NATIONAL OPEN UNIVERSITY OF NIGERIA

SCHOOL OF SCIENCE AND TECHNOLOGY

COURSE CODE: CHS 501

COURSE TITLE: INTRODUCTION TO HIV/AIDS

COURSE GUIDE

CHS 501
INTRODUCTION TO HIV/AIDS

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Introduction

CHS 501: Introduction to HIV/AIDS is a 2 unit course. The course is broken into 4 modules and 12 study units. It will introduce the students to definitions of HIV and AIDS, the differences between HIV and AIDS, HIV/AIDS timeline, origin and theories of HIV/AIDS, the biology of HIV, its clinical progression, symptoms manifestations, drug adherence, HIV counselling and management

At the end of this course, it is expected that students should be able to understand, explain and be adequately equipped with basic concepts and issues in HIV/AIDS.

The course guide tells you briefly what the course: CHS 501 is all about, the type of course materials to be used, what you are expected to know in each unit, and how to work through the course material. It suggests the general guidelines and also emphasises the need for self assessment and tutor marked assignment. There are also tutorial classes that are linked to this course and students are advised to attend.

What you will learn in this Course

The overall aim of this course, CHS 501, is to present an overview of HIV/AIDS. During this course, you will be equipped with definitions of HIV/AIDS and differences between HIV and AIDS. You will also learn about the origin and theories of HIV/AIDS, the biology, clinical progression, symptoms and management of HIV/AIDS.

Course Aim

This course aims to give students an in-dept understanding of HIV/AIDS. It is hoped that the knowledge would equip students with the basic issues in HIV/AIDS.

Course Objectives

Note that each unit has specific objectives. Students should read them carefully before going through the unit. You may want to refer to them during your study of the unit to check on your progress. You should always look at the unit objectives after completing a unit. In this way, you can be sure that you have done what is required of you by the unit.

However, below are overall objectives of this course.

On successful completion of this course, you should be able to:

- Define HIV and AIDS
- Identify the differences between HIV/AIDS
- Identify global HIV timeline

- Explain origin of HIV/AIDS
- Explain theories of HIV/AIDS
- Understand the biology of HIV
- Understand clinical progression of HIV
- Identify symptoms of HIV
- Explain HIV mode of Transmission
- Explain types of HIV test
- Illustrate understanding of treatment adherence
- Understand HIV counselling
- Discuss psychological issues in HIV management
- Discuss HIV nutrition management

Working through this Course

To complete this course, you are required to read the units, the recommended text books, and other relevant materials. Each unit contains some self assessment exercises and tutor marked assignments. There is also a final examination at the end of this course. Stated below are the components of this course and what you have to do.

Course Materials

The major components of the course are:

1. Course Guide
2. Study Units
3. Text Books
4. Assignment File
5. Presentation Schedule

Study Units

There are 12 study units and 4 modules in this course. They are:

MODULE I HIV/AIDS AND IMMUNE SYSTEM

Unit 1 HIV/AIDS: An Introduction

Unit 2 HIV/AIDS: Evolution of a Pandemic

Unit 3 HIV/AIDS Timeline

MODULE 2: THEORIES OF THE ORIGIN OF HIV/AIDS

Unit 1 Origin of HIV/AIDS

Unit 2 Origin of HIV/AIDS (Cont)

Unit 3 Theories of HIV/AIDS

MODULE 3 THE BIOLOGY OF HIV

Unit 1 Understanding the Biology of HIV

Unit 2 Clinical Progression of HIV

Unit 3 HIV: Symptoms, Testing and Medication

MODULE 4: HIV COUNSELLING AND MANAGEMENT

Unit 1 HIV Counseling
Unit 2 Psychological Issues in HIV Management
Unit 3 HIV management: The role of Nutrition

Recommended Texts

These texts will be of immense benefit to this course:

NIAID (2010) HIV and AIDS.
http://www.niaid.nih.gov/topics/hivaids/understanding/Pages/whatAreHIVAIDS.aspx

Pratt, R. J. (2003). HIV and AIDS: *A Foundation for Nursing and Healthcare Practice*, 5th Edition. London: BookPower.

UNAIDS (2011) 'World AIDS Day Report 2011' – 2011. Joint United Nations Programme on HIV/AIDS.

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Alta van Dyk (2005). *HIV/AIDS care and Counselling: A Multidisciplinary approach. Third edition.* South Africa: Maskew Miller Longman.

Hubley, J. (1995). *The AIDS Hangbook (Second Edition).* London: MacMillan

Levert, S. (1987). AIDS in search of a killer. New York: Julian Messner.

Medical News Today (2012). What is HIV and AIDS.

ww.medicalnewstoday.com/articles/17131.php

D'Cruz, Premilla (2004). Family Care in HIV. SAGE.

Neill McKee, Jane Bertrand and Antje Becker-Benton (2004). Strategic Communications in the HIV/AIDS Epidemic. SAGE.

Richard Nelson-Jones (2005). Practical Counselling and Helping Skills. SAGE.

Assignment File

The assignment file will be given to you in due course. In this file, you will find all the details of the work you must submit to your tutor for marking. The marks you obtain for these assignments will count towards the final mark for the course. Altogether, there are 12 tutor marked assignments for this course.

Presentation Schedule

The presentation schedule included in this course guide provides you with important dates for completion of each tutor marked assignment. You should therefore try to meet the deadlines.

Assessment

There are two aspects to the assessment of this course. First, there are tutor marked assignments; and second, the written examination.

You are thus expected to apply knowledge, comprehension, information and problem solving gathered during the course. Your TMA will be presented in e-format and this account for 30% of your exam score. At the end of the course, you will need to sit for a final written examination. This examination will account for 70% of your total score.

Tutor Marked Assignments (TMAs)

You are expected to attempt all the TMAs in your study material. However, 4 TMAs will be uploaded in your portal. The best 4 will count towards your final exam grade.

Final Examination and Grading

The final examination for CHS 501 will be of 2 hours and have a value of 70% of the total course grade. The examination will consist of questions which reflect the self assessment exercise and tutor marked assignments that you have previously encountered. Furthermore, all areas of the course will be examined. It is also better to use the time between finishing the last unit and sitting for the examination, to revise the entire course. You might find it useful to review your TMAs and comment on them before the examination. The final examination covers information from all parts of the course.

Course marking Scheme

The following table includes the course marking scheme

Table 1 Course Marking Scheme

Assessment	**Marks**
TMAs	30 %
Final Examination	70%
Total	100%

Course Overview

This table indicates the units, the number of weeks required to complete them and the assignments.

Table 2: Course Organizer

Unit	**Title of Work**	**Weeks Activity**	**Assessment (End of Unit)**
	Course Guide	Week 1	
Module 1	**HIV and the Immune System**		
Unit 1	HIV/AIDS: An introduction	Week 1	Assignment 1
Unit 2	Evolution of a pandemic	Week 2	Assignment 2
Unit 3	HIV/AIDS time line	Week 3	Assignment 3
Module 2	**Theories of the Origin of HIV/AIDS**		
Unit 1	Origin of HIV/AIDS	Week 4	Assignment 4
Unit 2	Origin of HIV/AIDS (Cont)	Week 5	Assignment 5
Unit 3	Theories of HIV/AIDS	Week 6	Assignment 6
Module 3	**The Biology of HIV/AIDS**		
Unit 1	Understanding the biology of HIV/AIDS	Week 7	Assignment 7
Unit 2	Clinical progression of	Week 8	Assignment 8

	HIV/AIDS		
Unit 3	HIV: Symptoms, Testing and Medication	Week 9	Assignment 9
Module 4	**HIV Counselling and Management**		
Unit 1	HIV counselling	Week 10	Assignment 10
Unit 2	Psychological issues in HIV management	Week 11	Assignment 11
Unit 3	HIV management: The role of nutrition	Week 12	Assignment 12

How to get the most out of this course

In distance learning, the study units replace the university lecturer. This is one of the huge advantages of distance learning mode; you can read and work through specially designed study materials at your own pace and at a time and place that suit you best. Think of it as reading from the teacher, the study guide tells you what to read, when to read and the relevant texts to consult. You are provided exercises at appropriate points, just as a lecturer might give you an in-class exercise.

Each of the study units follows a common format. The first item is an introduction to the subject matter of the unit and how a particular unit is integrated with the other units and the course as a whole. Next to this is a set of learning objectives. These learning objectives are meant to guide your studies. The moment a unit is finished, you must go back and check whether you have achieved the objectives. If this is made a habit, then you will significantly improve your chances of passing the course. The main body of the units also guides you through the required readings from other sources. This will usually be either from a recommended text book or from other sources.

Self assessment exercises are provided throughout the unit, to aid personal studies. Working through these self tests will help you to achieve the objectives of the unit and also prepare you for tutor marked assignments and examinations. You should attempt each self test as you encounter them in the units.

The following are practical strategies for working through this course

1. Read the course guide thoroughly
2. Organize a study schedule. Refer to the course overview for more details. Note the time you are expected to spend on each unit and how the assignment relates to the units. Important details, e.g. details of your tutorials and the date of the first day of the semester are available. You need to gather together all these information in one place such as a diary, a wall chart calendar or an organizer. Whatever method you choose, you should decide on and write in your own dates for working on each unit.
3. Once you have created your own study schedule, do everything you can to stick to it. The major reason that students fail is that they get behind with their course

works. If you get into difficulties with your schedule, please let your tutor know before it is too late for help.

4. Turn to Unit 1 and read the introduction and the objectives for the unit.
5. Assemble the study materials. Information about what you need for a unit is given in the table of content at the beginning of each unit. You will almost always need both the study unit you are working on and one of the materials recommended for further readings, on your desk at the same time.
6. Work through the unit, the content of the unit itself has been arranged to provide a sequence for you to follow. As you work through the unit, you will be encouraged to read from your set books.
7. Keep in mind that you will learn a lot by doing all your assignments carefully. They have been designed to help you meet the objectives of the course and will help you pass the examination.
8. Review the objectives of each study unit to confirm that you have achieved them. If you are not certain about any of the objectives, review the study material and consult your tutor.
9. When you are confident that you have achieved a unit's objectives, you can start on the next unit. Proceed unit by unit through the course and try to pace your study so that you can keep yourself on schedule.
10. When you have submitted an assignment to your tutor for marking, do not wait for its return before starting on the next unit. Keep to your schedule. When the assignment is returned, pay particular attention to your tutor's comments, both on the tutor marked assignment form and also written on the assignment. Consult you tutor as soon as possible if you have any questions or problems.
11. After completing the last unit, review the course and prepare yourself for the final examination. Check that you have achieved the unit objectives (listed at the beginning of each unit) and the course objectives (listed in this course guide).

Tutors and Tutorials

There are 16 hours of tutorial provided in support of this course. You will be notified of the dates, time and location together with the name and phone number of your tutor as soon as you are allocated a tutorial group.

Your tutor will mark and comment on your assignments, keep a close watch on your progress and on any difficulties you might encounter and provide assistance to you during the course. You must mail your tutor marked assignment to your tutor well before the due date. At least two working days are required for this purpose. They will be marked by your tutor and returned to you as soon as possible.

Do not hesitate to contact your tutor by telephone, e-mail or discussion board if you need help. The following might be circumstances in which you would find help necessary: contact your tutor if:

- You do not understand any part of the study units or the assigned readings.
- You have difficulty with the self test or exercise.

- You have questions or problems with an assignment, with your tutor's comments on an assignment or with the grading of an assignment.

You should try your best to attend the tutorials. This is the only chance to have face to face contact with your tutor and ask questions which are answered instantly. You can raise any problem encountered in the course of your study. To gain the maximum benefit from the course tutorials, prepare a question list before attending them. You will learn a lot from participating in discussion actively. GOODLUCK!

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NATIONAL OPEN UNIVERSITY OF NIGERIA

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MODULE I HIV/AIDS AND IMMUNE SYSTEM

UNIT 1: HIV/AIDS: AN INTRODUCTION

CONTENT

1.0 INTRODUCTION

People have been warned about HIV and AIDS for so many years now. AIDS has already killed millions of people, millions more continue to become infected with HIV, and there's no cure - so AIDS will be around for a while yet. However, some of us still do not know exactly what HIV and AIDS actually are. This unit defines HIV/AIDS; illustrate the anatomy of HIV; explain the differences between HIV and AIDS as well as how long it takes for HIV to become AIDS. It further sorts the myths from the facts about AIDS. Enjoy your studies.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- Define HIV
- Define AIDS
- Identify anatomy of HIV
- Illustrate the difference between HIV/AIDS
- Identify how long it takes for HIV to become AIDS
- Identify the HIV facts and myths

3.0 MAIN CONTENT

3.1 What is HIV?

HIV stands for: **H**uman **I**mmunodeficiency **V**irus

HIV is a virus. Viruses such as HIV cannot grow or reproduce on their own, they need to infect the cells of a living organism in order to replicate (make new copies of themselves). The human immune system usually finds and kills viruses fairly quickly, but HIV attacks the immune system itself - the very thing that would normally get rid of a virus.

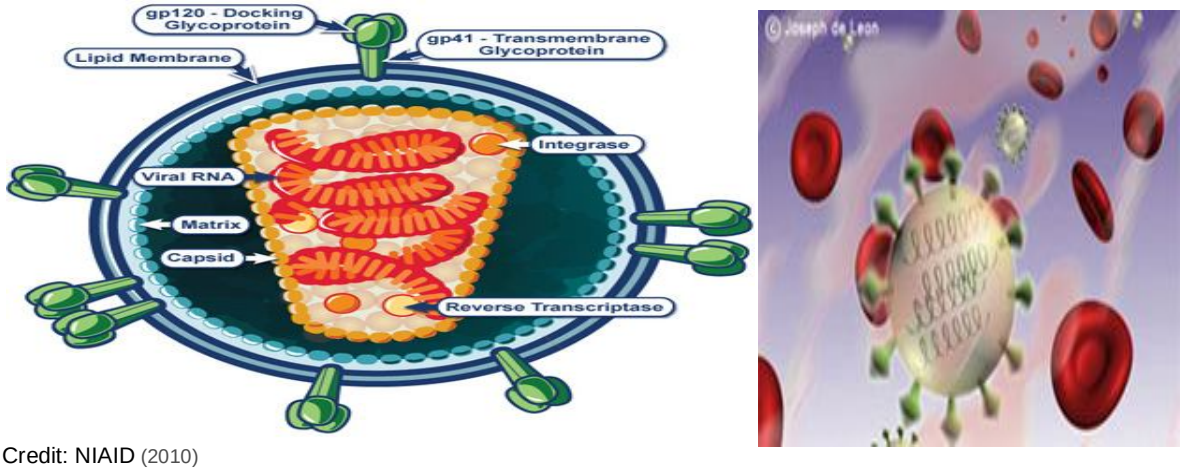
HIV is the virus that causes AIDS. People can become infected with HIV from other people who already have it, and when they are infected they can then go on to infect other people. Basically, this is how HIV is spread. Someone who is diagnosed as infected with HIV is said to be 'HIV+' or 'HIV positive'.

Over time, HIV can damage the immune system to such a degree that infections may begin to occur as a result of a weakened immune system. Eventually, one may acquire various illnesses due to the damage done by the virus. When this happens this is called AIDS or Acquired Immune Deficiency Syndrome. That is, a collection of illnesses.

AIDS is the final stage of HIV infection. A person infected with HIV is diagnosed with AIDS when he or she has one or more opportunistic infections, such as pneumonia or tuberculosis, and has a dangerously low number of CD4+ T cells (less than 200 cells per cubic millimeter of blood), (NIAID, 2010).

With around 2.7 million people becoming infected with HIV in 2010, there are now an estimated 34 million people around the world who are living with HIV, including millions who have developed AIDS (UNAID 2010)

3.1.1: Anatomy of HIV



Credit: NIAID (2010)

Note: Structure of HIV will be discussed in later units.

3.2: WHAT IS AIDS?

AIDS is a short for Acquired Immune Deficiency Syndrome. We say that this disease is acquired because it is not a disease that is inherited. It is caused by a virus (the human immunodeficiency virus or HIV), that enters the body from outside. *Immunity* is the body's natural ability to defend itself against infection and disease. A *deficiency* is a shortcoming – the weakening of immune system so that it can no longer defend itself against passing infections. A syndrome is a medical term for a collection of specific signs and symptoms that occur together and that are characteristic of particular condition (Alta van Dyk, 2005).

Although we use the term 'disease' when we talk about it, AIDS is not a specific illness. It is really a collection of many different conditions that manifest in the body (or specific parts of the body) because the HIV has so weakened the body's immune system that it can no longer fight the disease-causing agent that are constantly attacking it. It is therefore more accurate to define AIDS as a syndrome of opportunistic diseases, infections and certain cancers – each or all of which has the ability to kill the infected person in the final stage of the disease (Alta van Dyk, 2005).

3.3: What is the Difference between HIV and AIDS?

In simple terms - you cannot 'catch' AIDS. You can, however, catch HIV. Being infected with HIV does not mean that one has AIDS, but if left undiagnosed and/or untreated; HIV infection damages the immune system and can progress to AIDS.

AIDS results from the destruction of the immune system by HIV. The immune system's function is to fight off infections and other diseases.

If your immune system is damaged or not working well you are at risk of life-threatening infections and cancers.

HIV attacks and destroys the disease fighting cells of the immune system. The body is left with a weakened defense against disease.

In specific terms:

- HIV is the virus which attacks the T-cells in the immune system.
- AIDS is the syndrome which appears in advanced stages of HIV infection.
- HIV is a virus.
- AIDS is a medical condition.
- HIV infection causes AIDS to develop. However, it is possible to be infected with HIV without developing AIDS. Without treatment, the HIV infection is allowed to progress and eventually it will develop into AIDS in the vast majority of cases.
- HIV testing can identify infection in the early stages. This allows the patient to use prophylactic (preventive) drugs which will slow the rate at which the virus replicates, delaying the onset of AIDS.

- AIDS patients still have the HIV virus and are still infectious. Someone with AIDS can pass HIV to someone else (Medical News Today, 2012)

SELF ASSESSMENT EXERICE

Illustrate the difference between HIV and AIDS

3.4 How long does HIV take to become AIDS?

Without drug treatment, HIV infection usually progresses to AIDS in an average of *ten years*. This average, though, is based on a person having a reasonable diet. Someone who is malnourished may well progress to AIDS and death more rapidly.

Antiretroviral medication can prolong the time between HIV infection and the onset of AIDS. Modern combination therapy is highly effective and, theoretically, someone with HIV can live for a long time before it becomes AIDS. These medicines, however, are not widely available in many poor countries around the world, and millions of people who cannot access medication continue to die.

3.5 HIV facts and myths

Around the world, there are a number of different myths about HIV and AIDS. Here are some of the more common ones:

'You would have to drink a bucket of infected saliva to become infected yourself' . . . Yuck! This is a typical myth. HIV *is* found in saliva, but in quantities too small to infect someone. If you drink a bucket of saliva from an HIV positive person, you will not become infected. There has been only one recorded case of HIV transmission via kissing, out of all the many millions of kisses. In this case, both partners had extremely badly bleeding gums.

'Sex with a virgin can cure HIV' . . . This myth is common in some parts of Africa, and it is totally untrue. The myth has resulted in many rapes of young girls and children by HIV+ men, who often infect their victims. Rape will not cure anything and is a serious crime all around the world.

'It only happens to gay men / black people / young people, etc' . . . This myth is false. Most people who become infected with HIV did not think it would happen to them, and were wrong.

'HIV can pass through latex' . . . Some people have been spreading rumours that the virus is so small that it can pass through 'holes' in latex used to make condoms. This is untrue. The fact is that latex blocks HIV, as well as sperm - preventing pregnancy, too (Alta van Dyk, 2005).

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4.0 CONCLUSION

We hope you enjoyed your studies. We have defined HIV and AIDS as well as the differences between HIV and AIDS. We observed that it took a normal healthy body an average of 10years for HIV to become AIDS. Finally, we identified some the myths and facts of HIV. We encourage you to consult other text books for further reading.

5.0 SUMMARY

In this unit, we studied the following:

- What is HIV?
- What is AIDS?
- Brief Anatomy of HIV
- Difference between HIV/AIIDS
- How long it takes for HIV to become AIDS
- HIV facts and myths

Let us now attempt the assignment below.

6.0 TUTOR MARKED ASSIGNMENT

- What is HIV?
- What is AIDS?
- What is the difference between HIV and AIDS?

7.0 REFERENCES/FURTHER READINGS

Avert: Averting HIV/AIDS. HIV/AIDS information from Avert.org. *http://www.avert.org/* Site accessed on 15th January, 2008.

Alta van Dyk (2005). *HIV/AIDS care and Counselling: A Multidisciplinary approach. Third edition*. South Africa: Maskew Miller Longman.

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UNAIDS (2011) 'World AIDS Day Report 2011' – 2011. Joint United Nations Programme on HIV/AIDS.

UNIT 2 HIV/AIDS: EVOLUTION OF A PANDEMIC

CONTENT

1.0 INTRODUCTION

With time, the HIV/AIDS pandemic is unfolding and revealing its secrets. Paradoxically, the pandemic is becoming simultaneously more difficult and simpler to comprehend. A modern understanding of the pandemic requires two levels of awareness. First, it is essential to appreciate the enormously complex histories of individual HIV/AIDS epidemics at the community or national level. Yet this level of information is insufficient. It is also necessary to recognize the common features among the HIV/AIDS epidemic, which at a deeper level, provide insight into the natural history and the shape of the slowly maturing pandemic (Jonathan Mann & Tarantola, 1996).

Now in this third decade of our experiences with this pandemic, to many of us, Acquired Immune Deficiency Syndrome (AIDS) seem to have always been here, always stalking us, always part of our lives, always the principal focus of our personal and professional activities. Health workers working in this field today are involved in a rapidly accelerating spiral of dynamic developments: evolving science, new drugs, new prevention strategies, ever-moving political agendas, changing vulnerabilities and the restructuring of models for the provision of care. In our lifetime, HIV is perhaps the ultimate epidemic, reshaping our world beyond recognition, bringing out both the best and the worst in humankind (Pratt, 2003).

In this unit therefore, we shall provide a simple but in-depth history and evolution of HIV/AIDS, contributory factors to the rapid spread of HIV as well as common terms associated with describing epidemics.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- Define common terms associated with describing epidemics
- Give a brief description of the history and evolution of HIV/AIDS
- Identify the factors contributing to the rapid spread of HIV/AIDS

3.0 MAIN CONTENT

3.1 Definition of Terms: Prevalence, Incidence, Pandemic, Endemic. Epidemic

Prevalence is the term used to describe the *number of persons who have a specific disease or condition in a defined population at one specific point in time*, e.g. the total number of men and women between the ages of 12 and 18 residing in Nigeria, known to be infected with HIV and alive by December, 2012.

Incidence is the *rate at which a certain event occurs in a defined population during a specific period of time*, e.g. the number of new cases of HIV infection that occurred in Nigeria during 2012.

Endemic is the term used *to describe the disease present (or usually prevalent) in a population or geographical area all the time,* e.g. a certain level of polio is always present (endemic) in an area in Northern Nigeria.

An Epidemic is a *sudden increase in the incidence of an endemic disease (or condition), or the occurrence of a new disease with a high incidence introduced into a population,* e.g. a sudden and significant increase in the incidence of Tuberculosis during a specific period of time.

A Pandemic refers to an *epidemic disease distributed or occurring widely throughout a region, country, continent or globally*. HIV/AIDS is a global pandemic composed of a plethora of local epidemics, all with their own unique characteristics, driven nationally by varied but similar human behaviours and societal conditions.

SELF ASSESSMENT EXERCISE

Define the following:

- Prevalence
- Incidence
- Endemic
- Pandemic
- Epidemic

3.2 Brief History of HIV/AIDS

It is sometimes difficult to recall how it all started, how we arrived at this stage of our journey through a global disaster, engaging one of the most serious threat to public health in our lifetime. The story began a long time ago.

AIDS was to enter the world's consciousness and became part of the vocabulary of the human soul as a result of a dawning awareness of the advent of a new and strange disease first reported in California, in 1981. In July 1981, the New York Times reported an outbreak of a rare form of cancer among gay men in New York and California, first referred to as the "gay cancer"; but medically know as Kaposi Sarcoma. About the same time, emergency rooms in New York City began to see a rash on seemingly healthy young men presenting with fevers, flu like symptoms, and a pneumonia called Pneumocystis. About a year later, the CDC (Centers for Disease Control) link the illness to blood and coins the term AIDS (Acquired Immune Deficiency Syndrome). In that first year over 1600 cases were diagnosed with close to 700 deaths (CDC, 1981). Probably, no one actually expected the magnitude of the epidemic that was in the making. However, evidence of a gathering storm was soon arriving.

The presence of related retroviruses in African monkeys and apes and the close relationship of HIV to a Chimpanzee Immunodeficiency virus all suggest that Central Africa may have been the site of evolution of HIV.

Some people think that there are other possible origins of HIV. One of these is the suggestion that HIV was a deliberate or accidental product of biological warfare research. That is not possible, since the technology and the basic knowledge that would have been necessary to create such a virus had not been developed in 1975, when the epidemic began to grow (Avert, 2008; Bartlett & Finkbeiner, 2001; Pratt, 2003).

3.3 Spread of HIV: Contributory Factors?

This could be explained by scenarios below:

- Urbanization of this region of the world, particularly under British and French colonialism, brought young African males to the cities for education and to participate in the professions. The more relaxed morals of the urban setting bring the availability of multiple sexual partners and prostitution. In fact, prostitution was evidently created deliberately in certain circumstances to solve the problems of the separation of the urban professional men from their tribal families. Such events could furnish a greater spread of HIV, enough to possibly create a 'base' of HIV positive individuals for future expansion.
- Western approaches to health care in urban Africa, including the extensive use of blood transfusions for the treatment of Malaria and the frequent use and reuse of hypodermics for everything from immunizations and antibiotics to vitamin injections (a common practice in Central Africa) would contribute to the growth of this epidemic.
- With urbanization came increased air travel and increased contact with other parts of the world and with other societies: government supported exchange programs in education, policy development, agriculture, and the arts; increasing economic and business ties; all possible conduits for an infective virus that is not

visible in the early years after infection, although fully capable of being transmitted.

- When the virus arrived in Europe and in the United States, it was joined with two "cultural epidemics" that were instrumental in the rapid growth of the disease: the so-called sexual liberation movement, and the dramatic increase in recreational and addictive drug use. Multiple sexual partners and injection drug use (IDU) with shared needles are the two main ways to get this virus.
- As a final ecological twist, the virus arrived in America just as the sexual liberation shifted from the heterosexual to the homosexual community. As is often true, the disenfranchised are the last to receive the "benefits" of any societal change. So members of the gay community were the last to experience the open sexual freedom that occurred in the twentieth century (Pratt, 2003; Alta van Dyke, 2005; Avert, 2008; Bartlett & Finkbeiner, 2001; Medical News Today, 2012).

4.0 CONCLUSION

Seemingly coming from nowhere two decades ago, HIV/AIDS now poses one of the most significant threats to public health for this generation and perhaps for generation to come. In no country of this planet can it be said that this epidemic is under control. It continues as a volatile, unstable and escalating situation. This global pandemic will only be brought under control when all the issues that are factored into its continuing escalation are confronted and tackled (Pratt, 2003).

5.0 SUMMARY

In this unit, we studied the following:

- Common terms associated with describing epidemics
- Brief history of HIV/AIDS
- Contributory factors to the spread of HIV

6.0 TUTOR MARKED ASSIGNMENT

- Do a literature search on current HIV statistics (globally and locally)

7.0: REFERENCES

About.com (2012). History of HIV/AIDS. Retrieved on September 15 from http://aids.about.com/od/newlydiagnosed/a/hivtimeline_2.htm

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Unit 3: HIV/AIDS Timeline

CONTENT

1.0 INTRODUCTION

The history of HIV is filled with triumphs and failures; living and death. The HIV time line stretches before us, marking our past and reaching toward our future. But where will that future lead? What does the history of HIV show us? What have we learned throughout the history of HIV? (Cichocki, 2010). The timeline of HIV is elaborated in this unit.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- Identify HIV timeline

3.0 MAIN CONTENT

The HIV time line began early in 1981. In July of that year, the New York Times reported an outbreak of a rare form of cancer among gay men in New York and California. This "gay cancer" as it was called at the time was later identified as Kaposi's Sarcoma, a disease that later became the face of HIV/AIDS. About the same time, emergency rooms in New York City began to see a rash of seemingly healthy young men presenting with fevers, flu-like symptoms, and a rare pneumonia called Pneumocystis. This was the beginning of what has become the biggest health care concern in modern history. Thirty one years later the disease still plagues society. How did we get to this point? We will take a look back at 25 years of HIV/AIDS (Cichocki, 2010).

1959

While we talk about HIV/AIDS being about 25 years old, in actuality it is believed that the syndrome has been around far longer. In 1959, a man residing in Africa died of a mysterious illness. Only decades later, after examining some blood samples taken from that man, was it confirmed that he actually died from complications related to an HIV infection (Cichocki, 2010).

1981

As stated above, 1981 saw the emergence of Kaposi's Sarcoma and Pneumocystis among gay men in New York and California. When the Centers for Disease Control reported the new outbreak they called it "GRID" (gay-related immune deficiency), stigmatizing the gay community as carriers of this deadly disease. However, cases started to be seen in heterosexuals, drug addicts, and people who received blood transfusions, proving the syndrome knew no boundaries (Cichocki, 2010).

1983

Researchers at the Pasteur Institute in France isolate a retrovirus that they believe is related to the outbreak of HIV/AIDS. Thirty-three countries around the world have confirmed cases of the disease that was once limited to New York and California. Controversy arises a year later when the US government announces their scientist, Dr. Robert Gallo isolates a retrovirus HTLV-III, that he too claims is responsible for AIDS. Two years later it's confirmed that HTLV-III and the Pasteur retrovirus are indeed the same virus, yet Gallo is still credited with its discovery. An international committee of scientists, rename the virus HIV (Cichocki, 2010).

1984

A Canadian flight attendant, nicknamed "patient zero" dies of AIDS. Because of his sexual connection to several of the first victims of HIV/AIDS, it is believed that he is responsible for introducing the virus into the general population (Cichocki, 2010).

1985

The controversy surrounding the HIV/AIDS virus continues when Robert Gallo's lab patents an HIV test kit that later is approved by the FDA. The Pasteur Institute sues and is later awarded rights to half of the royalties from the new test. At the same time, HIV/AIDS enters the public eye when Rock Hudson dies of AIDS and Ryan White is barred from his elementary school in Indiana (Cichocki, 2010).

SELF ASSESSMENT EXERCISE

The global HIV time line began in ---------- year and where?

1987 - A Treatment Arrives

After 6 years of watching people die, a new treatment emerges that is hailed as the first huge step in beating HIV/AIDS. The drug Retrovir (AZT, Zidovudine) is FDA approved and begins to be used in high doses to treat people infected with HIV. And not a minute too soon. Politically, HIV/AIDS is a topic that most avoid. But in

response to public pressure, President Ronald Reagan finally acknowledges the HIV/AIDS problem and for the first time uses the term "AIDS" in a public speech (Cichocki, 2010).

1990

After years of fighting to stay in school, and raging an even harder battle against the ravages of HIV/AIDS, Ryan White dies at the age of 19. That year, The Ryan White Care Act is enacted by Congress to provide government sponsored funds for the care of HIV/AIDS infected people (Cichocki, 2010).

1992 - Combination Therapy Arrives

The FDA approves the first drug to be used in combination with AZT. The addition of the drug Hivid marks the beginning of HIV/AIDS combination therapies. But a more disturbing development centers around HIV tainted blood. Three French senior health officials knowingly sell HIV tainted blood, resulting in the infection of hundreds of transfusion recipients, most of whom have hemophilia (Cichocki, 2010).

1993

People who are infected and scientists alike are confused and concerned when a British study, the Concorde Trials, offers proof that AZT monotherapy does nothing to delay progression to AIDS in asymptomatic patients. As a result, the AZT debate emerges, with one side proclaiming AZT saves lives and the other denouncing AZT as useless; the "rethinker" movement is born (Cichocki, 2010).

1996 - Protease Inhibitors Arrive

Treatment options take another step forward with the introduction of power HIV-fighting drugs called Protease Inhibitors. The use of these drugs in combination with existing HIV/AIDS drugs proves effective in controlling HIV. These new "triple-therapies" give patients and scientists new hope in eliminating HIV/AIDS. But that hope is dashed when a year later, scientists find HIV/AIDS "hides" in reservoirs in the body, making total elimination of the virus virtually impossible (Cichocki, 2010).

1997

In late 1996 data from AIDS Clinical Trials Group study 076 (ACTG 076) made it clear that Retrovir (AZT) used during pregnancy and at the time of delivery drastically reduces transmission of HIV from mother to child. Those findings led to protocols that now drastically reduce transmission from mother to child from 1 in 4 to less than 3% (Cichocki, 2010).

1998

More than 15 years after the prediction there would be of an AIDS vaccine within 2 years, the first human trials in the United States of an HIV/AIDS vaccine begins. In a desperate attempt to get affordable HIV/AIDS drugs to the hardest hit areas of Africa, European drug companies ignore US patent laws and begin making generic versions of HIV/AIDS medications. In response, US drug companies file lawsuits to stop such practices. And sadly, 17 years after HIV/AIDS entered our culture, an African AIDS activist is beaten to death by neighbors after publicly admitting she was HIV infected (Cichocki, 2010).

2000

The AIDS "rethinker" movement gets international attention and support when South African president Thabo Mbeki questions the use and effectiveness of HIV medications as well as offering doubt that HIV causes AIDS. In response, the international scientific community issues the Durban Declaration, offering proof that HIV and AIDS are indeed connected (Cichocki, 2010).

2001

As scientists grow concerned over medication toxicity and effectiveness, US pharmaceutical companies drop their patent lawsuits, paving the way for European drug companies to manufacture and distribute cheaper HIV medications to the hardest hit areas of Sub-Saharan Africa. Cautious optimism emerges with the release of the first entry inhibitor, Fuzeon. Since 1981, 21 million people worldwide have died of AIDS, including 17 million from Sub-Saharan Africa (Cichocki, 2010).

- *31 million people are now living with HIV worldwide, the majority of whom are from African nations* (Cichocki, 2010).

2004

As the emphasis on simpler therapies continues, regimen pill burdens are greatly improved with the release of two new combination drugs, Truvada and Epzicom as well as two new protease inhibitors, Reyataz and Lexiva. In December, the first generic formulation of an HIV medication is approved by the FDA, instilling hope that HIV medication prices may soon come down (Cichocki, 2010).

2005

HIV statistics have become sobering to say the least.

- *4.9 million people were newly infected in 2005*

- *40.3 million People worldwide living with HIV/AIDS.*

And as the numbers continue to climb, work on an HIV vaccine has for the most part failed. Once thought to be "just around the corner" it has become obvious in 2005 that an HIV vaccine is still years away. Medication advances continue but long term side effects of HIV medication use are becoming more evident. So much so that experts now agree that for many patients, waiting to start HIV medications is the best course of action. Finally, 2005 saw a rise in HIV rates on college campuses and risky behavior among those people already infected is still a problem. Positive prevention messages are becoming a priority as syphilis and other STI rates of infection continue to rise sharply (Cichocki, 2010).

2006

Experts conclude that HIV has its origins in the jungles of Africa among wild chimps. Experts go on to report that evidence suggests that the simian form of HIV (SIV) entered the human species and became HIV by way of monkey bites or ingesting monkey meat and brains. While the origins of HIV are clearer, the means to pay for HIV care and medications has become more complicated. A revamping of the Medicare / Medicaid systems has made getting medications difficult for many. India surpasses South Africa as the world's largest HIV population and in the US infection rates of HIV are steady while STIs are on the rise (Cichocki, 2010).

2009

Scientists at the University of North Carolina at Chapel Hill announce they have decoded the structure of an entire HIV genome. How this will affect the future of HIV treatment, prevention, and education is not entirely known. What we do know is that the more we know about HIV, the better we can fight its affects on public health in the US and around the world (Cichocki, 2010).

4.0 CONCLUSION

Seemingly coming from nowhere two decades ago, HIV/AIDS now poses one of the most significant threats to public health for this generation and perhaps for generation to come. In no country of this planet can it be said that this epidemic is under control. It continues as a volatile, unstable and escalating situation. This global pandemic will only be brought under control when all the issues that are factored into its continuing escalation are confronted and tackled (Pratt, 2003).

5.0 SUMMARY

In this unit, we studied the following:

HIV Timeline (1956 – 2009)

6.0 TUTOR MARKED ASSIGNMENT

Highlight important events of HIV Timeline (1956 – 2009)

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MODULE 2: THEORIES OF THE ORIGIN OF HIV/AIDS

UNIT 1 ORIGIN OF HIV AND AIDS:

CONTENT

1.0 INTRODUCTION

The origin of AIDS and HIV has puzzled scientists ever since the illness first came to light in the early 1980s. For over twenty years it has been the subject of fierce debate and the cause of countless arguments, with everything from a promiscuous flight attendant to a suspect vaccine programme being blamed. So what is the truth? Just where did HIV come from?

The first recognized cases of HIV occurred in the USA in the early 1980s. A number of gay men in New York and San Francisco suddenly began to develop rare opportunistic infections and cancers that seemed stubbornly resistant to any treatment. At this time, AIDS did not yet have a name, but it quickly became obvious that all the men were suffering from a common syndrome.

The discovery of HIV, the Human Immunodeficiency Virus, was made soon after. While some were initially resistant to acknowledge the connection (and indeed some remain so today), there is now clear evidence to prove that *HIV causes AIDS.* So, in order to find the source of AIDS, it is necessary to look for the origin of HIV, and find out *how, when and where* HIV first began to cause disease in humans.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- Describe what type of virus HIV is

- Ascertain if HIV actually came from an SIV
- Explain what happened in 1999 as regards to HIV
- Identify how HIV could have crossed species

3.0 MAIN CONTENT

3.1 What type of virus is HIV?

HIV causes AIDS. So, in order to find the source of AIDS, it is necessary to look for the origin of HIV, and find out *how, when and where* HIV first began to cause disease in humans. So to the question: How and what type of virus is HIV, we will provide the following information.

HIV is a *lentivirus,* and like all viruses of this type, it attacks the immune system. Lentiviruses are in turn part of a larger group of viruses known as *retroviruses.* The name 'lentivirus' literally means *'slow virus'* because they take such a long time to produce any adverse effects in the body. They have been found in a number of different animals, including cats, sheep, horses and cattle. However, the most interesting lentivirus in terms of the investigation into the origins of HIV is the Simian Immunodeficiency Virus (SIV) that affects monkeys.

3.2 Did HIV come from an SIV?

It is now generally accepted that HIV is a descendant of a Simian Immunodeficiency Virus because certain strains of SIVs bear a very close resemblance to HIV-1 and HIV-2, the two *types of HIV.*

HIV-2 for example corresponds to *SIVsm*, a strain of the Simian Immunodeficiency Virus found in the sooty mangabey (also known as the green monkey), which is indigenous to western Africa.

The more virulent, pandemic strain of HIV, namely HIV-1, was until recently more difficult to place. Until 1999, the closest counterpart that had been identified was *SIVcpz*, the SIV found in chimpanzees. However, this virus still had certain significant differences from HIV.

SELF ASSESSMENT EXERCISE

The name 'lentivirus' literally means ----------

Lentiviruses are found in the following animals----------

SIV means ----------

3.3 What happened in 1999?

In February 1999 a group of researchers from the University of Alabama (Gao, et al., 1999), announced that they had found a type of SIVcpz that was almost identical to HIV-1. This particular strain was identified in a frozen sample taken from a captive member of the sub-group of chimpanzees known as Pan troglodytes troglodytes (*P. t. troglodytes*), which were once common in west-central Africa.

The researchers (led by Paul Sharp of Nottingham University and Beatrice Hahn of the University of Alabama) made the discovery during the course of a 10-year long study into the origins of the virus. They claimed that this sample proved that chimpanzees were the source of HIV-1, and that the virus had at some point crossed species from chimps to humans.

Their final findings were published two years later in *Nature* magazine (Bailes, et al., 2003). In this article, they concluded that wild chimps had been infected simultaneously with two different simian immunodeficiency viruses which had "viral sex" to form a third virus that could be passed on to other chimps and, more significantly, was capable of infecting humans and causing HIV.

These two different viruses were traced back to a SIV that infected red-capped mangabeys and one found in greater spot-nosed monkeys. They believe that the hybridisation took place inside chimps that had become infected with both strains of SIV after they hunted and killed the two smaller species of monkey.

They also concluded that all three 'groups' of HIV-1 - namely Group M, N and O - came from the SIV found in *P. t. troglodytes*, and that each group represented a separate crossover 'event' from chimps to humans.

3.4 How could HIV have crossed species?

It has been known for a long time that certain viruses can pass between species. Indeed, the very fact that chimpanzees obtained SIV from two other species of primate shows just how easily this crossover can occur. As animals ourselves, we are just as susceptible. When a viral transfer between animals and humans takes place, it is known as *zoonosis.*

4.0 CONCLUSION

The Human Immunodeficiency Virus (HIV) soon after discovery became a worldwide interest. While some were initially resistant to acknowledge the connection (and indeed some remain so today), there is now clear evidence to prove that *HIV causes AIDS.* This unit therefore provided information on strains of HIV, scientific findings of researches on HIV, with specifics to findings reported in 1999, as well as a brief insight on hybridization of HIV.

5.0 SUMMARY

We hope you enjoyed your studies. We achieved the following:

- We identified what type of virus HIV is?
- Ascertain the connection between HIV and SIV
- Reported HIV researches and findings of 1999
- Illustrated how HIV could have crossed species

6.0 TUTOR MARKED ASSIGNMENT

- HIV research: What happened in 1999?

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UNIT 2 ORIGIN OF HIV (Cont)

CONTENT

1.0 INTRODUCTION

During the last few years it has become possible not only to determine whether HIV is present in a blood or plasma sample, but also to determine the particular subtype of the virus. Studying the subtype of virus of some of the earliest known instances of HIV infection can help to provide clues about the time it first appeared in humans and its subsequent evolution.

The question of exactly where the transfer of HIV to humans took place, and where the 'epidemic' officially first developed has always been controversial. Some have suggested that it is dangerous to even try to find out, as HIV has frequently been blamed on an innocent person or group of individuals in the past. However, scientists remain keen to find the true origin of HIV, as most agree it is important to understand the virus and its epidemiology in order to fight it.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- Ascertain if HIV actually came from Africa
- Identify why Haiti is significant in the spread of HIV
- Explain factors that caused the HIV/AIDS epidemic to spread so suddenly

3.0 MAIN CONTENT

3.1 So did HIV definitely come from Africa?

Given the evidence we have already looked at, it seems highly likely that Africa was indeed the continent where the transfer of HIV to humans first occurred (monkeys from Asia and South America have never been found to have SIVs that could cause HIV in humans). In May 2006, the same group of researchers who first identified the Pan troglodytes troglodytes strain of SIVcpz, announced that they had narrowed down the location of this particular strain to wild chimpanzees found in the forests of Southern Cameroon (BBC, 2006). By analyzing 599 samples of chimp droppings (P. T. troglodytes are a highly endangered and thus protected species that cannot be killed or captured for testing), the researchers were able to obtain 34 specimens that reacted to a standard HIV DNA test, 12 of which gave results that were virtually indistinguishable from the reactions created by human HIV. The researchers therefore concluded that the chimpanzees found in this area were highly likely the origin of both the pandemic group M of HIV-1 and of the far rarer group N. The exact origins of group O however remain unknown.

HIV group N principally affects people living in South-central Cameroon, so it is not difficult to see how this outbreak started. Group M, the group that has caused the worldwide pandemic, was however first identified in Kinshasa, in the Democratic Republic of Congo. It is not entirely clear how it transferred from Cameroon to Kinshasa, but the most likely explanation is that an infected individual travelled south down the Sangha river that runs through Southern Cameroon to the River Congo and then on to Kinshasa, where the group M epidemic probably began.

Just as we do not know exactly who spread the virus from Cameroon to Kinshasa, how the virus spread from Africa to America is also not entirely clear. However, recent evidence suggests that the virus may have arrived via the Caribbean island of Haiti (BBC, 2006).

3.2 Why is Haiti significant?

The HIV epidemic in Haiti first came to light in the early 1980s, at around the same time that cases in the USA were being uncovered. Following the discovery of a number of Haitians with Kaposi's Sarcoma and other AIDS-related conditions, medical journals and books began to claim that HIV had come from Haiti, and that Haitians were responsible for the its epidemic in the United States.

These claims, which were often founded on dubious evidence, fuelled pre-existing racism in the US and many Haitians suffered severe discrimination and stigmatization as a result. A large number of Haitian immigrants living in the US lost their jobs and were evicted from their homes as Haitians were added to homosexuals, hemophiliacs and heroin users to make the 'Four-H Club' of groups at high risk of HIV (Farmer, 1992).

The emotionally-charged culture of blame and prejudice that surrounded HIV and AIDS in the early years meant that it soon became politically difficult to present

epidemiological findings in a neutral and objective way. For many years the link between Haiti and the US epidemic was therefore dropped as a subject.

In March 2007 however, it returned to the public eye at the Fourteenth Conference on Retroviruses and Opportunistic Infections (CROI) in Los Angeles. A group of international scientists presented data based on complex genetic analysis of 122 early samples of HIV-1, group M, subtype B (the most common strain found in the USA and in Haiti) showing that the strain had probably been brought to Haiti from Africa by a single person in around 1966; a time when many Haitians would have been returning from working in the Congo (Carter, 2007).

Genetic analysis then showed that subtype B spread slowly from person to person on the island, before being transferred to the US, again probably by a single individual, at some point between 1969 and 1972. A paper published in October 2007 by Worobey and colleagues gave a 99.7% certainty that HIV subtype B originated in Haiti before passing to the US (Change, 2007).

It is possible that HIV had entered the US several times before subtype B took a firm hold (which would explain the infection of the St. Louis teenager in the early to mid-1960s), but it was the late 1960s / early 1970s transfer that is believed to be responsible for the widespread epidemic seen in the US today. Once the virus had established itself in the gay community, it would have spread fairly rapidly (anal intercourse carries a very high transmission risk), with transmission occurring within and between the US and Haiti, and internationally, until the original route taken by the virus was largely obscured.

Dr Michael Worobey, lead researcher in the study, claimed that his data was not intended to place any blame on Haiti, or on Central Africans, and stressed that none of the people who first transmitted HIV would have been aware they were infected. His work still received strong protests from one Haitian delegate at the CROI conference however, demonstrating the extent to which tracing HIV's origins remains a politically sensitive exercise.

SELF ASSESSMENT EXERCISE

Why is Haiti prominent in the history of HIV?

3.3 What caused the epidemic to spread so suddenly?

There are a number of factors that may have contributed to the sudden spread of HIV, most of which occurred in the latter half of the twentieth century.

3.3.1 Travel

International travel has undoubtedly played a major role in the spread of HIV.

Both national and international travel undoubtedly had a major role in the initial spread of HIV. In the US, international travel by young men making the most of the gay sexual revolution of the late 70s and early 80s would certainly have played a large part in taking the virus worldwide. In Africa, the virus would probably have been spread along truck routes and between towns and cities within the continent itself. However, it is quite conceivable that some of the early outbreaks in African nations were not started by Africans infected with the 'original' virus at all, but by people visiting from overseas where the epidemic had been growing too. The process of transmission in a global pandemic is simply too complex to blame on any one group or individual.

Much was made in the early years of the epidemic of a so-called 'Patient Zero' who was the basis of a complex "transmission scenario" compiled by Dr. William Darrow and colleagues at the Centre for Disease Control in the US. This epidemiological study showed how 'Patient O' (mistakenly identified in the press as 'Patient Zero') had given HIV to multiple partners, who then in turn transmitted it to others and rapidly spread the virus to locations all over the world. A journalist, Randy Shilts, subsequently wrote an book (Shilts, 1987), based on Darrow's findings, which named Patient Zero as a gay Canadian flight attendant called Gaetan Dugas. For several years, Dugas was vilified as a 'mass spreader' of HIV and the original source of the HIV epidemic among gay men. However, four years after the publication of Shilts' article, Dr. Darrow repudiated his study, admitting its methods were flawed and that Shilts' had misrepresented its conclusions.

While Gaetan Dugas was a real person who did eventually die of AIDS, the Patient Zero story was not much more than myth and scaremongering. HIV in the US was to a large degree initially spread by gay men, but this occurred on a huge scale over many years, probably a long time before Dugas even began to travel.

3.3.2 The Blood Industry

As blood transfusions became a routine part of medical practice, an industry to meet this increased demand for blood began to develop rapidly. In some countries such as the USA, donors were paid to give blood, a policy that often attracted those most

desperate for cash; among them intravenous drug users. In the early stages of the epidemic, doctors were unaware of how easily HIV could be spread and blood donations remained unscreened. This blood was then sent worldwide, and unfortunately most people who received infected donations went on to become HIV positive themselves.

In the late 1960's haemophiliacs also began to benefit from the blood clotting properties of a product called Factor VIII. However, to produce this coagulant, blood from hundreds of individual donors had to be pooled. This meant that a single donation of HIV+ blood could contaminate a huge batch of Factor VIII. This put thousands of haemophiliacs all over the world at risk of HIV, and many subsequently became infected with the virus.

3.3.3 Drug Use

The 1970s saw an increase in the availability of heroin following the Vietnam War and other conflicts in the Middle East, which helped stimulate a growth in intravenous drug use. This increased availability and together with the development of disposable plastic syringes and the establishment of 'shooting galleries' where people could buy drugs and rent equipment, provided another route through which the virus could be passed on.

4.0 CONCLUSION

It is likely that we will never know who the first person was to be infected with HIV, or exactly how it spread from that initial person. Scientists investigating the possibilities often become very attached to their individual 'pet' theories and insist that theirs is the only true answer, but the spread of AIDS could quite conceivably have been induced by a combination of many different events. Whether through injections, travel, wars, colonial practices or genetic engineering, the realities of the $20^{th}/21^{st}$ Century have undoubtedly had a major role to play. Nevertheless, perhaps a more pressing concern for scientists today should not be how the AIDS epidemic originated, but how those it affects can be treated, how the further spread of HIV can be prevented and how the world can change to ensure a similar pandemic never occurs again.

5.0 SUMMARY

In this unit, we studied the following:

- We ascertained if HIV definitely came from Africa
- We ascertained why Haiti is significant in the spread of HIV
- We indentified few factors that might have triggered sudden spread of the epidemic

6.0 TUTOR MARKED ASSIGNMENT

- Explain the factors that triggered the spread of HIV.

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UNIT 3 THEORIES OF HIV/AIDS

CONTENT

1.0 INTRODUCTION

You will indeed find this unit very interesting. This unit provides different theoretical views on the origin of HIV. Views ranged from the Hunter Theory, the Oral Polio Vaccine OPV Theory, to the Conspiracy Theory among many. No doubt, you will find most of these theories controversial, but not to worry, such views are meant to be insightful and thought provoking. Enjoy your studies.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- Explain Hunters Theory of HIV
- Describe the Oral Polio Vaccine Theory of HIV
- Ascertain the fact behind the Contaminated Needle Theory
- Discuss the Colonialism Theory
- Discuss the Conspiracy Theory

3.0 MAIN CONTENT

Below are some of the most common theories about how this 'zoonosis' took place, i.e., how Simian Immunodeficiency Virus SIV became HIV in humans:

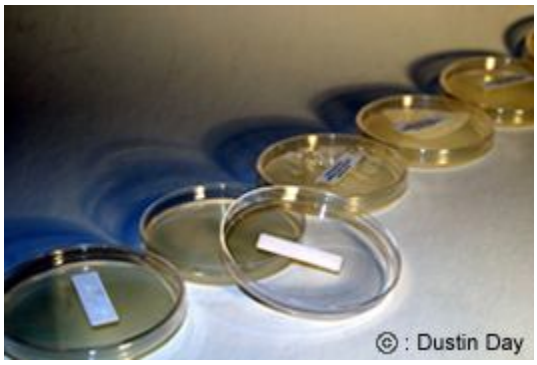
3.1 The 'Hunter' Theory

The most commonly accepted theory is that of the 'hunter'. In this scenario, SIVcpz was transferred to humans as a result of chimps being killed and eaten or their blood getting into cuts or wounds of the hunter. Normally the hunter's body would have fought off SIV, but on a few occasions it adapted itself within its new human host and

become HIV-1. The fact that there were several different early strains of HIV, each with a slightly different genetic make-up (the most common of which was HIV-1 group M), would support this theory: every time it passed from a chimpanzee to a man, it would have developed in a slightly different way within his body, and thus produced a slightly different strain.

An article published in The Lancet in 2004 (Wolfe, et al., 2004), also shows how retroviral transfer from primates to hunters is still occurring even today. In a sample of 1099 individuals in Cameroon, they discovered that (1%) were infected with SFV (Simian Foamy Virus), an illness which, like SIV, was previously thought only to infect primates. All these infections were believed to have been acquired through the butchering and consumption of monkey and ape meat. Discoveries such as this have led to calls for an outright ban on bushmeat hunting to prevent simian viruses being passed to humans.

3.2 The Oral Polio Vaccine (OPV) theory



Some other rather controversial theories have contended that HIV was transferred iatrogenically (i.e. via medical interventions). One particularly well-publicized idea is that polio vaccines played a role in the transfer.

In his book, *The River*, the journalist Edward Hooper suggests that HIV can be traced to the testing of an oral polio vaccine called Chat, given to about a million people in the Belgian Congo, Ruanda and Urundi in the late 1950s. To be reproduced, live polio vaccine needs to be cultivated in living tissue, and Hooper's belief is that Chat was grown in kidney cells taken from local chimps infected with SIVcmz. This, he claims, would have resulted in the contamination of the vaccine with chimp SIV, and a large number of people subsequently becoming infected with HIV-1.

Many people have contested Hooper's theories and insist that local chimps were not infected with a strain of SIVcmz that is closely linked to HIV. Furthermore, the oral administration of the vaccine would seem insufficient to cause infection in most people (SIV/HIV needs to get directly into the bloodstream to cause infection - the lining of the mouth and throat generally act as good barriers to the virus) (Cohen, 2000).

In February 2000 the Wistar Institute in Philadelphia (one of the original manufacturers of the Chat vaccine) announced that it had discovered in its stores a phial of polio vaccine that had been used as part of the program. The vaccine was subsequently analyzed and in April 2001 it was announced that no trace had been found of either HIV or chimpanzee SIV (Blancou et al., 2001). A second analysis confirmed that only macaque monkey kidney cells, which cannot be infected with SIV or HIV, were used to make Chat (Berry et al., 2001). While this is just one phial of many, it means that the OPV theory remains unproven.

The fact that the OPV theory accounts for just one (group M) of several different groups of HIV also suggests that transferral must have happened in other ways too, as does the fact that HIV seems to have existed in humans before the vaccine trials were ever carried out. More about when HIV came into being can be found below.

SELF ASSESSMENT EXERCISE

- Could production of the oral polio vaccine have contributed to the spread of HIV? What do you think?

3.3 The Contaminated Needle Theory

This is an extension of the original 'hunter' theory. In the 1950s, the use of disposable plastic syringes became commonplace around the world as a cheap, sterile way to administer medicines. However, to African healthcare professionals working on inoculation and other medical programmes, the huge quantities of syringes needed would have been very costly. It is therefore likely that one single syringe would have been used to inject multiple patients without any sterilisation in between. This would rapidly have transferred any viral particles (within a hunter's blood for example) from one person to another, creating huge potential for the virus to mutate and replicate in each new individual it entered, even if the SIV within the original person infected had not yet converted to HIV.

3.4 The Colonialism Theory

The colonialism or 'Heart of Darkness' theory is one of the more recent theories to have entered into the debate. It is again based on the basic 'hunter' premise, but more thoroughly explains how this original infection could have led to an epidemic. It was first proposed in 2000 by Jim Moore, an American specialist in primate behaviour, who published his findings in the journal AIDS Research and Human Retroviruses (Chitrin, Rawls and Moore, 2000).

During the late 19th and early 20th century, much of Africa was ruled by colonial forces. In areas such as French Equatorial Africa and the Belgian Congo, colonial rule was particularly harsh and many Africans were forced into labour camps where sanitation was poor, food was scare and physical demands were extreme. These factors alone would have been sufficient to create poor health in anyone, so SIV could easily have infiltrated the labour force and taken advantage of their weakened immune

systems to become HIV. A stray and perhaps sick chimpanzee with SIV would have made a welcome extra source of food for the workers.

Moore also believes that many of the laborers would have been inoculated with unsterile needles against diseases such as smallpox (to keep them alive and working), and that many of the camps actively employed prostitutes to keep the workers happy, creating numerous possibilities for onward transmission. A large number of laborers would have died before they even developed the first symptoms of AIDS, and those that did get sick would not have stood out as any different in an already disease-ridden population. Even if they had been identified, all evidence (including medical records) that the camps existed was destroyed to cover up the fact that a staggering 50% of the local population was wiped out there.

One final factor Moore uses to support his theory is the fact that the labour camps were set up around the time that HIV was first believed to have passed into humans - the early part of the 20th century.

3.5 The Conspiracy Theory

Some say that HIV is a 'conspiracy theory' or that it is 'man-made'. A recent survey carried out in the US for example, identified a significant number of African Americans who believe HIV was manufactured as part of a biological warfare programme, designed to wipe out large numbers of black and homosexual people (Fears, 2005). Many say this was done under the auspices of the US federal 'Special Cancer Virus Program' (SCVP), possibly with the help of the CIA. Linked in to this theory is the belief that the virus was spread (either deliberately or inadvertently) to thousands of people all over the world through the smallpox inoculation programme, or to gay men through Hepatitis B vaccine trials. While none of these theories can be definitively disproved, the evidence given to back them up is usually based upon supposition and speculation, and ignores the clear link between SIV and HIV or the fact that the virus has been identified in people as far back as 1959.

4.0 CONCLUSION

We learnt from the previous unit that when a viral transfer between animals and humans takes place, it is known as zoonosis. In this unit, we discussed some of the most common theories about how this 'zoonosis' took place, and how SIV became HIV in humans.

5.0 SUMMARY

We hope you found this unit insightful. In this unit, we explored the following theories of HIV:

- Hunters Theory
- Oral Polio Vaccine Theory

- Contaminated Needle Theory
- Colonialism Theory
- Conspiracy Theory

6.0 TUTOR MARKED ASSIGNMENT

- How could HIV have crossed-species? Discus using the Hunter and contaminated needle perspectives

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MODULE 3 THE BIOLOGY OF HIV

UNIT 1 UNDERSTANDING THE BIOLOGY OF HIV/AIDS

CONTENT

1.0 INTRODUCTION

This unit provides a basic review of the biology of HIV/AIDS viruses, preparing the learner for further readings.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- Understand the biology of HIV
- Identify structure of HIV
- Explain viral envelop
- Explain viral core
- Illustrate steps in HIV replication sample
- Understand how HIV evade the immune system

3.0 MAIN CONTENT

3.1 Biology of HIV – An Introduction

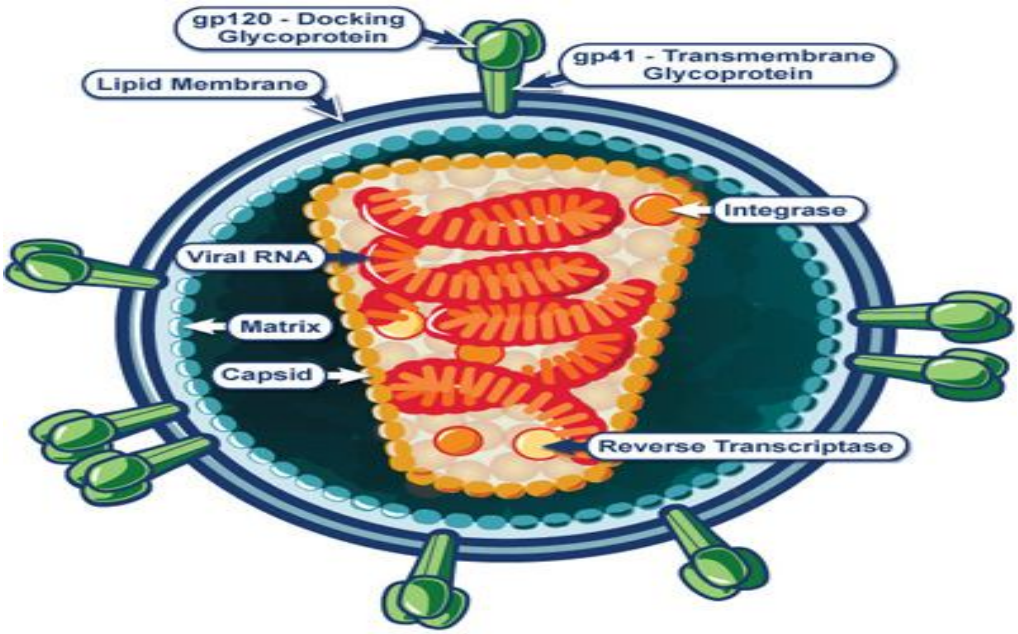
HIV belongs to a class of viruses known as retroviruses. Retroviruses are viruses that contain RNA (ribonucleic acid) as their genetic material. After infecting a cell, HIV uses an enzyme called reverse transcriptase to convert its RNA into DNA (deoxyribonucleic acid) and then proceeds to replicate itself using the cell's machinery.

Within the retrovirus family, HIV belongs to a subgroup known as lentiviruses, or "slow" viruses. Lentiviruses are known for having a long time period between initial infection and the beginning of serious symptoms. This is why there are many people who are unaware of their HIV infection, and unfortunately, can spread the virus to others.

Similar versions of HIV infect other nonhuman species, such as feline immunodeficiency virus (FIV) in cats and simian immunodeficiency virus (SIV) in monkeys and other nonhuman primates. Like HIV in humans, these animal viruses primarily infect immune system cells, often causing immune deficiency and AIDS-like symptoms. These viruses and their hosts have provided researchers with useful, although imperfect, models of the HIV disease process in people (NIAIDS, 2010).

Now let us take a look at the structure of HIV. You can refer to unit 1 for comparison.

3.2: Structure of HIV



Credit: NIAID (2010)

You need to study this diagram carefully because it will aid your understanding of subsequent units. Let us look at specific terms that can further elaborate the structure and mechanism of HIV.

3.3: The viral envelope

HIV is *spherical* in shape and has a *diameter of 1/10,000 of a millimeter*. The outer coat of the virus, known as the *viral envelope*, is composed of two layers of fatty molecules called lipids, taken from the membrane of a human cell when a newly formed virus particle buds from the cell. Embedded throughout the viral envelope are proteins from the host cell, as well as 72 copies (on average) of a complex HIV protein known as Env. These Env copies protrude or spike through the surface of the virus particle (called a "virion"). Env consists of a cap made of three molecules called glycoprotein 120 (gp120), and a stem consisting of three molecules called gyclycoprotein 41 (gp41) that anchor the structure in the viral envelope. Much of the research to develop a vaccine to prevent HIV infection has focused on these envelope proteins (NIAID, 2010).

3.4: The viral core

Within the viral envelope is a bullet-shaped core or capsid, made up of 2,000 copies of the viral protein, p24. The capsid surrounds two single strands of HIV RNA, each of which has a complete copy of the virus's genes.

HIV has three structural genes (*gag, pol, and env*) that contain information needed to make structural proteins for new virus particles. The env gene, for example, codes for a protein called gp160 that is broken down by a viral enzyme to form gp120 and gp41, the components of the env protein.

HIV has *six regulatory genes* (tat, rev, nef, vif, vpr, and vpu) that contain information needed to produce proteins that control the ability of HIV to infect a cell, produce new copies of virus, or cause disease. The protein encoded by nef, for instance, apparently is necessary for the virus to replicate efficiently, and the vpu-encoded protein influences the release of new virus particles from infected cells. Recently, researchers discovered that vif (the protein encoded by the vif gene) interacts with an antiviral defense protein in host cells (APOBEC3G), causing inactivation of the antiviral effect and enhancing HIV replication. This interaction may serve as a new target for antiviral drugs.

The ends of each strand of HIV RNA contain an RNA sequence called the long terminal repeat (LTR). Regions in the LTR act as switches to control production of new viruses and can be triggered by proteins from either HIV or the host cell.

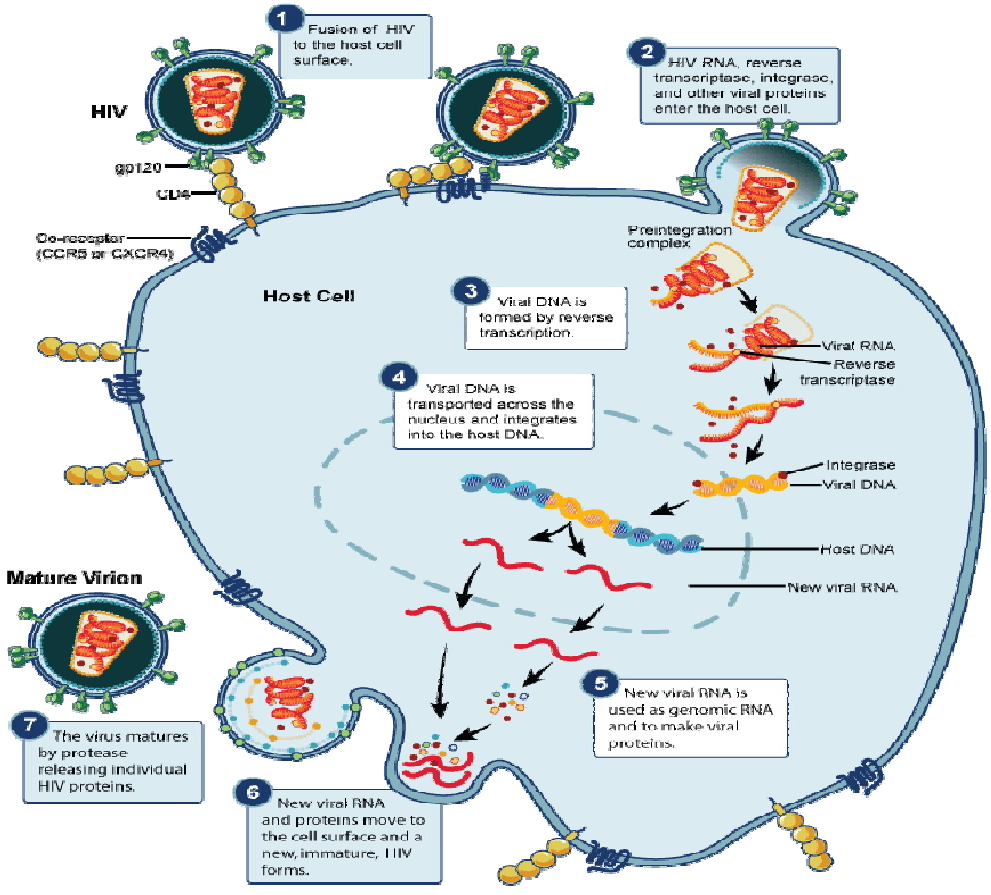
HIV's core also includes a protein called p7, the HIV nucleocapsid protein. Three enzymes carry out later steps in the virus's life cycle: reverse transcriptase, integrase, and protease. Another HIV protein called p17, or the HIV matrix protein, lies between the viral core and the viral envelope (NIAID, 2010).

3.5: HIV Replication Cycle

3.5.1: Steps in the HIV Replication Cycle

1. Fusion of the HIV cell to the host cell surface.
2. HIV RNA, reverse transcriptase, integrase, and other viral proteins enter the host cell.
3. Viral DNA is formed by reverse transcription.
4. Viral DNA is transported across the nucleus and integrates into the host DNA.
5. New viral RNA is used as genomic RNA and to make viral proteins.
6. New viral RNA and proteins move to cell surface and a new, immature, HIV virus forms.
7. The virus matures by protease releasing individual HIV proteins.

Find below a presentation of HIV replication cycle



Credit: NIAID (2010)

SELF ASSESSMENT EXERCISE

What are the steps in HIV replication cycle?

3.6: HIV Replication Cycle Glossary

CD4 – a large glycoprotein that is found on the surface of helper T cells, regulatory T cells, monocytes, and dendritic cells. Its natural function is as a co–receptor that assists the T cell receptor (TCR) to activate its T cell following an interaction with an antigen presenting cell. CD4 is a primary receptor used by HIV–1 to gain entry into host T cells.

Co–receptor (CCR5 or CXCR4) – protein molecules on the surface of lymphocytes or monocytes that bind to the gp120 protein of HIV and facilitate, usually with CD4, entry of viral nucleic acid and proteins into the cell.

DNA (deoxyribonucleic acid) – is a nucleic acid that contains the molecular basis of heredity for all known living organisms and some viruses and is found in the nuclei and mitochondria of eukaryotes. Chemically DNA consists of two polymer strands of units called nucleotides made up of one of four possible bases plus sugar and phosphate groups. The polymers are joined at the bases by hydrogen bonds to form a double helix structure.

Fusion of virus and cell membranes – a merging of cell and virus membranes that permits HIV proteins and nucleic acids to enter the host cell.

Genomic RNA – the nucleic acid that contains all of the hereditary information of a virus, and is found in a mature virion.

gp120 – an HIV glycoprotein having a molecular weight of 120 that protrudes from the outer surface of the virion. This glycoprotein binds to a CD4 receptor on a T cell to facilitate entry of viral nucleic acid and proteins into the cell.

HIV (human immunodeficiency virus) – is a lentivirus and a member of the retrovirus family. HIV infects and destroys helper T cells of the immune system causing a marked reduction in their numbers. Loss of CD4 cells leads to generalized failure of the immune system and susceptibility to life threatening opportunistic infections.

Integrase – An enzyme found in retroviruses including HIV that permits the viral DNA to be integrated into the DNA of the infected cell.

Preintegration complex (PIC) – It is composed of viral RNA and proteins (nucleocapsid, p6, Vpr, integrase, and matrix) as well as some host proteins. It functions to reverse transcribe genomic RNA into double stranded DNA prior to integration into the host genomic DNA.

Protease – an enzyme that hydrolyzes or cuts proteins and is important in the final steps of HIV maturation.

Nucleus – a membrane enclosed cellular organelle of eukaryotes that functions to contain the genomic DNA and to regulate gene expression.

Reverse transcriptase – an enzyme found in HIV that creates double stranded DNA using viral RNA as a template and host tRNA as primers.

RNA (ribonucleic acid) – a nucleic acid that differs from DNA in that it contains ribose and uracil as structural components.

RNA virus – a virus that uses RNA as its genetic material and belongs to either Group III, IV, or V of the Baltimore Classification System of Viruses. HIV belongs to Group III, double stranded RNA viruses.

Virion – a single and complete extracellular infective form of a virus that consists of an RNA or DNA core with a protein coat or "envelope" (NIAID, 2010).

3.7: How HIV Evolves to Evade the Immune System

HIV replicates rapidly with several billion new viruses made every day in a person infected with HIV. What makes HIV so difficult to stop, however, is its ability to mutate and evolve.

Reverse transcriptase, the enzyme that makes DNA copies of HIV's RNA, often makes random mistakes. As a result, new types or strains of HIV develop in a person infected with HIV. Some strains are harder to kill because of their ability to infect and kill other types of cells, while other strains replicate at faster rates. The more virulent and infectious strains of HIV are typically found in people who are in the late stages of infection. Different strains of HIV can also recombine to produce an even wider range of strains. In essence, HIV is constantly changing and trying to evade the immune system. Its ability to evolve rapidly is one of the major reasons why HIV is such a deadly virus (NIAID, 2010).

4.0: CONCLUSION

We hope you enjoyed your study. This unit provided general and specific information on the biology of HIV. Here, we learnt that HIV belongs to a class of viruses known as retroviruses while retroviruses are viruses that contain RNA (ribonucleic acid) as their genetic material. The structure of HIV further shows that HIV is spherical in shape, with a diameter of 1/10,000 of a millimeter. The outer coat of the virus is known as the viral envelop, and within the viral envelop is a bullet-shaped core or capsid. In this unit, we also identified steps in HIV replication cycle as well as how HIV evades the immune system.

5.0: SUMMARY

In this unit, we learnt the following:

- The biology of HIV
- Structure of HIV
- The viral envelop
- The viral core
- HIV replication cycle
- HIV replication cycle glossary
- How HIV evolves to evade the immune system

6.0 TUTOR MARKED ASSIGNMENT

Describe the following

- The viral envelop
- The viral core

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Unit 2: HIV: CLINICAL PROGRESSION

CONTENT

1.0 INTRODUCTION

We hope you enjoyed the previous unit. In unit 1 of module 3, we studied the biology of HIV, its structure and replication cycle. Unit 2 is a follow-up of unit 1 and we will look at the clinical progression of HIV.

2.0: OBJECTIVES

At the end of this unit, you are expected to:

- Illustrate how HIV devastates the immune system
- Explain how HIV hides from the immune system
- Identify factors that affect HIV disease progression
- Explain HIV risk factors
- Take note of quick facts about HIV Transmission

3.0 MAIN CONTENT

3.1: How HIV Devastates the Immune System

Scanning electron micrograph of HIV-1, colored green, budding from a cultured lymphocyte.
Credit: CDC

Every day, HIV destroys billions of CD4+ T cells in a person infected with HIV, eventually overwhelming the immune system's capacity to regenerate or fight other infections. Below are different ways this may occur.

3.1.1 Killing Cells Directly

CD4+ T cells infected with HIV may be killed when a large amount of virus is produced and buds out from the cell surface. The budding process disrupts the cell membrane and causes the cell to die. The cell can also expire when the virus excessively uses the cell's machinery for its own purposes, disrupting normal activities needed for the survival of the cell.

Apoptosis (cell suicide)

An HIV-infected cell undergoing apoptosis. Credit: Institute of Cell and Molecular Science

When the regulation of a cell's machinery and functions become grossly distorted because of HIV replication, the infected cell may commit suicide by a process known as programmed cell death or *apoptosis*. There is evidence that apoptosis occurs most frequently in the bloodstream and lymph nodes of people infected with HIV.

3.1.2: The Death of Innocent Bystander Cells

Cells that are not infected with HIV may also die as a direct result of the effects of HIV infection.

- HIV may bind to the cell surface, making it appear as if the cell is infected. After antibodies attach to the virus on the cell, killer T cells, which serve to protect the immune system by killing infected cells, may mistakenly destroy the cell. This process is called *antibody-dependent cellular cytotoxicity*.

- CD8 T cells, also known as "killer T cells," may mistakenly destroy uninfected cells that have consumed HIV particles and display HIV fragments on their surfaces.
- Because some HIV envelope proteins bear some resemblance to certain molecules on CD4+ T cells, the body's immune responses may mistakenly damage these cells.
- Uninfected cells may undergo apoptosis. Scientists have demonstrated in laboratory experiments that the HIV envelope alone or when bound to antibodies sometimes sends an inappropriate signal to CD4+ T cells. This can cause the cells to undergo apoptosis, even if not infected by HIV (NIAID, 2010).

3.1.3: Destruction of Immune Precursor Cells

Studies suggest that HIV also destroys precursor cells (young cells that have not yet fully developed) that later mature into cells with special immune functions. HIV can also damage the bone marrow and the thymus, which are needed for developing precursor cells. The bone marrow and thymus probably lose their ability to regenerate, further compounding the suppression of the immune system.

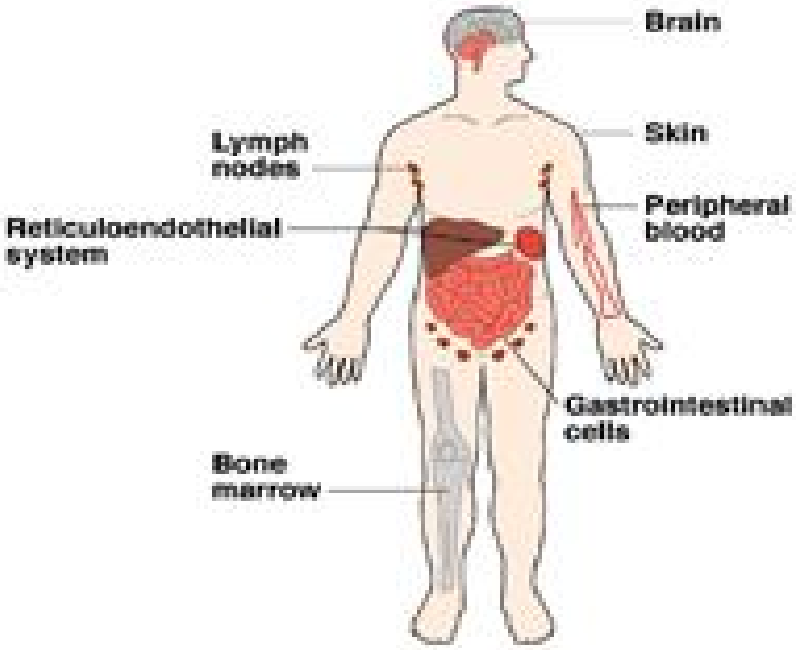
3.2: How HIV Hides from the Immune System

When HIV infects a cell, the virus can hide within the cytoplasm (the jelly-like fluid that fills the cell) or integrate into the cell’s genetic material (chromosomes). Shielded from the immune system, HIV can lie dormant in an infected cell for months or even years. These cells serve as a latent reservoir of the virus.

Antiretroviral drugs are capable of suppressing HIV, even to undetectable levels in the blood, but they cannot eliminate the virus hiding in these latent reservoirs. A key NIAID (2010) research priority is to learn how HIV establishes these latent reservoirs and to develop strategies to purge the virus from the body.

SELF ASSESSMENT EXERCISE

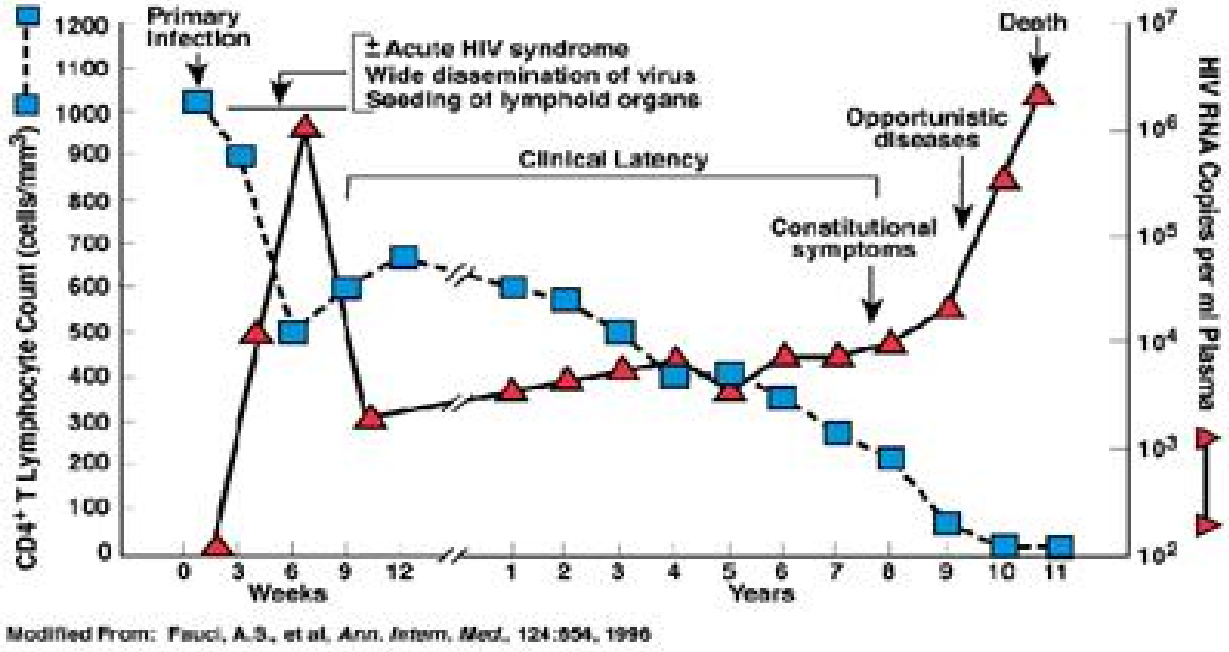
Explain how HIV devastates the immune system



Credit: (NIAID, 2010)

HIV can hide in the brain, lymph nodes, skin, peripheral blood, reticuloendothelial system, bone marrow, and gastrointestinal cells

3.3: Clinical Progression of HIV



Typical course of HIV infection that shows the relationship between the levels of HIV (viral load) and CD4+ T cell counts over the average course of untreated HIV infection.

3.3.1: Acute Primary Infection

Once HIV enters the body, the virus infects a large number of CD4+ T cells and replicates rapidly. During this acute phase of infection, the blood has a high number of HIV copies (viral load) that spread throughout the body, seeding in various organs, particularly the lymphoid organs such as the thymus, spleen, and lymph nodes. During this phase, the virus may integrate and hide in the cell's genetic material. Shielded from the immune system, the virus lies dormant for an extended period of time. In the acute phase of infection, up to 70 percent of HIV-infected people suffer flu-like symptoms (NIAID, 2010).

3.3.2: The Immune System Strikes Back

Two to 4 weeks after exposure to the virus, the immune system fights back with killer T cells (CD8+ T cells) and B-cell-produced antibodies. At this point, HIV levels in the blood are dramatically reduced. At the same time, CD4+ T cell counts rebound, and for some people the number rises to its original level (NIAID, 2010).

3.3.3: Clinical Latency

During this phase, a person infected with HIV may remain free of HIV-related symptoms for several years despite the fact that HIV continues to replicate in the lymphoid organs where it initially seeded (NIAID, 2010).

3.3.4: Progression to AIDS

The immune system eventually deteriorates to the point that the human body is unable to fight off other infections. The HIV viral load in the blood dramatically increases while the number of CD4+ T cells drops to dangerously low levels. An HIV-infected person is diagnosed with AIDS when he or she has one or more opportunistic infections, such as pneumonia or tuberculosis, and has fewer than 200 CD4+ T cells per cubic millimeter of blood (NIAID, 2010).

3.4: Factors that Affect HIV Disease Progression

3.4.1: Mutations in HIV Co-receptors

Most strains of HIV use a co-receptor molecule called CCR5, in addition to the CD4 molecule, to infect a cell. Other HIV strains use a different co-receptor known as CXCR4 to attack cells. Both receptors enable the virus to enter a cell during the initial stage of HIV infection.

Studies have shown that people infected with HIV who have specific genetic mutations in one of their two copies of the CCR5 gene progress to AIDS slower than people with two normal copies of the CCR5 gene. There are also rare individuals with two mutant copies of the CCR5 gene who appear, in most cases, to be completely

protected from HIV infection. Gene mutations in other HIV co-receptors such as CXCR4 also may influence the rate of disease progression.

3.4.2: High Viral Load

HIV patient getting blood drawn. Credit: NIAID (2010)

The amount of HIV in a person's blood often is called his or her viral load. People with high viral loads are more likely to progress to AIDS faster than people with lower levels of the virus. In addition, research has shown that the level of HIV in a person's blood after the first few months of infection, known as the viral set point, also influences the speed of progression to AIDS. Those with higher viral set point are much more likely to get sick faster than those with lower viral set point.

Highly active antiretroviral therapy (HAART), which is a potent combination of three or more antiretroviral drugs belonging to at least two different antiretroviral drug classes, can help lower the viral load and viral set point for those infected with HIV. For many people, HAART delays the progression to AIDS for a prolonged period of time.

3.5: HIV Risk Factors

HIV is found in the blood, semen, or vaginal fluid of someone who is infected with the virus. You may be at increased risk of becoming infected with HIV if you

- Engage in anal, vaginal, or oral sex with men who have sex with men, multiple partners, or anonymous partners without using a condom
- Inject drugs or steroids where needles/syringes are shared
- Have a sexually transmitted infection, such as syphilis, genital herpes, chlamydia, gonorrhea, bacterial vaginosis, or trichomoniasis
- Have been diagnosed with hepatitis, tuberculosis, or malaria
- Exchange sex for drugs or money
- Are exposed to the virus as a fetus or infant before or during birth or through breastfeeding from a mother infected with HIV
- Received a blood transfusion or clotting factor in the United States anytime from 1978 to 1985
- Engage in unprotected sex with someone who has any of the risk factors listed above (NIAID, 2010)

3.6: Quick Facts about HIV Transmission

- HIV cannot survive for very long outside of the body
- HIV cannot be transmitted through routine daily activities such as using a toilet seat, sharing food utensils or drinking glasses, shaking hands, or through kissing.
- The virus can only be transmitted from person to person, not through animals or insect bites
- People infected with HIV who are taking antiretroviral therapy can still infect others through unprotected sex and needle-sharing (NIAID, 2010)

4.0 CONCLUSION

In this unit, we studied how HIV devastates the immune system, specifically through killing of cells (apoptosis), the death of innocent bystander cell and destruction of immune precursor cells. We also studied how HIV evades the immune system and observed that HIV hides in the brain, lymph nodes, skin, and bone marrow and so on. Clinical progression of HIV was illustrated as: acute infection phase, clinical latency phase and the progression to AIDS phase.

5.0 SUMMARY

In this unit, we studied the following:

- HIV devastates the immune system
- How HIV hides from the immune system
- Clinical progression of HIV
- Factors that affect HIV disease progression
- HIV risk factors
- Quick facts about HIV Transmission

6.0 TUTOR MARKED ASSIGNMENT

Explain the clinical progression of HIV

7.0 REFERENCES AND FURTHER READINGS

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Unit 3: HIV: SYMPTOMS, TESTING AND MEDICATION

CONTENT

1.0 INTRODUCTION

This is the last unit of module 3. Recall that in unit 1, we studied the biology of HIV, and while unit 2 presented the clinical progression of HIV. In this unit, which naturally draws insight from previous ones, we will study HIV symptoms, testing, diagnosis, classes of antiretroviral drugs, its side effects and the challenges of designing HIV vaccine. Enjoy your studies.

2.0 OBJECTIONS

At the end of this unit, you should be able to:

- List symptoms of HIV
- Explain HIV testing and diagnosis
- List types of HIV tests
- List classes of HIV/AIDS Antiretroviral Drugs
- Understand the concepts of adherence and Drug resistance
- Explain HIV complications and side effects
- Understand the challenges in designing HIV

3.0 MAIN CONTENT

3.1: HIV Symptoms

3.1.1: Early Symptoms

In the first stages of HIV infection, most people will have very few, if any, symptoms. Within a month or two after infection, they may experience a flu-like illness, including:

- Fever
- Headache
- Tiredness
- Enlarged lymph nodes in the neck and groin area

These symptoms usually disappear within a week to a month and are often mistaken for another viral infection, such as flu. However, during this period people are highly infectious because HIV is present in large quantities in genital fluids and blood. Some people infected with HIV may have more severe symptoms at first or symptoms that last a long time, while others may have no symptoms for 10 years or more.

3.1.2: Later Symptoms

During the late stages of HIV infection, the virus severely weakens the immune system, and people infected with the virus may have the following symptoms:

- Rapid weight loss
- Recurring fever or profuse night sweats
- Extreme and unexplained tiredness
- Prolonged swelling of the lymph glands in the armpits, groin, or neck
- Diarrhea that lasts for more than a week
- Sores of the mouth, anus, or genitals
- Pneumonia
- Red, brown, pink, or purplish blotches on or under the skin or inside the mouth, nose, or eyelids
- Memory loss, depression, and other neurologic disorders.

Each of these symptoms can be related to other illnesses. The only way to find out if you are infected with HIV is to get tested (NIAID, 2010).

3.2: Testing and Diagnosis

3.2.1: Routine HIV Testing

HIV testing involves taking a blood sample. Credit: NIAID (2010).

People who have been infected recently with HIV often have few to no symptoms yet are extremely infectious and may unknowingly transmit the virus to others. Therefore, the Centers for Disease Control and Prevention (CDC) recommends HIV testing for adults, adolescents, and pregnant women during routine medical care (Revised recommendation of HIV testing, 2006). Regular HIV screenings allow healthcare providers to identify people who are not aware that they are infected with HIV, so that they can be counseled on the need to avoid high-risk behaviors, instructed on safe-sex practices, and given information about starting antiretroviral therapy. HIV testing can also be performed anonymously if a person is concerned about confidentiality (NIAID, 2010).

3.3: Types of HIV Tests

Healthcare providers can test a sample of blood to see if it contains human antibodies (disease-fighting proteins) specific to HIV.

The two key types of HIV antibody tests are the:

- Enzyme-linked immunosorbent assay (ELISA)
- The Western blot.

However, these antibody tests may not detect HIV antibodies in someone who has been recently infected with HIV (within 1 to 3 months of infection). In these situations, healthcare providers can test the blood for the presence of HIV genetic material. This test is extremely critical for identifying recently infected people who are at risk for unknowingly infecting others with HIV.

3.3.1: HIV Testing in Infants

CDC (2006) recommends that all pregnant women get tested for HIV before and/or during delivery. Knowing the HIV status of the mother allows physicians to prevent mother-to-child HIV transmission by providing antiretroviral treatment to both mothers infected with HIV and their newborn infants. However, it is difficult to determine if a baby born to a mother infected with HIV is actually infected because babies carry their mothers' HIV antibodies for several months. Today, healthcare providers can conduct an HIV test for infants between ages 3 months and 15 months. Researchers are now evaluating several blood tests to determine which ones are suitable for testing babies younger than 3 months.

3.4: Treatment of HIV Infection

Photo of a variety of different drug treatments Credit: NIAID (2010).

In the early 1980s when the HIV/AIDS epidemic began, people with HIV were not likely to live longer than a few years.

Today, there are 31 antiretroviral drugs (ARVs) approved by the Food and Drug Administration to treat HIV infection. These treatments do not cure people of HIV or AIDS. Rather, they suppress the virus, even to undetectable levels, but they do not completely eliminate HIV from the body. By suppressing the amount of virus in the body, people infected with HIV can now lead longer and healthier lives. However, they can still transmit the virus and must continuously take antiretroviral drugs in order to maintain their health quality.

3.5: Classes of HIV/AIDS Antiretroviral Drugs

The antiretroviral medications used to treat HIV/AIDS currently are organized into five major drug classes.

1 **Reverse Transcriptase (RT) Inhibitors** interfere with the critical step during the HIV life cycle known as reverse transcription. During this step, the HIV enzyme RT converts HIV RNA to HIV DNA. There are two main types of RT inhibitors.

a) Nucleoside/nucleotide RT inhibitors are faulty DNA building blocks. When these faulty pieces are incorporated into the HIV DNA (during the process when HIV RNA is converted to HIV DNA), the DNA chain cannot be completed, thereby blocking HIV from replicating in a cell.
b) Non-nucleoside RT inhibitors bind to RT, interfering with its ability to convert the HIV RNA into HIV DNA.

2. **Protease Inhibitors** interfere with the protease enzyme that HIV uses to produce infectious viral particles.

3. **Fusion/Entry Inhibitors** interfere with the virus' ability to fuse with the cellular membrane, thereby blocking entry into the host cell.

4. **Integrase Inhibitors** block integrase, the enzyme HIV uses to integrate genetic material of the virus into its target host cell.

5. **Multidrug Combination Products** combine drugs from more than one class into a single product. To combat virus strains from becoming resistant to specific antiretroviral drugs, healthcare providers recommend that people infected with HIV take a combination of antiretroviral drugs known as highly active antiretroviral therapy (HAART). Developed by NIAID-supported researchers, the HAART strategy combines drugs from at least two different antiretroviral drug classes.

In Development

Another HIV/AIDS drug class known as maturation inhibitors is still in development. If successful, they could potentially prevent HIV from properly assembling and maturing. For example, these treatments could block HIV from forming a protective outer coat or from emerging from human cells (NIAID, 2010).

3.6: Adherence and Drug Resistance

Photo of a weekly drug organizer, Credit: NIAID, 2010

People infected with HIV who take antiretroviral treatments sometimes find it difficult to adhere to their drug regimens. This can be caused by the complexity of having to

take several drugs each day or the unpleasant side effects that may result from some medicines, such as nausea and vomiting. However, when patients fail to take their medicines, HIV has an opportunity to create more variations of itself, including strains that are resistant to antiretroviral drugs. Therefore, it is important for people to continue taking their medicines as prescribed by their healthcare providers.

SELF ASSESSMENT EXERCISE

Explain classes of HIV/AIDS Antiretroviral drugs

3.7: Antiretroviral drugs: complications and side effects

Below are side effects of antiretroviral drugs (Johnson, 2012)

1. Lactic acidosis leads to high levels of acid in the blood, which can be fatal. It can result from the use of NRTIs.

Symptoms of lactic acidosis include:

- Long-lasting nausea, vomiting, and abdominal pain
- Unusual fatigue
- Shortness of breath
- Rapid breathing
- Enlarged or tender liver
- Cold or blue hands and feet
- Abnormal heart beat
- Weight loss

Treatment of lactic acidosis may include:

- Changing your drug regimen, but only under the guidance of your doctor
- Intravenous fluids, possibly in the hospital
- Vitamin supplements

2. Hyperglycemia occurs with higher-than-usual levels of blood sugar, called glucose. It is a symptom of diabetes. However, you can have hyperglycemia without having diabetes. Protease inhibitors, growth hormone drugs, and hepatitis C infection can increase the risk of this side effect.

Symptoms of hyperglycemia include:

- Increased urination
- Excessive thirst or hunger
- Unexplained weight loss

Treatment of hyperglycemia includes:

- Stopping protease inhibitors, but only under the guidance of your doctor
- Hypoglycemic drugs (to lower blood sugar) taken by mouth
- Insulin injected under the skin

3. Hyperlipidemia is an increase of fat in the blood. These fats include cholesterol and triglycerides. This condition can lead to heart disease and pancreatitis, an inflammation of the pancreas. Some protease inhibitors can increase this side effect.

Symptoms of hyperlipidemia do not exist. The only way to know if you have this condition is to have lab tests at least once a year.

Treatment of hyperlipidemia includes taking cholesterol-lowering drugs, such as statins or fibrates.

4. **Lipodystrophy** is also called fat redistribution. If you have it, your body produces, uses, and stores fat differently. This side effect is associated with the use of both NRTIs and PIs as well as the HIV virus itself. It is less common with the newer medications.

Symptoms of lipodystrophy include:

- A buildup of fat in the neck or upper shoulders, belly, or breasts
- A loss of fat in the face, arms, legs, or buttocks

Treatment of lipodystrophy may include:

- A change in HIV drugs, but only under the guidance of your doctor
- Egrifta is a drug given daily by injection. Side effects include include joint pain, redness and rash at the site of injection, stomach pain, swelling, and muscle pain. The drug may also cause increases in blood sugar.
- Exercise and diet changes
- Glucophage (metformin), a drug to lower high blood sugar and help reduce abdominal fat
- Hormone treatment (such as human growth hormone), injections of fat or synthetic material, or implants

6. Hepatotoxicity is liver damage. It may result from several classes of HIV drugs, including NNRTIs, NRTs, and PIs. Liver damage may include inflammation, death of liver cells, or too much fat in the liver.

Symptoms of liver damage include:

- Increased liver enzymes in the blood
- Nausea or vomiting
- Abdominal pain
- Loss of appetite or diarrhea
- Fatigue
- Yellowing of skin and eyes (jaundice)

- Enlarged liver
- Abnormal fat distribution
- Abnormal lipid and glucose metabolism
- Bone loss

Treatment of liver damage includes stopping or changing HIV drugs, but only under the guidance of your doctor.

7. Skin rashes may range from mild to severe, covering at least 30% of the body's skin surface area. Some are life-threatening. All classes of HIV drugs, including fusion inhibitors, may cause this side effect.

Symptoms of severe rashes include:

- Flat or raised red spots with blisters in the center
- Blisters in the mouth, eyes, genitals, or other moist areas
- Peeling skin that causes painful sores
- Fever
- Headache

Treatment of skin rashes includes:

- A change in medications, but only under the guidance of your doctor
- Antihistamine drugs
- Hospitalization and intravenous fluids and medications for severe skin rashes (Johnson, 2012)

Monitoring for these complications and side effects is the responsibility of patients and their healthcare providers.

3.8: HIV Prevention

Consistent use of male latex condoms can help protect against HIV infection. Credit: NIAID, 2010

Currently, there is no vaccine to prevent HIV infection nor is there a cure for HIV/AIDS. To reduce your risk of becoming infected with HIV or transmitting the virus to others:

- Get tested regularly for HIV
- Practice abstinence

- Remain faithful to your spouse or partner
- Consistently use male latex or female polyurethane condoms
- Do not share needles

Topical microbicides, such as gels, creams, and foams, are designed to be applied to the vagina or rectum prior to sexual intercourse. Credit: NIAID (2010)

Antiretrovirals as an HIV prevention tool

3.9: Challenges in Designing HIV Vaccines

Vaccines teach the immune system to recognize a specific harmful organism and fight off the disease when the body faces the real thing. Despite extraordinary advances in understanding both HIV and the human immune system, a fully successful HIV vaccine continues to elude researchers.

The most difficult challenges today for HIV vaccine researchers are:

- HIV attacks CD4+ T cells, the most important part of the immune system that coordinates and directs the activities of other types of immune cells that combat intruding microbes. For a vaccine to be effective, it will need to be able to activate these cells - a difficult feat if they are being infected and destroyed by the virus.
- Scientists have not identified the correlates of immunity, or protection, for HIV and are still trying to design vaccines to induce the appropriate immune responses necessary for protection. Unlike other viral diseases for which investigators have made successful vaccines, there are no documented cases of complete recovery from HIV infection. Therefore, HIV vaccine researchers have no human model of recovery from infection and subsequent protection from re-infection to guide them.
- In an infected person, HIV continually mutates and recombines to evolve into new strains of virus that differ slightly from the original infecting virus. This extensive diversity of HIV poses a challenge to vaccine design as an HIV vaccine would need to protect against many different strains of the virus circulating throughout the world. Conventional vaccines have had to protect against one or a limited number of strains.

- Ideally, an HIV vaccine will marshal two kinds of immune responses to fight HIV: T cells and antibodies secreted by B cells. These immune responses would prevent the establishment and spread of the virus from the original site of infection and decrease the effects of the disease in those who do become infected. However, scientists have not yet been able to stimulate both types of responses. To date, researchers have only stimulated T cell responses weakly with experimental HIV vaccines and have had difficulty stimulating the production of antibodies that protect against a broad range of HIV strains.
- Researchers lack the knowledge about which HIV immunogens, pieces of HIV used to construct an experimental HIV vaccine, will get the immune system to recognize HIV during an actual encounter and protect against disease.
- Lack of a practical animal model to predict the effectiveness of an HIV vaccine in people hampers HIV vaccine development. Currently, researchers rely on experiments using non-human primate models infected with the simian cousin of HIV, known as SIV, and an engineered combination of SIV and HIV, known as SHIV, to somewhat mimic disease progression. Evaluating experimental vaccines in these animals requires an SIV or SHIV analog instead of the actual HIV vaccine candidate used in clinical trials in humans (NIAID, 2010)

4.0 CONCLUSION

Did you enjoy your studies, we guess you did. This unit started by introducing you to symptoms of HIV/AIDS of which some are rapid weight loss, sores in mouth, genitals and anus, etc. The unit briefly presented insight on testing and diagnosis of HIV and management. It further illustrates classes of HIV/AIDS drugs, side effects, prevention of HIV and challenges of designing HIV vaccines.

5.0 SUMMARY

In this unit, we studied:

- Early and later symptoms of HIV
- HIV testing and diagnosis
- HIV symptoms
- Types of HIV tests
- HIV testing in infants
- Treatment/management of HIV infection
- Classes of HIV/AIDS Antiretroviral Drugs
- Adherence and Drug resistance
- Complications and side effects of HIV drugs
- HIV prevention
- Challenges in designing HIV vaccine

6.0 TUTOR MARKED ASSIGNMENT

Explain side effects of HIV antiretroviral drugs

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MODULE 4: HIV COUNSELLING AND MANAGEMENT

UNIT 1 HIV COUNSELLING

CONTENT

1.0: INTRODUCTION

In the previous unit, we identified early and later symptoms of HIV, HIV testing and diagnosis, types of HIV tests, classes of HIV/AIDS antiretroviral drugs, adherence and drug resistance, complications and side effects of HIV drugs and HIV prevention. In this unit, we will study basic elements of HIV counseling.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- Define HIV Counseling
- Explain aims of counseling in HIV infection prevention
- Identify different HIV counseling programmes and services

- Illustrate points in Pre-test discussion
- Identify points for counselor and/or physician to cover for Pre-test counseling
- Illustrate points in Post-test discussion
- Explain causes of uncertainty in the case of HIV positive result
- Describe counseling during combination antiretroviral therapy

3.0 MAIN CONTENT

3.1 What is HIV counseling?

Counseling in HIV and AIDS has become a core element in a holistic model of health care, in which psychological issues are recognized as integral to patient management.

HIV and AIDS counseling has two general aims:

(1) The prevention of HIV transmission and

(2) The support of those affected directly and indirectly by HIV

It is vital that HIV counseling should have these dual aims because the spread of HIV can be prevented by changes in behaviour. One to one prevention counseling has a particular contribution in that it enables frank discussion of sensitive aspects of a patient's life —such discussion may be hampered in other settings by the patient's concern for confidentiality or anxiety about a judgmental response. Also, when patients know that they have HIV infection or disease, they may suffer great psychosocial and psychological stresses through a fear of rejection, social stigma, disease progression, and the uncertainties associated with future management of HIV. Good clinical management requires that such issues be managed with consistency and professionalism, and counseling can both minimize morbidity and reduce its occurrence. All counselors in this field should have formal counseling training and receive regular clinical supervision as part of adherence to good standards of clinical practice.

3.2 Aims of Counseling in HIV Infection Prevention

To:

1. Determine whether the lifestyle of an individual places him or her at risk
2. Work with an individual so that he or she understands the risks
3. Help to identify the meanings of high risk behaviour
4. Help to define the true potential for behaviour change
5. Work with the individual to achieve and sustain behaviour change

3.3 Different HIV counseling programmes and services

They are:

1. Counseling before the test is done
2. Counseling after the test for those who are HIV positive and HIV negative
3. Risk reduction assessment to help and prevent transmission
4. Counseling after a diagnosis of HIV disease has been made
5. Family and relationship counseling
6. Bereavement counseling
7. Telephone "hotline" counseling
8. Outreach counseling
9. Crisis intervention
10. Structured psychological support for those affected by HIV
11. Support groups

SELF ASSESSMENT EXERCISE

- What is HIV Counseling?
- Identify Different HIV counseling programmes and services

3.4 HIV Pre-test discussion

A discussion of the implications of HIV antibody testing should accompany any offer of the test itself. This is to ensure the principle of informed consent is understood and to assist patients to develop a realistic assessment of the risk of testing positive to HIV antibody. This process should include accurate and up to date information about transmission and prevention of HIV and other sexually transmitted infections. Patients should be made aware of the "window period" for the HIV test—that a period of 12 weeks since the last possible exposure to HIV should have elapsed by the time of the test.

3.4.1 Pretest discussion checklist: Indications for further counseling and referral to counselor

1. People who have been sexually active in areas of high HIV prevalence
2. Men who have sex with men
3. Current or previous sexual partners HIV positive
4. Client presenting with clinical symptoms of HIV infection
5. High risk sexual behaviour
6. High risk injecting drug practices
7. Learning or language difficulties

3.4.2 Points for Counselors and/or Physician to cover for Pre-test Counseling

1. What is the HIV antibody test (including seroconversion)

2. The difference between HIV and AIDS
3. The window period for HIV testing
4. Medical advantages of knowing HIV status and treatment options
5. Transmission of HIV
6. Safer sex and risk reduction
7. Safer injecting drug use
8. If the client were positive how would the client cope: personal resources, support network of friends/partner/family
9. Who to tell about the test and the result
10. Partner notification issues
11. HIV status of regular partner: is partner aware of patient testing?
12. Confidentiality
13. Does client need more time to consider?
14. Is further counseling indicated?
15. How the results of the test are obtained (in person from the physician or counselor)

Patients may present for testing for any number of reasons, ranging from a generalized anxiety about health to the presence of HIV related physical symptoms. For patients at minimal risk of HIV infection, pre-test discussion provides a valuable opportunity for health education and for safer sex messages to be made relevant to the individual. For patients who are at risk of HIV infection, pre-test discussion is an essential part of post-test management. These patients may be particularly appropriate to refer for specialist counseling expertise. In genitourinary medicine clinics where HIV antibody testing is routinely offered as a part of sexual health screening, health advisers should provide counseling to patients who have been identified as high risk for testing HIV positive.

The importance of undertaking a sensitive and accurate sexual/and or injecting drug risk history of both the patient and their sexual partners cannot be overstated. If patients feel they cannot share this information with the physician or counselor then the risk assessment becomes meaningless; patients may be inappropriately reassured, for example, and be unable to disclose the real reason for testing. Counseling skills are clearly an essential part of establishing an early picture of the patient and his/her history and of how much intervention is needed to prepare him or her for a positive result, and to further reinforce prevention messages. It is at this stage that potential partners at risk are identified which will become an important part of the patient's management if HIV positive.

3.5 Post-test Discussion

HIV results should be given simply, and in person. For HIV negative patients this may be a time where the information about risk reduction can be "heard" and further reinforced. With some patients it may be appropriate to consider referral for further work on personal strategies to reduce risks—for example one to one or group interventions. The window period of 12 weeks should be checked again and the

decision taken about whether further tests for other sexually transmitted infections are appropriate.

3.6 Causes of Uncertainty in the Case of HIV Positive Result

- The cause of illness: Progression of disease
- Management of dying, Prognosis
- Reactions of others (loved ones, employers, social networks)
- Effects of treatment
- Long term impact of antiretroviral therapy
- Impact of disclosure and how this will be managed

HIV positive patients should be allowed time to adjust to their diagnosis. Coping procedures rehearsed at the pre-test discussion stage will need to be reviewed in the context of the here and now; what plans does the patient have for today, who can they be with this evening? Direct questions should be answered but the focus is on plans for the immediate few days, when further review by the counselor should then take place. Practical arrangements including medical follow up should be written down. Overloading the patient with information about HIV should be avoided at this stage. Sometimes this may happen because of the health professional's own anxiety rather than the patient's needs. Counseling support should be available to the patient in the weeks and months following the positive test results.

3.7 Counseling during Combination Antiretroviral Therapy

Significant developments in combination antiretroviral therapy have led to a surge of optimism about long term medical management of HIV infection, and people are now living much longer with HIV. *Patient adherence* is an important factor in the efficacy of drug regimens. However, taking a complicated drug regimen—often taking large numbers of tablets several times a day—is a constant reminder of HIV infection. The presence of side effects can often make patients feel more unwell than did the HIV and some may be unable to cope with the side effects.

Counseling may be an important tool in determining a realistic assessment of individual adherence and in supporting the complex adjustment to a daily routine of medication.

4.0 CONCLUSION

Counseling in HIV and AIDS is thus very vital in prevention and management programmes. It aims at preventing the HIV transmission as well supporting those infected and affected by HIV. Different HIV counseling services includes pre-test and post-test counseling, bereavement counseling, crisis intervention, Telephone ‘hotline’ counselling, outreach counseling, etc.

5.0 SUMMARY

In this unit, we have been able to:

- Define HIV Counseling
- Explain aims of counseling in HIV infection prevention
- Identify different HIV counseling programmes and services
- Illustrate points in Pre-test discussion
- Identify points for counsellor and/or physician to cover for Pre-test counseling
- Illustrate points in Post-test discussion
- Explain causes of uncertainty in the case of HIV positive result
- Describe counseling during combination antiretroviral therapy

Let us attempt the question below

6.0 TUTOR MARKED ASSIGNMENT

- Identify points for counselor and/or physician to cover for pre-test counseling
- Explain causes of uncertainty in the case of HIV positive result

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UNIT 2 PSYCHOLOGICAL ISSUES IN HIV COUNSELING AND MANAGEMENT

CONTENT

1.0 INTRODUCTION

Many reactions to an HIV positive diagnosis are part of the normal and expected range of responses to news of a chronic, potentially life threatening medical condition. Many patients adjust extremely well with minimal intervention. Some will exhibit prolonged periods of distress, hostility, or other behaviours which are difficult to manage in a clinical setting. It should be noted that serious psychological maladjustment may indicate pre-existing morbidity and will require psychological/psychiatric assessment and treatment. Depressed patients should always be assessed for suicidal ideation.

Effective management requires allowing time for the shock of the news to sink in; there may be a period of emotional "ventilation", including overt distress. The counselor should provide an assurance of strict confidentiality and rehearse, over time, the solutions to practical problems such as who to tell, what needs to be said, discussion around safer sex practices and adherence to drug therapies. Clear information about medical and counseling follow up should be given. Counseling may be of help for the patient's partner and other family members.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- Identify psychological issues in HIV/AIDS counseling and management
- Highlight the importance of counseling patients and partners together
- Explain HIV coping strategies

3.0 MAIN CONTENT

3.1 Psychological issues in HIV/AIDS counseling and management

Psychological issues to take note of in HIV counseling and management include:

Shock

- of diagnosis
- recognition of mortality
- of loss of hope for the future

Fear and anxiety

- uncertain prognosis
- effects of medication and treatment/treatment failure
- of isolation and abandonment and social/sexual rejection
- of infecting others and being infected by them
- of partner's reaction

Depression

- in adjustment to living with a chronic viral condition
- over absence of a cure
- over limits imposed by possible ill health
- possible social, occupational, and sexual rejection
- if treatment fails

Anger and frustration

- over becoming infected
- over new and involuntary health/lifestyle restrictions
- over incorporating demanding drug regimens, and possible side effects, into daily life

Guilt

- interpreting HIV as a punishment; for example, for being gay or using drugs
- over anxiety caused to partner/family

3.2 Counselling Patients and Partners Together

This should only take place with the patient's explicit consent, but it may be important for the following *reasons*:

- Adjustments to sexual behaviour and other lifestyle issues can be discussed and explained clearly to both.
- If the patient's partner is HIV negative (ie a serodiscordant couple) particular care and attention must be paid to emotional and sexual consequences in the relationship.
- Misconceptions about HIV transmission can be addressed and information on safer sex given.
- The partner's and the patient's psychological responses to the diagnoses or result, such as anxiety or depression, can be explained and placed in a manageable perspective
- There may be particular issues for couples who have children or who are hoping to have children or where the woman is pregnant
- Partners and family members sometimes have greater difficulty in coming to terms with the knowledge of HIV infection than the patients do themselves. Individual counseling support is often required to manage this, particularly role changes within the relationship, and other adjustment issues that may lead to difficulties. This is part of a holistic approach to the patient's overall health care.
- In many cases the need for follow up counseling may be episodic and this seems appropriate given the long term nature of HIV infection and the different challenges a patient may be faced with. The number of counseling sessions required during any of these periods largely depends on the individual presentation of the patient and the clinical judgment of the counselor.

SELF ASSESSMENT EXERCISE

- Identify the importance of counseling patients and spouses together

3.3 HIV coping strategies

The importance of encouraging and working towards coping strategies involving *active participation* (to the extent the patient can manage) in planning of care and in *seeking appropriate social support* has been demonstrated clinically and empirically. Such an approach includes encouraging problem solving, participation in decisions about their treatment and care, and emphasizing self worth and the potential for personal control over manageable issues in life.

Many patients diagnosed with HIV some years ago are now feeling well enough to return to work and to study and are, paradoxically, learning to readjust to living, as they had formally adjusted to the possibility of dying. Patients also have to deal with the uncertainty which remains about the long term efficacy of current medical treatment, and there are some who will fail on combination therapy. Even with the significant medical advances in patient management, counseling remains an integral part of the management of patients with HIV, and their partners and family.

4.0 CONCLUSION

We have seen that reactions to an HIV positive diagnosis is part of the normal and expected range of responses to news of a chronic, potentially life threatening medical condition. Effective management requires allowing time for the shock of the news to sink in; there may be a period of emotional "ventilation", including overt distress. Also counseling may be of help for the patient's partner and other family members.

5.0 SUMMARY

In this unit, we studied:

- Psychological issues in HIV counseling and management
- The values of counseling patients and partners together
- HIV coping strategies

6.0 TUTOR MARKED ASSIGNMENT

- Why is it important to counsel HIV patient and partner together?

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UNIT 3 HIV MANAGEMENT: THE ROLE OF NUTRITION

CONTENT

1.0 INTRODUCTION

This unit seeks to provide an overview of the role of nutrition in the management of HIV/AIDS.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- Discuss the primary nutrition problem caused by HIV/AIDS
- Describe the nutrition problems associated with combination anti-retroviral therapy
- List practical suggestions for improving nutritional status in symptomatic AIDS

3.0 MAIN CONTENT

3.1 Nutrition Problems Associated with HIV

There are minor disturbances in metabolic and nutritional status in asymptomatic HIV infection but these do not influence energy balance or disease progression (Sharpston, et al, 1996; 1999). Malnutrition is not a complication of asymptomatic HIV disease. There is limited need for intervention with diet at this stage of disease although patients sometimes request healthy eating advice.

Weight loss and malnutrition are common manifestations of HIV, and are a major cause of morbidity and mortality. There is a progressive depletion of body weight and

lean body mass (LBM) as patients near death. Kotler et al demonstrated that at time of death body weight was depleted to 66% of ideal and lean body mass 54% of ideal (Kottler, 1989). There was no relationship between time of death and body fat content. This data suggests that lean body mass rather than fat affects survival in AIDS wasting.

The most common risk factors for malnutrition are anorexia, acute infection, fever and diarrhoea (Macallan, et al, 1995). Weight loss occurs when the energy (calorie) intake from food and drink is lower than the minimum amount of calories required for basic metabolic functions of your body.

Weight loss in HIV infected patients is not a continuous process. It is often episodic, coinciding with secondary infection, especially Pneumocystis carinii pneumonia (PCP), cytomegalovirus (CMV), gastrointestinal infection and bacterial infections (Macallan, et al. 1993; Grunfield, et al, 1992). During such episodes, profound reduction of calorie intake (Grunfield, et al, 1992; Macallan, et al, 1995), and metabolic alterations are likely to act synergistically to promote rapid loss of lean tissue.

Opportunistic infection will alter resting energy expenditure, body composition (with losses of fat and fat free mass) and reduce food intake to different degrees depending on the specific infection.

The distinction between a nutritional starvation response, (as seen in patients with protozoal diarrhoea), and a nutritional cachectic response, (as seen in patients with systemic Mycobacterium avium intracellulare) is an important determinant over success with nutritional intervention. (Sharpstone et al, 1996) In the situation of cachexia increasing nutrition will not replete lean body mass until the underlying stimulus driving the cachexia is treated.

The nutritional management of patients with symptomatic disease is therefore best co-ordinated with knowledge of current infection, because of this varied metabolic response in different opportunistic diseases. Increased knowledge about the cause of nutritional problems will allow the clinician to advise the patient on the reason for nutritional intervention and the likelihood of intervention being successful.

Optimal nutritional management of patients with opportunistic infections should include aggressive therapy both of opportunistic infection and associated weight loss. Nutritional intervention should therefore be started at diagnosis of any events to minimise nutritional losses.

3.2 Nutrition problems associated with combination Anti-retroviral therapy

Introduction of combination anti-retroviral therapy has led to malnutrition no longer being a major complication of HIV disease. However this has not resulted in normalisation of nutritional status.

Side effects possibly associated with therapy such as a fat redistribution syndrome and metabolic complications are reported. This is known as *Lipodystrophy*. In this scenario we see abnormal redistribution of body fat, with accumulation in the abdominal area, in the axillary pads, and in the dorsocervical pads. In contrast there is a decrease in body fat in the legs, arms and nasolabial and cheek pads. Coupled with the body composition change we see metabolic alterations such as hyperlipidaemia and insulin resistance. (Ketler 2000)

Treatment is based on the increased morbidity likely to be associated with atherosclerotic disease. The precise nature of the risk is uncertain and investigators argue that the risk of morbidity is low in relation to the benefits from anti-retroviral therapy.

SELF ASSESSMENT EXERCISE

What is Lipodystrophy?

3.3 Improving Nutritional Status in Symptomatic AIDS

The complex relationship between the factors involved in HIV wasting complicates the design of nutritional approaches. Dietary advice needs to be individualised to maximise the chance of effectiveness. Specific advice should be offered to the patient if they experience a profound loss of appetite, vomiting, diarrhoea, or a sore mouth. Patients experiencing acute symptoms are anxious. Individualising advice will allow the advice to be kept as simple as possible, and provide the best chance of the patient totally understanding the purpose of the advice.

Dietary advice should begin with suggestions about food intake. In some patients with very severe eating problems it is necessary to consider liquid instead of solids, as these will be consumed easier. Sometimes specialised supplement drinks are helpful if problems with eating persist. In situations where it is difficult to access ready-made drinks it is possible to make up soups and drinks at home using cooled boiled water, fruit juice, soy products, fruits or vegetables.

3.4 Practical suggestions for food intake in symptomatic AIDS (Ross, 2001)

Anorexia	Investigate cause of anorexia Encourage foods without strong smell Encourage cold foods Provide foods of choice	
Vomiting	Ice cubes from cooled boiled water Fluids, cooled boiled water, green tea, diluted fruit juices Chilled foods Soups, puddings Light foods Try use of ginger	
	Increase	**Decrease**
Sore mouth	Try to eat soft foods soups, puddings, mash foods Encourage fluids Use a straw if this helps	Avoid spicy foods Avoid very hard foods Avoid acid foods Avoid extremes of temperatures
Diarrhoea	Encourage fluids, increase low fibre starchy foods, rice, noodles, potatoes Increase protein foods eggs, pork, chicken, tofu	Avoid heavily spiced foods Avoid very fatty foods Avoid green vegetables

4.0 CONCLUSION

Studies in HIV wasting have demonstrated that opportunistic illness is associated with gross nutritional depletion. Dietary intervention should take place early to minimise nutritional losses. Patients who can access combination therapy face different nutritional challenges. Future research will more clearly define the mechanism behind lipodystrophy. Assessment of nutritional status and attention to diet ideally should be prioritised at onset of opportunistic illness. Advice should be relevant to the individual, to local need and resources.

5.0 SUMMARY

In this unit, we studied:

- Nutrition problem associated with HIV
- Nutrition problems associated with combined anti-retroviral therapy
- Improving nutritional status in symptomatic AIDS
- Practical guidelines for food intake in symptomatic AIDS

6.0 TUTOR MARKED ASSIGNMENT

- Give practical suggestions for food intake in symptomatic AIDS

7.0 REFERENCES/FURTHER READINGS

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