-effect system.

Immune response in subclinical disease situations, as well as the processes of coping with other stressors, may require a considerable amount of resources and therefore constitute an important source of fitness constraints (Colditz, Chapter 11, this volume). Knap and Bishop (2000) distinguished four processes of particular interest in this field: (i) the way a population's genetic potential for immunocompetence can be changed by breeding, (ii) the way an animal's immunocompetence is influenced by its production potential, in combination with the environmental resources that are available to it at a given time, (iii) the way the disease status of an animal (and a population of animals) is influenced by its immunocompetence, and (iv) the way the production level of an animal is influenced by activation of its immune system.' This is illustrated in Fig. 12.5, with the main relationships among the animal's genetic potential (for immunocompetence and production traits), its environment (in terms of the available resources and the epidemiology of any prevalent infectious diseases) and its phenotypic expression of genetic potential (in terms of immunocompetence and production traits).

The factor that connects the four processes is the influence of environmental conditions on the relation between the genetic potentials for production traits and for immunocompetence. The most striking effect of this is environmental sensitivity of genetic correlations, as in Figs 12.3 and 12.4. Ultimately, all four processes influence the realized level of production. A main task for the future is to fit the four together into a comprehensive framework.

At present, the serious literature provides very little quantitative information on the above issue of '(ii) the way an animal's immunocompetence is influenced by its production potential, in combination with the environmental resources that are available to it at a given time' in pigs, but tendencies towards unfavourable relations between immunocompetence and lean growth capacity have been reported by Stahly *et al.* (1994), Frank *et al.* (1997), McComb *et al.* (1997) and Schinckel *et al.* (1998, 1999). These results are difficult to interpret because there is little information about standard errors in the reports, and the described trends are not always consistent between traits. The most striking result is perhaps the mortality of the pigs

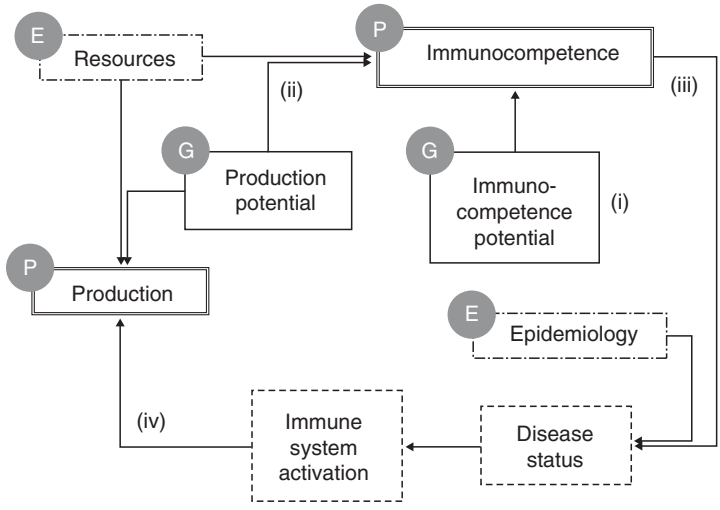

Fig. 12.5. The main genetic (G), environmental (E) and phenotypic (P) entities that play a role in the relation between genetic change and infectious disease. (From Knap and Bishop, 2000.)

of Frank *et al.* (1997). Their two genotypes (with average 51% and 57% carcass lean, respectively) showed 2.8% and 3.6% mortality, respectively, in a 'low immunostimulation' environment and 5.6% and 18.5% mortality in 'high immunostimulation' conditions.

Adipose tissue has been found to have endocrine functions: it produces the hormone leptin, which mediates energy allocation to humoral immune functions (Drazen *et al.*, 2001), and Demas *et al.* (2003) suggested that 'reductions in energy availability in the form of reduced body fat [in rodents] can reduce humoral immune functions […] these findings support the idea that reduced energy availability can lead to increased disease susceptibility in a variety of mammalian species'. In these studies, body fat was 'reduced' surgically (by lipectomy); how this would translate to the much more gradual reduction of body lipid mass through selective breeding (with the possibility for adaptation) remains open, but the concept is interesting enough. The question that follows from it is if selection for reduced body fatness reduces the amount of (lipid-free) adipose tissue as such (which must be the endocrine part), or reduces the tendency of the organism to deposit lipid in this tissue.

The experimental evidence on interactions between lean growth potential and immunocompetence in livestock is largely rudimentary and sometimes anecdotal. Most studies derived their genetic contrasts from comparisons of different breeds or, at best, selection lines that were developed from the same base population. For the animal breeding industry to take relevant action on this issue would require information on the within-population variance and covariance of these traits. Given the current lack of quantitative data, research emphasis should be on the quantification of genetic variation and covariation within genotypes, rather than on revealing contrasts between breeds. The strong increase of genomics work in the past decade should lead to much more useful information.

7. Environmental Support of High-performance Genotypes

It may seem self-evident that changes in genetic potential should be accompanied by changes in the production environment that is supposed to support it, but in practical pig production this is often not the case; Chapter 17 gives a few examples. The literature is confusing regarding this issue, and this may serve here as an illustration of a much more general confusion about the side effects of genetic change in production traits. Genetic contrasts or developments get often confounded with environmental ones.

Williams (1998) makes a comparison between: (i) experimental results of around 1970 (e.g. Elsley *et al.*, 1969; O'Grady *et al.*, 1973), which show that the weaning-to-mating interval following the first-parity litter of 'pre-modern' sow genotypes is hardly influenced by the lactational feeding level; and (ii) later results (e.g. King and Williams, 1984; King and Dunkin, 1986; Mullan and Williams, 1989), which show that this interval becomes substantially prolonged at lower feeding levels. This is further interpreted as an increased environmental sensitivity of the 'modern' genotypes because of their diminished body reserves.

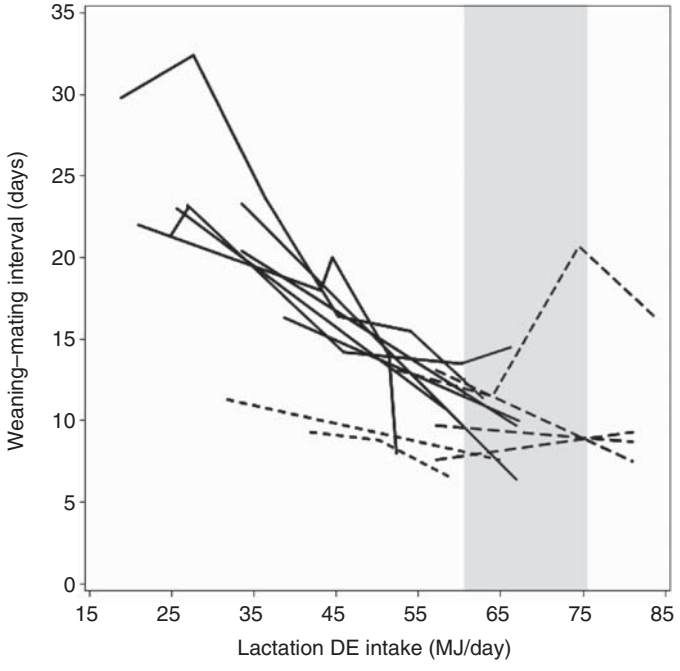

Fig. 12.6. Weaning–mating interval in primiparous sows in relation to preceding lactational DE intake. Solid lines: King and Williams (1984), King and Dunkin (1986), Koketsu *et al*. (1996), Mullan and Williams (1989), Reese *et al*. (1982a,b) and Yang *et al*. (1989). Broken lines: Elsley *et al*. (1969) and O'Grady *et al*. (1973). Dotted lines: Nelssen *et al*. (1986) and Prunier *et al*. (1993). The shaded area represents ARC (1981) and NRC (1988) recommendations.

But when these data are plotted together, and results from further experiments are added (as in Fig. 12.6), two things become clear. First, two of the nine 'modern' genotypes (from Nelssen, and Prunier) show as little influence of the lactational feeding level on weaning-to-mating interval as the older ones did (although this influence is now statistically significant due to improved experimental design). Second, the genotypes that *do* show a large influence do so only at grossly inadequate feeding levels. All these 'modern' genotypes show essentially the same weaning-to-mating interval as the earlier ones when fed at adequate levels. These adequate levels coincide with the *lowest* levels fed in the earlier experiments, and with the *lower bounds* of the recommendations for lactating sows made by councils such as ARC (1981) and NRC (1988).

A much more valid point raised by Williams (1998) is that the voluntary feed intake capacity of sows of some of the 'modern' genotypes may have been reduced as a consequence of selection for high lean growth in the pre-pubertal period. The consequence of this would be that these sows are not able to ingest the nutrients required to avoid excess mobilization of body reserves during lactation (see Section 5). This was supported by experimental results from Apeldoorn and Eissen (1999). There are two issues here: (i) a possible reduction of pre-pubertal voluntary feed intake capacity; and (ii) a 'carry-over' effect of this reduction to the reproductive age.

The first plot of Fig. 12.7 shows that the voluntary feed intake of growing pigs has indeed decreased considerably since the late 1960s. But much of this decrease is simply due to reduced energy requirements that follow from the reduced lipid deposition rates shown in Fig. 12.1. Sound experimental evidence for a reduced intake *capacity* in high lean growth genotypes is fragmentary. McCracken *et al.* (1994) subjected such pigs to forced feeding and failed to

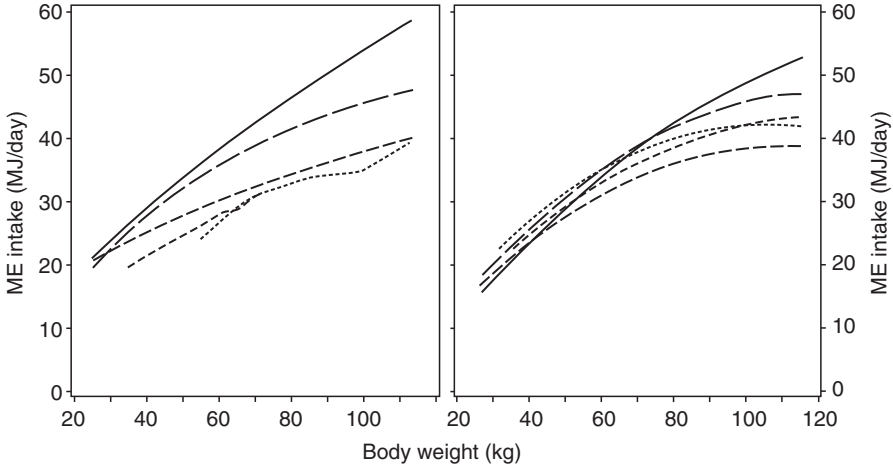

Fig. 12.7. *Ad libitum* metabolizable energy (ME) intake of growing pigs of different genotypes. Left: observed data reported by Cole *et al*. (1967), NRC (1988), Cole and Chadd (1989), Labroue (1996) and Von Felde (1996), top to bottom. Right: simulated 'desired ME intake' of the genotypes in Fig. 12.2, located in 1969 (———), 1976 (—— ——), 1984 (— —), 1990 (– – –) and 1993 (---).

measure an increased protein deposition rate in these overfed pigs relative to *ad libitum*-fed controls.

The second plot in Fig. 12.7 shows the simulated 'desired feed intake' (i.e. the intake required to satisfy the energy and protein demands of the genotype's growth potential, and its maintenance) curves of the five pig genotypes of Fig. 12.2. Although the data in these two plots represent different sets of genotypes, there is a considerable overlap between the two in the time range covered. Comparison of these plots while ignoring their disconnection suggests that the observed reduction of *ad libitum* feed intake in genotypes from the late 1960s to the late 1980s (the three upper lines in the first plot) is accompanied by a reduction of the simulated 'desired feed intake' during the same period in the second plot. Hence, these early genotypes consume less feed because they 'desire' less of it, which is because they grow less fat.

The first plot shows an ongoing reduction of observed feed intake during the 1990s (the lower two lines), but the simulated genotypes from that period in the second plot show an *increase* of 'desired feed intake' relative to the 1984 one. This mismatch may be caused by failure of the simulation model to take account of two factors: (i) reduced maintenance requirements independent from body composition; and (ii) reduced feed intake capacity. There is very little concrete evidence for or against either option, apart from McCracken *et al.*'s (1994) results mentioned above, which would count against option (ii). A reduction in maintenance requirements during this period with its intensified housing conditions (some of which would have *increased* maintenance) may be an indirect effect of selection for feed efficiency.

Much of the evidence for a 'carry-over effect' of pre-pubertal to lactational feed intake capacity is again anecdotal, and is awaiting proper genetic confirmation. For many years, the only published genetic correlation estimate (Van Erp *et al.*, 1998) was based on a grossly inadequate number of observations so that its dubiously high $+0.92 \pm 0.50$ value was not significantly different from zero. Bunter *et al.* (2007) reported an equally confusing estimate of -0.26 ± 0.33, together with low positive genetic correlations among lactational feed intake records in first versus second and third parity (0.4 ± 0.3 and 0.4 ± 0.4; Hermesch, 2007). The -0.26 value was recently updated for first-parity records only, with preliminary estimates at $+0.3$ to $+0.6 \pm 0.14$ (K.L. Bunter, 2008, personal communication) depending on the statistical model and the data subset used for lactational feed intake. Whittemore (1998) made the obvious comment that if the required nutrient intake cannot be realized during lactation, feeding levels during pregnancy will have to be increased.

Whatever the possible causes of impaired fertility in modern pig genotypes, the above only illustrates that there seems to be a clear demand in the industry for dam-line products that are not constrained by either limited body reserves or limited feed intake capacity, if only to compensate for inadequate environmental conditions. As argued above, this is contradictory to ongoing selection for high lean growth, with the logical consequence that dam-line breeding must be steered away from this. In accordance with this, many commercial dam-line breeding goals nowadays aim at an increase of feed intake and no further reduction of body fatness. See Bergsma *et al.* (2008) for a detailed analysis of the energetic efficiency of sows during lactation and its relation to reproductive performance.

8. Improving Robustness

It should be clear from all the above that sensible pig breeding objectives have to formally include fitness traits in order to retain (or restore) the required balance. In the Western world, pig breeding objectives used to be dominated by lean growth rate, but the economically optimum levels for these traits were being approached in the mid-1990s. For example, Ducos and Bidanel (1996) wrote:

> [T]he selection objective defined some years ago for the maternal breeds [in France], which leads to an improvement in the prolificacy and to a decrease in the adiposity of the carcasses, should enhance the energetic competition phenomena between the growth and the reproductive functions, and/or to insufficient mobilization of the body's lipid reserve ability during the suckling period, and, finally, to increased genetic antagonisms between production and reproductive traits. These reported cogitations, combined with unfavourable changes in the French carcass payment rules, have recently led to an important decrease in the economic weight of the carcass lean content as a selection objective in Large White and French Landrace breeds.

In addition, pig breeders have been made acutely aware of the need for genetically robust animals as a result of the rapid expansion of pig production in: (i) the USA, with its low-cost and large-scale (and hence increasingly intensive) production systems; (ii) South-east Asia and Latin America with their hot climatic and health-related complications; and (iii) eastern Europe with its problems with adequate housing, cold climate control and nutrient supply (see Knap, 1998). This is rapidly moving the attention towards fitness-related traits, and the breeding goals and selection strategies of at least the internationally operating breeding companies have been following that move (see Chapter 17 for more detail).

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13 Selection for High Production in Poultry

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The allocation of resources by an individual may be viewed in the context of the history of the population in concert with life cycle stages. Thus, it is instructive to review some of the behaviours that initially favoured domestication of the fowl, which commenced during Neolithic times. In addition to a positive reaction to humans were general dietary habits, an ability to adapt to a range of environments and a social structure that allowed intermingling of the sexes, promiscuity and precocial young with rapid hen–chick attachment (Hale, 1969). Although today's chickens may appear different from their junglefowl ancestors (Al-Nasser *et al.*, 2007) and current husbandry practices make some of the behaviours favouring domestication no longer relevant, domestic chickens have retained to a large extent the repertoire of natural behaviours connected with surviving, growing and producing viable offspring (Duncan, 1998). Also, domestic chicken and junglefowl interbreed successfully. Accordingly, domestication of the fowl may be considered minor when viewed in an evolutionary context.

1. Resources Are Finite

Resources available to an individual are finite. When they are or are no longer allocated to one function, be it through prior selection, phase of life or human manipulation, there will be more or fewer resources available for other processes (e.g. Siegel, 1995; Rauw *et al.*, 1998, 1999; Siegel and Gross, 2007). For example, the artificial incubator allowed rearing of young as a class separate from their parents, which facilitated feeding of specialized diets and extended the breeding season. Resources required for incubation behaviour and care of young became available and could be redirected to the production of more eggs. The development of the trap nest made it possible to identify the hen that laid the egg, which then allowed artificial selection for those hens that laid more eggs. Thus, there was a

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cascading pattern for selection for increased egg production. These inventions, plus advances in disease control and nutrition, set the stage for the post-World War II era that saw acceptance by the poultry community of negative genetic and phenotypic relationships between body weight and reproduction. The result was the demise of dual-purpose fowl for commercial production (Siegel *et al.*, 2007). An emerging poultry industry provided a ready market whereby the dual-purpose chicken was replaced by specialized stocks bred for either rapid growth or high egg production (Hunton, 2006). Understanding the value of hybrid vigour, coupled with large populations and intense selection, allowed for developing stocks that excelled in production of either meat or eggs (Siegel, 1989). The result has been great strides in enhancing expression of growth and egg production traits (e.g. Havenstein *et al.*, 2003; Flock *et al.*, 2005) as well as global production of meat and eggs (Windhorst, 2006).

In parallel with the development of specialized stocks of chickens for meat and for eggs was enhanced feed efficiency. Feed efficiency per se is a heritable trait that responds to genetic selection (Pym, 1990). In addition, nutritionists developed diets that further enhanced the efficient conversion of feedstuffs to meat or eggs. Feeding these specially formulated diets in concert with immunization programs plus genetic selection allowed chickens to reach market weights at younger ages or have high intensities of egg production. Hatching mass is nearly three times higher and adult body mass more than four times higher for broilers than for junglefowl (Jackson and Diamond, 1996). To reach a 2–2.5 kg market weight took about 110 days in the 1940s. Today this weight is accomplished in about 35 days, reducing the costs of labour, heating, lighting and housing. Layers resemble broilers in their large hatching mass, but resemble junglefowl in their lower growth rate and smaller pectoral muscles (Jackson and Diamond, 1995). Adult *ad libitum* fed broiler hens may reach three to four times the mature weight of laying hens while producing less than half the number of eggs per year. Moreover broiler hens are rarely recycled for a second laying cycle.

The time frame post-World War II during which these great changes in performance occurred is only about 0.5% of the period from when the fowl was first domesticated. This suggests that of the characteristics that initially favoured domestication of the fowl, perhaps an ability to adapt was most important during this brief window of time. This characteristic becomes even more relevant when viewed in the context of modern transportation that allowed for a global production of similar stocks: the jet aircraft facilitated rapid movement of elite stocks throughout the world. Selection for efficient production of meat or eggs in the fowl is goal-oriented and should be viewed as a continuation of the domestication process.

There is, however, a caveat whereby individuals with high feed efficiency for growth or reproduction may, in turn, have fewer resources available to respond to chronic and acute genetic and/or environmental insults. That is, there are consequences of selection for high production. Breeders of chickens are cognizant that selection for changes in one trait may not be independent of changes in other traits. These changes may have consequences that could be positive or negative (Bessei, 2006).

2. Consequences of Selection

2.1 Feed intake and digestion

In the development of the commercial meat chicken of today, breeders have emphasized selection for rapid growth to market age with enhanced feed efficiency and more recently, low levels of body fat. In turn, nutritionists have formulated complete diets to maximize these traits during the growing period. Growth is a complex process directed by the genetic code of the individual. There is a definite sequence and synchrony to growth with formation of skeletal, muscle and adipose tissue. Competing for resources available for growth are body functions including maintenance, health and reproduction. Allocation of resources at any point in time is not independent of an individual's past history and stage in life as well as the past history of the population (Katanbaf *et al.*, 1988b). In the fowl, resources during embryonic development are fixed by what is available from the egg. Post-hatch there is transition to utilization of external feed. During the early post-hatch period, supply organs (e.g. intestine and liver) grow faster than demand organs (e.g. muscle, fat and ovary). This pattern of growth is critical because the 'engine' must have adequate capacity before carrying 'cargo'.

A consequence of selection for either rapid growth to market weight or for high egg production is an earlier age at sexual maturity. Onset of lay requires a convergence of age, body weight and body composition (Zelenka *et al.*, 1986). To achieve this convergence, feeding and husbandry programmes are designed to reduce growth during various ages. When released from these restrictions there is frequently compensatory growth whereby growth rate is accelerated. Katanbaf *et al.* (1988a) provided feed *ad libitum* (AL) from 0 to 28 days of age to one group of broiler chickens and fed another on alternate days (AD). At that age when AL chicks were almost twice as heavy as AD chicks, half of the AD chicks were then released to *ad libitum* feeding (ADR). By day 42, the body weight gains were 771, 696 and 448 g for ADR, AL and AD chicks, respectively. Clearly, the ADR chicks were exhibiting compensatory growth (i.e. resources were being directed to growth). On day 48, half of the chicks from each group were given an *Escherichia coli* challenge. Mortalities 72 h post-challenge were 7%, 20% and 50% for AD, AL and ADR groups, respectively. The consequence of the diversion of resources to growth was that fewer resources were available to respond quickly to the *E. coli* environmental insult.

Rapid growth in broilers is fuelled by increased feeding rates and supported by increased sizes of the small intestine, the gizzard, proventriculus and possibly the ceca, thus increasing the nutrient transporter capacities. However, broilers appear to deviate further from the linear double-logarithmic relationship between organ mass and body mass than the red junglefowl for all gut compartments (Jackson and Diamond, 1995). Mitchell and Smith (1991) indicated that genetic selection for rapid growth was associated with increases in absolute weight and length of the small intestine, but with marked reductions in relative weight and length. In broiler chickens, gut compartments are relatively small at hatching and then undergo a steep growth spurt relative to body mass (Nir *et al.*, 1996). The size of the chick at hatch is largely determined by the size of the egg, which is similar for broilers and

layers. Because broilers grow to a much larger adult body size than layers the proportion of hatch to final body weight is much lower.

Forbes (1995) reviewed numerous factors, including genotype, that influence dietary choices made by chickens. Meat-type chickens from three genetic stocks known to differ in growth potential were fed either a single diet or a choice of two diets that differed in protein and energy (Siegel *et al.*, 1997). The choice diets were formulated so that when mixed in specific proportions they provided a single diet with recommended levels of protein and energy to enhance growth and feed efficiency (body weight/feed consumed). From a range of criteria, chickens fed the single diet were heavier with enhanced feed efficiency and more breast meat and less fat than those provided choice diets (Table 13.1). There was no difference between groups in immunocompetence as measured by response to sheep red blood cell antigen. The results indicated that intense selection for growth and feed efficiency to market weight did not rule out the chickens' ability to discriminate among diets. Moreover, diets formulated to be sound economically are not consistent with dietary preferences. These results suggested that meat-type chickens may innately, when given a choice, select diets with long-term survival rather than economic benefits.

Work by Siegel and co-workers, comparing broilers from a high line with those from a low line selected for body weight at 56 days of age, showed that that a correlated improved feed efficiency resulted from several physiological factors, including a decreased metabolic rate (Owens *et al.*, 1971), a higher food passage and digestion and higher enzymatic activities in the small intestine (Dunnington and Siegel, 1995). Mitchell and Smith (1991) noted that selection for rapid growth rate resulted in animals with a reduced relative amount of mucosa in the small intestine, suggesting an increase in net efficiency of digestion and absorption per unit of mucosa. In addition, selection for fast growth appeared to have damaged the hypothalamic satiety mechanisms leading to a failure to diminish the hunger drive and consequently to hyperphagia or overconsumption (Burkhart *et al.*, 1983; Dunnington and Siegel, 1996). Chickens from the slow-growing line could be force-fed to eat substantially above *ad libitum* feed intake, whereas this was possible to a significantly lesser extent in the fast-growing line. These results suggest that selection for fast growth in broilers resulted in individuals that consumed feed

Table 13.1. Comparisons of broilers fed a single diet with those allowed to balance protein and energy.

	Diet	
Trait	Single	Choice
Body weight	+	−
Feed efficiency	+	−
SRBC antibody	=	=
% Abdominal fat	−	+
% Breast meat	+	−

+, Superior response; −, inferior response; =, no advantage.

above their metabolic requirements until they reached a limit set by their gastrointestinal capacity (Nir *et al.*, 1978; Barbato *et al.*, 1984). Because broiler breeders, i.e. the parent stock who produce broilers, have the same huge appetites as their progeny, human intervention is required by means of implementation of feed restriction programmes to reduce the propensity for obesity in breeders and control hyperphagia (O'Sullivan *et al.*, 1991; Decuypere *et al.*, 2006; Renema *et al.*, 2007). If those animals would be allowed free access to food, they would soon become obese and suffer from obesity-related problems, such as low fertility and reduced life expectancy. A restricted feeding regime may result in acceptable levels of egg and semen production, and reduce the incidence of multiple ovulations. The numerous caveats involved with propensity for broiler growth potential and feed consumption coupled with feed restriction programmes were recently reviewed by Renema *et al.* (2007).

2.2 Ascites

Ascites is a condition caused by pulmonary hypertension resulting in valvular insufficiency and right ventricular failure, which increases hydraulic pressure in the vena cava and portal system resulting in leakage of fluids from the liver into the abdominal cavity (Squires and Julian, 2001). Ascites was initially observed at high altitudes, under hypoxic conditions. However, since approximately 1980, ascites in broiler chickens is also observed at lower altitudes, its incidence running parallel with a faster growth rate, improved feed conversion and an increased metabolic rate (Scheele, 1996; Julian, 1998).

Rapid growth in broilers requires a high metabolic requirement for oxygen that requires a high volume of blood flow through their lungs (Julian, 1989). Julian (1989) observed a drop in lung volume as a percentage of body weight from 2.0 at day 1 to 1.4 at day 144 in commercial broilers, from 1.8 to 1.5 in domestic laying-strain fowl and from 2.3 to 1.8 in red junglefowl, constituting a drop in lung volume of 32%, 20% and 20%, respectively. As Balog states: 'Their hearts and lungs have to work harder to keep up with the rapid rate of growth, and they just can't do it.' (Weaver-Missick, 2000). Ascites, which had become a prominent cause of illness, death and carcass condemnation in meat-type chickens, is less an issue today because of genetic research and intense selection against this condition by commercial breeders (Druyan and Cahaner, 2007; Pavilidis *et al.*, 2007; Druyan *et al.*, 2008).

2.3 Muscle pathology

Muscle mass is determined by the number of muscle fibres and the size of those fibres. This, in turn, is related to the capacity of fibres to adapt to activity-induced demands and may be associated with stress susceptibility and meat quality (Rehfeldt *et al.*, 2000). Fast-growing meat-type chickens have more muscle fibres with larger diameters than slower-growing strains (Dransfield and Sosnicki, 1999; Macrae *et al.*, 2006). Moreover, muscles of fast-growing chickens contain a particularly low

proportion of oxidative and a high proportion of glycolytic muscle fibres in different muscles (Henckel, 1992). Oxidative muscle fibres are adapted to aerobic metabolism for rapid, fatigue-resistant activity, whereas glycolytic muscle fibres are adapted to anaerobic metabolism, fatigue faster and are used for brief bursts of activity (Dransfield and Sosnicki, 1999). Henckel (1992) suggested that an increase in glycolytic fibres may result from selection for improved meat yield, growth rate and feed conversion rate, as glycolytic muscle fibres have higher growth potentials than the other fibre types. Macrae *et al.* (2006) suggest that an increase in the number of glycolytic fibres result because there may be an upper size constraint on the oxidative muscle fibres as they need to employ an efficient oxidative metabolism requiring relatively small diameters so that oxygen diffusion into the fibre does not become limiting. In turn, glycolytic fibres can function metabolically with large diameters, and as a result, may be more responsive to selection for increased fibre size. Large muscle fibres may be reaching the maximum functional size constraints when the increased oxygen diffusion distances of large fibres reduce oxidative capacity, resulting in metabolic stress associated with increased diffusion distances for oxygen, metabolites and waste products in larger fibres (Macrae *et al.*, 2006). Because the oxidative capacity determines the ability of a fibre to sustain physical stress, it was suggested that most commercial chickens may be very sensitive to environmental stresses, resembling the situation observed in pigs, in which those muscles would be classified as PSE (pale, soft and exudative meat; Henckel, 1992; Rehfeldt *et al.*, 2000).

An increase in growth rate has resulted in an increased incidence of deep pectoral and focal myopathy (Dransfield and Sosnicki, 1999). Meat-type chickens rather than layer-type birds were shown to have a higher prevalence of myopathological changes in muscle sections (Soike and Bergmann, 1998; Macrae *et al.*, 2006) and impaired adaptability to exertion of skeletal muscle (Soike and Bergmann, 1998). Because of the markedly increased muscle size in heavy breeds, muscles become strangulated and ischemic, such that the increased pressure within the muscle occludes the blood vessels, causing a necrosis of the muscle (Bianchi *et al.*, 2006). In the study of Castellini *et al.* (2002), good adaptation to extensive rearing conditions was better in slower-growing poultry genotypes, while faster-growing genotypes showed unbalanced muscle response to the greater activity and the oxidative stability of the meat was reduced. Wilson (1990) suggested that selection for rapid growth has contributed to muscles that outgrow their life support systems and bring about muscle damage.

2.4 Skeletal disorders

Rapid growth and heavy body weight have been implicated in musculoskeletal disorders in meat-type poultry (Julian, 1998). Sanotra *et al.* (2001) estimated that more than 30% of broilers have high-moderate to severe gait impairment. An increased incidence of skeletal deformities in rapidly growing strains compared with slower-growing strains may result from nutrient deficiencies (rapidly growing birds have a higher requirement for specific nutrients), inadequacies in the production of growth factors or signalling mechanisms needed for rapid tissue growth or cell proliferation, and from a higher frequency of mechanically induced or

trauma-associated problems (Julian, 1998; Whitehead *et al.*, 2003). Up to about 4 weeks of age, rapid growth may contribute to leg problems, resulting from bone, cartilage, tendon and ligament production of poor structural quality and inadequate tensil strength. This is compounded by heavy body weights producing stress on the bones, tendons and ligaments (Whitehead *et al.*, 2003). According to Leach and Gay (1987), genetic selection for improved muscularity has resulted in birds with a posture that is inappropriate for extended periods of walking and standing. Tibial dyschondroplasia, a specific form of growth plate abnormality, is becoming less common in meat-type poultry because of recent non-invasive procedures for its detection, which allows for breeders to select against it. Rare or absent in other birds, it is most specifically related to rapid growth, likely because of a requirement for very rapid long bone growth (Julian, 1998). Another growth-related skeletal deformity is osteochondrosis, resulting from a vascular degeneration (Julian, 1998). There is evidence that some skeletal deformities are painful as evidenced by studies with analgesics (Danbury *et al.*, 2000).

2.5 Behaviour

In addition to selection for commercial purposes, there is a history of selection experiments with the fowl in experimental settings. For decades we have had an interest in direct, as well as correlated responses to short- and long-term selection for specific traits (e.g. Siegel, 1979, 1989; Rauw *et al.*, 1998). In an attempt to study genetic variation in adaptive responses, a selection experiment was designed (Gross *et al.*, 1984) to measure plasma corticosterone levels in response to social strife. The selection procedure was simple in that chicks from a common base population were reared in battery brooders as sex-intermingled flocks until 5 weeks of age. They were then transferred to other cages and maintained in eight-bird unisexual flocks where they developed a stable social hierarchy. To disrupt the stable hierarchies, beginning at 9 weeks of age chickens were subjected to social strife by moving them daily into new unisexual flocks according to a plan, which precluded contact with previous flock mates. After a period of about 2 weeks, plasma corticosterone levels were measured. From a common population, the high and low tails for plasma corticosterone response became the base populations for high and low response lines. Responses to selection were rapid, and by the sixth generation there was hardly any overlap between the divergently selected high and low lines (Fig. 13.1). There were, however, consequences to this selection as chickens from the low response line were less active, had poorer sensory discrimination, enhanced feed efficiency and heavier body weights than those from the high line. Resources were reallocated as a consequence of their responsiveness to the social strife. When these lines were compared under different environmental settings with physical rather than social stress, each population had advantages and disadvantages (Gross and Siegel, 1985) and such is also the case for other populations (Siegel and Gross, 2007).

Selection for improved growth, feed conversion and conformation traits has resulted in correlated effects on behaviour (Kjaer and Mench, 2003). Fast-growing strains showed a lower activity level than slow-growing broilers (Bizeray *et al.*, 2000;

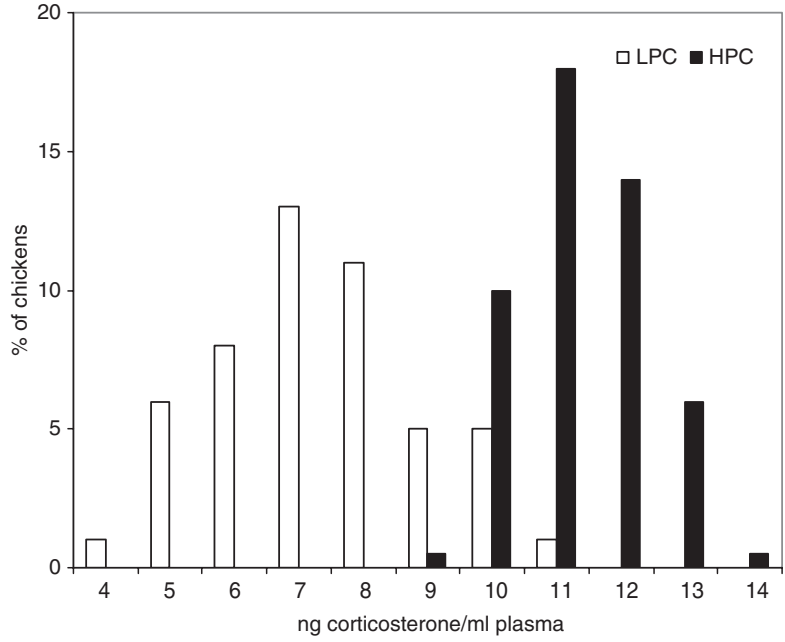

Fig. 13.1. Distribution of plasma corticosterone levels for low plasma corticosterone (LPC) and high plasma corticosterone (HPC) lines in the sixth generation of selection.

Reiter and Kurtritz, 2001), perched, walked and scratched less, performed more sitting on the floor, eating and drinking (Bokkers and Koene, 2003) and performed only limited dust-bathing (Vestergaard and Sanotra, 1999). Broilers perform more behaviours, such as feeding and preening while sitting rather than standing (Sørensen *et al.*, 2000). Reduced movement in broiler chickens may be related to an increased incidence of skeletal problems with age (Kjaer and Mench, 2003). It also has been reported that broiler breeder males lack certain elements of courtship behaviour (Duncan, 2001). Duncan suggested that courtship deficiency and hyper-aggressiveness may be linked genetically to production traits for which breeders have been selecting, or alternatively that 'the breeding companies may have been selecting males who approach females very quickly in the mistaken belief that they are very sexy. In fact, these males are aggressive.' Mating behaviour responds to artificial selection (Siegel, 1979) and aggressive and sexual behaviour may or may not be sequential (Balander *et al.*, 1980).

Väisänen and Jensen (2003) suggest that the adaptability of layers to their social and physical environment may have been influenced by means of selection for increased production capacity, as results suggested that White Leghorns may have greater problems in adapting to a new environment (Väisänen and Jensen, 2003) and have poorer social learning capacity with a weaker ability to cope with group disruptions (Väisänen *et al.*, 2005) than the red junglefowl. Increased egg production has resulted in more aggressive hens and higher social dominance leading to higher levels of feather pecking and cannibalism (Kjaer and Mench, 2003).

3. To Where from Here?

It is evident from our discussion to this point that there are numerous scenarios for consequences of selection in the allocation of resources. We have reported that these may occur in a range of biological settings (e.g. Siegel, 1995; Rauw *et al.*, 1998, 1999; Siegel and Gross, 2007) and conclude this chapter with examples summarized in Fig. 13.2. Classification for adult meat-type and egg-laying stocks show considerably more resources for maintaining the large body size at a cost of resources for reproduction in the former. Contrariwise, the high reproduction has the consequence of smaller body size and thus reduced resources necessary for maintenance. Stage in life cycle, feeding programme and health status are further examples of resource allocations. All reflect consequences of selection in the fowl throughout essentially 10,000 years of domestication. Figure 13.2 shows that the sum of the weights of the parts add up to the total. Increased growth must be supported by sufficient feed intake, which must be supported by a digestive system including the associated glands and organs, which increases metabolism and oxygen demands, which requires a larger respiratory system and a heart and circulatory system with an increased capacity (Emmans and Kyriazakis, 2000). Also needed is a skeletal foundation to withstand the increased weight load.

The poultry sector has reacted to fitness problems by adapting breeding strategies to counteract unfavourable trends (Knap and Wang, 2006). For example, Thorp and Luiting (2000) indicate that the field incidence of skeletal disorders has been diminishing because of successful selection strategies. With implementation of changes in breeding programmes, the incidence of ascites has also declined during the recent past. From the genetic point of view the redistribution of resources to address the 'down side' issues involves greater emphasis in breeding programmes

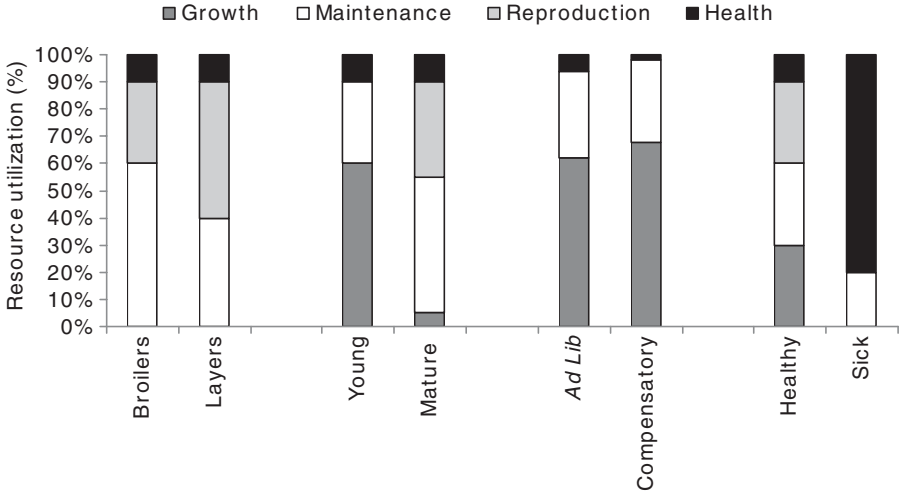

Fig. 13.2. Allocation of resources for growth, maintenance, reproduction and health in mature broilers versus layers, in young versus mature chickens, in broilers fed *ad libitum* versus broilers undergoing compensatory growth, and in healthy versus sick chickens.

on parameters associated with these skeletal and metabolic traits. This increased emphasis automatically redirects the emphasis from associated traits such as body weight and yield in the zero-sum paradigm.

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14 Selection for High Production in Dairy Cattle

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1. Introduction

Increasing productivity has been one of the major goals in dairy cattle breeding. The necessity to increase productivity comes from the increasing cost of labour and land, compared to the major source of income: the price of milk. Labour costs increased 16 times between 1960 and 1995, the price of land increased 15 times; after adjustment for inflation, both increased by a factor of 3 in this time period. However, inflation was higher than the increase in milk price and therefore the adjusted price in 1995 was only half of the price in 1950 (47%; Data LEI, 1998). This example demonstrates that increasing the amount of milk produced per man and per hectare has been of major importance for the economic survival of the dairy farm.

The contribution of genetic improvement to higher productivity has been spectacular. Predictions show that conventional dairy cattle breeding schemes are expected to increase milk yield with 1.5% (Smith, 1985), i.e. around 100 kg milk per lactation. Modern breeding schemes allow even more progress due to the use of multiple ovulation and embryo transfer (Meuwissen, 1991) or genomics selection (Meuwissen *et al.*, 2001). Typical annual increases of around 100–125 kg milk per lactation have been achieved in The Netherlands, the USA and Canada.

Apart from increased genetic merit, phenotypic yield also increases due to improved environmental factors, i.e. better nutrition, housing, health management, etc. In Canada, for example, phenotypic milk yield increased with 155 kg/year, whereas genetic merit increased with 92 kg/year. In the UK (ADC, 1998) milk yield has increased with 3.7% per year in Holstein heifers over the past 5 years and the increase in genetic merit for milk yield was 2% per year. Hence, in both countries more than 50% of the total progress in yield can be attributed to genetic improvement alone. Similar results were obtained in experiments where selection lines with high and low genetic merit animals are kept under the same management circumstances. For example, in the Langhill experiment in Scotland, yields

 Resource Allocation Theory Applied to Farm Animal Production (ed. W.M. Rauw)

increased by 4% per year, and half of this increase could be attributed to the difference between selected and control animals (Simm *et al.*, 1994). Hence, milk yield is increasing by about 3–4% per year and approximately half of this improvement is due to improved genetic selection.

The objective of this chapter is to address possible effects of this increase in milk yield on health and welfare of dairy cows. First, the effects solely due to breeding, i.e. the correlated response of selection, will be reviewed, and second, the interaction with management will be considered. The latter is important because selection practices may force irreducible changes in management, feeding or husbandry, and eventually there may be a mismatch between genetic merit of cows and the capabilities and constraints of dairy farmers to manage these cows. The question is: 'Will average or more extensive dairy farmers still be able to manage dairy cows that are selected predominantly for production?'

2. Effects of Selection for Yield Only

2.1 Health

In comprehensive reviews of genetic parameters for health traits (Pryce *et al.*, 1997b; Rauw *et al.*, 1998) the average genetic correlation between mastitis on milk yield was reported to be 0.38 across 16 studies. More recent studies report values between 0.15 and 0.49 (Pryce *et al.*, 1997a, 1998a; Luttinen and Juga, 1997; Van Dorp *et al.*, 1998; Carlen *et al.*, 2004; Negussie *et al.*, 2008). Sire genetic evaluations for protein yield from the USA were negatively correlated to clinical mastitis in Denmark and Sweden (Rogers *et al.*, 1998). Unfavourable genetic correlations were estimated between 305-d protein yield and clinical mastitis (De Haas *et al.*, 2002; Hansen *et al.*, 2002; Heringstad *et al.*, 2005). In 11 studies, a small unfavourable correlation between yield and somatic cell count was reported (on average 0.14) in first parity (for review see Mrode and Swanson, 1996); however, in later parities the sign of the correlation was less consistent probably due to selection on yield and somatic cell count in first lactation heifers. Also, it can be expected that the incidence of clinical mastitis caused by *Staphylococcus aureus* and *Streptococcus dysgalactiae* will increase, while clinical *Escherichia coli* mastitis seems to be less affected (De Haas *et al.*, 2002).

Recent reviews on genetics of feet and legs (Boelling and Pollott, 1997; McDaniel, 1995; McDaniel, 1997) reported a negative association between 'feet and leg traits' and milk production (Foster *et al.*, 1989; Brotherstone and Hill, 1991a; Short and Lawlor, 1992; Groen *et al.*, 1994). However, 'feet and legs traits' do not have a one-to-one relationship with health of foot and legs. Genetic correlations between production and 'culling for leg problems' ranged from 0.20 to 0.27 (Uribe *et al.*, 1995) and unfavourable correlations were reported between feet and leg disorders and yield (by Lyons *et al.*, 1991; Groen *et al.*, 1994; Pryce *et al.*, 1997a; Pryce *et al.*, 1998a; Van der Waaij *et al.*, 2005).

Recording diseases is less common in most countries. Sometimes data from management information systems or smaller recording schemes are available to estimate genetic parameters, but the relatively small number of estimates does not

give a clear picture. For example, the genetic correlation between yield and ketosis is reported to be 0.17 in the first lactation, but close to zero in the second lactation (Mantysaari *et al.*, 1991), and a value of around 0.77 was reported by others (Uribe *et al.*, 1995; Tveit *et al.*, 1992). Genetic correlations of milk yield with observations on mastitis, ketosis and presence of disease (coded 0 or 1) were >0.5 (Emanuelson, 1988; Simianer *et al.*, 1991; Mrode and Swanson, 1996; Heringstad *et al.*, 2000). Significant correlations of the sire breeding value for milk yield with the incidence of endometritis (0.57), ovarian cysts (0.61), ketosis (0.57), mastitis (0.46) and hoof disorders (0.62) in their daughters were also reported (Klug *et al.*, 1988). Genetic correlations between yield and disease traits (udder oedema, milk fever, retained placenta, metritis, displaced abomasum, ketosis, cystic ovary, mastitis and lameness) were mostly positive (0.02–0.44; Van Dorp *et al.*, 1998). Only 'retained placenta' had a negative genetic correlation with milk yield (−0.28) in this study (Van Dorp *et al.*, 1998). Overall, genetic correlations indicate an unfavourable association between yield and most disease traits. This is supported by results from selection experiments for a range of health traits and overall health costs (Shanks *et al.*, 1977, 1978; Hansen *et al.*, 1990; Dunklee *et al.*, 1994), though it was difficult to find significant effects for individual diseases (Dunklee *et al.*, 1994; Wautlet *et al.*, 1990; Jones *et al.*, 1994; Pryce *et al.*, 1999).

2.2 Fertility

Genetic correlations between yield and fertility are undesirable, indicating poorer fertility with increasing genetic merit for yield. For example, a large number of estimates are available for the genetic correlation between yield and calving interval, days open, days till first service and first service conception (for reviews see Pryce and Veerkamp, 2001; Veerkamp *et al.*, 2003; Rydhmer and Berglund, 2006; Flint, 2006). Estimates with calving interval range from 0.22 to 0.59 (Short *et al.*, 1990; Pryce *et al.*, 1997a, 1998a; Hoekstra *et al.*, 1994; Campos *et al.*, 1994; Grosshans *et al.*, 1997), with days open from 0.16 to 0.64 (Van Arendonk *et al.*, 1989; Bagnato and Oltenacu, 1993; Campos *et al.*, 1994; Grosshans *et al.*, 1997; Seykora and McDaniel, 1983), with days till first service from 0.22 to 0.44 (Van Arendonk *et al.*, 1989; Hoekstra *et al.*, 1994; Grosshans *et al.*, 1997; Pryce *et al.*, 1997a, 1998a) and with first service to conception from −0.62 to 0.05 (Hoekstra *et al.*, 1994; Boichard and Manfredi, 1994; Bagnato and Oltenacu, 1993; Pryce *et al.*, 1997a, 1998a).

These genetic correlations between yield and fertility may be biased upwards, i.e. suggesting a stronger negative effect than caused by actual genetic effects (Philipsson, 1981; Jansen, 1985) because of farmers' inseminations decisions. However, even when progesterone profiles are used to determine days till first heat, negative correlations were found (Darwash *et al.*, 1997; Veerkamp *et al.*, 2000). Negative effects of selection were reported between selection lines, for example, for oestrus not observed, conception at first service, days to first heat, calving interval, days open and days to first service (Kelm *et al.*, 1997; Hageman *et al.*, 1991; Pryce *et al.*, 1998a), the number of days till first oestrus (66 versus 43 days) and number of ovulations before first visual oestrus (1.6 versus 0.7) (Harrison *et al.*, 1990). No significant differences were found between lines for traits measured in heifers except

for the interval from first service to conception, which approached significance (controls superior; Bonczek *et al.*, 1992).

2.3 Longevity

Reviews on longevity in dairy cattle have been published by Essl (1998) and Vollema (1998). Longevity has had a long-standing interest from dairy farmers, fuelled partly by the economic importance of longevity (e.g. Rendel and Robertson, 1950) and partly by concerns about the effects of increasing milk yield (e.g. Miller *et al.*, 1967). Longevity is seen as an overall measure of health and fitness traits.

Estimates of the genetic correlation between yield and longevity are inconsistent with the unfavourable genetic association between yield and health or fertility as discussed above. For example, Strandberg and Shook (1985) reported 30 of 31 quoted estimates of the genetic correlation to be within the range of 0.3–0.9, consistent with values reported since this review (e.g. Short and Lawlor, 1992; VanRaden *et al.*, 1992; Visscher *et al.*, 1994). Hence, the conclusion could be that selection for higher yield would improve longevity. However, Strandberg (1992), Essl (1989) and Dekkers (1993) developed a theoretical model that showed how voluntary culling for low milk yield is likely to cause this positive genetic correlation between yield and longevity. Thus, because of the strong effect of culling for low milk production, the genetic correlation between yield and longevity is a bad indicator for the genetic association between yield and involuntary culling. To approximate an estimate for involuntary culling, longevity is often adjusted for differences in milk yield. Heritability estimates for adjusted longevity traits are low (around 0.05), but there is important genetic variation, because differences between daughter groups may be more than one lactation for expected lifespan (Jairath *et al.*, 1998; Brotherstone *et al.*, 1998).

2.4 Energy resources

It is natural for mammals to be in negative energy balance after parturition; however, the extent to which animals are in negative energy balance differs, and this difference between animals is heritable. Energy balance is defined as the energy intake minus energy requirements for a given yield and maintenance. The concern with energy balance is that the magnitude of the negative energy balance indicates the robustness of cows and their vulnerability/resistance to disease or poor fertility. For example, genetic correlations between fertility and energy balance were reported to range from −0.40 to −0.80 (Veerkamp *et al.*, 2000). There is evidence that selection for yield will increase the magnitude of the negative energy balance (Veerkamp, 1998). This is not surprising, because given that the genetic standard deviation for intake is approximately half of that for milk yield, intake is expected to increase by 0.28 kg/day on average, when genetic merit for milk yield is increased by 1 kg/day. When a high concentrate diet contains about 11.8 MJ metabolizable energy (ME), then this would be sufficient to deliver $0.28 \times 11.8 = 3.3$ MJ milk yield. However, typically, about 5.3 MJ ME is required for 1 kg milk; therefore, the

genetic correlation between intake and milk yield suggests that the correlated response in feed intake from selection on yield alone cannot cover the extra requirements for the increased yield. In recent years there have been several studies confirming this scenario, using indicators for energy balance like dairy form (angularity), live weight change and body condition (Veerkamp, 1998) or using experimental feed intake data (Gordon *et al.*, 1995; Veerkamp *et al.*, 1995, 1997, 2000; Buckley *et al.*, 2000). The extra loss in condition, which is associated with the increased energy deficit in high genetic merit cows, is apparently not compensated when the energy density of the diet is increased (Koenen and Veerkamp, 1997). Veerkamp *et al.* (2003) proposed, on the basis of a review on the effects of selection on hormones and metabolites, that this resource allocation is an important cause of poorer fertility with increased genetic merit for yield.

3. Interaction of Genetics and Management

To address concerns that current selection practices force irreducible changes in management, feeding or husbandry, or that genetic merit of cows outstrips the capabilities of dairy farmers to manage cows, it is important to investigate effects of genotype × environment (G × E) interactions.

3.1 Genetic parameters

Interactions between genotype and environment can be investigated by estimation of variance components to identify one or more of the following G × E effects: heterogeneous variances across environments, the genetic correlation of a trait expressed in different environments being smaller than 1.0 (re-ranking) and heterogeneous genetic correlations between traits across environments. In such analyses, herd characteristics – usually calculated as an average across all animals – are used to reflect the herd environment and the management style of the farmer. In the cases where G × E is reported for health and fertility, large differences in genetic variances were observed across environments: genetic variances for fertility traits increased in some situations more than twofold, and a threefold increase for genetic variances of somatic cell scores was found (Calus *et al.*, 2005). For yield, the variances at most doubled across environments (Calus and Veerkamp, 2003; Kolmodin *et al.*, 2004). Genetic correlations of a trait across environments were as low as 0.65 for survival (Calus *et al.*, 2005), while for somatic cell scores, on a test-day level, the lowest genetic correlation was as low as 0.72 (Calus *et al.*, 2006). Within country, literature values of genetic correlations for health and fertility ranged from 0.74 (Petersson *et al.*, 2005) to unity (e.g. Carlen *et al.*, 2005; Castillo-Juarez *et al.*, 2000; Raffrenato *et al.*, 2003). Genetic correlations between yield traits across environments are, however, all close to unity (e.g. Calus and Veerkamp, 2003; Kolmodin *et al.*, 2004).

Moreover, the underlying genetic correlations between milk production and health and fertility also change over herd environment. The correlation between milk production and 'number of inseminations before pregnancy' in heifers varied

from 0.21 on low-production farms to 0.49 on high-production farms. These varying correlations also lead to a varying response to selection. For example, selection in the average herd environment with regard to production to decrease the number of services to conception with 0.1 services will lead to a decrease in milk production of 35 kg per lactation in a low-production environment, but to a decrease of 178 kg in a high-production environment. The consequence is that measures taken to improve health and fertility in management decisions can have unexpected consequences if genetics is not taken into account (Windig *et al.*, 2006).

3.2 Experiments

The effects of feeding system, genotype and genotype by feeding system interactions on a range of health and fertility traits were investigated in Holstein Friesian cows at the Langhill Dairy Cattle Research Centre. Feeding system had a significant effect on milk fever, days to first service and days to first heat, all in favour of the forage-based system (Pryce *et al.*, 1999). Differences between genetic lines were significant for fertility traits, but not for health traits (with the exception of mastitis). There were no genetic lines by feeding system interactions, indicating that the observed line differences applied to both dietary treatments (Pryce *et al.*, 1999). If high genetic merit animals are given the low concentrate system, no differences are expected for fertility traits compared with low genetic merit animals fed the high concentrate diet.

A few studies have investigated the combined effect of feeding system and genotype on feed intake, feed efficiency and body tissue mobilization. Richardson *et al.* (1971) performed an experiment with 228 Jerseys heifers sired by 13 different bulls and found a significant interaction between ration and sire for gross efficiency, measured over the whole lactation period. Lamb *et al.* (1977) found evidence for a sire by ration interaction for milk energy production, gross efficiency over 305 days and fat yield. In their analysis, the estimate of the variance components for interaction of sire with ration ranged in relative magnitude from 15% to 99% of the sire variance component. However, most of the interaction was accounted for by a single bull from New Zealand. Wang *et al.* (1992) reported a breed by concentrate feeding interaction for milk production (56 and 112 days milk) and feed efficiency measured in Ayrshire and Holstein cows. These authors also observed a re-ranking of sires on different concentrate feeding levels. However, in all of these studies animals were fed concentrates according to milk production, which makes biological interpretation of the results difficult.

Several studies have been performed where animals were not fed according to production (Korver, 1982; Oldenbroek, 1988; Veerkamp *et al.*, 1994, 1995; Dillon *et al.*, 2006). Korver (1982) compared Dutch Friesians and crosses between Holstein Friesians and Dutch Friesians for several feed intake, milk production and liveweight traits and did not observe a significant feeding system–breed interaction. In that experiment two fixed amounts of concentrates were fed and cows had *ad libitum* access to roughage. Oldenbroek (1988) reported breed by diet interactions for intake (over 39 weeks of lactation) and production characteristics for a group of Jersey heifers compared with groups of Holstein Friesian, Dutch Friesian and Dutch Red and White heifers.

There was some evidence that selected cows were able to maintain a higher milk yield on forage-based diets primarily because of body tissue mobilization and a more negative energy balance (Veerkamp *et al.*, 1995). High genetic merit animals also stayed for a longer period in negative energy balance on a forage-based diet (Veerkamp and Emmans, 1995), but this effect was not more than was expected on the basis of the diet and genetic effect together, i.e. there was no interaction. Although significant feeding effects and genetic line effects were observed for plasma metabolites and (responses in) hormone concentrations, interactions between the two were not significant (Xing *et al.*, 1991).

Beerda *et al.* (2007) assessed the effects of G × E on milk yield and energy and protein balances in a study with 100 heifers with high or low genetic merit for milk yield that were milked two or three times a day that received rations of low or high caloric density. This 2 × 2 × 2 factorial arrangement modelled different management strategies and showed the effects of genotype, environment and G × E interactions on energy balances. The different groups varied considerably in milk production levels, which ranged from 21.8 to 35.2 kg (mean daily energy-corrected milk (ECM) production in the first 100 days in milking) with the experimental factors influencing milk production in the direction as was expected. Only in the groups that received high caloric density rations was milk production significantly increased in high genetic merit compared with low genetic merit cows (mean difference of 3.5 kg of ECM/day), as was the case for three times milking compared with two times milking (mean difference of 4.1 kg of ECM/day). Interestingly, high genetic merit cows, like low genetic merit cows, reduced yield in response to low nutrient supply. The G × E interactions are in line with the notion that high genetic merit cows have increased tissue mobilization, dry matter intake, and, as a result, milk production, especially under conditions that promote high yield (Hoogendoorn *et al.*, 1990; Veerkamp *et al.*, 1995). Post-partum body condition scores were significantly lower in high genetic merit cows than in low genetic merit cows and high genetic merit cows had higher fat content in milk than low genetic merit cows, with the latter being restricted to the groups that received low caloric density rations: an G × E interaction that seems to reflect the high genetic merit animals' trait to mobilize fat reserves for fat production even though conditions (read: diets) are suboptimal.

High genetic merit for milk yield seems intrinsically connected to increased risks for negative energy balance-related disorders, but in this study the high genetic merit cows did adapt milk output when energy intake was suboptimal and management factors, such as feed caloric density and milking frequency, had the stronger impact on calculated energy and protein balances. Commencement of luteal activity was generally later with a more negative protein balance for high genetic merit animals, but not for low genetic merit animals (Windig *et al.*, 2008). Some high genetic merit animals, however, had still an early commencement of luteal activity despite a negative protein balance, but these animals showed prolonged progesterone cycles, indicative of other fertility problems. In the same experiment, a high genetic merit for milk yield was related to higher somatic cell scores. The effects of feeding and milking frequency on udder health were similar for animals with low and high genetic merit for milk production, although high genetic merit animals seemed slightly more sensitive to low energy supply (Ouweltjes *et al.*, 2007).

Breed × diet interactions for the interval from calving to first oestrus were observed, as differences in the interval between low and high diets were more pronounced for the Holsteins than for Herefords. Because diet influenced reproduction more in Holstein Friesian than in Hereford cows, the use of dairy breeds in beef production may necessitate diets higher in energy to achieve maximum reproductive performance (Hansen *et al.*, 1982).

4. Discussion

Overall, there is clear evidence that there are negative genetic associations between milk yield and most other traits. An important question to discuss is 'What are the practical implications of this genetic association?', but first we will discuss possible reasons for this overall negative association.

4.1 Why is there a negative association with milk yield?

Different mechanisms may underlie the clear negative genetic correlation between yield and health or fertility, e.g. pleiotropic gene effects, linkage or complex physiological associations. It would be nice to understand why there is a negative genetic association between milk yield and all the other traits. A very popular line of thought has been that milk yield is simply getting too high, and that cows cannot cope with such a high milk yield. However, there is evidence to suggest that this is not the right explanation. For example, if the height of the milk yield was an important factor, then the expectation is that phenotypic correlations are even stronger than genetic correlations, i.e. the absolute milk yield (before disease event) becomes very important. Phenotypic correlations have not been reviewed here, but these are generally small. For example, phenotypic correlations between yield and many different health traits did not differ substantially from zero (Pryce *et al.*, 1997b, 1998a; Van Dorp *et al.*, 1998). A slightly higher phenotypic correlation is observed with fertility traits, but these correlations are still below 0.2 (Pryce *et al.*, 1997a, 1998a). This suggests that phenotypic yield of a cow is poorly associated with health.

It is also important to note that the phenotypic association between milk production and fertility/health varies from herd to herd (Windig *et al.*, 2005). These authors found that, within farms with a high average production per cow, the highest producing cows had the poorest udder health and lowest fertility. Within farms with a low average production, the difference in health and fertility between high and low producing cows was small. Interestingly, relationships across farms were different: in farms with a low average production, fertility and health were on average poorer than in farms with a high average production. Apparently, management in high producing farms is such that production increases simultaneously with improved health and fertility. One explanation is that high milk yield is starting to be an issue in high, but not in low, producing herds. However, others found that the strength of negative associations between

yield and fertility in high-production herds is equal to or lower than that in low-production herds (Castillo-Juarez *et al.*, 2000; Kearney *et al.*, 2004; Oltenacu and Algers, 2005).

Also, in our G × E experiment we found that signs for health risks, i.e. severe negative energy balances, protein balances and measures of udder health and fertility, were not directly associated with a high milk production itself (Beerda *et al.*, 2007; Ouweltjes *et al.*, 2007; Windig *et al.*, 2008). For example, feeding had a large effect on milk yield, but hardly affected udder health. Achieving a higher production through increased milking frequency did impair teat condition, but also resulted in lower somatic cell scores, whereas a high genetic merit for milk yield was related to higher somatic cell scores. Fertility problems were not restricted to the cows having the highest milk production. Thus, whereas high milk yield per se will increase allostatic load, this does not necessarily compromise the health or fertility status of relatively young cows. All these results together support the suggestion that the unfavourable associations with yield are not necessarily the consequence of the increase in yield per se (Gutierrez *et al.*, 2006; Weigel, 2006).

Another explanation for the negative association with yield is that highly selected animals lose homeostatic balance (Rauw *et al.*, 1998), based on the ideas of the resources allocation theory (Beilharz *et al.*, 1993). As presented previously, there are strong indications that genetic selection affects energy partitioning in lactating dairy cows, and causes a genetically induced negative energy balance and a lower body condition score (Veerkamp and Brotherstone, 1997; Veerkamp *et al.*, 2003; Gutierrez *et al.*, 2006; Friggens *et al.*, 2007). Also, after adjusting fertility for yield, there is still a correlation of 0.5 with both feed intake and energy balance (Veerkamp *et al.*, 2000). Calculations based on genetic parameters show that when including feed intake with a positive weight in the breeding goal, i.e. a larger proportion of the extra yield from selection comes from intake, energy balance is not worsened during selection for yield (Veerkamp and Koenen, 1999). Using feed intake and energy balance measures can also help to reduce the negative effects on fertility while still 75% of the gain in yield is maintained (Veerkamp *et al.*, 2000). Hence, there are clear indications that energy availability and partitioning play an important role in explaining the negative associations with yield, and in many countries research has commenced to use body condition score as a predictor of energy balance in breeding programmes (Koenen *et al.*, 2001; Pryce *et al.*, 2001; Berry *et al.*, 2002; Lassen *et al.*, 2003; Banos *et al.*, 2004, 2006; Dechow *et al.*, 2004a,b; Kadarmideen, 2004; Mao *et al.*, 2004; Pryce and Harris, 2006; Dal Zotto *et al.*, 2007; De Haas *et al.*, 2007; Friggens *et al.*, 2007). Still it is too simple to assume that the only explanation is that energy resources have become limiting. For example, if energy availability is the limiting factor for fertility in high producing cows, then it is expected that on a low caloric diet, fertility is dramatically reduced. However, cows reduce their milk yield instead. Similarly, feeding extra concentrate does often not improve fertility or health; cows increase their milk yield instead. An alternative way of reasoning is that animals that are genetically able to switch off fertility or health are selected, in order to have more energy available for a higher milk yield. In this case, no G × E effects are

expected, and it is unlikely that a change in management (e.g. feeding) can compensate for the genetically induced reduction in fertility.

4.2 Practical implications of the genetic associations with yield

All these negative associations between yield and other traits may look quite dramatic. However, when put in perspective it is good to realize that the magnitude of the effects is relatively small. For example, the incidence of mastitis and lameness is expected to increase with 4% and 2% with single-trait selection for milk yield in 10 years (Pryce *et al.*, 1998a): rather than 12 out of 100 cows, 16 out of 100 cows will get mastitis in 10 years' time. Because of the impact of the genetic correlation between yield and fertility, with single-trait selection for yield and an expected yearly increase of genetic merit of 132 kg milk per year, calving interval is expected to increase between 5 and 10 days in 10 years of selection for yield (Short *et al.*, 1990; Pryce *et al.*, 1998b), assuming that there are no changes in management, and single-trait selection for yield is practised.

In addition, 'multi-trait' selection may overcome the adverse effects of genetic selection for increased yield. For example, when the economic consequences of increased mastitis and somatic cell scores were included in the selection criteria, the economic gain improved with nearly 1%, and increased somatic cell scores and mastitis due to selection were reduced by 40–60% (Colleau *et al.*, 1995). When more emphasis is given to mastitis it is possible to constrain the genetic trend for mastitis to zero. The relative weight to reduce somatic cell scores was twice the weight used on increasing yield (De Haas *et al.*, 2002). However, the reduction in genetic progress for yield ranges between −5% and −36%, depending on what measures are available to predict mastitis. Suggestions for measures to predict mastitis are given by, e.g. Detilleux (2002) at animal, cellular/hormonal and DNA levels. By making better use of the information on variation and fluctuations in somatic cell counts, it is possible to predict mastitis genetically more accurately (De Haas *et al.*, 2008). When incidence of mastitis, lameness and calving interval measures are available on 100 daughters of a bull, it is possible to constrain the genetic trend for these traits to zero, while the reduction in genetic trend for yield is only 11% (Pryce *et al.*, 1998b). Hence, it is possible with multi-trait selection to improve yield genetically without adversely affecting health, welfare or fertility. This is because the genetic correlation with yield is often below 0.5, and hence natural genetic variation ensures that there are animals that have a high yield without compromising health, fertility or welfare.

In several countries, genetic trends show hardly any negative trend (except for fertility). This may have been due to multi-trait selection. In most dairy cattle breeding schemes there is attention for other traits than milk production, in particular conformation traits. Initially, these were the *descriptive* traits that were too difficult to handle to genetically select for, but in the 1980s linear-type traits have been introduced that represent a biological scale. Linear-type traits have proven to be useful, as some of the linear-type traits are associated with mastitis (e.g. Thomas *et al.*, 1984; Seykora and McDaniel, 1986; Monardes *et al.*, 1990; Rogers *et al.*, 1991, 1998; Van Dorp *et al.*, 1998; Rupp and Boichard, 1999), reproductive or

calving performance (e.g. Dadati *et al.*, 1986; Cue *et al.*, 1990; Shapiro and Swanson, 1991), longevity (Rogers *et al.*, 1989; Brotherstone and Hill, 1991a,b; Boldman *et al.*, 1992; Short and Lawlor, 1992), energy balance (Veerkamp and Brotherstone, 1997), and locomotion or lameness (Boelling and Pollott, 1997). Selection for some linear-type traits may have reduced the negative effects of selection for a higher yield, in particular for udder health. At the same time, selection for some type traits may have had negative effects for health and welfare. For example, selection for dairy form (angularity) has inflated the negative effects on energy balance, and selection for size has often been reported to result in negative effects on longevity.

Although the moderate responses found in practice (compared with sudden effects from management failure) may seem a relief for animal breeders, at the same time it is the biggest risk factor. Over many years, these effects accumulate, and slowly increase the pressure on management. Trends in management, like cost reduction, increasing herd size, shortages of (skilled) labour or reduced investments due to high pressure on cost control, may suddenly reveal the negative genetic trend since the late 1980s. At that moment in time, cross-breeding and other breeds become more popular in practice, and a drastic revision of the breeding goal is required (Veerkamp and Beerda, 2007).

5. Conclusion

Overall, the findings indicate that mismanagement is a more potent risk factor for animal health than genetic merit for milk yield, although there is reason to assume that one-sided selection for high milk yield makes cows more vulnerable to poor fertility and disease. The biggest concern is fertility, and both poor genes for fertility and reallocation of resources may play an important role. The risk of one-sided selection has been addressed by the dairy cattle breeders by contemporary multiple trait selection indices (Miglior *et al.*, 2005). However, attention for functional traits in breeding may be of growing importance, because drastic changes in management may cause a mismatch between genetics and management in the future, even when small negative effects on health and fertility are compensated for. This makes the efforts to breed more robust cattle even more important.

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15 Consequences of Biological Engineering for Resource Allocation and Welfare

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1. Introduction

One of the major aspects of the functioning of all animals, including humans, is that they have to attempt to cope with a wide range of actual and potential adversities (Lazarus and Folkman, 1984; Broom, 2001a). In order to do this they have an array of coping systems with components including organ physiology, cellular mechanisms such as the immune system, brain function and behaviour (Broom and Johnson, 2000). Some of the brain mechanisms involve the cognitive and emotional components of positive and negative feelings. Feelings, such as pain, fear and the various forms of pleasure, are important parts of coping systems. Hence, they are generally adaptive and, like other biological mechanisms, they have evolved by natural selection (Broom, 1998). The extent to which the various mechanisms helping individuals to adapt to their environment (Broom, 2006) are successful and the degree to which the coping is easy or difficult has a major effect on the welfare of the individual (Broom and Fraser, 2007). The welfare of an individual is its state as regards its attempts to cope with its environment (Broom, 1986). Welfare ranges from very good, when needs are satisfied (Hughes and Duncan, 1988a,b; Dawkins, 1990; Toates and Jensen, 1991) and there are usually positive feelings, to very poor when some needs are not met and there are indicators of harms or coping difficulty or suffering. A question considered in this chapter is how the welfare of animals might be affected by biological, largely genetic, engineering.

One possible reason why welfare could be poorer in some animals changed by one of the forms of genetic engineering is that the change in the animal involves more utilization of resources for one part of its functioning and, since resources are limited, this results in less resource availability for other functioning. The possible links between resource availability, genetic change and welfare is a specific question considered here.

In the following sections on genetic engineering and its effects on welfare, some of the changes involve impacts on the possible limits of resource availability,

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while others do not. The general issue of how such limits may have important effects is therefore considered in more detail after the various examples have been presented.

2. Conventional Breeding and Welfare

Conventional breeding methods need not affect welfare, but they can sometimes change animals in such a way that they have more difficulty in coping or are more likely to fail to cope (Broom, 1994, 1995, 2001b). Examples of such effects are the sensory, neurological or orthopaedic defects found commonly in certain breeds of dog. Others are the effects of the genes promoting obesity in mice, double muscling linked to parturition problems in cattle and many examples of selection promoting fast growth and large muscles in farm animals. Modern strains of pigs have relatively larger muscle blocks, more anaerobic fibres and smaller hearts than have the ancestral strains (Dämmrich, 1987). They are more likely to die or to become distressed during any activity. Modern broiler strains grow to a weight of 2–2.5 kg in 35 days as compared with 12 weeks in the late 1970s. Their muscles and guts grow very fast, but the skeleton and cardiovascular system do not. Hence, many of the birds have leg problems, such as tibial dyschondroplasia or femoral head necrosis, or cardiovascular malfunction, such as that which gives rise to ascites.

It is clear that for meat-producing animals that are growing too fast for their legs and heart, the welfare is becoming poorer and poorer because of this genetic selection and the continuation of this trend is morally wrong. The competitive nature of the industry makes it difficult for individual producers to take action to reverse the trend. There is pressure on those concerned with genetic engineering to make such animals grow even faster.

An example of conventional breeding leading to a substantial change in production in a farm animal, with consequential risks of poor welfare for the animals, is the dairy cow. The average energy corrected milk yield for Swedish dairy cows increased from 4200 to 9000 kg between 1957 and 2003 (see Pryce and Veerkamp, 2001). On many farms the average production per cow is over 10,000 kg of milk and individual cows may produce twice as much. The beef cattle average is 1000–2000 kg (Webster, 1993). The dairy animal is producing considerably more than its ancestor would have. This raises questions of whether it is at or beyond its maximum production level and the extent of any consequent welfare problems. The peak daily energy output of the dairy cow per unit body weight is not very high in comparison with some other species such as seals or dogs, but the product of daily energy output and duration of lactation is very high indeed. Hence, long-term problems are the most likely to occur (Nielsen, 1998). This is what we see because, although some cows seem to be able to produce at high levels without welfare problems, the risk of poor welfare indicated by lameness, mastitis or fertility problems is greater as milk yield increases.

Data from National Milk Records in the UK show an increase in average yields of dairy cows of about 200 kg/year from 1996 to 2002 and 50% of the increase in milk yield is attributed to genetics (Pryce and Veerkamp, 2001). The situation is similar in the USA where, between 1993 and 2002, the average milk

production per cow increased by 1287 kg, and 708 kg of this increase, or 55%, was due to genetics. This increase in dairy cow productivity has been associated with increases over the expected levels resulting from veterinary progress, in leg and foot problems, mastitis, reproductive problems and metabolic disorders (Broom, 2004).

For a review of lameness, including the extent to which it is a welfare problem, see Greenough and Weaver (1996). Almost all animals that walk with a limp, reduce walking to a low level or avoid walking whenever possible suffer from some leg or foot pain. Their ability to carry out various preferred behaviours is generally impaired and there may be adverse consequences for other aspects of their normal biological functioning. Lameness always means some degree of poor welfare and sometimes means that welfare is very poor indeed. Measurements of the extent to which some degree of lameness occurs in dairy cows include 35–56 cases per 100 cows per annum in the USA, 59.5 cases per 100 cows per annum in the UK and more than 83% of examined cows in The Netherlands. The actual figures depend upon the method of assessment and most of these cases were not treated by veterinary surgeons, but there is no doubt that lameness is often a severe welfare problem.

Mastitis in mammals is a very painful condition. The sensitivity to touch of affected tissues is clearly evident and there is obvious damaging of normal function. Mastitis prevalence in dairy cows should have declined greatly with improved methods of prevention and treatment, but it has not declined as much as it should have done. Webster (1993) reports 40 cases of mastitis per 100 cows per year as an average for the UK.

The steady increase in reproductive problems of dairy cows as milk yields have increased is well known. As Studer (1998) states, 'despite programmes developed by veterinarians to improve reproductive herd health, conception rates have in general declined from 55–66% 20 years ago to 45–50% recently (Spalding *et al.*, 1975; Foote, 1978; Ferguson, 1988; Butler and Smith, 1989)'. Reproductive problems in dairy cows result in large numbers of cows being culled because of failure to get in calf. In a study of 50 dairy herds in England, Esslemont and Kossaibati (1997) found that farmers reported failure to conceive as the predominant reason for culling with 44% of first lactation, 42% of second lactation and 36.5% of cows in total being culled for this reason. However, mastitis, feet and leg problems, ketosis and other disease conditions can lead to reproductive problems and it is difficult to discover their initial cause from farmers' records. A report by Plaizier *et al.* (1998) concerning Canadian herds indicated that reproductive culling risk varied between 0% and 30% with a mean of 7.5%. Studies showing that milk yield is positively correlated with the extent of fertility problems have come from a range of different countries (van Arendonk *et al.*, 1989; Oltenacu *et al.*, 1991; Nebel and McGilliard, 1993; Hoekstra *et al.*, 1994; Pösö and Mäntysaari, 1996; Pryce *et al.*, 1997, 1998). Studer (1998) explains that high-producing cows that are thin and whose body condition score declines by 0.5–1.0 during lactation often experience anoestrus. A loss of condition score of about 1.0 during lactation was normal in the review presented by Broster and Broster (1998). Many published studies (Oltenacu *et al.*, 1991; Dematawewa and Berger, 1998; Royal *et al.*, 2000; Pryce and Veerkamp, 2001; Roxstrom, 2001; Veerkamp

Table 15.1. Positive correlations between milk production level in England and indicators of poor welfare. (From Pryce *et al.*, 1997.)

Milk yield from 33,732 lactation records	
Calving interval	0.50 ± 0.06
Days to first service	0.43 ± 0.08
Mastitis	0.21 ± 0.06
Foot problems	0.29 ± 0.11
Milk fever	0.19 ± 0.06

Table 15.2. Positive correlations between milk production level in Scotland and indicators of poor welfare. (From Pryce *et al.*, 1998.)

Milk yield from 10,569 lactation records	
Calving interval	0.28 ± 0.06
Days to first service	0.41 ± 0.06
Mastitis	0.29 ± 0.05
Somatic cell count	0.16 ± 0.04
Foot problems	0.13 ± 0.06

et al., 2003) show negative correlations between milk yield and fertility measures, indicating that the decline in fertility observed on dairy farms is, at least in part, an unwanted consequence of successful selection for higher yields. Data on the relationships between milk yield and production measures from two large-scale studies are presented in Tables 15.1 and 15.2. The decline in fertility, reflected in increased calving interval and in longevity, measured by proportion of cows alive at 48 months of age in Holstein cows in the North-eastern USA from 1957 to 2002, are shown in Fig. 15.1.

The review by Ingvartsen *et al.* (2003) examined the relationship between milk production and production-related diseases as defined by Kelton *et al.* (1998): dystocia, parturient paresis, ketosis, displaced abomasum, retained placenta, ovarian cyst, metritis, mastitis and lameness. The review of 11 epidemiological

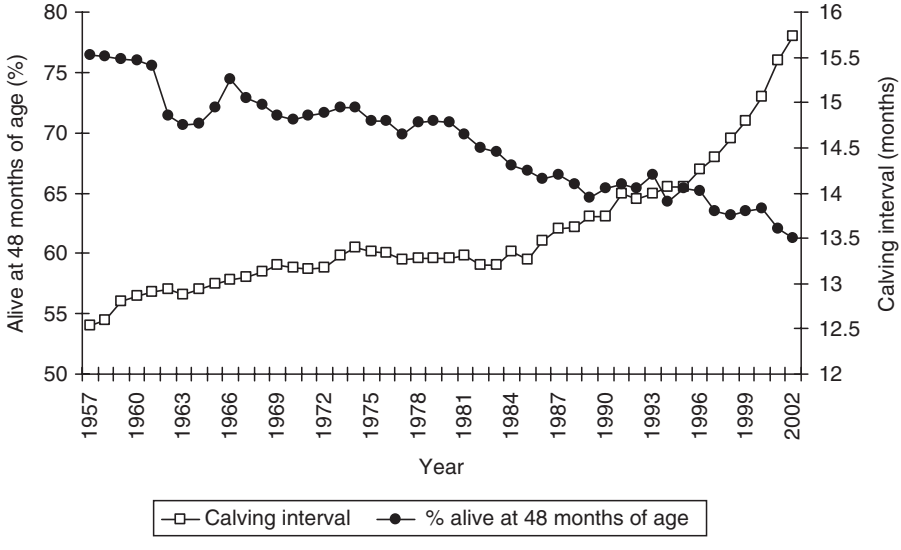

Fig. 15.1. Average calving interval and proportion of cows alive at 48 months of age between 1957 and 2002 for Holstein cows in the North-eastern USA. (After Oltenacu and Algers, 2005.)

studies showed clear evidence that cows with high yield in the previous lactation are at increased risk of mastitis and ovarian cysts in the subsequent lactation, but for other diseases the phenotypic association was weak because of the large variability between studies. It was concluded that cows producing more milk are also likely to eat more and make greater use of their body reserves in early lactation (Veerkamp, 1998).

3. Embryo Transfer

There are two areas for investigation in relation to embryo transfer. The first is the immediate effects of the procedures themselves and the second is the effects during pregnancy, at parturition and soon afterwards.

The collection of eggs and the insertion of eggs into another female animal can be carried out without the necessity for surgery in a large animal like a cow. The procedure in cattle is mainly carried out by superovulation and non-surgical recovery and involves the transfer of embryos, which may have been fertilized *in vivo* or *in vitro*. Ovaries may also be collected from dead animals in the abattoir and the ova grown-on and fertilized in the laboratory before transfer. These embryos may be transferred directly or frozen for storage and future use. The procedure for transferring single embryos to carefully selected recipients does not normally cause welfare problems. The continued use of superovulatory drugs can result in subsequent fertility problems. However, in animals of the size of sheep, pigs or smaller, an incision must be made in the abdominal cavity to carry out the procedures. This will always cause a greater degree of poor welfare in the animals than would occur in cattle. The effects of these procedures in all mammals can be monitored in the same ways as those described for transgenic animals.

In cattle, embryo transfer is carried out at 7 days after the onset of oestrus, so the technique is more difficult than artificial insemination and requires considerable training and experience. Caution must be exercised if this practice is to become widely available in the commercial field, as embryos fertilized *in vitro* have been implicated in the production of oversize calves. The technique must be carried out using epidural anaesthesia. When an egg is inserted into a female mammal that results in the growth of a fetus, which is larger or a different shape from the fetus that the mother would produce after mating with a male of similar type, problems may occur during pregnancy and at parturition. Some problems during pregnancy and most problems at parturition result in poor welfare of the mother, the young animal or both.

4. Xenotransplantation

Xenotransplantation involves the transfer of tissues or organs from one kind of animal to another. In some cases (i) the material transferred is not cellular, for example, the placing of a pig heart valve into a human. In this case, the valve has no

blood or other cells with it and can be cleaned so that no rejection of foreign proteins by the cells of the recipient animal will occur. In other cases (ii) whole organs may be transferred, so measures to prevent immunological rejection processes occurring are necessary. The example for case (i) is now frequent and involves the killing of the donor animal. Since this can be done in a humane way, no welfare problem is likely. The ethical issue requires consideration but is similar to that when the ethics of eating animals is considered. Case (ii) is much more risky and difficult. There could be substantial animal welfare problems associated with the immuno-modification of the donor, but donor animals would normally be kept in very good conditions. At present, there is substantial public resistance to the use of such xenotransplantation to humans because of the risk that new viral or other diseases may be passed to people.

5. Cloning

Cloning of vertebrates, i.e. the production of genetically identical animals by nuclear transfer, has been carried out since 1952 with frogs and is now used in the farming of fish for food (Gurdon, 1974). It was not until 1986 that the first cloned mammals (mice) were produced by transferring nuclear material from embryonic cells, and this was rapidly followed by successful nuclear transfer in sheep and cattle (Gurdon and Byrne, 2002). The techniques involved in the first production of clones in sheep by nuclear transfer required that oocytes were recovered by laparotomy from donor ewes, ewes were superovulated by the administration of hormone by injection and by insertion of a vaginal tampon and the oocyte DNA was removed by microsuction (Wilmut *et al.*, 1997). Oocyte donor ewes were used for only one surgical donation. Other sources of cells were fetuses taken post-mortem from ewes and mature cells grown in culture. Following nuclear transfer, the oocytes were cultured *in vivo* in the ligated oviduct of a live sheep for a period of 7 days when the ewe was humanely killed and two or three developed blastocysts transferred to synchronized recipient sheep by laparotomy. These were then allowed to develop to full term to be delivered. The success rates of these various methods have not been high.

There may be poor welfare associated with cloning for various reasons including: the procedures described above, adverse effects on the mothers carrying the cloned young, the production of extra-large offspring, reduced life expectancy of the cloned animals and the possibility of adverse effects on the cloned animals unless they receive extra care.

'Large offspring syndrome' or 'fetal oversize' is a phenomenon found occasionally in calves and lambs that are born following embryo manipulations. There may be increased incidence of developmental abnormality in cloned animals. The ability of stockpeople to meet the particular needs of animals produced by cloning is an important issue. These needs may be associated simply with the greater performance achieved by the animals, and the special care associated with such high performance. Alternatively, abnormalities may be generated through cloning, which go unrecognized initially yet may be stressful for the animals.

However, such adverse effects of cloning techniques may be counterbalanced by a reduction in the number of animals used in research, for at present, some of this research relies on more random genetic modification techniques.

6. The Welfare of Transgenic Animals

Transgenesis can result in: (i) better welfare; (ii) no change from the average for unmodified animals; or (iii) poorer welfare:

1. Some genetic manipulations can be beneficial to the modified animals. If genes conferring disease resistance are inserted into the genome of an individual, for example, by making it possible for the modified animal to produce antibodies to bacterial toxins (Clark, 2001) or conferring avian leucosis virus resistance, then the welfare of the modified individual is better than that of the unmodified individual. If the animal can cope with disease challenge better, its welfare is slightly improved for most of the time and very much improved in the circumstance where disease challenge occurs.

2. When the transgenic animal is modified so that it can produce a novel protein in its blood or milk, there may be no effect at all on its welfare. No evidence of adverse effects on the behaviour of transgenic sheep was found (Hughes *et al.*, 1996). However, there could be some other adverse effect and the predictability of that effect will vary according to the precision of the transgenesis procedure. Gene transfer by introducing embryonic stem cells into a blastocyst are more predictable in their effects than the introduction of genetic material by microinjection.

3. The production of disease-susceptible animals by transgenesis, so that the animals can be used in medical research, will result in poorer welfare whenever the gene is expressed. The extent of the poor welfare will differ considerably according to the level of expression and the disease state. If the animals produced as a result of transgenesis were modified in a way that increased their growth rate, the growth of a particular organ, or differential growth in such a way that an already productive genetic strain was made even more productive, there is a serious risk that the welfare of the animals would be worse as a direct consequence of the manipulation. Those carrying out such work should consider whether the animals are already close to some biological limit to adaptability before proceeding. When Pursel *et al.* (1989) produced transgenic pigs with the human growth hormone gene added, the resulting animals had major joint and other limb disorders as they grew and so the study could not be continued.

Genetic manipulation could affect sensory functioning, the structure of bones or muscles, hormone production, detoxification ability, neural functioning, etc. In one line of transgenic mice, the production of oxytocin was altered (Crawley, 1999). The question which must be considered is not whether or not there is a change, but whether there is a change that affects the animal's welfare. In some cases, any effects of the genetic modification on the welfare of other individuals must be considered.

In a study of the effects on welfare of transgenesis or treatment with biotechnology products, control animals which have not been modified or treated

should also be used. A wide range of welfare measures are necessary because the actual effects on the individual will seldom be known and also because species and individuals vary, both in the methods that they use to try to cope with adversity and in the measurable signs of failure to cope. A simple welfare indicator could show that welfare is poor, but absence of an effect on one indicator of poor welfare does not mean that the welfare is good. For example, if the major effect of a manipulation was a behavioural abnormality or an increase in disease susceptibility but only growth rate was measured, a spurious result could be obtained. The choice of measurements should include the main methods of assessing poor welfare (Broom and Johnson, 2000; Broom and Fraser, 2007), but often it will be obvious from a preliminary study of morphology, or a clinical examination, which measurements of function or of pathology will be most relevant.

The effects of genetic manipulation or treatment with biotechnology products may not be apparent at all stages of life, so the animal must be studied at different stages including the oldest age likely to be reached during usage. Some effects may be evident in the second generation but not in the first, so modified animals should be studied for two generations.

7. The Welfare of Animals Treated with Biotechnology Products

Biotechnology products could be identical with naturally occurring chemicals such as hormones. However, since they are often produced by bacteria they may not be identical. For example, most of the commercially available recombinant bovine somatotrophin (BST) differs slightly from the natural BST. Some biotechnology products may be completely different from any chemical normally found in the species. In addition to this possible difference, the quantities of the products, which can be given to animals are often much greater than normal physiological levels. As a consequence of these important possibilities for difference, the effects of biotechnology products on welfare should be assessed in the same way as the effects of transgenesis and should be subject to the same legislative controls. Somatotrophins have effects on tissue growth, hence the name 'trophin', which refers to growth, not 'tropin', which refers to direction of movement.

Work on the effects of recombinant bovine (BST) and porcine somatotrophin (PST) injections has also been directed almost entirely towards finding out how to improve productivity in dairy cows and pigs. Any results, which indicate what the effects on the welfare of the animals might be, have been derived largely as an incidental by-product of the main study.

Since BST occurs naturally, low levels are unlikely to have any adverse effects on welfare, but even at low levels the effects need to be checked because of any differences in amino acid sequence from the natural form. BST injection results in increases in the amount of insulin-like growth factor-1 (IGF-1) in the blood and in milk (Prosser and Mepham, 1989; Prosser *et al.*, 1989, 1991). These increases can be substantial and it has been shown that high levels of IGF-1 can affect rat bone growth (Juskevich and Guyer, 1990). Low levels of IGF-1 are likely to have no

adverse effect, but it is a potent mitogen and it is not known what effects high levels of it have on the cow, or on the calf that consumes the milk, or indeed on people who do so (Mepham, 1991).

The most clearly documented side effects of BST and PST are on disease incidence and reproduction (Broom, 1993; Simonsen, 1993; Willeberg, 1993). The effects of BST injection are similar to changes that occur during the rising phase of lactation; high-yielding cows, which are not treated with BST are particularly susceptible to disease at this time. Kronfeld (1988) states that high levels of BST result in subclinical hypermetabolic ketosis, which can lead to reduced reproductive efficiency and a higher incidence of mastitis and other production-related diseases. However, studies reviewed by Phipps (1989) provide no evidence for increased incidence of ketosis following BST treatment. Several of the studies of cows treated with BST, so that milk yields are particularly high, report that the incidence of mastitis can increase, while others do not. There are also some reports of increased incidence of lameness (Phipps, 1989; Craven, 1991). High production levels are associated with greater incidence of both mastitis and lameness (Broom, 1994), and BST use can result in high production levels, so effects will depend upon how great were the maximal production levels using BST. Increases in disease following BST use may be directly related to the metabolism associated with high production levels, but welfare is obviously poorer if mastitis and lameness occur, whatever the exact reason for it. Meta-analyses of studies of BST effects and studies using large data sets showed substantial increases in both mastitis and lameness (Willeberg, 1997). The increase in the risk of clinical mastitis above the risk in non-treated cows in five studies was 15–45%, 23%, 25%, 42% and 79%. In studies of foot disorders, a large-scale study with multiparous cows showed 2.2 times more cows affected and 2.1 times more days affected in BST-treated cows than in cows not treated with BST (EU Scientific Committee on Animal Health and Animal Welfare, 1999).

Surveys of the results of several studies of BST-treated animals by Epstein (1990) and Epstein and Hardin (1990) showed that the conception rates of treated and control cows were 89%:59% and 95%:50%, respectively. Assuming that the attempts to get the cows to conceive were equivalent, these results also indicate poorer welfare in BST-treated cows. Phipps (1989), in reviewing the evidence for effects of BST on reproduction, distinguishes, first, between the use of BST early in lactation and late in lactation and, second, between higher and lower doses of BST. If the BST is administered early in lactation and at higher dose levels, the reductions in pregnancy rate reported by Epstein can be produced. However, it seems that administration of lower dose levels of BST later in lactation are less likely to have any adverse effects on welfare. However the meta-analysis (EU Scientific Committee on Animal Health and Animal Welfare, 1999) showed that with BST usage, the pregnancy rate dropped from 82% to 73% in multiparous cows and from 90% to 63% in primiparous cows. In addition, multiple births substantially increased.

A further point, which may be very important to the cows, is that each injection has some effect on a cow and repeated injections may cause swollen and tender injection sites (Comstock, 1988). The EU Scientific Committee on Animal Health and Animal Welfare Report (1999) showed that there were severe injection site reactions in at least 4% of cows.

More general effects of BST use are, first, that higher mastitis incidence may result in more antibiotic treatment and greater risk of the development of pathogen resistance and, second, that the possible change from smaller to larger dairy farms, which could result from widespread BST usage, could lead to poorer average stockmanship and less individual care of cows.

8. Resource Limitations, Genetic Selection and Welfare

The possible limitations to adaptation have been considered by evolutionary biologists in relation to natural selection ever since the writings of Darwin. A development of this approach is the idea presented by Beilharz *et al.* (1993) that selection of domesticated animals for certain characteristics that led to the utilization of a substantial proportion of available resources, could have consequences for how well other systems could function. The negative collateral consequences of selection for increased production were presented by Goddard and Beilharz (1997) who suggested the 'Resource Allocation Theory'. The resources an animal has are limited and as a result, if output is increased through one biological process, such as producing more milk, other functions such as fertility, maintenance, movement, immune defence, etc. will be affected. The resources that one process demands can be increased to a certain extent. Management factors, such as increasing access to feed and nutrients, could increase fitness of the animal until resources become limited again. Any further increase in fitness would imply a reallocation of resources and thus modify other outputs such as disease resistance or behaviour (Beilharz *et al.*, 1993). Reviewing the negative side effects of selection for high production, Rauw *et al.* (1998) concluded that 'when a population is genetically driven towards high production, … less resources will be left to respond adequately to other demands like coping with unexpected stressors; i.e. buffer capacity is negatively affected'.

The actual limitations that might exist could involve the total quantity of a resource such as energy that is available, the amount of a raw material such as a micronutrient, the rate at which particular rate-limiting enzymes can be produced or any of several other kinds of biochemical and physiological limitations.

9. Can We Produce New Animals Whose Welfare Is Never Poor?

Domestication is the process by which a population of animals becomes adapted to man and to the captive environment by some combination of genetic changes occurring over generations and environmentally induced developmental events recurring during each generation (Price, 1984, 2002). Adaptation is discussed further by Broom (2004) and Broom and Fraser (2007). The widespread existence of poor welfare in domestic animals, however, shows that there are limits to how much animals can adapt to conditions imposed on them by humans. Genetic engineering could change animals further than has been possible so far with conventional breeding in this same direction. However, there will always be limits to change in animals that are required to actively feed themselves and otherwise regulate their interactions with their environment. If tissue culture is used, animal cells

might be cultured without the need for a nervous system and supracellular regulatory systems. The welfare of such cell masses might never be poor.

10. Legislation Required

There is legislation about animal experimentation in the European Union (EU), which requires that some account should be taken of the welfare of the animal during experimentation on transgenesis, or on treatment with biotechnology products. Research workers need to consider the welfare of the animal carefully and should be able to justify all of their actions to a member of the general public. However, after the animal ceases to be experimental, or if a genetically modified animal or product of biotechnology for treatment of animals are brought in from another country, the animals are not covered by the animal experimentation legislation.

It will not be adequate to depend upon the moral consciences of those who use transgenic animals, and specific legislation is needed concerning testing before usage. There is EU legislation relating to human health and preservation of the environment. There should also be legislation requiring that no genetically modified animals or animals treated with biotechnology products should be used commercially unless their welfare has been assessed using an adequate range of measures at suitable intervals throughout life and on through the next generation. If there is a net benefit for the welfare of animals, including humans, then the genetic manipulation should be permitted. This is a stricter criterion than just to say that any harm to the animal must be weighed against any benefit because this latter criterion could allow severe effects solely for financial gain. Modifications of animals which are carried out for commercial purposes only but which result in poor welfare should not be permitted. There is legislation in The Netherlands stating that genetically modified animals cannot be used unless specific permission is given. The EU and other countries should be following that lead. If such action does not occur quickly it will become more difficult as economic pressures build up.

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16 Breeding Goals to Optimize Production Efficiency

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1. Introduction

The aim of genetic improvement in livestock is to increase efficiency of production. Increase in efficiency of production means saving on the input of production factors by setting up a new production function. By an alternative use, the saved production factors get a value in terms of a (market) price or an opportunity cost.

Genetic improvement in livestock is obtained by differential use of parental stock – by facilitating that superior parental stock has a higher chance to contribute genes to offspring, relative to non-superior stock. A major decision to be taken in breeding programmes is the definition of 'superiority', the breeding goal, involving: (i) the definition of genotype traits for which genetic improvement will contribute to savings in production factors; and (ii) the relative weighting of those genotype traits. The scientific basis for this relative weighting was developed for plants and first applied for livestock by Hazel (1943). Theoretically, this relative weighting includes a current market economic value (the only component in the original definition of selection index theory; Hazel, 1943), a component that reflects the benefit of using an animal with a superior genotype (cumulative discounted expression; McClintock and Cunningham, 1974) and a non-current market component (social value; Olesen *et al.*, 2000).

This chapter describes general aspects of breeding goal definition with emphasis on the derivation of 'economic values' and the definition of genotype traits.

2. An Aggregate Genotype as True Breeding Value

The breeding goal lists genotype traits subject to genetic improvement and gives each trait a value. This way a weighted summation of traits is made: an aggregate genotype to be improved (Hazel, 1943). The values used for weighting traits are generally called 'economic values', 'economic weights' or 'goal values'. In this chapter, the

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term goal value, *a*, is used as a combined value including a market or economic component, a non-market or social component and discounting aspects. In matrix notation:

H = **a'g** US$/animal;

a = **c'** (**ev** + **sv**) US$/animal/unit;

where:

g ($m \times 1$) vector with genetic superiorities of m genotype traits (unit);
a ($m \times 1$) vector with goal values of m genotype traits (US$/animal/unit);
c ($m \times m$) diagonal matrix with cumulative discounted expressions of m genotype traits (animal × year/animal);
ev ($m \times 1$) vector with economic values of m genotype traits (US$/animal/year/unit);
sv ($m \times 1$) vector with social values of m genotype traits (US$/animal/year/unit); (nb, animal year is the unit of counting the number of expressions of the trait).

Economic and social values directly and indirectly relate to savings of production factors and the price or opportunity cost given to the saved production factors. Economic and social values heavily depend on the livestock production system (consider, e.g. opportunities for alternative use on a farm) and in broader terms on natural, social and economic circumstances at the regional, national and international level, in the foreseeable future when genetic improvement is to be realized (Groen *et al.*, 1997).

In the original selection index theory by Hazel (1943), the weighting of genotype traits only included an economic component. The social component was introduced by Olesen *et al.* (2000). Expansion of the selection index theory was done by the introduction of the gene flow methodology, which incorporated a cumulative discounted expression in the weighting of aggregate genotype traits (McClintock and Cunningham, 1974). The principal idea of the incorporation of the cumulative discounted expression was that genotype traits that have a faster flow of genes in offspring, and thus a larger and earlier level of expression, should have a higher weighting. In other words, the value of identified genetic superiority depends on the time and frequency of expression of the superiority in offspring. Time and frequency of expression differ between traits, and therefore, it is important to include cumulative discounted expressions in goal values. McClintock and Cunningham (1974) proposed the use of a 'standard discounted expression' of an individual's genotype in the progeny in the year in which the mating took place. To deal with more complex design of mating systems, the discounted gene flow method was proposed by Elsen and Mocquot (1974) and Hill (1974). The discounted gene flow is expressed as a number of cumulative discounted expressions as a consequence of one mating. 'Cumulative' refers to an accumulation of expressions over generations or years; 'discounted' implies that future return is discounted to today's values by a discounting factor (Brascamp, 1978). Cumulative discounted expressions are specified per selection path included in the genetic improvement scheme.

Relative levels of goal values of different traits are important for an accurate definition of the breeding goal giving optimum levels of genetic improvement, i.e.

a technological change as a change in the production function, optimally addressing predicted future societal needs. To obtain an accurate calculation of revenues from the breeding programme (in order to optimize its structure), primarily the absolute levels of goal values are important.

An important implicit effect of the breeding goal definition is the quantification of the (true) genetic variance among animals subject to selection (σ_H^2).

3. An Information Index as a Predicted Breeding Value

The breeding goal is defined as the aim of genetic improvement at the population level ('superiority') and at the same time serves the identification of individual superior stock. For this latter perspective, identification of individual genetic values is required. As (true) genetic values of individuals are unknown, these are predicted indirectly. For this purpose, current genetic improvement strategies in livestock widely adopted 'selection index theory' (Hazel, 1943), or its advanced version in Best Linear Unbiased Prediction of breeding values (Henderson, 1973). The tool used in deciding on which males and females will become parents of the next generation is the 'selection index' or predicted breeding value (*I* or PBV). The PBV is a summary of observations, information on measurements and scores, weighted such to account for (Hazel, 1943):

1. The genetic possibilities of improvement (by considering the genetic and phenotypic covariances in the population);
2. The number of observations on the animal and its relatives (by considering the covariances on the observations); and
3. The relative importance of traits (given by goal values) in the aggregate genotype.

In matrix notation:

$$I = \mathbf{b'X} \text{ US\$/animal;}$$

$$\mathbf{b} = \mathbf{P^{-1}Ga} \text{ US\$/animal/unit;}$$

where:

- **b** ($n \times 1$) vector with regression coefficient on phenotypic observations (US$/animal/unit);
- **X** ($n \times 1$) vector with phenotypic observations (unit);
- **P** ($n \times n$) matrix with covariance among n phenotypic observations;
- **G** ($n \times m$) matrix with covariance between m genotype traits and n phenotypic observations;
- **a** ($m \times 1$) vector with goal values of m aggregate genotype traits (US$/animal/unit).

Originally, the selection index is a single-step procedure with a multi-trait approach; I is the PBV for the (true) multi-trait aggregate genotype (PBV_{MT}). In practice, however, a two-step procedure is generally applied with first, a prediction of breeding values per genotype trait (PBV_i) and second, weighting PBVs to an overall index. $PBV_{MT} = \Sigma(PBV_i)a_i$, where a_i is the goal value for trait i and summation is

over all genotype traits. The error of this simplification is dependent on the accuracy of (single trait) PBVs and the correlation structure among genotype traits.

The extent to which the (true) genetic variance (σ_H^2) can indeed be exploited in selection depends on the accuracy of the breeding value estimation procedure (r_{IH}, i.e. the correlation between the index and aggregate genotype: $\sigma_I^2 = r_{IH}^2\ \sigma_H^2$). Selection and mating strategies determine the intensity of selection (i). The revenue of the genetic improvement scheme in terms of genetic gain per year is calculated by these factors $\delta g = \Sigma(i_1 \times r_{IH,l} \times \sigma_{H,l}) = \Sigma(i_l \times \sigma_{I,l})$, where summation is over all selection paths.

Where the original selection index theory assumed a situation of a steady-state programme (in other words, an infinite time horizon applied in gene flow) and the absence of inflation (i.e. zero interest rate, indicating no preference for earlier expression allowing for ignoring the discounting), it in fact simplified the definition of the breeding goal by ignoring its dependence on the structure of the breeding programme. The gene flow methodology allowed for including this interdependence, but only for simplified situations of breeding value estimation, as in the selection index theory. In a steady-state genetic improvement strategy and in the absence of inflation, the cumulative discounted expressions for all traits in all selection paths is $1/\Sigma L$, where L is the sum of generation intervals over all selection paths. In this situation, the calculation of genetic gain per year equals the formula of Rendel and Robertson (1950). Assumed simplifications in breeding value estimation and breeding programme in essence consider the same r_{IH}, i and generation interval for all individual (potential) breeding animals. A theoretical step was made by the introduction of the contribution theory (Woolliams and Thompson, 1994), which directly related the accuracy of the breeding value estimation procedure at the level of an individual parental stock animal to the contribution of the individual to the gene flow (i.e. to predict genetic gain and inbreeding trend) of the breeding programme. The contribution theory has no additional effect on the weighting of genotype traits.

4. Methodology to Derive Economic Values

This section focuses on the derivation of economic values only and is based on earlier contributions by Groen (1989, 2003) and Groen *et al.* (1997). For more details and applications on the derivation of cumulative discounted expressions and social values, see, e.g. Jiang *et al.* (1999) and Olesen *et al.* (2000).

Economic values can be assigned non-objectively or derived objectively by a normative approach. Non-objective methods assign values in order to achieve a desired or restricted amount of genetic gain for some genotype traits (Kempthorne and Nordskog, 1959; Brascamp, 1984). These methods are useful to examine the borders of the possible solution area for genetic improvement. In defining breeding goals, it is assumed that a below average performance for one trait can be compensated by an above average performance for another. Commercial breeding companies, however, experience problems in marketing animals, which are too far behind for some traits even if they excel in others. Therefore, they tend to put more emphasis on poor traits and less on superior ones (Schultz, 1986). The method

of De Vries (1988) modifies the economic value based on the performance level of each trait for the stock concerned, the average performance levels of all traits of all competitors, a compensation factor expressing to what extent a low level for one trait may be compensated by a high level for another trait, and the minimum trait level required for acceptance by farmers. The purpose of this strategy is to avoid decreases in sales for the commercial company, although at the population level it obviously leads to less optimal genetic improvement and lower savings in production factors than would be achieved applying normative, objective economic values.

Figure 16.1 illustrates the general principle in objectively, normatively deriving economic values. An equation or a set of equations (a model) represents the behaviour of a system. The equations identify modelling elements and their relationships (i.e. regression coefficients or class effects). Genotypes for animal traits are defined unambiguously by modelling parameters.

Economic efficiency of production is a function of costs and revenues of the production system. Costs can be defined as the total value of production factors (input) required for production within the system; revenues can be defined as the total value of products (output) resulting from production within the system. A single-equation model is often called a 'profit function'. However, regarding the strict definition of profit as output minus input, the more general term 'efficiency function' better represents this type of modelling. A multi-equation simulation model is referred to as a bio-economic model (e.g. Groen *et al.*, 1997). Using simulation models, economic values are derived by partial budgeting; with efficiency functions, these can also be derived by partial differentiation. Bio-economic modelling offers opportunities to consider large numbers of elements and their relationships. Thereby, bio-economic modelling allows for the implementation of mathematical programming techniques to optimize production systems. Mathematical programming allows for finding (given farm characteristics) the best use of saved production factors, or in other words, the highest opportunity cost (Steverink *et al.*, 1994).

The economic value of a genotype trait expresses to what extent economic efficiency of production is improved at the moment of expression of one unit of genetic superiority for that trait. Derivation of economic values, therefore, involves:

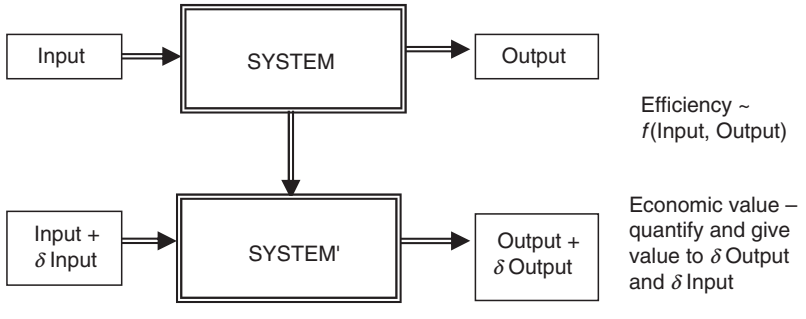

Fig. 16.1. The general principle in deriving economic values. The apostrophe denotes a marginal change in genetic merit of one genotype trait.

- quantifying changes in physical amounts (and qualities) of each production factor required and product produced, in terms of δ Input and δ Output;
- giving a value, i.e. a price or opportunity cost to the changes in production factors required and product produced.

In performing these quantifying and valuing changes, three modelling assumptions are to be made:

1. Level (animal, farm, national, etc.) and planning term (operational, tactical or strategic) of the system modelled.
2. Size of the system: fixed number of animals, fixed input of a production factor or fixed output of a product.
3. Interest of selection: maximize profit (Output − Input), minimize cost price (Input/Output) or maximize revenues on investment (Output/Input).

The theoretically appropriate level to be used in deriving goal values in animal breeding is the one for which limited resources, prices of products and production factors are influenced by an improvement of a trait (Fewson, 1982). A good example is given in a dairy industry with a milk quota system limiting the amount of product at farm level. Improvement of genetic merit for milk production per cow will have to result in a reduction in the number of cows at a farm. To include the effects of a reduction in the number of cows (reduced costs of housing, feeding, labour, etc.), derivation of economic values should be performed at farm or higher level.

In fact, the system level and planning term together determine for which saved production factors an alternative use is feasible in the time frame considered. Alternative use implies a (market) price or opportunity cost; with no alternative use the value of a saved production factor is zero. Fixed costs are not subject to changes while variable costs are. In other words, input of production factors considered fixed does not have an alternative use, while input of production factors considered variable has.

Groen *et al.* (1997) presented the concepts of economic production theory regarding different combinations of assumed size of the system and assumed interest of selection in deriving economic values. For example, with a fixed number of animals and profit maximization, the economic value is positive when the marginal revenues of increased output per animal exceed the marginal costs of increased output per animal (see also Melton *et al.*, 1993). In this case, breeding for increased output per animal will be beneficial. What does this teach us? It reminds us that:

1. The essence of improvement in efficiency of a production system is saving inputs of production factors per unit product and/or a change towards use of cheaper production factors.
2. Saved production factors get a value (price or opportunity cost) by an alternative use.

Now, saved production factors can either be used in the system where they are saved from (and thus extend product output of this system) or can be transferred to another system (via the market; Willer, 1967). Likewise, additionally required production factors are either to be drawn from the market or from an alternative use in the system. Concepts of production theory as presented by Groen *et al.*

(1997) express differences in assumed use of saved production factors. In other words, choices of level and size of the system and of interest of selection will influence the outcome of the derivation of economic values when the value of production factors differentiate for assumed different possibilities of alternative use.

For the 'profit, fixed number' perspective, saved production factors are sold at the market. Market prices will equal average total costs of production assuming that: (i) markets of products and production factors are purely by competitive, and (ii) industry and all individual firms are in equilibrium (Stonier and Hague, 1964). These assumptions imply an equivalence of economic values on the basis of a fixed number of animals when derived within profit and cost price interests. This equivalence was denoted as the 'zero' or 'normal' profit theory by Brascamp *et al.* (1985). On the basis of fixed output, economic values within a profit interest are equivalent to economic values within a cost price interest. These economic values will also be equivalent to economic value 'fixed number, cost price' when (iii) all costs of the farm are considered to be variable per unit product. This equivalence was denoted by Smith *et al.* (1986) when introducing the rescaling theory. Table 16.1, in a simplified way, explains the scientific background of long-standing discussions in literature on breeding goal definition.

Now the discussions tend to get another perspective: the question is no longer whether the normal profit theory and rescaling theory are correct or not (in fact, Table 16.1 proves these theories to be correct), but the question is whether or not the production circumstances explicitly required for the equivalences do actually hold in practical situations for which breeding goals are derived.

In agricultural industries, products and production factors are commonly heterogeneous and not fully divisible. Heterogeneity of products and production factors leads to division of markets (Dahl and Hammond, 1977) and cause the average costs of production to be different for individual firms. Given (equilibrium) market prices, some firms will have a lot of profit; other firms will be just efficient enough to continue production (Stonier and Hague, 1964). As an important result, the equivalence of perspectives may hold under certain conditions for the sector as

Table 16.1. Economic values expressed in concepts of economic production theory. (After Groen, 1989; see also Groen *et al.*, 1997; Groen, 2003.)

	Interest of selection	
Size of the system	Profit maximization	Cost price minimization
Fixed number of animals	Marginal revenues[a] – marginal costs[b]	Average total costs[a] – marginal costs[b]
Fixed input	Marginal revenues[a] – average (revenues-fixed costs per animal)[c]	Average total costs[a] – average fixed costs farm[c]
Fixed output	Average variable costs[a] – marginal costs[b]	Average variable costs[a] – marginal costs[b]

[a]Per δy units of product.

[b]Per δy units of product, corresponding to δx_v units production factor.

[c]Per δx_v units production factor.

a whole, but may not be valid from an individual producer's point of view. In defining breeding goals, the definition of efficiency function has to correspond to the individual livestock producer's interest of selection. The producer's primary reason for buying a certain stock at a certain price will be based upon his assessment of how animals will perform.

The point of assuming fixed costs per animal or per farm to be variable per unit product is also subject to debate. Costs may be fixed (constant or discontinuously variable) with respect to the size of the farm (Horring, 1948). Considering these fixed costs to be variable per unit of product requires an assumption on the (continuously optimum) size of the farm. Smith *et al.* (1986) proposed to express all fixed costs per animal or per farm per unit of output, thereby assuming a given optimum farm structure or size, with efficient use of resources. The condition of fixed cost to be constant per unit of product is arithmetically correct when assuming that: (i) all farms have the same size; and (ii) changes in output and input are accomplished by a change in the number of farms. However, structural developments in the industry are detached from improvements in the efficiency of production, which is not correct considering long-term effects of the implementation of new techniques (Zeddies *et al.*, 1981; Amer and Fox, 1992).

A similar discussion is regarding the system subdivision: Are economic values different for different levels of the production system, e.g. nucleus breeder, hatchery, multiplier and commercial grower? In fact, the normal profit theory by Brascamp *et al.* (1985) also holds true here. Jiang *et al.* (1998), for a broiler example, illustrated that in a situation where trading between system levels is based on cost prices (e.g. in a fully integrate enterprise), breeding goals are the same for each system level perspective. However, when assuming that market prices substantially differ from cost prices (i.e. one system level is making an 'assess' of profit), breeding goals start to differentiate between system levels.

Although the normal profit theory and rescaling theory may not hold for (all) practical production circumstances for which economic values are derived, it is advised to routinely check differences between (relative) economic values when derived using different perspectives. Large differences may indicate non-sustainability of assumed (predicted) price ratios: price ratios that hold for a current market situation, but are expected not to hold on a strategic planning term. For example, when modelling a single farm, one should check the level of farm profit, probably for different farming systems. A (too) high level of farm profit might indicate that product prices are assumed to be (too) high relative to prices or opportunity costs of production factors.

5. Definition of Genotype Traits

Livestock production is a means of producing human food, other products for human consumption, and intermediates to be used as inputs for plant or energy production. Livestock also serves human interests in many other ways, like banking and social status. In general, livestock production is a means to create human welfare and well-being by converting resources (production factors, labour, land and capital) into higher valued products. Depending on many factors, for example, the

state of development of the country, key issues in the use of saved production factors are to increase food security or food quality, to enhance (economic and social) well-being of the producer, consumer and citizen or to increase sustainability of the system.

Ideally, all animal characteristics, i.e. all genotype traits that influence this broad scope of efficiency of livestock production, should be included in the aggregate genotype. However, in practice the number of genotype traits selected for is limited, only including a relevant part of reality. A number of theoretical and practical aspects to be considered in the choice of genotype traits is discussed below.

A model is always less than reality – a model only includes parts of reality relevant to the purpose of modelling. This seems to be a trivial point, but I include it here to stress that a breeding goal definition in terms of an aggregate genotype is a conceptual model. On one hand, breeding goal definition should aim at doing an optimal job in terms of building a scientifically sound, heuristically logic and practically implementable aggregate genotype. On the other hand, implementing an aggregate genotype in a breeding programme always gives results or 'side effects' not aimed for, not expected, probably undesired. This should not be automatically considered a failure, but can be considered a good reason for redefinition of the breeding goal.

Genotypes of animal traits are defined unambiguously by modelling parameters. This is a clear advantage of normative, objective modelling. Rather than defining, e.g. milk production as an average production level of an average cow, modelling for the purpose of deriving economic values requires an unambiguous definition of a milk production level of, e.g. a first parity cow, calving in February at an age of 2.2 years, in a production period of 305 days. The unambiguous definition of genotype traits should be associated with an unambiguous calculation of phenotypic and genetic population parameters for index derivation or breeding value prediction.

In practice, the choice of breeding goal traits is often based on the potential for genetic improvement of the trait and costs (in labour, facilities and time) of accurate breeding value prediction of the trait (Harris, 1970). This, however, is to be considered as theoretically not sound. The potential for genetic improvement should not be an aspect of the choice of genotype traits, but is an explicit element of the derivation of regression coefficients for observations or information sources in the selection index or PBV ($b = P^{-1}Ga$). The consideration of the costs of including information sources also should not be an aspect of choice of genotype traits, but should be a part of the optimization of the breeding programme. Higher costs in a breeding programme associated with new or more observations for more accurate breeding value prediction (increased $\sigma_I^2 = r_{IH}^2 \, \sigma_H^2$) should be compared with higher returns from more, or more optimal genetic improvement at the population level ($\delta g = \Sigma(i_l \times r_{IH,l} \times \sigma_{H,l}) = \Sigma(i_l \times \sigma_l)$).

Dickerson (1970) advocated the improvement of a biological efficiency rather than economic efficiency; breeding for economic efficiency might lead to biologically less-efficient livestock, and this is in the long term not sustainable. Differences between biological and economic efficiencies are restricted to differences in defining costs and revenues. In the biological definition, costs and revenues are expressed in energy and/or protein terms; in the economic definition this is in terms of

money. The major problem arising with the biological definition is that not all costs and revenues can be expressed in terms of energy and/or protein. The economic definition largely deals with this problem. However, a disadvantage of the economic expression is weakness in stability in time and place of monetary units (Schlote, 1977). Notwithstanding imperfectness, money is 'the standard for measuring value' (Stonier and Hague, 1964), and therefore, efficiency of production is usually considered to be economic efficiency. Nevertheless, it is very relevant to check if genetic improvement strategies based on (current market) economic prices and opportunity cost of production factors and products indeed also leads to improvement of biological efficiency. An example is given by Jiang *et al.* (1998) and also Hirooka *et al.* (1998). A large discrepancy between the relative weighting based on current market economic efficiency and biological efficiency could indicate that assumed market prices and opportunity cost will not last on a strategic term.

In modern Western breeding programmes, for a long time, breeding goals were generally limited to a restricted number of production and reproduction traits. Fortunately, there is a strong tendency to complement production and reproduction traits with functional traits such as workability, health and longevity. There certainly is a point in limiting the number of traits in the breeding goal: it gives focus and explicitly defines the priorities of the desired genetic improvement. However, as also mentioned above, one should always be aware of side effects of selection for high production due to correlated responses for functional traits (Rauw *et al.*, 1998). A good solution to prevent undesired side effects is simply to include these traits in the breeding goal (e.g. Groen *et al.*, 1997). This brings us to the point that derivation of goal values for functional traits is not as straightforward as it is for production and reproduction traits. This is certainly true, but there now are ample examples in literature available for modelling efforts to derive goal values for animal characteristics in all kinds of livestock species. Still, normative modelling to derive goal values for functional traits is an important domain for further scientific research both for economic and social values.

Especially in lower input production environments, livestock serves many more roles than only food production: fibre, power, fertilizer, fuel, transportation, insurance, social status and banking, and also these roles are to be considered when defining breeding goals. Benefits of part of these roles can be modelled normatively. Olesen *et al.* (2000) also considered non-food production roles of livestock to be included in breeding goal definition for sustainable production systems, but they emphasized that current market validation procedures do not account for important cultural/social aspects, like concerns about animal welfare and loss of historical breeds. According to Olesen *et al.* (2000) these 'non-current market values' of animal traits (e.g. ethical values of improved animal welfare through less suffering from diseases or stress and a higher quality of life) are to be quantified.

In relation to points mentioned above (unambiguous definition of traits, attention for biological efficiency and more emphasis on functional traits and animal welfare) a more recent trend is to use new conceptual frameworks for defining genotype traits. Examples are residual feed intake by Luiting (1991), thermal comfort and welfare by Kanis *et al.* (2004) and also resource allocation (Beilharz, 1998). A major advantage is the focus on underlying physiological processes. This will probably lead to a definition of genotype traits that has a better physiological

basis and is more directly related to direct gene regulation. With an ongoing trend towards identification of superiority at the gene level, this is a positive development. However, on the other side, genotype traits defined according to these new conceptual frameworks are more difficult to (phenotypically) observe, measure or score. Of course, implementation of underlying physiological traits as genotype traits does not require routine observations in a breeding programme, but experimental genetic and phenotypic covariances for the physiological traits and correlated traits for routine observations are to be defined in order to facilitate breeding value prediction on a routine basis.

6. Discussion

Genetic improvement is not aiming at an optimum; it is dynamically searching for improvements. Given animal genetic variation (within or between breeds), there is always a means of improvement. In fact, this approach is originating from the historical and continued natural process of re-establishing genetic variation (i.e. mutations). This approach is also an incentive to conserve genetic variation during the process of selection.

So, there is not 'a worldwide standard' or even 'a country standard' breeding goal. Differences in economic, social and ecological production environments give rise to different approaches and wishes in what human welfare and well-being is. These differences give rise to different development objectives. A diversification of breeding goals is important to serve farmers facing different local situations. A diversification of breeding goals according to local production environments will support genetic improvement for locally adapted breeds, and thus help to conserve genetic resources.

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17 Robustness

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1. Introduction

Animal robustness is rapidly developing into a key area in farm animal breeding. Sustainable breeding goals combine robustness traits with production traits to such an extent that selection balances genetic change in production potential with genetic change in environmental sensitivity. This should maximize the genetic change of sustainable production in the commercial production environment. This chapter describes the options for including robustness traits in breeding goals, and for exploiting commercial performance data for quantifying the environmental sensitivity of the animal's production potential.

The production potential of domestic livestock has increased considerably since the 1960s. There are many ways to illustrate this. Figure 12.1 gives examples for body lipid-to-protein mass and protein deposition rate, and for litter size, in pigs. Increasing genetic potential for production traits requires improved animal nutrition and management to support its expression, but such changes often lag behind. At the end of the 1990s, a user's guide for terminal sire lines of a Dutch pig breeding company said of the line with the most extreme lean growth capacities: '[Its] progeny are more muscled and, therefore, somewhat less robust. This is only noticeable in case of health problems and unstable conditions' (Anonymous, 1999; translated). The text followed with the sensible advice for pig producers to use this line only if the production environment is sufficiently under control. Similarly, Siegel and Dunnington (1998) suggested that:

> genetic selection to improve growth […] has resulted in [animals] that are extremely proficient in utilization of feed for body weight. […] The costs exacted from these practices include voids in ability to maintain a successful balance of other biological needs, including immune responses. An industry demanding rapidly growing, high yielding, feed efficient [animals] must recognize the effects of limited resources and rely on improved husbandry to minimize expression of these imbalances.

 Resource Allocation Theory Applied to Farm Animal Production (ed. W.M. Rauw)

In highly productive farm animals, productive functionality may become reduced when the development of supportive tissues is compromised (leading to physical damage, infection, morbidity, reduction of food intake and mortality) or when the reproductive endocrine system is disturbed (leading to reduced fertility). Productive functionality may also be reduced when production processes come to demand so many resources that functions for coping with external stressors become constrained in resource-limited conditions. The latter scenario assumes that the metabolic drive of production processes is so strong that it dominates resource allocation at the expense of coping processes, but it may also work the other way around. Although changes in genetic potential should logically be accompanied by changes in the production environment, in practice it often does not happen. A classical example is the UK 'thin sow syndrome' of around 1980, when many (essentially underfed) sows had to mobilize so much body lipid and protein during lactation that they became subsequently infertile. And up to about 1990, sows in the UK commonly showed a 'second-parity dip', where average litter size in parity 2 was *lower* (instead of half a piglet higher) than in parity 1. Compared to more conventional genotypes, the leaner gilts from the more advanced breeding programmes of the 1970s had less body reserves available for mobilization in late pregnancy and lactation, so they had to be fed at a much higher level, especially in winter. At the end of the 1990s, a textbook on sow lactation still stressed the fact that the 'novel' genotypes need an adapted nutritional regime in order to function properly (Whittemore, 1998; Williams, 1998), illustrating that the mental lag in the livestock industry can be very long.

The allocation of resources (Van Noordwijk and De Jong, 1986; Beilharz *et al.*, 1993; Van der Waaij, 2004; Friggens and Van der Waaij, Chapter 18, this volume) to production processes may then be constrained by: (i) *insufficient provision of resources* by the environment (e.g. inadequate nutrition and hot climate); and/or (ii) *additional demands for resources* by the environment (e.g. cold climate, subclinical disease and overcrowding). This leads to environmental sensitivity, i.e. loss of adaptive capacity to cope with limiting conditions. From the point of view of the production trait under concern, it leads to genotype × environment interaction.

Pig production is rapidly moving towards large-scale systems where the infectious, climatic, nutritional and social environment is often challenging due to low-cost design, and where attention to the individual animal is often limited. This introduces the feature 'robustness', defined here as the ability to combine a high production potential with resilience to stressors, allowing for unproblematic expression of a high production potential in a wide variety of environmental conditions (Knap, 2005). This unproblematic expression may be compromised by reduced functionality of the system, as above:

> [F]ast growing [broiler chicken] lines are not suitable for standard [middle-Eastern] management and sanitation conditions. Their successful rearing is possible only under conditions that are not always economically feasible. Breeders must find economic solutions for this problem, which will become more acute in the future. (Nir, 1998)

Hence the increasingly wide variety of environmental conditions in which livestock is required to perform is rapidly moving attention to robustness traits. Transnational pig breeding companies are following that move (e.g. Knap and Luiting, 1999; Landsutvalget for Svin, 2004; Koeleman, 2005), similar to the poultry breeding

sector (Flock *et al.*, 2005). For the breeding industry, the relevant issues are: (i) if it is commercially worthwhile to breed robust livestock; and (ii) to what extent it is possible to do so.

2. Robustness as a Breeding Goal Trait

Animal breeding goals comprise traits that play a key role in the profit equation. A classical profit equation for slaughter pig production (from Knap, 1989) is:

$$\begin{aligned}\text{Profit} = {} & \text{CWT} \times (V_{\text{kg}} + \text{LEAN} \times V_{\text{lean}}) \\ & - \text{DAYS} \times (C_{\text{day}} + \text{DFI} \times C_{\text{feed}}) \\ & - C_{\text{SY}}/(\text{LSY} \times \text{LSW})\end{aligned} \tag{17.1}$$

The first term represents income: carcass weight (CWT, in kg) multiplied by the standard carcass price (V_{kg}, in money per kg) plus the extra value of lean content (LEAN, in %), dependent on the bonus value for lean (V_{lean}, in money per % per kg). The second term gives the cost of growing the pig: it takes a number of DAYS to reach slaughter weight, and each day has a fixed cost (C_{day}, in money per day, due to housing, management, health care, etc.) plus feed costs: daily feed intake (DFI, in kg per day) multiplied by the feed price (C_{feed}, in money per kg). The third term gives the sow-related cost of producing the pig: LSY (litters per sow per year) multiplied by LSW (average litter size at weaning) gives the number of pigs weaned per sow per year, C_{SY} is the cost per sow per year.

This leads to five breeding goal traits: LEAN, DAYS, DFI, LSY and LSW. The marginal economic value (MEV) of each trait follows classically from differentiating the profit equation with respect to it. Now add three robustness traits to the profit equation:

$$\begin{aligned}\text{Profit} = {} & \text{GPS} \times \text{CWT} \times (V_{\text{kg}} + \text{LEAN} \times V_{\text{lean}}) \\ & - [\text{GPS} \times (\text{DAYS} - D_{\text{mort}}) + D_{\text{mort}}] \times (C_{\text{day}} + \text{DFI} \times C_{\text{feed}}) \\ & - C_{\text{SD}} \times \frac{\text{NLITT} \times 28 + (\text{NLITT} - 1) \times 115 + \text{WCI})}{\text{NLITT} \times \text{LSF} \times \text{PWS}}\end{aligned} \tag{17.2}$$

GPS is growing pig survival rate; carcass income is received only from the proportion of pigs that actually survive up to slaughter, and growing costs are accrued only as long as the pig survives (up to D_{mort} days here). The third line still represents the sow-related cost of producing the weaner pig. C_{SD} is the cost per sow per day: the rest of the term gives the number of sow days per piglet weaned. NLITT is the number of litters per sow lifetime (sow longevity), and each cycle is assumed to have 28 days for lactation, 115 days for gestation and WCI days from weaning to conception. LSW is now represented by litter size at farrowing (LSF) multiplied by preweaning survival rate (PWS). This gives three novel breeding goal traits related to robustness: PWS, GPS and NLITT.

Using parameter values for a typical Western market situation, and differentiating, the resulting MEVs are in Table 17.1, standardized into money per genetic standard deviation of each trait. The familiar production traits have MEVs of 2–4 money units per genetic standard deviation; the three robustness traits have MEVs of a similar magnitude: 1–3 money units per genetic standard deviation. Hence, the

Table 17.1. Marginal economic values of pig breeding objective traits, standardized into money per genetic standard deviation (σ_G) of each trait.

Trait	Money/σ_G
Lean content	+3.45
Days to reach slaughter weight	−4.31
Daily feed intake	−3.89
Litter size at farrowing	+2.16
Days from weaning to conception	−0.64
Preweaning survival rate	+1.08
Growing pig survival rate	+2.21
Number of litters per sow lifetime	+3.01

profitability of pig production can be changed by genetic improvement of a robustness trait at a similar rate as by improvement of a conventional production trait. Similar information on dairy cattle was provided by Groen *et al.* (1997), Pedersen *et al.* (2002) and various contributions to Interbull (1999). This shows that, strictly from a profitability point of view, it seems indeed worthwhile to produce robust livestock.

3. Neutralizing Antagonisms Between Robustness and Production Traits

Thus, robustness traits should be part of livestock breeding goals, but can we expect significant genetic progress from selection? The question is how robust livestock can be produced by breeding. Much of the discussion here is about antagonisms between robustness and production performance, which would compromise the 'unproblematic expression of high production potential in a wide, this volume variety of environmental conditions' (as above) more strongly at a higher production potential. The reviews by Rauw *et al.* (1998; Chapter 12, this volume) give good evidence for this in pigs. But much of the debate in this area is dominated by decades-old information that is long overdue for reinterpretation. We give an example of this and then consider more recent developments.

Dämmrich and Unshelm (1975) raised the concept that muscle mass is outgrowing bone mass in fast-growing livestock. They found less disturbance of bone and joint growth in pigs fed restrictedly (with a growth rate of 350 g/day up to 125 kg body weight) than when fed unrestrictedly (with a growth rate of 610 g/day, referred to as 'excessively heavy'), and suggested that 'skeletal growth [...] does not keep pace with the capacity to gain weight and so there is a proneness to damage by overloading'. It is perfectly normal for current (2008) commercial slaughter pigs to grow at that 'excessive' level of 610 g/day, and for current nucleus breeding stock to grow more than 50% faster, so this is a worrying statement. Since then, however, skeletal soundness has been seriously targeted in livestock breeding.

Figure 17.1 gives the results of a selection experiment in broiler chickens (Leenstra *et al.*, 1984). This study shows four things: (i) selection for increased

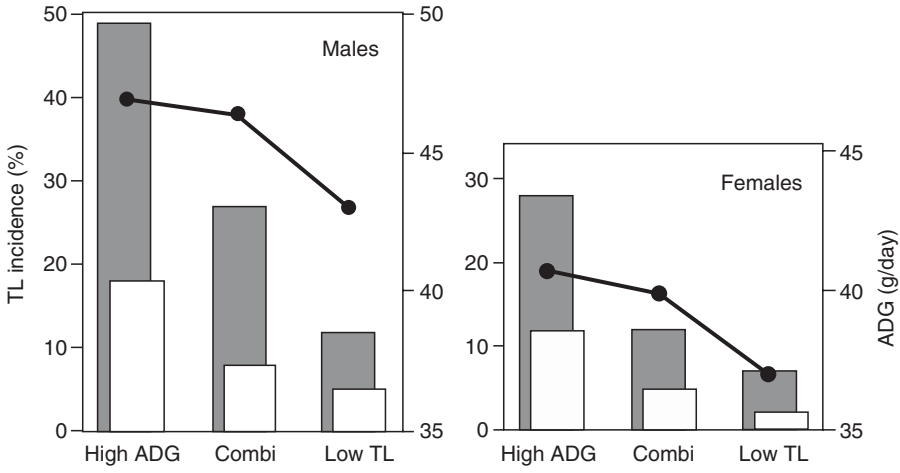

Fig. 17.1. Incidence of twisted legs (TL, bar plots, left vertical axes) and average daily gain (ADG, line plots, right vertical axes) in three broiler chicken sire strains selected for high ADG, low TL incidence or a combination of both for three generations. Grey bars, in cages; white bars, on litter. (Data from Leenstra *et al.*, 1984.)

growth rate had an unfavourable correlated response in the incidence of the skeletal defect 'twisted legs'; (ii) there was a strong effect of environmental factors (i.e. floor type) and gender (confounded with growth rate) on twisted legs incidence; (iii) selection for reduced twisted legs incidence was very successful across environments and gender, but it had an unfavourable correlated response in growth rate; and (iv) selection for a combination of the two traits resulted in a considerable reduction of twisted legs incidence while keeping growth rate close to the maximum level (see also Siegal *et al.*, Chapter 13, this volume). Leg soundness is an important selection criterion in pig breeding. In the past, selection was often practised by independent culling on own performance of young selection candidates, but because of low heritabilities, a best linear unbiased prediction (BLUP) approach is more effective for achieving genetic progress. This is illustrated in Fig. 17.2, which shows 10-year genetic trends for growth rate (lifetime average daily gain), and for Rothschild and Christian's (1988) leg soundness score in seven PIC pig lines. These lines are bred according to breeding goals that place different emphasis on growth rate and leg soundness, among many other traits.

Figure 17.2 shows that: (i) in spite of possibly antagonistic correlations between the two traits, it is perfectly realistic to achieve a positive genetic trend in both traits at the same time; (ii) the lines with the highest genetic trend in one trait do *not* show the lowest genetic trend in the other trait – the inset gives the correlation plot of the slopes of the trendlines and the correlation is by no means negative; and (iii) apart from such considerations, leg soundness shows a consistent pattern of genetic improvement in each line, on average by three residual standard deviations in this period. This leg soundness score has genetic correlations with production traits between −0.28 (unfavourable) and +0.26 (favourable). Different emphasis on traits in the breeding goals of the lines in Fig. 17.2 results in different rates of genetic change per trait in different lines; this is not a coincidence (and for robustness

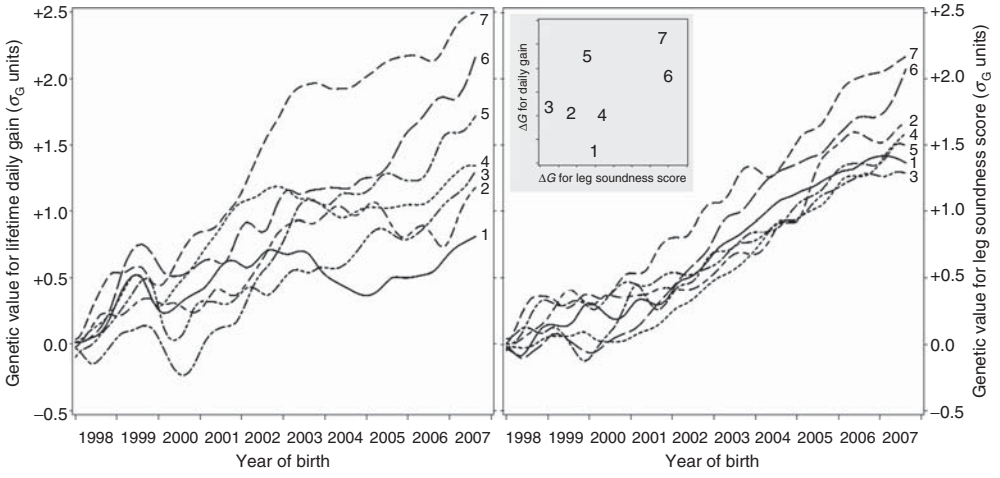

Fig. 17.2. Genetic trends in lifetime daily gain (left) and leg soundness score (right) in seven PIC pig lines. Both traits are expressed in genetic standard deviation units, and the trendlines were forced to start from the same point in 1998. The inset shows the relationship between the linear slopes of the trendlines.

traits not left to unfavourably correlated effects), but a deliberate strategy. As in Leenstra *et al.*'s (1984) broiler example, antagonisms such as signaled by Dämmrich and Unshelm (1975) can be neutralized by deliberate selection. The critical issue is proper inclusion of robustness traits in the breeding goal, and in the selection criterion.

A disadvantage of leg soundness scores such as described above is the subjective nature of the observations. This may lead to inconclusive and anecdotal evidence, which is common in the older literature on livestock robustness. Osteochondrosis diagnostics (based on physical or radiological examination of cartilage lesions) and mortality rates are more informative. Because of the more quantitative nature of such traits, genetic correlations with production traits are easier to interpret. Several pig breeding programmes record osteochondrosis in leg joints, or have done so until recently. Table 17.2 gives a summary of heritability estimates of osteochondrosis traits, and of the genetic correlations of these with production traits. Resource allocation theory would predict unfavourable correlations to occur particularly in leaner, faster-growing animals housed in more adverse conditions. But the results in Table 17.2 (and in its sources) do not support this. Hence, either osteochondrosis incidence in growing pigs is triggered by other mechanisms than disturbed resource allocation, or the data of these studies were not sufficient to detect such trends (standard errors of the genetic correlation estimates were ≤0.3 for reference 3 and mostly ≤0.1 in the other studies). But the various osteochondrosis measurements are clearly heritable, and the range of genetic correlations is just as inconclusive as mentioned above for leg soundness scores. Genetic antagonism between production and robustness can be neutralized by inclusion of the robustness trait in the breeding objective, combined with proper selection methods.

At the nucleus level, where the genetic production potential is very high and conditions are intensive (but favourable in terms of health and nutrition), perinatal,

Table 17.2. Genetic parameter estimates for osteochondrosis incidence in growing pigs.

Country[a], breed[b]	h^2	r_G (<0 is favourable) with		Floor, group size	Reference[c]
		Growth	Lean %		
DK; Y	0.08–0.32	0.24	–0.08	Straw, 12	1
DK; L	0.16–0.38	0.34	–0.07	Straw, 12	1
DK; D(LY)	0.02–0.22	–0.45–0.27	–0.07–0.35	Full slats, 8–15	2
CH; Y,L,D	0.00–0.39	–0.44–0.31	–0.11–0.32	Part slats, 10	3
SE; L,Y	0.16–0.28	0.17–0.39	0.03–0.13	Straw, 2	4
NO; L	0.20–0.22	0.11–0.12	–0.03–0.01	Slats/straw, 10	5
NO; D	0.12–0.15	0.04–0.05	–0.10–0.08	Slats/straw, 10	5

[a]DK, Denmark; CH, Switzerland; SE, Sweden; NO, Norway.
[b]D, Duroc; L, Landrace; Y, Yorkshire.
[c]1, Jørgensen and Andersen (2000); 2, Jørgensen and Nielsen (2005); 3, Kadarmideen *et al*. (2004); 4, Lundeheim (2005); and 5, B. Holm, Norsvin (2006, personal communication).

preweaning, flatdeck, grower-finisher and sow mortality rates in PIC pig lines have estimated heritabilities ranging from 0.04 to 0.13 (the same order of magnitude as for fertility traits), and weak genetic correlations with production traits. There are therefore good possibilities for a genetic reduction of mortality rates while maintaining genetic improvement of production traits. Again, what is required is a proper multivariate BLUP system (which will need large data volumes, especially for categorical traits like mortality) and proper weighting of the resulting estimated breeding values into the selection index, based on MEVs such as from Equation 17.2. Of course, it is more useful to work with mortality records collected in commercial conditions rather than in the nucleus: data volume can be much larger, and genetic parameters are likely to be different in more adverse conditions. This provides a logistical challenge: it is a very demanding task to record mortality among individually identified and pedigreed crossbred pigs in commercial farms. Such systems have now been established in various parts of the world (Knap, 2005; Casey *et al.*, 2006).

Perhaps the most striking case of pig mortality traits that are subject to a correlated increase through selection for increased production is perinatal and preweaning piglet mortality as related to litter size. Genetic improvement of litter size in pigs has been very successful since the worldwide implementation of BLUP around 1990, and this has raised concerns about antagonistic correlations with piglet mortality traits, mainly mediated through the weight and stage of development of the newborn piglet (Roehe and Kalm, 2000; Lund *et al.*, 2002; Grandinson *et al.*, 2002; Simianer *et al.*, 2003; Foxcroft *et al.*, 2006; Foxcroft, 2007; Canario *et al.*, 2007; Rosendo *et al.*, 2007).

For example, the Danish pig breeding organization Danavl (internationally known as Danbred) wrote in 1997:

> A breeding value for litter size at birth […] has been part of the breeding goal from 1991. Since then we have achieved an annual genetic progress of 0.2 to 0.3 piglets per litter. It almost seems that we have been too successful, because the large improvement of litter size also leads to a lower birth weight per piglet. (Petersen, 1997; translated)

A year later, Danavl reported 'concern about the fast development of litter size [...] the strong one-sided progress in litter size can have unexpected side effects, for example in the form of increased numbers stillborn and lower survival levels', and announced the start of recording perinatal and preweaning mortality, with the plan to 'revise the breeding goal with the inclusion of number liveborn per litter and survival levels' (Andersen and Palmø, 1998; translated). The analysis of these data produced unfavourable estimates of the genetic correlation between litter size and piglet survival rates (Su *et al.*, 2007) and therefore revealed that

> there is no linear relationship, but mortality rate increases with increased litter size [...] the relationship is shown in Figure 3. Mortality rates [...] increase continuously with more than 11 pigs per litter. [...] There are now two options for increasing the number of liveborn piglets per litter. Either we continue with breeding for an increased total number born per litter [...] more piglets born will steadily increase the number born alive per litter. Or we can try to increase the survival rate of piglets born. (Nielsen and Henriksen, 2004; translated)

The latter option was chosen, and the dam-line breeding goal was revised that same year: '[T]o counter a continuing increase in mortality [...], the number of piglets per litter alive at five days after birth was included in the breeding goal instead of litter size at birth' (Landsudvalget for Svin, 2004; translated).

Likewise, the Dutch pig breeding company Topigs studied preweaning survival and its relationships with birth weight and developmental stage at birth (Knol *et al.*, 2002; Leenhouwers *et al.*, 2002), and included the trait in its dam-line breeding goals as one of the aspects of 'balanced breeding' that 'allow the sow to actually rear the increased number of piglets born per litter' (Pigture Group, 2006; translated), to the extent that traits related to piglet survival now comprise more than half of the breeding goal, compared to litter size at just over a quarter. As a consequence, preweaning survival (which seems to have been on a similar decline as in the Danavl lines of Fig. 17.3) shows a favourable genetic trend since 2002, while

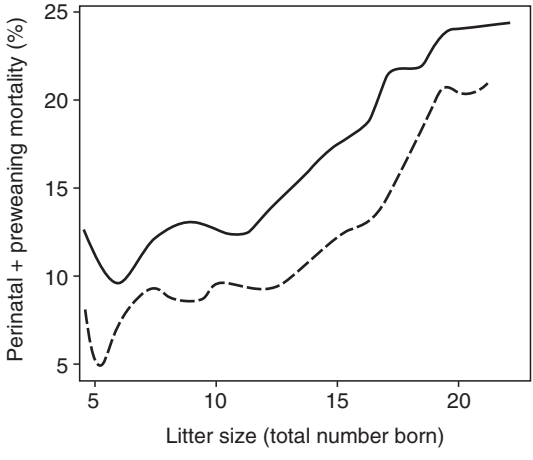

Fig. 17.3. Perinatal plus pre-weaning mortality in two pig lines, in relation to litter size. Litter size levels <5 were omitted due to small numbers of records. (Data from Nielsen and Henriksen, 2004, Fig. 3; B. Nielsen, 2008, personal communication.)

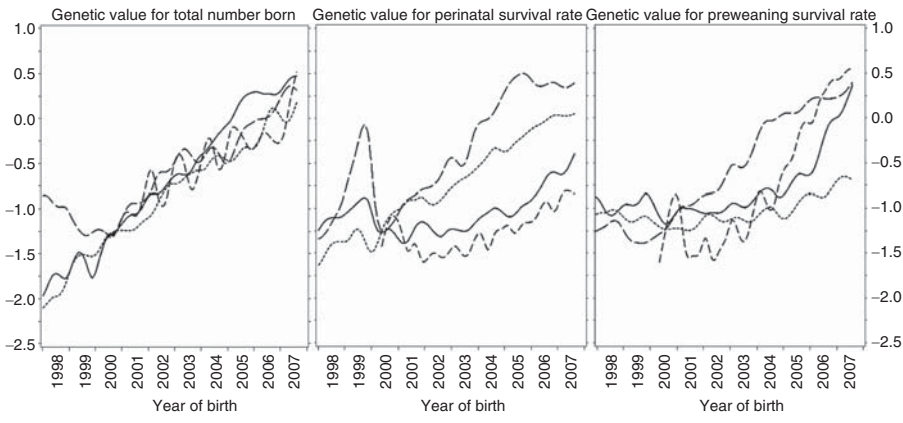

Fig. 17.4. Genetic trends in litter size, perinatal survival rate and preweaning survival rate in four PIC pig lines. Each trait is expressed in terms of the standard deviation of its EBVs, and the trendlines were forced to start from the same point in 2000.

litter size is still increasing as well (Pigture Group, 2005; translated). The same principle is illustrated in Fig. 17.4, which gives 10-year genetic trends for litter size (total number born), perinatal survival rate (the complement of stillbirth) and preweaning survival rate in four PIC pig lines.

Figure 17.4 shows two things: (i) thanks to BLUP and extensive data recording, selection for traits with low heritability such as reproductive and mortality traits can be very successful; and (ii) the two survival rate traits show a clear improvement while litter size is increasing at the same time; piglet survival traits (in one form or another) have been part of the breeding goal of these PIC lines since 1997. The transferability of genetic progress such as in Fig. 17.4 to commercial conditions was confirmed by Roehe *et al.* (2008). Hence, in spite of possibly antagonistic correlations between the traits, and functionally dependent on the emphasis given to each trait in the breeding goal of each line, it is perfectly realistic to achieve a positive genetic trend in all three traits simultaneously (as in Fig. 17.2). Antagonisms such as signaled by Foxcroft (2006) and Foxcroft *et al.* (2007), and shown in Fig. 17.3 can be neutralized by deliberate selection. The critical issue is proper inclusion of robustness traits in the breeding goal, and in the selection criteria.

4. Selection Strategies for Improved Robustness

There are two options for breeding for animal robustness. These can be implemented simultaneously in an evaluation system for *performance-relevant robustness*. Section 3 illustrated a *direct approach*, which simply involves the inclusion of directly measurable robustness traits in the breeding objective and in the selection index. Because of low heritabilities, the selection criteria should be based on BLUP as opposed to phenotypic performance. The estimated breeding values can be formally weighted into the breeding goal by MEVs such as from Equation 17.2, which results in a

combination of robustness and production traits in the breeding goal: many markets today would welcome robust livestock, but not at the expense of production levels. Although this seems very obvious from evidence like that presented in Table 17.1, it is often overlooked.

An *indirect approach* involves the use of reaction norms analysis (Lynch and Walsh, 1998, pp. 672–685) to estimate breeding values for the environmental sensitivity of the genetic potential for production performance. Environmental sensitivity in livestock was studied through this method by Schinckel *et al.* (1999; pigs), Calus *et al.* (2002; cattle), Kolmodin *et al.* (2002; cattle), Pollott and Greeff (2004; sheep), Hermesch *et al.* (2006; pigs), Knap and Wang (2006; pigs) and Maricle *et al.* (2007; cattle). Figure 17.5 shows reaction norms for litter size in pigs, as estimated by Knap and Su (2008). This approach requires that progeny of specific sires is identified as such, is spread across a wide environmental range (usually through AI) and is recorded for the production trait of interest. The production performance (vertical axis in Fig. 17.5) is then regressed on a descriptor of the environment (e.g. a herd-year-season effect as on the horizontal axis of Fig. 17.5), which would be expected to produce a positive slope overall: better environments lead to better production. This can be done not only across the whole dataset, but also separately for the progeny group of each sire, and if there is genetic variation in environmental sensitivity of the production potential, this will produce regression lines ('reaction norms' in population genetics) with different slopes for different sires, whereas the intercept is equivalent to the conventional breeding value for the trait of interest.

Such an analysis quantifies an animal's requirements for environmental support of its genetic potential. It therefore detects robustness as defined above: animals 'that combine a high production potential with resilience to external stressors, allowing for the expression of the high production potential in a wide variety of environmental

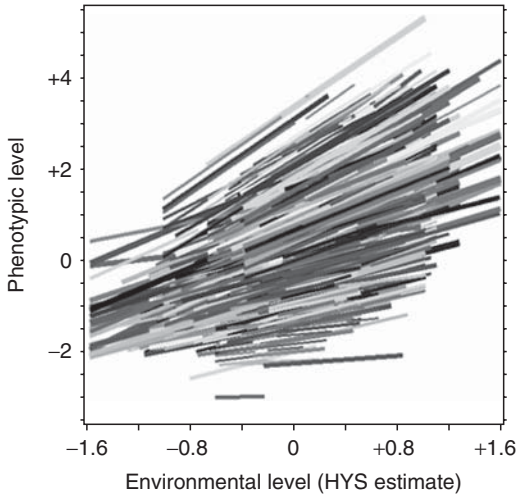

Fig. 17.5. Reaction norms for litter size in pigs. Each of the 2040 trendlines represents the performance of daughters of a particular sire, in relation to the production environment. (From Knap and Su, 2008.)

conditions'. An animal with a reaction norm with a steep slope is environmentally sensitive; from the point of view of sustaining production, it is non-robust. In the dataset behind Fig. 17.5, the reaction norm slopes have a genetic correlation to the intercepts of 0.7. Based on this, a routine breeding programme that selects for increased production levels only (equivalent to high intercepts here) will result in a gradual increase of the slopes, i.e. of environmental sensitivity. The same phenomenon was signaled by Kolmodin *et al.* (2003) and Van der Waaij (2004) and is further discussed by Friggens and Van der Waaij in Chapter 18. Because the reaction norm slopes have a very low heritability (h^2 = 0.02 in the data of Fig. 17.5), this increase in environmental sensitivity will be very slow. And, as in Section 3, it can be neutralized by including the reaction norms slope into the breeding goal (with a negative MEV, that is a function of the environmental spread among commercial conditions, see Knap, 2005 for details) and the selection index.

As noted by Friggens and Van der Waaij (Chapter 18), a single trait's reaction norm is equivalent to 'a single-trait definition of robustness' and, as such, not a simple and foolproof recipe that can be applied without proper care for the system as a whole. As they emphasize, an animal

> that maintains […] production in the face of decreasing [environmental support] is deemed to be robust but in order to maintain this production in the face of declining resources [it] must be diverting nutrients away from other life functions. […] When robustness can be understood as the ability to cope with environmental challenges […] an animal that maintains milk production by suppressing its immune function is clearly less robust than an animal that maintains milk production by reducing growth rate.

Which means that: (i) it becomes really interesting to what extent the environmental sensitivity rates of the various production traits are correlated among each other; and (ii) the breeding goal has to be designed and monitored with care, as always.

The combination of both approaches leads to a breeding objective that gives weighting to direct robustness traits in proportion to their impact on overall productivity, and to environmental sensitivity in proportion to the environmental spread among commercial conditions. Both approaches have strong demands for data collection on individually identified and pedigreed animals in commercial conditions. Fitness traits, with their low heritabilities and problematic data recording, provide the classical case for DNA marker-assisted selection schemes, and this applies equally well to reaction norm slopes. Wherever marker-assisted selection is useful for genetic improvement of production traits, it is currently so in terms of the intercept of the reaction norm, and it seems logical to shift the attention to the slope.

5. Conclusions

The above leads to the following conclusions:

1. Incorporation of robustness traits in the profit function for pig production shows that genetic improvement of these traits would have an economic value similar to the value of genetically improving production traits.

2. Breeding for robustness can be arranged in two ways. First, fitness-related traits such as leg soundness, mortality rates, longevity and disease resistance can

be included in the selection criterion in addition to production traits. Second, the environmental sensitivity of the expression of production traits can be quantified through reaction norms, and this sensitivity can be included in the breeding objective in addition to the genetic potential for those same production traits.

3. Both options require extensive data collection and sophisticated data processing to be successful. Marker-assisted selection will be most useful in this particular area with its low heritabilities and strong influences of the production environment.

4. The second option is potentially more powerful than the first one because it allows (when properly parameterized) for an economically optimal balance between robustness and production potential, dependent on the demands for robustness of any particular production environment. This should allow for unproblematic expression of high production potential in a wide variety of environmental conditions.

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18 Modelling of Resource Allocation Patterns

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1. Introduction

The aim of this chapter is to provide an introduction to the modelling of resource allocation, and to demonstrate that this is a powerful way to generate insights and to make predictions. Resource allocation models provide a simple and elegant framework for considering genotype × environment (G × E) interactions. In these models, one is forced to consider G × E in terms of multiple traits rather than the traditional single-trait approach exemplified by the slopes of reaction norms. The reaction norm approach, examining for different genotypes the relation between environmental quality (EQ) and the phenotypic level of a given trait, is in itself an elegant tool for describing G × E in single traits (David *et al.*, 2004). However, the reaction norm slopes are often *interpreted* as being a measure of the animals' robustness. This interpretation, a single-trait definition of robustness, is simply inadequate. Under this interpretation, a cow that maintains milk production in the face of decreasing nutrient availability is deemed to be robust, but in order to maintain this production in the face of declining resources she must be diverting nutrients away from other life functions. The single-trait definition of robustness completely ignores this consequence: an animal that maintains milk production by suppressing its immune function is clearly less robust than an animal that maintains milk production by reducing growth rate – when 'robustness' can be understood as the 'ability to cope with environmental challenges' (see also Chapter 17). Resource allocation models avoid this problem because they explicitly examine the partition of resources between different life functions: they provide a framework for a multi-trait definition of robustness. They also provide a framework for exploring trade-offs: the situation where selection pressure or the environment directly causes a change in the partition of resources.

Given that resource allocation models examine the partition of resources it is easy to see their practical relevance in animal nutrition. Being able to predict the proportion of dietary intake that is partitioned to production is an issue to which a substantial research effort has been devoted (Yearsley *et al.*, 2001; Friggens and Newbold, 2007). Indeed, some of the nutritional models used in this endeavour at

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first glance seem to be the same as resource allocation models. However, there is a crucial difference: resource allocation models as discussed here combine considerations of resource partitioning with considerations of the (resulting) fitness of the animal. Consideration of resources alone (as is commonly the case in nutritional models) does not allow optimum patterns of resource allocation to be deduced, nor does it allow the prediction of G × E. The models discussed here are designed to allow incorporation of the underlying genetic drives and other adaptations that animals use, through the consideration of their effects on fitness.

2. The Basis of Resource Allocation Models

In a basic resource allocation model, the actual resources obtained by the animal (R_O) are partitioned between two functions: in this example these are production (R_{Prod}) and the sum of other life functions, i.e. survival plus reproduction (R_{SR}). In this model, it is assumed that the partition of resources to 'survival/reproduction' equals 'c', the resource allocation coefficient, and that to 'production' '$1 - c$'. R_O, R_{SR} and R_{Prod} are all expressed in the same units (because $R_O = R_{SR} + R_{Prod}$), for example, MJ/lifetime or MJ/day. The extensions from Fig. 18.1A shown in

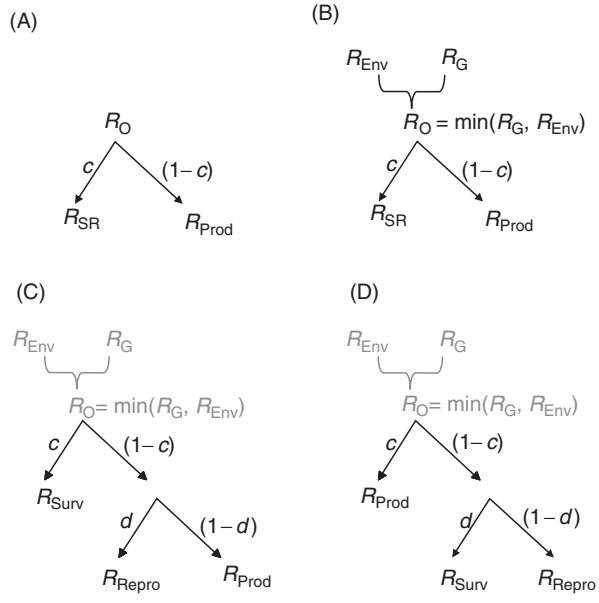

Fig. 18.1. (A) The base element of a resource allocation model showing allocation of resources to production (R_{Prod}) and survival/reproduction (R_{SR}). The development of the base element is described in detail in the text of following sections. (B) Extension of the base element of a resource allocation model to include the effect of the environment on resources obtained. R_G is the genetic ability to acquire resources (resource units/time unit; e.g. MJ/day). R_{ENV} is the availability of resources within the environment (e.g. MJ/day). (C and D) Two possible hierarchical structures for expanding the resource allocation model to include three traits.

Fig. 18.1B–D are discussed later in the chapter. First there is a need to consider the base model.

Figure 18.1A shows only the resources part of the model. It cannot, on its own, predict how c will change or indeed what an optimal c is. If $c = 0$, no resources are allocated to survival and producing offspring; if $c = 1$, there are no resources allocated to production. Both are unsuccessful strategies. Therefore, fitness values need to be ascribed to the life functions survival/reproduction and production. Solving resource allocation problems requires quantification of the contribution to fitness of the two traits. This is discussed in greater detail in Section 3.

For now, let us assume that we can quantify the contributions to fitness and that the total fitness of the animal is the product of the fitness value of survival/reproduction and production. Isolines of equal fitness can now be drawn, as shown in Fig. 18.2A. It is often the case that fitness values combine in a multiplicative way, e.g. number of offspring per litter and number of litters (Roff, 2002; Friggens, 2003). In contrast, the total amount of resources used is the sum of the resources used for each of the two traits. Thus, isolines of equal resource usage are as shown in Fig. 18.2B. If we know the energy required to increase production by one fitness unit and that of increasing survival/reproduction by one fitness unit, then we can combine the resource and fitness graphs. In Fig. 18.2C, it is assumed that both traits require the same amount of resources per fitness unit. Under these assumptions, it can be shown that the amount allocated that maximizes fitness is 50%, i.e. $c = 0.5$. Clearly, these outcomes depend upon the relationship between fitness and the two traits in question, but this graphical representation of the model illustrates the principles involved.

It seems intuitive to take this basic resource allocation idea, start adding in other life functions, and build up a hierarchy of pairs of life functions. But we first need to consider the issues involved in incorporation of fitness in the model, and incorporation of the environment. In both cases, the time-step of the model has important consequences.

3. Incorporating Fitness

A prerequisite for optimizing resource allocation is that the traits being examined contribute to fitness. A trait that makes no contribution (positive or negative) to fitness is not amenable to selection pressure and cannot be optimized within the model. Likewise, a trait that has become fixed, i.e. shows no variability, cannot be accommodated within the model.

In addition, it is important that the definition of the component traits is adequate to the part of fitness being examined. For example, one could model the optimum resource allocation between the traits 'number of offspring per reproductive cycle' and 'size of offspring'. In this case, that what is being optimized is the contribution to fitness of litter mass and not the total fitness of the animal. An associated issue is that of the relevant time-step for the model. Any useful definition of (some level of) fitness invokes a time period, e.g. *lifetime* reproductive success or litter mass per *reproductive cycle*. As discussed later in this chapter, there are some issues associated with the choice of the time-step being modelled. In this section we examine incorporation of fitness within a model using a time-step of a generation.

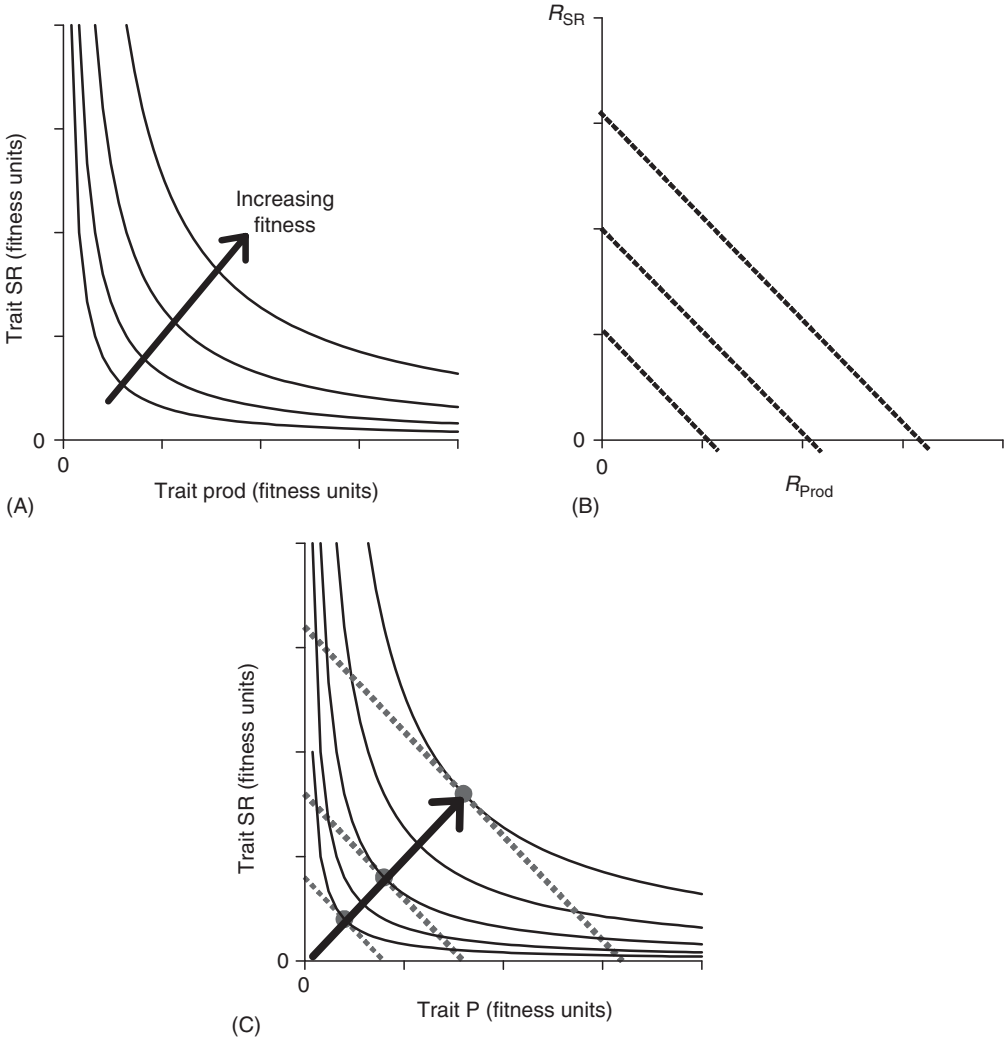

Fig. 18.2. The relationship between resources and fitness. Panel A shows an example of two component traits of fitness: survival/reproduction (SR) and production (Prod), where total fitness = SR × Prod. When considered purely in terms of fitness, i.e. when the resources required are ignored, many different combinations of SR and Prod can give the same level of fitness. Panel B shows the relationship between SR and Prod in terms of resources (R) required. A line of equal total $R = R_{SR} + R_{Prod}$. In an environment that can only supply this amount of resources, only those combinations of trait *R* and *S* that lie within the triangle between the axes and the resource isoline are possible. Panel C shows that there is only one partition of resources between SR and Prod that maximizes fitness (solid circles, arrow). The slope of the arrow is '*c*', the resource allocation coefficient.

In order to incorporate fitness on this timescale, we need to put our resource allocation model within some genetic structure. Given that we can calculate the probability of contributing to the next generation, then the genetic structure permits the model to iterate through generations showing the long-term consequences of a particular situation. As shown in Fig. 18.3, this requires a selection gate, i.e. criteria

for which animals actually contribute to the next generation. The selection gate controls the probabilities of being selected, i.e. the probability of contributing genes to the next generation. In the context of artificial selection, this allows number of sires and number of dams, as well as mating strategies to be included. In the context of natural selection, this allows population size (stable or expanding) and pairing probabilities to be included. It also implies that there is genetic variability in some of the model parameters. A stochastic element needs to be included that describes the variation that occurs due to the re-combination of genes that occurs with mating.

The genetic architecture could in itself be represented in increasingly complex ways and there are a number of examples of this (Kristensen *et al.*, 2005; Mulder *et al.*, 2007), but as it is not the purpose of this chapter to describe this, we will only highlight those issues that are exclusively pertinent to our resource allocation model. In Fig. 18.3, examples of fitness traits are 'lifetime reproductive success' and 'production'. These are not the same as R_{SR} and R_{Prod}, so we need functions that relate *the resources used* for survival/reproduction and production to the *fitness outcomes* of these two processes.

The notion of fitness outcomes holds for both natural and artificial selection, the only difference being the extent to which the different traits contribute to fitness. In the farm animal context, the trait under artificial selection pressure, i.e. production, makes a big contribution to fitness since production level directly affects the probability of being selected to contribute to the next generation. This probability is obviously a theoretical entity, but has a direct measurable outcome, which is number of offspring in the next generation. Clearly, in the context of natural selection, production is not a virtue in itself and a more meaningful model would start by considering the trade-off between survival and reproduction.

The relativity between R_{SR} and R_{Prod} in fitness terms must be controlled by two slopes, which are the relative fitness values of an extra unit of resources used for survival/reproduction and production. Here, for simplicity, we assume that the number of offspring produced is linearly related to R_{SR}, between the lower limit L_{SR} and upper limit U_{SR} with a slope α. L_{SR} implies that there is a level of investment below which there can be no survival and therefore number of offspring is 0; U_{SR}

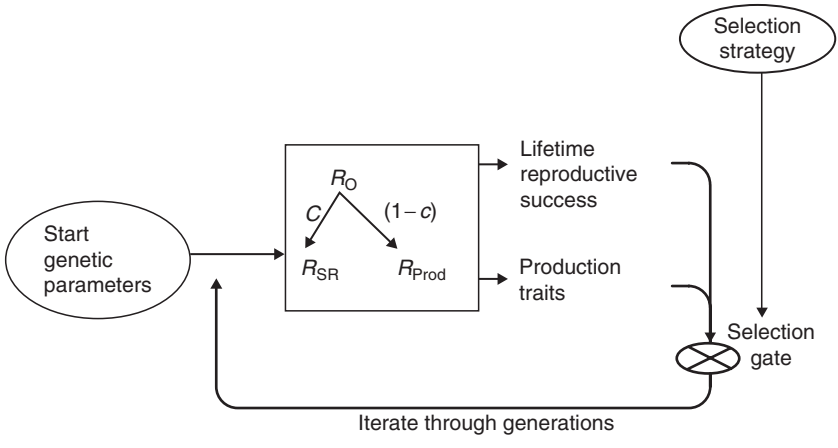

Fig. 18.3. A model architecture for resource allocation with a time-step of a generation.

implies that there is a level of resources beyond which investment in survival/reproduction has no further value. This could represent a maximum reproductive capacity within the time unit being modelled. The fitness value of R_{SR}, i.e. the probability of contributing genes to the next generation is then:

$$p(\text{SR}) = \alpha \times (\text{R}_{\text{SR}} - \text{L}_{\text{SR}}), \text{ when } \text{R}_{\text{SR}} \text{ is between } \text{L}_{\text{SR}} \text{ and } \text{U}_{\text{SR}} \quad (18.1)$$

$$p(\text{SR}) = 1, \text{when } R_{\text{SR}} > U_{\text{SR}} \text{ and}$$

$$p(\text{SR}) = 0, \text{when } \text{R}_{\text{SR}} < L_{\text{SR}}$$

When the fitness value of R_{SR} is expressed as a probability of contributing genes to the next generation, $\alpha = 1/(U_{SR} - L_{SR})$. Even when sufficient resources are allocated to survival/reproduction, the animal may not necessarily survive or reproduce (accidental death etc.). This can be simulated by drawing a random number from a standard normal distribution. If the draw has a value below p(SR) the animal is assumed to survive and be able to reproduce, whereas if the value is above p(SR) this will not be the case. It is possible that an animal will survive, and will be selected, but is not able to reproduce due to insufficient resources allocated to survival/reproduction.

A similar process can be applied to R_{Prod} when production is the trait under direct selection. Low production, relative to the rest of the population, will result in a low probability of being selected. Whether or not the animal will contribute genes to the next generation on the basis of its production level will depend on two factors: the population size and the number of animals to be selected. We have deliberately avoided discussing the selection intensity because both the population size and the number of animals selected can vary according to the selection criteria (Mulder and Bijma, 2005) and resource availability (RA).

The ratio between R_{Prod} and the actual product amount (e.g. kg milk or kg lean meat) can be described by a coefficient γ. The fitness value of R_{Prod} is then:

$$p(\text{Prod}) \ \delta \times \gamma \times \text{R}_{\text{prod}} \quad (18.2)$$

where δ is the relative importance of the production level in the selection criteria applied at the selection gate, i.e. the relationship between R_{Prod} and the probability of contributing genes on the basis of production level. In this case, lower and upper limits are not relevant (maintenance costs are a part of survival and not production).

Given that we have defined the relationships between resources and the probability of contributing genes to the next generation, i.e. fitness, then the next step is to define how the two traits combine to give overall fitness. Clearly, the shape of the function that combines the two traits is crucial for finding the optimum trade-off as it controls the shape of the fitness isolines. The rationale for, and different ways of, combining fitness traits has been discussed extensively by Stearns (1992) and Roff (2002).

Assuming that the probabilities are multiplicative, the overall probability of being selected is (when R_{SR} is between L_{SR} and U_{SR}):

$$p(\text{Select}) = p(\text{SR}) \times p(\text{Prod}) = (\alpha \times \text{R}_{\text{SR}})(\delta \times \gamma \times \text{R}_{\text{prod}}) \quad (18.3)$$

For each iteration of the model, this allows the animals that can reproduce (those that have allocated a sufficient amount of resources to survival/reproduction) to be

ranked according to p(Select) at the selection gate and thus provide the next generation in the model run.

4. Incorporating Environmental Factors

The above equation, and indeed the model in Fig. 18.1A, does not explicitly invoke the environment other than referring to resources *obtained* (R_O). The word 'obtained' clearly distinguishes R_O from RA and *ability to acquire* resources. It indicates that R_O is an outcome. What determines R_O?

The environment plays a role by causing variation in availability of resources, so we postulate an R_{Env}, the resources available within the environment. We also know that there is between-animal variation in intake when kept under *ad libitum* feeding (at the same physiological stage; Van Arendonk *et al.*, 1991; Von Felde *et al.*, 1996), i.e. we can postulate a genetic component, R_G, i.e. the animal's ability to acquire resources. We then need some function for determining R_O; for simplicity we choose the minimum of the two (Fig. 18.1B). This is merely stating that when the environment is sufficiently abundant, the animal eats according to its potential, and when the environment does not provide a sufficient amount of resources, intake is constrained to that which the environment can provide. Recent developments by, e.g. Tolkamp *et al.* (2006) propose refinements to this that are worthy of serious consideration, but which we will not discuss further here.

R_{ENV} can be generated from an RA which ranges from 0 to 1 as follows:

$$R_{ENV} = y(RA^b) \tag{18.4}$$

where y gives the resource units per day when RA = 1, and b controls the curve shape (when $b = 1$, this is a linear function). This last equation allows flexibility in implementing the relationship between the resources available from the environment and the level of resource intake that this environment allows. There is no a priori reason for assuming a linear relation between the two. Given that intake of bulk limiting feeds is related to size, it is tempting to consider making R_{ENV} size-dependent. This could be done by making 'y' a function of live weight, but as size-scaling is a topic in its own right (see Martin *et al.*, Chapter 9, this volume) this is not further discussed.

The above describes the environment in terms of its effects on RA, which can affect life functions such as survival through a reduced R_O. However, the environment is more than just 'resource availability'. Factors such as, e.g. predator or pathogen density can affect the probability of survival and reproduction independently of RA. To deal with this in the model, the environment is split into two quantities: RA and EQ. These can then be treated as independent factors allowing direct effects of the environment on the fitness functions. Evading predators, combating pathogen challenges and maintaining body temperature in extreme temperatures all require resources. Thus, it can be envisaged that the lower limit of R_{SR} required for survival/reproduction, L_{SR}, depends on EQ. Assuming that EQ ranges from 0 (hell) to 1 (paradise) then $1 - EQ^b$ reverses the scale and allows a non-linear relationship between L_{SR} and EQ:

$$L_{SR} = n * (1 - EQ^b) + m \tag{18.5}$$

where m is the value of L_{SR} when EQ = 1 and $(m + n)$ is the value of L_{SR} when EQ = 0. A logical extension of this would be to make U_{SR} a function of L_{SR} since any EQ effects will also impose a 'tax' on the total fitness per unit resource function.

Finally, we can expect variation across time in environmental conditions (i.e. EQ and RA). As will become apparent from consideration of the above graphs (and the following scenarios), without an unpredictability in the environment (RA and/or EQ) it becomes rather easy to find one optimum resource allocation that suits a wide range of environments. In other words, such a model predicts that genetic variation in resource allocation will be selected against. This is not the case in the real world (Stearns, 1992).

One way of including random environmental variation would be to introduce stochasticity not just in the animal parameters, but also in the environmental ones. R_{ENV} can be made stochastic between generations by drawing RA from a distribution characterized by a mean and standard deviation such that RA = mean + τ (standard deviation), where τ is a model constant that controls the degree of stochasticity. This could be a random draw from a standard normal distribution. A similar approach could be applied to EQ. Clearly, the way in which RA and EQ change with time can be described in many ways, the choice which ultimately depends on the purpose of the model and the time-step chosen.

5. Selection in Different Environments

Having developed the conceptual equations for relating fitness to resources and for incorporating environmental effects, we can examine this very simple model. We can independently vary the average genotype and the average environment: we have created a framework within which it is possible to generate G × E effects. In this section, we explore the effects of different environments in two ways. First, we examine our framework using first principles, deterministically, to check whether our assumptions seem reasonable. Second, we simulate the same scenarios within a stochastic model, a version of the Van der Waaij model (2004) modified to contain the above equations.

5.1 Constant environmental quality, non-limiting resource availability

Assume that the EQ is constant and RA is non-limiting, i.e. $R_G < R_{Env}$ and thus $R_O = R_G$. The graph in Fig. 18.4A shows the effects of selection under these conditions, assuming that there are upper and lower limits to the probability of contributing genes to the next generation. If the initial values of either R_G or c are such that $R_G \times c$ is less than L_{SR}, then the animal dies, i.e. fitness below this level = 0. If $R_G \times c$ is between L_{SR} and U_{SR} and, as shown in Fig. 18.4A, c is already at the optimum, then selection will increase R_G (but not c because it is already optimal) until $R_G \times c = U_{SR}$. However, if at the start of selection c is not optimal, then c is expected to move towards a stable optimum value defined by the relativity between the two slopes described above, and then to remain unchanged. In this context, optimal means the combination of investment in production and survival/

reproduction that maximizes the probability of contributing genes to the next generation under the prevailing selection pressure and environmental conditions. If the relative selection weights for production and survival/reproduction change, then *c* will no longer be optimal. Likewise, if environmental conditions change, e.g. to a higher disease burden, and thus require a greater resource investment per unit fitness, then *c* will also no longer be optimal. This means that if we observe, under non-limiting RA conditions, that selection changes *c*, then this implies that the

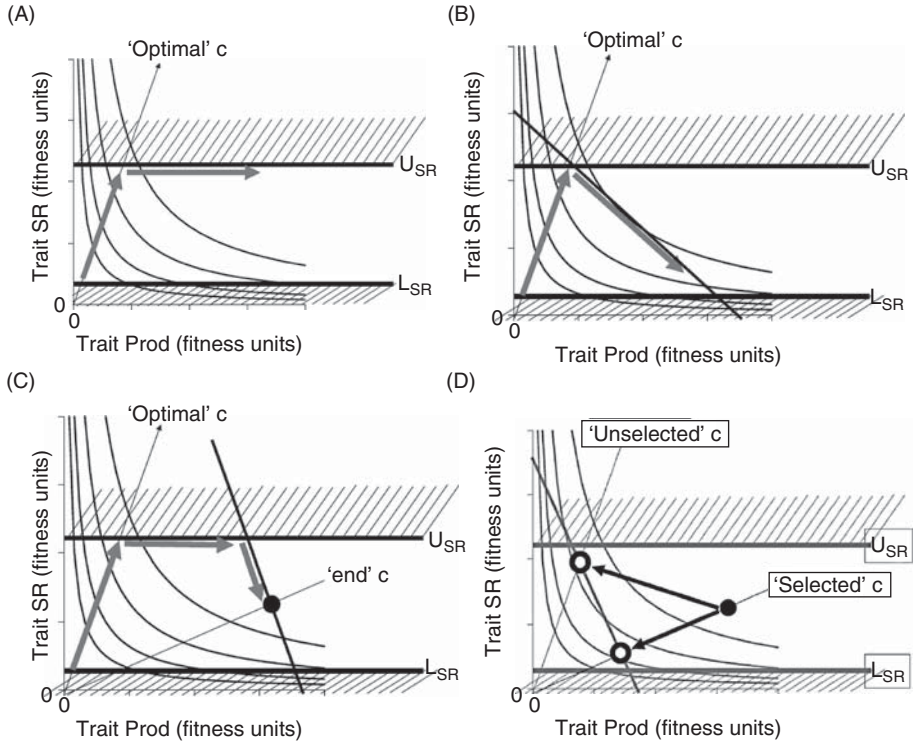

Fig. 18.4. (A) Selection for production under constant, non-limiting resource availability (RA). The arrows indicate the selection trajectory. The hatched areas indicate that above (for U_{SR}) and below (for L_{SR}) these limits fitness cannot change (and thus the fitness isolines no longer apply). (B) The selection trajectory when selecting for production under constant, limiting RA. (C) A selection scenario that genetically modifies the resource allocation coefficient, *c*, because eventually RA becomes limiting. (D) Possible consequences of selection for production on plasticity. If the environment now becomes poorer (indicated by the RA line sloping diagonally from upper left to lower right), then two extremes of partition are possible: no change in *c* indicating low plasticity (open circle on the line 'Selected'), and a complete reversion to the resource allocation of the unselected animal indicating high plasticity (open circle on the line 'Unselected'). Given that the partition of the unselected animal reflects the optimum fitness, it can be seen that the cost of selection for increased production in the low plasticity, 'Selected', animal is a substantial reduction in fitness when placed in a limiting environment.

current selection pressure or environmental challenge is different than that to which the animal was previously adapted – provided that $R_G \times c < U_{SR}$.

If $R_G \times c$ is greater than U_{SR}, then there is no benefit in further investing in survival/reproduction, therefore, selection on R_{Prod} will proceed by decreasing c and simultaneously increasing R_G so that $R_G \times c$ decreases to U_{SR}. In practice, it can be shown that $R_G \times c$ approaches U_{SR}, but does not actually reach it completely. This is because there is the stochastic element to the transfer of genes across generations, i.e. there will always be some offspring that have values of, e.g. R_G lower than the R_G values of both parents (stochasticity is explicitly discussed in Section 6). Thus, in this scenario, selection for production initially modifies R_G, but ultimately puts selection pressure on both R_G and c. Consequently, animals will be selected that have high intake capacity *and* allocate a large proportion of the resources obtained to production. The assumption that there is an upper limit to the fitness benefit of allocating resources to survival/reproduction (U_{SR}) is rather important. Without it, there would be one universal optimum value of c across all environments. Accepting U_{SR} results in a non-linear change in c across environments.

One might envisage that selection for production, if strong enough, would eventually result in animals that invest less in survival/reproduction than the maximum (U_{SR}). This follows from the notion that, for a given level of resources obtained, allocation of fewer resources to survival/reproduction will allow more resources to be allocated to production. However, because we are examining a situation of non-limiting RA, there is always an animal that achieves this higher level of production and also (through an even higher intake) can have maximum investment in survival/reproduction. Although this is so in our theoretical situation, consideration of the consequences of a truly non-limiting environment leads us to question the limitations imposed by the basic design of the animal. Evolution, across species and genera, has been able to alter basic designs to a remarkable degree to suit different situations, but within species there are design limits (see Naya and Bacigalupe, Chapter 4), and some traits have become fixed and are no longer amenable to selection. This is an important point that must be considered when simulating future consequences of selection.

Previously, we stated that the fitness function for R_{Prod} did not need any limits. For some species such as cattle, current evidence relating to the metabolic efficiency and production abilities suggests that this may be a reasonable assumption (Bauman, 2000). However, there must, ultimately, be limits to rates of production. For example, the supply of resources and disposal of waste products of metabolism is ultimately limited by the capacity of the vascular system. There is evidence from highly selected broiler chickens of an increasing vulnerability to ascites, a necrotic condition of the muscles caused by lack of oxygen (Beker *et al.*, 1995). This suggests that an upper limit to rates of production is worthy of careful consideration, especially if the model is to be used for predicting long-term consequences of selection.

5.2 Constant environmental quality, limiting resource availability

Clearly, a non-limiting environment cannot exist forever. As animals adapt to the higher RA, population sizes increase. At some point it will become limiting again, so we need to consider what we can expect to happen in a situation of limiting RA.

If RA is limiting for all animals, i.e. $R_G > R_{Env}$, and we have a model where U_{SR} and L_{SR} are constants not affected by fluctuation in the EQ, then selection will cause a real trade-off, as the only way to increase R_{Prod} is to decrease R_{SR} ($R_{Prod} + R_{SR} = R_O = R_{Env}$, which is constant). As shown in Fig. 18.4B, c will decrease and R_G is not expected to change as it is not in play (assuming that there is no a priori genetic correlation between R_G and c). If $R_O \times c > U_{SR}$, then the decrease in c has no cost. When $R_O \times c < U_{SR}$, then for a decrease in c to occur there will need to be a sufficient selection pressure applied.

We can imagine selection under controlled conditions where the animals involved start out being unconstrained by the environment, but then after a sufficient number of generations become constrained by the environment and reach an end point c value. Given a constant environment, no further change in c will occur. Such a scenario is shown in Fig. 18.4C. The net result is that the selected population now has a different average c as a result of selection: selection has favoured those animals with the highest production, and thus those with a relatively low resource investment in survival/reproduction. What happens if, after selection has occurred, we introduce a systematic change in RA, for example, a decrease in RA?

If selection has occurred in an abundant environment, then the partition of the selected animals in that environment is indicated by the solid black circle. If the environment now becomes poorer, then, as shown in Fig. 18.4D, two extremes of resource partitioning are possible: no change in partition indicating low plasticity (open circle on the line 'Selected'), and a complete reversion to the partition of the unselected animal indicating high plasticity (open circle on the line 'Unselected'). If the partition coefficient c is reduced by selection but the animal retains the plasticity to increase c in response to resource limitations, then we have both higher producing and more robust animals (open circle on the line 'Unselected'). This scenario has been postulated by some as being the situation in modern dairy cows (Collier *et al.*, 2005). Unfortunately, in the long term, this seems to be an unlikely outcome for a number of reasons. Simulation studies (Kolmodin *et al.*, 2003; Van der Waaij, 2004) and considerations of the costs of plasticity (West-Eberhard, 2003), i.e. maintaining plasticity, is itself a life function and thus subject to trade-off, suggesting that selection for increased production will reduce plasticity. Thus, it is likely that if the partition coefficient c is reduced by selection, the animal loses the plasticity to increase c in response to resource limitations and is less robust (open circle on the line 'Selected', Fig. 18.4D). Selected animals become increasingly adapted to the environment in which they were selected, and at the same time become increasingly sensitive to environmental changes.

6. Results from Stochastic Simulation

From first principles, the above conceptual framework seems adequate, but the anticipated consequences of selection under these controlled conditions ignore the stochasticity inherent in the process of sexual reproduction. Using the model of Van der Waaij (2004), we can simulate these scenarios with this stochasticity included.

In Fig. 18.5, three situations are represented in a single figure: selection for increased production in both a non-limiting and a limiting environment, and natural selection. At first, there are no limitations for most animals with respect to either

RA or EQ. Close to all animals survive and reproduce, and selection for production results in an increase in resource intake (R_G), and because of the increased selection pressure on production, a decrease in c (i.e. more resources allocated towards production, away from survival/reproduction). Selection thus drives the population average of $R_G \times c$ towards the upper threshold (U_{SR}). Then, as the environment

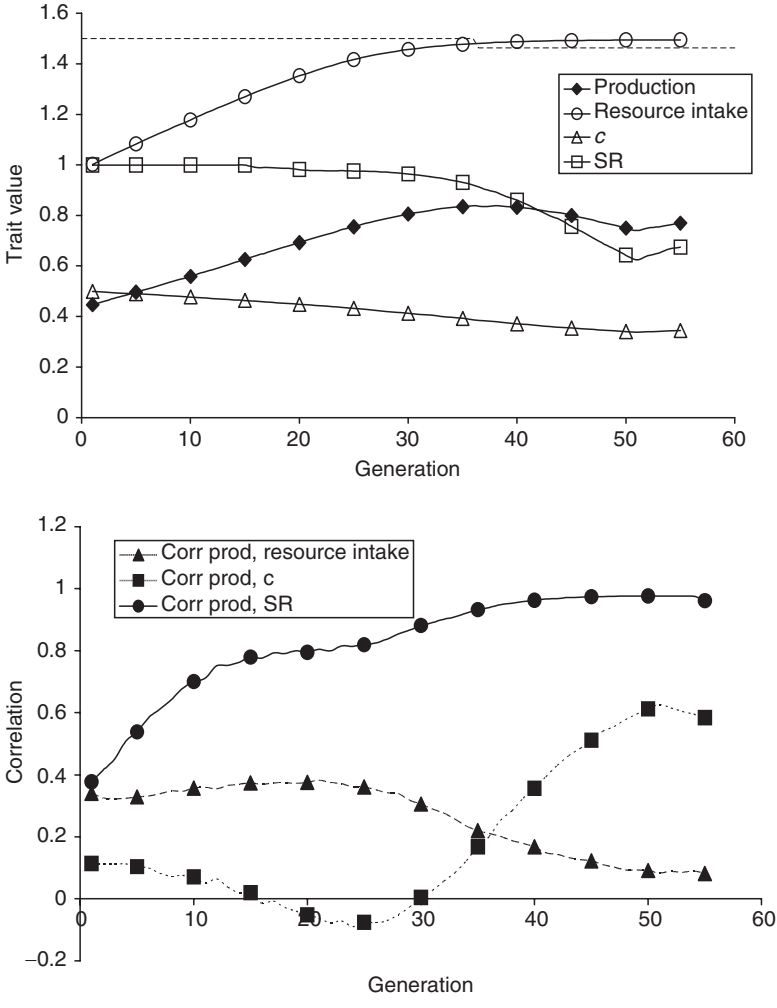

Fig. 18.5. Consequences of selection for production across generations, with a resource intake restricted at 150% of the intake at the start of selection, indicated by the horizontal dotted line in panel A. Initially, average R_G was set to be such that resources obtained (R_O) were 3 SD lower than the limit, so for most animals there was no limitation with respect to resource availability (RA) and environmental quality (EQ) was optimal. The population averages for production (in trait units), resource intake, survival/reproduction expressed as a proportion and the resource allocation coefficient c are shown in panel A. Panel B shows the correlations over generations between production and resource intake, survival/reproduction and c.

becomes limiting, progressively more animals start to run into problems, first represented by the correlation between survival/reproduction and production becoming stronger. From generation 15 onwards, the correlation between *c* and production becomes negative, indicating that a further decrease in *c* no longer has a positive influence on production because an increasing number of animals die (and thus have zero production). This occurs because we have imposed a biological design limit to intake capacity (R_G) of 150% of the initial value. Some of the animals will have reached the maximum resource intake capacity of 150% and the only possibility of increasing production is by a decrease in *c*. From generation 25 onwards, most of the animals have hit that resource intake ceiling. Eventually, only those animals with large enough *c* still manage to stay alive and produce and the correlation between *c* and production shifts to positive again (from generation 30 onwards). This is also represented by a decline in the increase in production and the decrease in survival/reproduction. To show the influence of natural selection, selection for production in the simulation was ceased from generation 50 onwards and all animals were allowed to reproduce according to their capability. As a consequence, those animals that are best capable of reproducing will have offspring that have higher probability of surviving and producing, etc. In other words, natural selection for higher fitness automatically occurs, resulting in increased survival/reproduction, *c* and production, and a decreased correlation between production and *c*.

These results agree with results of long-term selection experiments, where selection for increased body weight resulted in reduced reproductive performance in quail (Marks, 1996) and Turkey (Nestor *et al.*, 1996). Also, in farm animal breeding, similar consequences of selection for increased production have been observed (e.g. Rauw *et al.*, 1998; Kolmodin *et al.*, 2003).

This model is not able to deal with within-generation changes in, for example, *c* to deal with a different EQ. However, by doing simulations where animals are selected in one environment and then transferred to another, one can estimate how they manage with fixed values of *c* and Rg. Thus, some indication can be obtained on consequences of selection. Here we ignore the possible capacity of an animal to temporarily shift resource allocation to deal with environmental changes (plasticity).

Figure 18.6 shows the results of stochastic simulation, using the same model as that used in Fig. 18.5. Animals were selected in an intermediate environment (2) and moved into a better (3) and a worse (1) environment every so many generations for testing. Because of selection in environment (2), animals are adapted to the conditions (U_{SR} and L_{SR} and RA) in environment (2), gradually obtaining a level for *c* that is acceptable in environment (2). When the quality of the environment changes but the RA is unlimited (Fig. 18.6, first panel), the amount of resources required per unit fitness for survival/reproduction changes, and thus the optimal value of *c* is no longer equal to that in which selection in environment (2) resulted in. When transferred to the better environment, these animals have a higher than optimal value for *c*. If there was mortality in environment (2), this will now be reduced and thus production will be increased. This favours those animals with a lower value of *c*. Because, with increasing generations, the selection in environment (2) was gradually decreasing *c* (see Fig. 18.5), the more generations of selection in environment (2), the greater the gain in production when moved into the better

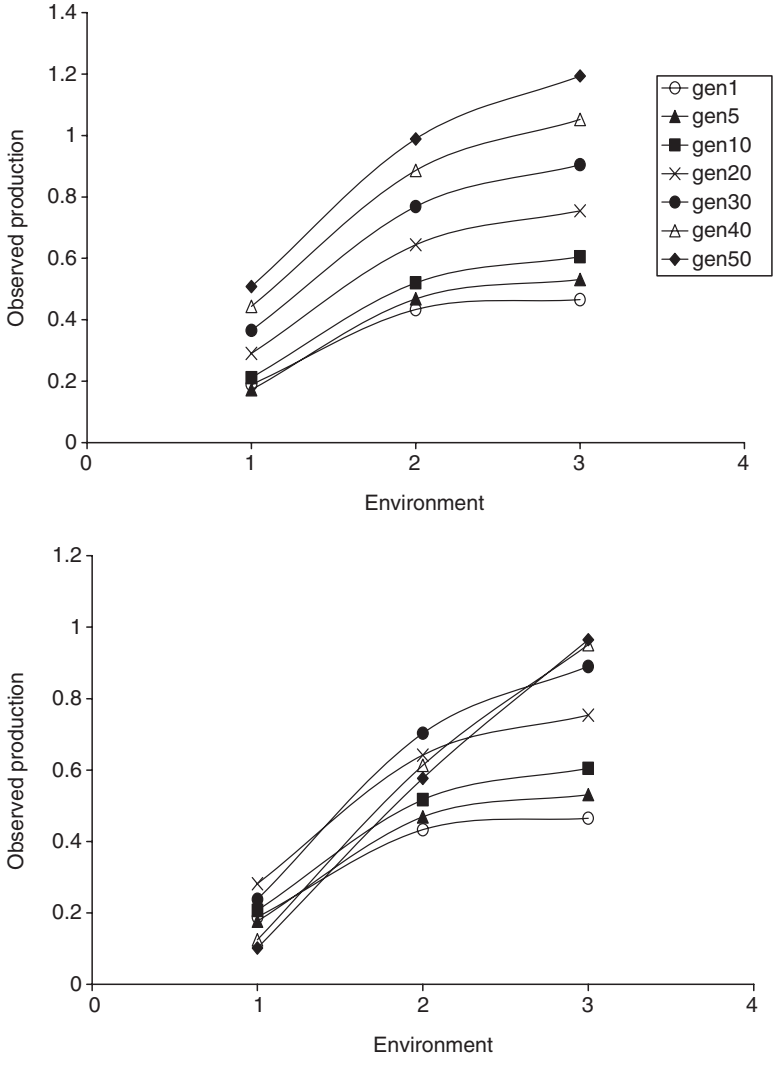

Fig. 18.6. Reaction norms for animals selected in an intermediate environment (2), who in generations 1, 5, 10, 20, 30, 40 and 50 were placed in a poor (1) or good (3) environment. The first panel shows this for a situation where resource availability (RA) is unlimited, and the second panel shows this where resource intake is restricted to 150% of the resource intake in generation 1. Environmental quality (EQ) was varied by shifting the thresholds L_{SR} and U_{SR}.

environment. For the same reason, the later generations have a greater drop in production when moved into the poorer environment. In the poorer environment the resource requirement per unit fitness in survival/reproduction is greater and thus animals with a higher value of c cope better.

In a situation where resources are not freely available, but limited to, for example, 150% of the original average intake, the consequences are slightly

more extreme (Fig. 18.6, second panel). After some generations of selection in environment (2), resources become limiting and selection pressure moves from Rg × *c* to *c* alone (Rg cannot change anymore; see Fig. 18.5, second panel). This trade-off has large consequences for mortality rates. Moving these animals to a better environment results in reduced mortality and thus a greater increase in average production than when selection occurred without resource constraint. Because selection pressure moves towards selection on *c* alone, moving the animals to a worse environment results in a higher mortality rate and thus a lower production level. Improving the environment as soon as the animals are getting into trouble results in animals that are less capable of dealing with a harsher environment. This is in agreement with results from Nielsen and Anderson (1987) who described the consequences of selection for growth in mice that were fed two different diets (two environments), and were transferred to the other diet after seven generations. Transgenerational adaptation to the environment of selection seems also to occur in practical farm animal breeding. Selection for increased milk production in a temperate climate has resulted in heat sensitive cows (Ravagnolo and Misztal, 2002; López-Gatius, 2003). Similary, fast-growing broilers show less heat tolerance than slow-growing ones (Yalcin *et al.*, 2001).

7. A Framework for Describing Plasticity

The graphical representation of resource allocation and trade-offs does two important things. It makes it very easy to see what the possible consequences of a change in environment are. And, crucially, it provides a way to describe plasticity that relates to total fitness. This implies that plasticity is defined in terms of more than one trait.

In agricultural science, a rather limited definition of plasticity is usually invoked: the rate of change in the level of a *single* trait when measured across different environments. This narrow definition of plasticity is often referred to as environmental sensitivity. When plasticity is defined in this way, it is unclear whether it is high or low plasticity that is desirable. It could be argued that we should select for animals that have low plasticity for milk production, i.e. those that have the least drop in production when exposed to a limiting environment (open circle on the line 'Selected' in Fig. 18.4D). On the other hand, it could be argued that we should select animals with high plasticity because low plasticity for milk production means that the gains that can be expected from improvements in the environment will be less than for cows with high plasticity (open circle on the line 'Unselected' in Fig. 18.4D). In contrast to this situation, as soon as the argument is extended to include other traits, in this case survival, then a clear difference between the two plasticity extremes emerges in terms of total fitness.

Plasticity and G × E, although often discussed in terms of one trait only, have limited biological meaning (Lewontin, 1974) unless they are interpreted in the context of the total fitness of the animal. In other words, a broader definition of plasticity than that usually used in agricultural science is necessary. Plasticity can be defined, in biological terms, as the combined physiological mechanisms by which

the animal copes with environmental challenge, i.e. it is inherently a multiple trait process. We can see that one way to approach this may be to describe plasticity in terms of the flexibility in the resource allocation coefficient, *c*.

8. Timescales

Answering questions about plasticity leads inexorably to the issue of the timescale of our resource allocation model. In the above discussion, we have chosen a time-step of a generation. This appears to be a logical time-step from a genetic standpoint and in many cases may be sufficient. However, the time-step of a generation can be a major limitation of the model. The consequence of choosing a time-step of a generation is that the resource part of the model comes to represent total or average lifetime values of the environment. It is not difficult to imagine two environments with same average R_{Env}, one which is constant over time and one which involves a period of superabundance together with a period where R_{Env} is zero. Clearly, the period of zero RA does not need to be very long before it has dramatic effects on survivability. It is precisely in such a situation of environmental 'challenge' that differences in plasticity come into play. The animal that has evolved the strategy of amassing body reserves prior to periods of nutritional challenge will be favoured. Capturing this effect requires some way of including these short-term challenges in the lifetime performance measures. When it is further considered that the efficiency of many responses to environmental challenge is enhanced by prior exposure (within that lifetime) to the challenge, e.g. immune responses, then it becomes obvious that capturing these qualities of animals requires resource allocation models that operate with much shorter time-steps than a generation.

Using a shorter time-step raises some modelling challenges. For instance, when using within lifespan time-steps it is necessary to represent the temporal expression of genes with age. In animals that are sexually immature, resource investment in reproduction is minimal and at the same time there is major investment in growth. Once the animal is mature the opposite applies. Clearly, the partition of resources between growth and reproduction changes dramatically as the animal ages, reflecting the temporal expression of the genes involved. This can be modelled by describing that part of these processes that is genetically determined or driven. For some processes such as growth (e.g. Emmans, 1997) and milk and egg production (Emmans and Fisher, 1986; Friggens *et al.*, 1999) this has been relatively well described. However, there is increasing evidence that there is a genetic component to the time-patterns of many of the processes that are involved in plasticity. This is not surprising since maintaining the capability to respond to environmental challenges has a cost (Diamond, 1998; West-Eberhard, 2003) and we can expect that evolution has favoured those animals that modulate this expenditure on plasticity according to the fitness consequences of not having this plasticity. One example of this relevant to farm animals is the genetically driven usage of body reserves to safeguard reproductive investment (Friggens, 2003; Friggens *et al.*, 2004). Although these plasticity processes are increasingly being described in terms that allow quantification of their temporal expression (Martin

and Sauvant, 2008), their incorporation into shorter time-step resource allocation models still remains a challenge.

9. Including a Hierarchy of More Than Two Life Functions

So far, we have kept the resource allocation model simple – although even this raises a number of questions and offers insights into the limitations of the single trait, univariate view. Now we consider briefly the expansion of our model to become a hierarchy of trade-offs. An obvious step would be to split survival from reproduction, which can be done in a number of ways. Given that negative genetic correlations between these traits have been found (Veerkamp *et al.*, 2001; Pryce *et al.*, 2002), it seems intuitive to do this by creating a second level with a trade-off between reproduction and production (Fig. 18.1C). However, because each level of the hierarchy must be related to fitness, this requires that we describe the relative fitness values of reproduction and production, and how they combine. An alternative would be to create the hierarchy shown in Fig. 18.1D, where the highest-level trade-off is between production and 'other fitness' and the next level is between survival and reproduction.

Deciding which of these structures is the most suitable depends to some extent on the selection context. If production is a major part of the selection pressure being applied, then it makes sense to implement this at a higher level than when selection pressure on production is small. Likewise, it is necessary to consider what is meant by production. Meat production invokes growth and size, which are often related with survival, whereas milk production is a component of reproduction and as such more likely to be correlated with other reproductive traits.

It may at first sight seem as if these questions about the model structure are an unnecessary and unwanted complication. After all, one could avoid this issue by allowing more than two traits to directly compete at the same level for resources. However, parameterization requires data that fully express all combinations of the traits, i.e. the full three-way interaction in the case of three traits. In practice, simplifying assumptions tend to be made to reduce the complexity and increase the chances of finding suitable data. Using a hierarchy of pairs of traits is a good way of forcing the modeller to simplify the model structure in a way that considers the key interactions between traits in terms of the underlying biology.

10. Concluding Remarks

Modelling resource allocation is a powerful way to generate insight and predictions. Resource allocation models have a wide range of applicability with examples of their use in predicting the effects of genetic selection (Roff *et al.*, 2002; Van der Waaij, 2004), nutrient partitioning (Friggens and Newbold, 2007; Martin and Sauvant, 2008), effects of early life performance on longevity (Stearns *et al.*, 1998; Ricklefs, 2006) and many other aspects of life history biology (Reznick *et al.*, 2000; Worley *et al.*, 2003). Thus, although models that explicitly use resource allocation theory have only recently been introduced in the context of agricultural science

(Beilharz *et al*., 1993) there is a long tradition of developing resource allocation theories within life history biology (Stearns, 1992; Roff, 2002). The prospect of combining the strengths of these two disciplines in the context of resource allocation is an exciting one. This approach has the potential to allow robust, quantitative, long-term prediction of G × E, a tool that is much needed in the context of adapting selection and management strategies to improve the robustness of modern livestock.

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