NATIONAL OPEN UNIVERSITY OF NIGERIA

SCHOOL OF SCIENCE AND TECHNOLOGY

COURSE CODE: HEM 702

COURSE TITLE: BASICS OF HIV/AIDS

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MODULE I EVOLUTION OF A PANDEMIC

UNIT 1 DEFINING HIV/AIDS

CONTENT

1.0 INTRODUCTION

People have been warned about HIV and AIDS for over twenty 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 don't know exactly what HIV and AIDS *actually are*. This unit defines HIV/AIDS and sorts the myths from the facts about AIDS.

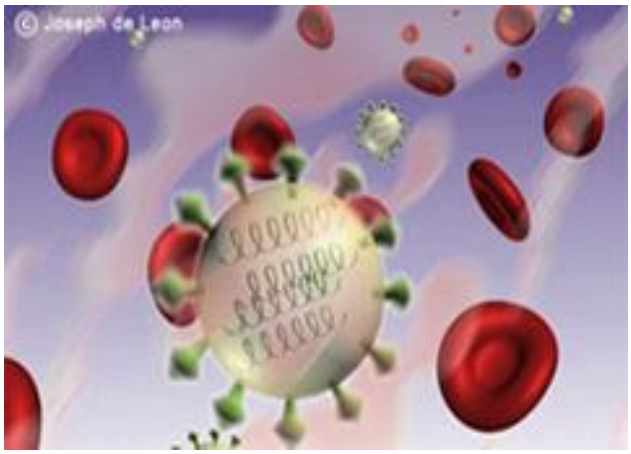
2.0 OBJECTIVES

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

- Define HIV and AIDS
- Identify timetable of important events in the history of AIDS
- Ascertain why HIV is describe as a dangerous virus
- Identify how long it takes for HIV to become AIDS
- Identify the HIV facts and myths
- Explain what 'safe sex' and ‘safer sex’ mean
- Ascertain if anything can create HIV
- Ascertain if there is a cure for AIDS

3.0 MAIN CONTENT

3.1 What is HIV?



HIV (Human Immunodeficiency Virus)

HIV is a virus. Viruses infect the cells of living organisms and replicate (make new copies of themselves) within those cells. A virus can also damage human cells, which is one of the things that can make an infected creature become ill.

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.

HIV stands for the '*Human Immunodeficiency Virus*'. Someone who is diagnosed as infected with HIV is said to be 'HIV+' or 'HIV positive'.

3.2 What is AIDS?

The full name of AIDS is Acquired Immune Deficiency Syndrome. As the name implies, it is a disease caused by a deficiency in the body’s immune system. It is a *syndrome* because there are a range of different symptoms which are not always

found in each case. It is *acquired* because AIDS is an infectious disease caused by a virus which is spread from person to person through a variety of routes. This makes it different from immune deficiency from other causes such as treatment with anti-cancer drugs or immune system suppressing drugs given to persons receiving transplant (Hubley, 1995).

A damaged immune system is not only more vulnerable to HIV, but also to the attacks of other infections. It will not always have the strength to fight off things that would not have bothered it before.

As time goes by, a person who has been infected with HIV is likely to become ill more and more often until, usually several years after infection, they become ill with one of a number of particularly severe illnesses. It is at this point that they are said to have AIDS - when they first become seriously ill, or when the number of immune system cells left in their body drops below a particular point. Different countries have slightly different ways of defining the point at which a person is said to have AIDS rather than HIV.

AIDS (*Acquired Immune Deficiency Syndrome*) is an extremely serious condition, and at this stage the body has very little defense against any sort of infection.

3.3 Timetable of Important Events in the History of AIDS

Pre 1980 silent period: HIV being transmitted before AIDS was recognised as a new disease.

1981 Epidemic of Pneumonocystis carinii infection in Los Angeles, USA

1981 Epidemic of Kaposi's sarcoma in New York, USA

1982 Case definition produced for AIDS by Center for Disease Control, Atlanta

1982 Slim disease encountered in Rakai, Uganda

1983 Increase in Kaposi's sarcoma found in Lusaka, Zambia

1983 Isolation of virus by Luc Montagnier in France

1985 ELISA blood test development

1986 Dr Halfdan Mahler, then Director of WHO, addresses United Nations on AIDS

1987 WHO special programme on AIDS formed (becoming a Global Programme on AIDS in 1988).

1987 HIV2 virus found in AIDS patients in West Africa

1988 First global meeting of health ministers on AIDS

1990 First conference on AIDS in Asia and the Pacific held in Canberra, Australia.

3.4 Why is HIV dangerous?

The immune system is a group of cells and organs that protect the body by fighting disease. The human immune system usually finds and kills viruses fairly quickly.

So if the body's immune system attacks and kills viruses, what's the problem?

Different viruses attack different parts of the body - some may attack the skin, others the lungs, and so on. The common cold is caused by a virus. What makes HIV so dangerous is that it attacks the immune system itself - the very thing that would normally get rid of a virus. It particularly attacks a special type of immune system cell known as a *CD4 lymphocyte*.

HIV has a number of tricks that help it to evade the body's defenses, including very rapid mutation. This means that once HIV has taken hold, the immune system can never fully get rid of it.

There is no way to tell just by looking if someone's been infected by HIV. In fact a person infected with HIV may look and feel perfectly well for many years and may not know that they are infected. But as the person's immune system weakens they become increasingly vulnerable to illnesses, many of which they would previously have fought off easily.

The only reliable way to tell whether someone has HIV is for them to take a blood test, which can detect infection from a few weeks after the virus first entered the body.

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.

3.6 HIV facts and myths

People with HIV look just like everybody else

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.

3.7 What does 'safe sex' mean?

Safe sex refers to sexual activities which do not involve any blood or sexual fluid from one person getting into another person's body. If two people are having safe sex then, even if one person is infected, there is no possibility of the other person becoming infected. Examples of safe sex are cuddling, mutual masturbation, 'dry' (or 'clothed') sex . . .

In many parts of the world, particularly the USA, people are taught that the best form of safe sex is no sex - also called 'sexual abstinence'. Abstinence is not a form of sex at all - it involves avoiding *all* sexual activity. Usually, young people are taught that they should abstain sexually until they marry, and then remain faithful to their partner. This is a good way for someone to avoid HIV infection, as long as their husband or wife is also completely faithful and does not infect them.

SELF ASSESSMENT EXERICES

Why is HIV dangerous?

3.8 What is 'safer sex'?

Safer sex is used to refer to a range of sexual activities that hold *little* risk of HIV infection.

Safer sex is often taken to mean using a condom for sexual intercourse. Using a condom makes it very hard for the virus to pass between people when they are having sexual intercourse. A condom, *when used properly*, acts as a physical barrier that prevents infected fluid getting into the other person's body.

3.9 Is kissing risky?

Kissing someone on the cheek, also known as social kissing, does not pose any risk of HIV transmission.

Deep or open mouthed kissing is considered a very low risk activity for transmission of HIV. This is because HIV is present in saliva but only in very minute quantities, insufficient to lead to HIV infection alone.

There has only been one documented instance of HIV infection as a result of kissing out of all the millions of cases recorded. This was as a result of infected blood getting into the mouth of the other person during open mouthed kissing, and in this instance both partners had seriously bleeding gums.

3.10 Can anything 'create' HIV?

No. Unprotected sex, for example, is only risky if one partner is infected with the virus. If your partner is not carrying HIV, then no type of sex or sexual activity between you is going to cause you to become infected - you can not 'create' HIV by having unprotected anal sex, for example.

You also can not become infected through masturbation. In fact nothing you do on your own is going to give you HIV - it can only be transmitted from another person who already has the virus.

3.11 Is there a cure for AIDS?

Worryingly, surveys show that many people think that there's a 'cure' for AIDS - which makes them feel safer, and perhaps take risks that they otherwise shouldn't. These people are wrong, though - there is still no cure for AIDS.

There is antiretroviral medication which slows the progression from HIV to AIDS, and which can keep some people healthy for many years. In some cases, the antiretroviral medication seems to stop working after a number of years, but in other cases people can recover from AIDS and live with HIV for a very long time. But they have to take powerful medication every day of their lives, sometimes with very unpleasant side effects.

There is still no way to cure AIDS, and at the moment the only way to remain safe is not to become infected.

4.0 CONCLUSION

We hope you enjoyed your studies. This unit provided basic introductory fact on HIV/AIDS and they were also presented in a simple and straightforward way for easier comprehension. Note the question and answer format used in the presentation. This enables the reader to consciously provide answers to the questions as he or she reads. This also aids in test for proper understanding of the concepts.

5.0 SUMMARY

In this unit, we touched on the following: definitions of HIV/AIDS, how long it takes for HIV to become AIDS and how HIV/AIDS is passed on. We also asked a most relevant question: is HIV dangerous? and went further to illustrate the myths and facts of HIV/AIDS.

6.0 TUTOR MARKED ASSIGNMENT

- Identify the myths and facts of HIV/AIDS

ANSWER TO SELF ASSESSMENT EXERCISE

- HIV is dangerous because there is no way to tell just by looking if someone's been infected by the virus. In fact a person infected with HIV may look and feel perfectly well for many years and may not know that they are infected. But as the person's immune system weakens they become increasingly vulnerable to illnesses, many of which they would previously have fought off easily.

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UNIT 2 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 and Tarantola, 1996).

Now in this third decade of our experiences with this pandemic, to many of us, Acquired Immune Deficience Syndrom (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, AIDS 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, as well as the dynamics of the continuing growth of the epidemic in different regions of the world.

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: Common Terms Associated with Describing Epidemics

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, 2005.

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 2005.

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.

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 of 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.

SELF ASSESSMENT EXERCISE

A strange new disease, HIV was first reported where:---------- and in ---------year

3.3 How did HIV/AIDS Spread So Quickly?

This could be explained by scenarios below:

- HIV infection existed at low levels for a long period of time in small tribal communities in Africa. The pool of available, uninfected individuals in such a community is simply too small. In addition, especially in small communities, sexually transmitted diseases seldom become epidemic. Multiple sexual partners would not be accepted in such communities, so transmission would be limited
- 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 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.

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

This unit looked at several common terms associated with describing epidemics. It also provided us with a brief description of the history of HIV/AIDS and lastly attempted an insight on the question: how did HIV/AIDS spread so quickly?

6.0 TUTOR MARKED ASSIGNMENT

Describe the following terms:

- Prevalence
- Incidence
- Endemic
- Epidemic
- Pandemic

7.0 REFERENCES/FURTHER READINGS

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UNIT 3 DEVELOPING GLOBAL PANDEMIC OF HIV/AIDS – REGIONAL STATISTICS

CONTENT

1.0 INTRODUCTION

Each year, the Joint United Nations Programme on HIV/AIDS (UNAIDS), in collaboration with the World Health Organization (WHO), publishes a report on the status of the global HIV/AIDS pandemic**.**

Acquired Immune Deficiency Syndrome (**AIDS**) has led to the deaths of more than 25 million people since it was first recognized in 1981, making it one of the most destructive epidemics in recorded history. Despite recent improved access to antiretroviral treatment and care in many regions of the world, the AIDS epidemic claimed approximately 3.1 million (between 2.8 and 3.6 million) lives in 2005 (an average of 8,500 per day), of which 570,000 were children.

UNAIDS and the WHO estimate that the total number of people living with the **human immunodeficiency virus** (**HIV**) has reached its highest level. There are an estimated 40.3 million (estimated range between 36.7 and 45.3 million) people now living with HIV. Moreover, almost 5 million people have been estimated to have been infected with HIV in 2005 alone.

Regarding the social effects of the HIV/AIDS pandemic, there has been since the 1980s a "profound re-medicalization of sexuality" (UNAIDS, 2005, 2006).

The pandemic is not homogeneous within regions with some countries more afflicted than others. Even at the country level there are wide variations in infection levels between different areas. The number of people living with HIV continues to rise in most parts of the world, despite strenuous prevention

strategies. Sub-Saharan Africa remains by far the worst-affected region, with 23.8 million to 28.9 million people living with HIV at the end of 2005, 1 million more than in 2003. Sixty-four percent of all people living with HIV are in sub-Saharan Africa (UNAIDS, 2006), as are more than 77% of all women living with HIV. South & South East Asia, are second most affected with 15%.

The key facts surrounding this origin of AIDS are currently unknown, particularly where and when the pandemic began, though it is said that it originated from the apes in Africa (UNIADS, 2005).

2.0 OBJECTIVES

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

- Identify the regional statistics of HIV/AIDS in 2005, and 2007.

3.0 MAIN CONTENT

3.1 HIV Statistics - Regional Statistics for HIV & AIDS (UNAIDS, 2005)

World region	Estimated adult prevalence of HIV infection (ages 15–49)	Estimated adult and child deaths during 2005
Worldwide	1.0% to 1.3%	2.8 to 3.6 million
Sub-Saharan Africa	6.6% to 7.8%	1.95 to 2.7 million
Middle East and North Africa	0.1% to 0.7%	25,000 to 145,000
South and South-East Asia	0.4% to 1.0%	290,000 to 740,000
East Asia	0.05% to 0.2%	20,000 to 60,000
Latin America	0.5% to 0.8%	52,000 to 86,000
Caribbean	1.1% to 2.7%	16,000 to 40,000
Eastern Europe and Central Asia	0.6% to 1.3%	39,000 to 91,000
Western and Central Europe	0.2% to 0.4%	12,000 to 15,000
North America,	0.4% to 1.1%	9,000 to 30,000
Oceania	0.5%	1,700 to 8,200

Source: UNA IDS and the WHO 2005 estimates. The ranges define the boundaries within which the actual numbers lie based on the best available information.

SELF ASSESSMENT EXERCISE

- Identify variations in HIV regional statistics and briefly explain reasons for the variations.

3.2 HIV Statistics – Regional Statistics for HIV & AIDS (UNAID, 2007)

The latest HIV statistics were published by UNAIDS in June 2007. The report gives the latest AIDS/HIV statistics from around the world, regional statistics for HIV/AIDS as well as AIDS data for individual countries and regions.

Sub-Saharan Africa

- Adults & Children Living with HIV/AIDS*: 24.7
- Adult Infection Rate(%): 5.9
- Deaths of Adults & Children*: 2.1

East Asia

- Adults & Children Living with HIV/AIDS*: 0.75
- Adult Infection Rate(%): 0.1
- Deaths of Adults & Children*: 0.04

Oceania

- Adults & Children Living with HIV/AIDS*: 0.08
- Adult Infection Rate(%): 0.4
- Deaths of Adults & Children*: 0.004

South & South-East Asia

- Adults & Children Living with HIV/AIDS*: 7.8
- Adult Infection Rate(%): 0.6
- Deaths of Adults & Children*: 0.6

Eastern Europe & Central Asia

- Adults & Children Living with HIV/AIDS*: 1.7
- Adult Infection Rate(%): 0.9
- Deaths of Adults & Children*: 0.084

Western Europe

- Adults & Children Living with HIV/AIDS*: 0.74
- Adult Infection Rate(%): 0.3
- Deaths of Adults & Children*: .012

North Africa & Middle East

- Adults & Children Living with HIV/AIDS*: 0.46
- Adult Infection Rate(%): 0.2
- Deaths of Adults & Children*: 0.036

North America

- Adults & Children Living with HIV/AIDS*: 1.4
- Adult Infection Rate(%): 0.8
- Deaths of Adults & Children*: 0..018

Caribbean

- Adults & Children Living with HIV/AIDS*: 0.25
- Adult Infection Rate(%): 1.2
- Deaths of Adults & Children*: 0.019

Latin America

- Adults & Children Living with HIV/AIDS*: 1.7
- Adult Infection Rate(%): 0.5
- Deaths of Adults & Children*: 0.065

Global Totals

- Adults & Children Living with HIV/AIDS*: 39.5
- Adult Infection Rate(%): 1.0
- Deaths of Adults & Children*: 2.9

Note: ***Figures in millions***

4.0 CONCLUSION

The UNAIDS report of 2005 described a world in which 2.8 – 3.6 million people had already died from this infection, and where HIV, the virus that causes AIDS, continued to spread in all countries of the world. In 2007, UNAIDS gave a global

total of 39.5million people living with HIV/AIDS. This is indeed a worrisome statistics that needs continued check.

5.0 SUMMARY

This unit provided us with HIV/AIDS statistics of 2005 and 2007. Hope you took note of the statistics and monitored the trend and variation of the pandemic. Hope you enjoyed your studies, now let us attempt the questions below.

6.0 TUTOR MARKED ASSIGNMENT

- Draw a table, identifying the regional HIV/AIDS statistics on 2007.
- Comment on death of adult and children in each region.
- Try to identify contributory factors underlining variations in regional HIV/AIDS statistics.
- Comment on possible solutions to HIV/AIDS Scourge

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UNIT 4 HIV/AIDS Epidemiology: HIV/AIDS Prevalence Across the World and Contributory Factors

CONTENT

1.0 Introduction

1.0 INTRODUCTION

UNAIDS and the WHO estimate that AIDS has killed more than 25 million people since it was first recognized in 1981, making it one of the most destructive pandemics in recorded history. Despite recent improved access to antiretroviral treatment and care in many regions of the world, the AIDS pandemic claimed an estimated 2.8 million (between 2.4 and 3.3 million) lives in 2005 of which more than half a million (570,000) were children (UNAIDS 2006 Report on Global AIDS).

Globally, between 33.4 and 46 million people currently live with HIV. In 2005, between 3.4 and 6.2 million people were newly infected and between 2.4 and 3.3 million people with AIDS died, an increase from 2004 and the highest number since 1981.

Sub-Saharan Africa remains by far the worst-affected region, with an estimated 21.6 to 27.4 million people currently living with HIV. Two million [1.5–3.0 million] of them are children younger than 15 years of age. More than 64% of all people living with HIV are in sub-Saharan Africa, as are more than three quarters of all women living with HIV. In 2005, there were 12.0 million [10.6–13.6 million] AIDS orphans living in sub-Saharan Africa 2005. South & South East Asia are second-worst affected with 15% of the total. AIDS accounts for the deaths of 500,000 children in this region. Two-thirds of HIV/AIDS infections in Asia occur in India, with an estimated 5.7 million infections (estimated 3.4–9.4 million) (0.9% of population), surpassing South Africa's estimated 5.5 million (4.9–6.1 million) (11.9% of population) infections, making India the country with the highest number of HIV infections in the world. In the 35 African nations with the highest prevalence, average life expectancy is 48.3 years—6.5 years less than it would be without the disease (UNIADS, 2006)

2.0 OBJECTIVES

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

- Describe HIV/AIDS Prevalence across the globe

3.0 MAIN CONTENT

3.1 HIV/AIDS Prevalence across the Globe

HIV prevalence across the world (UNAIDS)

Sub-Saharan Africa

Sub-Saharan Africa remains the hardest-hit region. HIV infection is becoming endemic in sub-Saharan Africa. It is home to just over 10% of the world's population but more than 60% of all people living with HIV worldwide reside here. The adult (15-49) HIV prevalence rate is 7.2% (range: 6.6 - 8.0%) with between 23.8 and 28.9 million people currently living with HIV. However, it must be noted that the actual prevalence does vary between regions. Presently, Southern Africa is the hardest hit region, with adult prevalence rates exceeding 20% in most countries in the region, and even 30% in Swaziland and Botswana. Eastern Africa also experiences relatively high levels of prevalence with estimates above 10% in some countries, although there are signs that the pandemic is declining in this region, notably in Uganda which previously recorded one of the highest prevalence rates on the continent. West Africa on the other hand has been much less affected by the pandemic, several countries reportedly have prevalence rates around 2-3% and no country has yet rates above 10%, although in two of the region's most populous countries, Nigeria and Côte d'Ivoire, between 5 and 7% of adults are reported to carry the virus.

Across Sub-Saharan Africa, more women are infected with HIV than men, with 13 women living with HIV for every 10 infected men and the gap continues to grow. Throughout the region, women are being infected with HIV at earlier ages than men. The differences in infection levels between women and men are most pronounced among young people (aged 15–24 years). In this age group, there are

36 women living with HIV for every 10 men. The widespread prevalence of sexually transmitted diseases, the practice of scarification, transfusion, and the poor state of hygiene and nutrition in Africa may all be facilitating factors in the transmission of HIV-1 in this region (Bentwich et al., 1995). In 2000, the World Health Organization estimated that 25% of the units of blood transfused in Africa were not tested for HIV, and that 5–10% of HIV infections in Africa, were transmitted via blood (WHO/AFRO, 2001).

Poor economic conditions (leading to the use of dirty needles in healthcare clinics) and lack of sex education contribute to high rates of infection. In some African countries, 25% or more of the working adult population is HIV-positive; in Botswana the figure was 35.8% (1999 estimate *World Press Review*), the highest reported infection rate in the world.

In South Africa, President Thabo Mbeki has questioned in the past the connection between HIV and AIDS - instead hinting at the possibility of factors such as undernourishment being one of the causes of the disease. While South Africa has created preventative programs and research initiatives to address its HIV problem (HIV/AIDS in South Africa, 2008), critics charge that the South African government has been slow to create antiretroviral programs and take other effective medical steps to stop the epidemic (Whiteside, 2003). UNAIDS estimates that in 2005 there were 5.5 million people in South Africa living with HIV — 12.4% of the population. This was an increase of 200,000 people since 2003 (UNAIDS, 2006).

Although HIV infection rates are much lower in Nigeria than in other African countries, the size of Nigeria's population meant that by the end of 2003, there were an estimated 3.6 million people living with HIV. On the other hand, Uganda, Zambia, Senegal, and most recently Botswana have begun intervention and educational measures to slow the spread of HIV, and Uganda has succeeded in actually reducing its HIV infection rate.

Middle East and North Africa

The prevalence in this area is 0.2% (0.1-0.7%), with between 230,000 and 1.4 million people infected. In this area, the routes of transmission of HIV is diverse, including paid sex, sex between men and injecting drug use. Among young people 15–24 years of age, 0.3% of women [0.1–0.8%] and 0.1% of men [0.1–0.3%] were living with HIV by the end of 2004.

SELF ASSESSMENT EXERICE

- Identify the contributory factors of HIV/AIDS in Sub-Saharan Africa

South and South-East Asia

The HIV prevalence rate across this region is less than .35 percent. Due to the population size this brings the total of HIV infections to 4.2 - 4.7 million adults and children. More AIDS deaths (480,000) occur in this region than any other region except sub-Saharan Africa. This sprawling region is not just vast but diverse, with the nature, pace and severity of HIV epidemics differing across the region. The AIDS picture in South Asia is dominated by the epidemic in India, but new data released by UNAIDS shows that India as of 2007 has a relatively low AIDS prevalence rate. With an estimated 2-3.1 million infections, India has the third largest number of people with aids after South Africa and Nigeria. In South and Southeast Asia, the HIV epidemic remains largely concentrated in injecting drug users, men who have sex with men, sex workers, and clients of sex workers and their immediate sexual partners. New infections occur in Thailand and Cambodia at a steady rate. Prevention strategies in these populations are, for the most part, inadequate (Wikipedia, 2007).

East Asia

The national HIV prevalence level in East Asia is 0.1% in the adult (15-49) group. However, due to the large populations of many East Asian nations, this low national HIV prevalence still means that large numbers of people are living with HIV. The picture in this region is dominated by China. Much of the current spread of HIV in China is through injecting drug use and paid sex. In China, the number was estimated at between 430,000 and 1.5 million by independent researchers, with some estimates going much higher. In the rural areas of China, where large numbers of farmers, especially in Henan province, participated in unclean blood transfusions; estimates of those infected are in the tens of thousands. In Japan, just over half of HIV/AIDS cases are officially recorded as occurring amongst homosexual men, with the remainder occurring amongst heterosexuals and also via drug abuse, in the womb or unknown means (Wikipedia, 2007).

Latin America

In this region, only Guatemala and Honduras have national HIV prevalence of over 1%. In these countries, HIV-infected men outnumber HIV-infected women by roughly 3:1. Lower prevalence in other countries disguises serious, localized epidemics. In Mexico, Brazil, Colombia and Argentina, drug injection and homosexual activity are the main modes of transmission, but there is concern that heterosexual activity may soon become a primary method of spreading the virus. Brazil accounts for more than a third of all HIV infections in Latin America, with the routes of transmission including paid sex, sex between men and injecting drug use. Brazil began in the 1980s a comprehensive AIDS prevention and treatment

programme to keep AIDS in check, including the production of generic versions of anti-retroviral drugs (Wikipedia, 2007).

Caribbean

The Caribbean is the second-most affected region in the world. Among adults aged 15–44, AIDS has become the leading cause of death. The adult prevalence rate is between 1.1% and 2.7%. HIV transmission occurs largely through heterosexual intercourse, with two thirds of AIDS cases in this region attributed to this route. Sex between men is also a significant route of transmission, even though it is heavily stigmatized and illegal in many areas. HIV transmission through injecting drug use remains rare, except in Bermuda and Puerto Rico (Wikipedia, 2007).

Eastern Europe and Central Asia

There is also growing concern about a rapidly growing epidemic in Eastern Europe and Central Asia, where an estimated 0.99-2.3 million people were infected in December 2005, though the adult (15-49) prevalence rate is low (0.9%). The rate of HIV infections began to grow rapidly from the mid-1990s, due to social and economic collapse, increased levels of intravenous drug use and increased numbers of prostitutes. By 2004 the number of reported cases in Russia was over 257,000, according to the World Health Organization, up from 15,000 in 1995 and 190,000 in 2002; some estimates claim the real number is up to five times higher, over 1 million. There are predictions that the infection rate in Russia will continue to rise quickly, since education there about AIDS is almost non-existent. Ukraine and Estonia also had growing numbers of infected people, with estimates of 500,000 and 3,700 respectively in 2004. The epidemic is still in its early stages in this region, which means that prevention strategies may be able to halt and reverse this epidemic. However, transmission of HIV is increasing through sexual contact and drug use among the young (<30 years). Indeed, over 80% of current infections occur in this region in people less than 30 years of age (Wikipedia, 2007).

Western Europe

In most Western countries, AIDS cases have fallen to levels not seen since the original outbreak; many attribute this trend to aggressive educational campaigns, screening of blood transfusions and increased use of condoms. Also, the death rate from AIDS in Western Europe has fallen sharply, as new AIDS therapies have proven to be an effective (if expensive) means of suppressing HIV.

In this area, the routes of transmission of HIV is diverse, including paid sex, sex between men, injecting drug use, mother to child and heterosexual sex. However, many new infections in this region occur through contact with HIV-infected individuals from other regions. The adult (15-49) prevalence in this region is 0.3% with between 570,000 and 890,000 people currently living with HIV. Due to the availability of antiretroviral therapy, AIDS deaths have stayed low since the lows of the late 1990s. However, in some countries, a large share of HIV infections remain undiagnosed and there is worrying evidence of antiretroviral drug resistance among some newly HIV-infected individuals in this region. Also, there has been a recent increase in risky behavior among men who have sex with men (Wikipedia, 2007).

North America

United States

The adult prevalence rate in this region is 0.7% with over 1 million people currently living with HIV. In the United States, sex between men (49%), heterosexual sex (32%) and needle sharing by intravenous drug users (14%) remain prominent sources of new HIV infections (CDC, 2005). Currently, rates of HIV infection in the US are highest in the eastern and southern regions, with the exception of California. Currently, between 35,000 to 40,000 new infections occur in the USA every year. AIDS is one of the top three causes of death for African American men aged 25–54 and for African American women aged 35–44 years in the United States of America. In the United States, African Americans make up about 47% of the total HIV-positive population and make up more than half of new HIV cases, despite making up only 12% of the population. AIDS continues to be a problem with illegal sex workers and injecting drug users. The main route of transmission for women is through heterosexual sex, and the main risk factor for them is non-protection and the undisclosed risky behaviour of their sexual partners. African American women are 19 times more likely to contract HIV than white women. Experts attribute this to "AIDS fatigue" among younger people who have no memory of the worst phase of the epidemic in the 1980s and early 1990s, as well as "condom fatigue" among those who have grown tired of and disillusioned with the unrelenting safer sex message. This trend is of major concern to public health workers (Wikipedia, 2008).

In the United States in particular, a new wave of infection is being blamed on the use of methamphetamine, known as crystal meth. Research presented at the 12th Annual Retrovirus Conference in Boston in February 2005 concluded that using crystal meth or cocaine is the biggest single risk factor for becoming HIV+ among US gay men, contributing 29% of the overall risk of becoming positive and 28% of the overall risk of being the receptive partner in anal sex. In addition, several

renowned clinical psychologists now cite crystal as the biggest problem facing gay men today, including Michael Majeski, who reckons meth is the catalyst for at least 80% of seroconversions currently occurring across the United States, and Tony Zimbardi, who calls crystal the number one cause of HIV transmission, and says that high rates of new HIV infection are not being found among non-crystal users. In addition, various HIV and STD clinics across the United States report anecdotal evidence that 75% of new HIV seroconversions they deal with are crystal-related; indeed, in Los Angeles, crystal is regarded as the main cause of HIV seroconversion among gay men in their late thirties. The First National Conference on Methamphetamine, HIV and Hepatitis took place in Salt Lake City in August of 2005.

On the other hand, as in Western Europe, the death rate from AIDS in North America has fallen sharply, as new AIDS therapies have proven to be an effective (if expensive) means of suppressing HIV.

Oceania

There is a very large range of national situations regarding AIDS and HIV in this region. This is due, in part, to the large distances between the islands of Oceania. The wide range of development in the region also plays an important role. The prevalence is estimated at between 0.2% and 0.7%, with between 45,000 and 120,000 adults and children currently living with HIV (Wikipedia, 2008)

4.0 CONCLUSION

In this unit, we provided a very detail picture of HIV/AIDS prevalence across the globe as well as its contributory factors. Sub-Saharan African was identified as the hardest hit area, followed by the Caribbean. Contributory factors therefore include poor health education, poverty, discrimination against women and use of contaminated object, sex between men among many.

5.0 SUMMARY

We hope you enjoyed your studies. Here we identified HIV/AIDS prevalence across the globe and contributory factors. Now let us attempt the questions below.

6.0 TUTOR MARKED ASSIGNMENT

- Compare and contrast the HIV/AIDS prevalence in Sub-Sahara Africa and the Caribbean.
- Identify the causative factors of HIV/AIDS in these regions

ANSWER TO SELF ASSESSMENT EXERCISE

- Poor economic conditions (leading to the use of dirty needles in healthcare clinics) and lack of sex education contribute to high rates of infection in sub-Saharan Africa. Others include cultural practices, inability of women to negotiate safer sex, etc.

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MODULE 2 HIV/AIDS: ORIGIN, THEORIES AND SPREAD

Unit 1 Origin of AIDS and HIV: The First Cases of AIDS
Unit 2 Theories of HIV/AIDS
Unit 3 HIV: Earliest Known Instances of Infection/Spread of HIV

UNIT 1 ORIGIN OF AIDS AND HIV: THE FIRST CASES OF 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 AIDS come from?

The first recognised cases of AIDS 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----------

---------- ----------

The Accronym 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 AIDS.

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 world wide 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.* In order to find the source of AIDS, this unit provided information on the origin of HIV, by describing the virus as well views on Simian Immunodeficiency Virus (SIV) that affects monkeys in order to further explain the origins of HIV

5.0 SUMMARY

We hope you enjoyed your studies. In this unit, we described what type of virus HIV is, we tried to ascertain if HIV actually came from an SIV, we further explained what happened in 1999 as regards to HIV and finally, we attempted to identify how HIV could have crossed species.

6.0 TUTOR MARKED ASSIGNMENT

- HIV research: What happened in 1999?

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UNIT 2 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

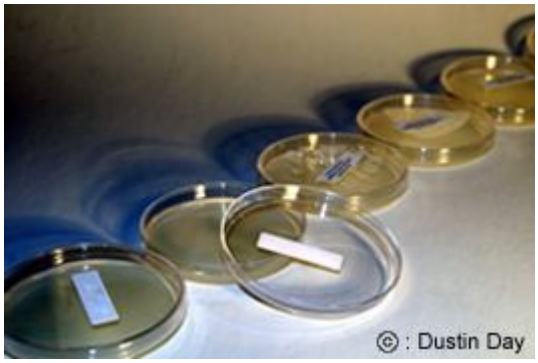
Below are some of the most common theories about how this 'zoonosis' took place, i.e., how Simian Immunodeficiency Virus SIV became HIV in humans:

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 analysed 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 labourers 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 labourers 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 were 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 different theoretical views on the origin of HIV. Specifically, we looked at the Hunters Theory, Oral Polio Vaccine Theory of HIV, the contaminated Needle Theory, the Colonialism Theory and the Conspiracy Theory.

6.0 TUTOR MARKED ASSIGNMENT

- How could HIV have crossed species? Discus using the Hunter Theory perspective

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UNIT 3 HIV/AIDS: EARLIEST KNOWN INSTANCES OF INFECTION/SPREAD OF HIV

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 AIDS 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:

- Identify the earliest known instances of HIV infection
- Ascertain if HIV actually came from Africa
- Identify why Haiti is Significant in the spread of HIV

- Explain and identify the factors that caused the HIV/AIDS epidemic to spread so suddenly

3.0 MAIN CONTENT

3.1 Earliest Known Instances of HIV Infection

Three of the earliest known instances of HIV infection are as follows:

1. A plasma sample taken in 1959 from an adult male living in what is now the Democratic Republic of Congo (Zhu, 1959).
2. HIV found in tissue samples from an American teenager who died in St. Louis in 1969 KOLATA, 1987).
3. HIV found in tissue samples from a Norwegian sailor who died around 1976 (Froland, 1988).

A 1998 analysis of the plasma sample from 1959 has suggested that HIV-1 was introduced into humans around the 1940s or the early 1950s; much earlier than previously thought. Other scientists have dated the sample to an even earlier period - perhaps as far back as the end of the 19th century.

In January 2000, the results of a new study presented at the 7th Conference on Retroviruses and Opportunistic Infections, suggested that the first case of HIV-1 infection occurred around 1930 in West Africa . The study was carried out by Dr Bette Korber of the Los Alamos National Laboratory. The estimate of 1930 (which has a 15 year margin of error) was based on a complex computer model of HIV's evolution. If accurate, it means that HIV was in existence before many scenarios (such as the OPV and conspiracy theories) suggest.

3.2 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 analysing 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.

SELF ASSESSMENT EXERCISE

- Identify three of the earliest known instances of HIV infection

3.3 Why is Haiti significant?

The AIDS 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 AIDS had come from Haiti, and that Haitians were responsible for the AIDS 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 stigmatisation 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, haemophiliacs and heroin users to make the 'Four-H Club' of groups at high risk of AIDS (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, in 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.

3.4 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.

3.4.1 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.

3.4.2 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.

3.4.3 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 have been able to identify the earliest known instances of HIV infection and stimulate argument on the genesis of HIV. We also tried to ascertain why Haiti is Significant in the spread of HIV and lastly we identified factors that triggered sudden spread of the epidemic.

6.0 TUTOR MARKED ASSIGNMENT

- Explain and identify the factors that triggered the spread of HIV.

ANSWER TO SELF ASSESSMENT EXERCISE

Three of the earliest known instances of HIV infection are as follows:

- A plasma sample taken in 1959 from an adult male living in what is now the Democratic Republic of Congo (Zhu, 1959).
- HIV found in tissue samples from an American teenager who died in St. Louis in 1969 KOLATA, 1987).
- HIV found in tissue samples from a Norwegian sailor who died around 1976 (Froland, 1988).

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MODULE 3 THE BIOLOGY OF HIV/AIDS

Unit 1 Understanding the Biology of HIV/AIDS
Unit 2 Koch Postulates - Evidence That HIV Causes AIDS
Unit 3 Koch Postulates Of HIV/AIDS (2, 3, and 4) Cont.

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 reader for a more detailed introduction to retroviruses, particularly the human immunodeficiency viruses.

2.0 OBJECTIVES

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

- Describe structure and genome of HIV/AIDS
- Explain viral tropism
- Describe entry into cell of HIV virus
- Explain replication and transcription of HIV virus
- Describe ‘assembly’ and ‘release’ of HIV virus

3.0 MAIN CONTENT

3.1 Viruses: HIV Structure and Genome – An Introduction

HIV primarily infects vital cells in the human immune system such as helper T cells (specifically $CD4^+$ T cells), macrophages and dendritic cells. HIV infection leads to low levels of $CD4^+$ T cells through three main mechanisms:

- Firstly, direct viral killing of infected cells
- Secondly, increased rates of apoptosis in infected cells
- Thirdly, killing of infected $CD4^+$ T cells by CD8 cytotoxic lymphocytes that recognize infected cells.

When $CD4^+$ T cell numbers decline below a critical level, cell-mediated immunity is lost, and the body becomes progressively more susceptible to opportunistic infections. If untreated, eventually most HIV-infected individuals develop AIDS (Acquired Immunodeficiency Syndrome) and die, however about one in ten remains healthy for many years, with no noticeable symptoms (Buchbinder, 1994). Treatment with anti-retrovirals, where available, increases the life expectancy of people infected with HIV. It is hoped that current and future treatments may allow HIV-infected individuals to achieve a life expectancy approaching that of the general public.

3.2 HIV: Structure and genome

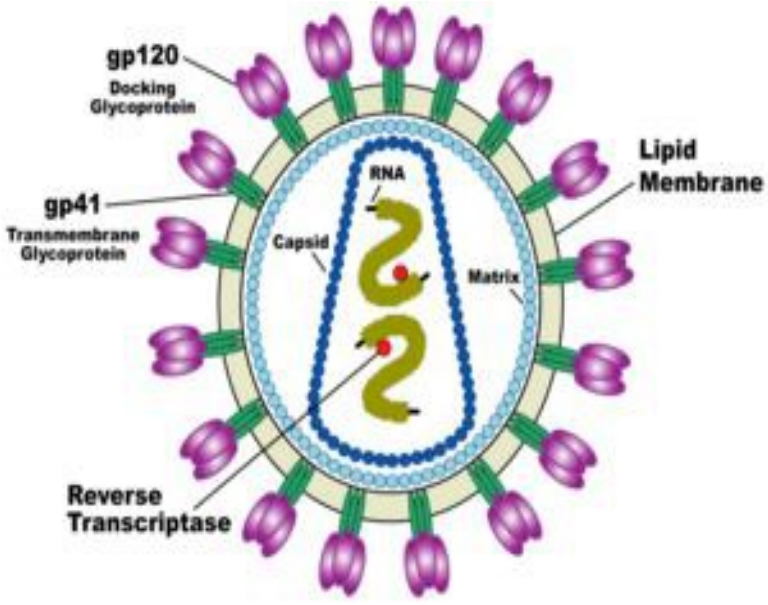


Diagram of HIV

HIV is different in structure from other retroviruses. It is about 120 nm in diameter (120 billionths of a meter; around 60 times smaller than a red blood cell) and roughly spherical (McGovern, 2002).

It is composed of two copies of positive single-stranded RNA that codes for the virus's nine genes enclosed by a conical capsid composed of 2,000 copies of the viral protein (HIV Sequence Compendium, 2005). The single-stranded RNA is tightly bound to nucleocapsid proteins, and enzymes needed for the development of the virion such as reverse transcriptase, proteases, ribonuclease and integrase. A matrix composed of the viral protein surrounds the capsid ensuring the integrity of the virion particle. This is, in turn, surrounded by the viral envelope which is composed of two layers of fatty molecules called phospholipids taken from the membrane of a human cell when a newly formed virus particle buds from the cell. Embedded in the viral envelope are proteins from the host cell and about 70 copies of a complex HIV protein that protrudes through the surface of the virus particle. This protein, known as Env, consists of a cap made of three molecules called glycoprotein (gp) 120, and a stem consisting of three gp41 molecules that anchor the structure into the viral envelope (Chan et al, 1997). This glycoprotein complex enables the virus to attach to and fuse with target cells to initiate the infectious cycle. Both these surface proteins, especially gp120, have been considered as targets of future treatments or vaccines against HIV (NIH, 1998).

Of the nine genes that are encoded within the RNA genome, three of these genes, *gag*, *pol*, and *env*, contain information needed to make the structural proteins for new virus particles. *env*, for example, codes for a protein called gp160 that is broken down by a viral enzyme to form gp120 and gp41. The six remaining genes, *tat*, *rev*, *nef*, *vif*, *vpr*, and *vpu* (or *vpx* in the case of HIV-2), are regulatory genes for proteins that control the ability of HIV to infect cells, produce new copies of virus (replicate), or cause disease. The protein encoded by *nef*, for instance, appears necessary for the virus to replicate efficiently, and the *vpu*-encoded protein influences the release of new virus particles from infected cells. 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 Sequence Compendium, 2005).

3.3. Viral Tropism

The term *viral tropism* refers to which cell types HIV infects. HIV can infect a variety of immune cells such as $CD4^+$ T cells, macrophages, and microglial cells. HIV-1 entry to macrophages and $CD4^+$ T cells is mediated through interaction of the virion envelope glycoproteins (gp120) with the CD4 molecule on the target cells and also with chemokine coreceptors (Chan et al, 1997).

Macrophage (M-tropic) strains of HIV-1, or non-syncitia-inducing strains (NSI) use the β-chemokine receptor CCR5 for entry and are thus able to replicate in macrophages and $CD4^+$ T cells (Cockley, et al, 2005). This CCR5 coreceptor is used by almost all primary HIV-1 isolates regardless of viral genetic subtype. Indeed, macrophages play a key role in several critical aspects of HIV infection. They appear to be the first cells infected by HIV and perhaps the source of HIV production when $CD4^+$ cells become depleted in the patient. Macrophages and microglial cells are the cells infected by HIV in the central nervous system. In tonsils and adenoids of HIV-infected patients, macrophages fuse into multinucleated giant cells that produce huge amounts of virus.

T-tropic isolates, or syncitia-inducing (SI) strains replicate in primary $CD4^+$ T cells as well as in macrophages and use the α-chemokine receptor, CXCR4, for entry (Cocklea et al, 2005; Deng et al, 1996; Feng et al, 1996). Dual-tropic HIV-1 strains are thought to be transitional strains of the HIV-1 virus and thus are able to use both CCR5 and LESTR as co-receptors for viral entry.

The α-chemokine, SDF-1, a ligand for CXCR4, suppresses replication of T-tropic HIV-1 isolates. It does this by down-regulating the expression of CXCR4 on the surface of these cells. HIV that use only the CCR5 receptor are termed R5, those that only use CXCR4 are termed X4, and those that use both, X4R5. However, the use of coreceptor alone does not explain viral tropism, as not all R5 viruses are able to use CCR5 on macrophages for a productive infection (Cocklea et al, 2005), and HIV can also infect a subtype of myeloid dendritic cells (Knight et al, 1990), which probably constitute a reservoir that maintains infection when $CD4^+$ T cell numbers have declined to extremely low levels.

Some people are resistant to certain strains of HIV (tang et al, 2003). One example of how this occurs is people with the CCR5-Δ32 mutation; these people are resistant to infection with R5 virus as the mutation stops HIV from binding to this coreceptor, reducing its ability to infect target cells.

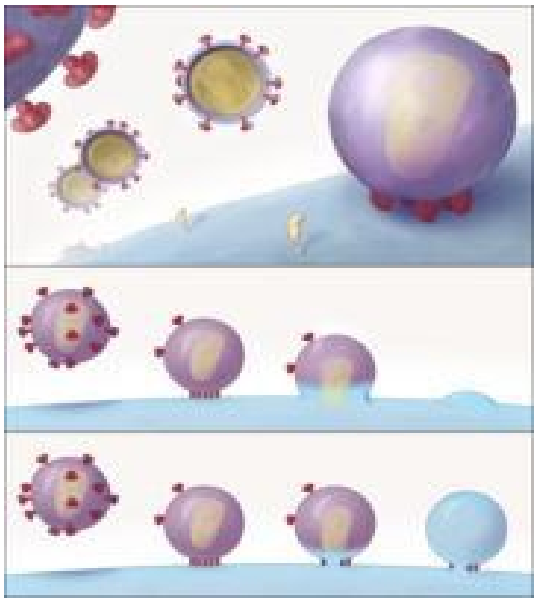
Sexual intercourse is the major mode of HIV transmission. Both X4 and R5 HIV are present in the seminal fluid which is passed from partner to partner. The virions can then infect numerous cellular targets and disseminate into the whole organism. However, a selection process leads to a predominant transmission of the R5 virus through this pathway (Zhu, et al, 1993; Van't Wont, et al, 1994; Zhu et al, 1996). How this selective process works is still under investigation, but one model is that spermatozoa may selectively carry R5 HIV as they possess both CCR3 and CCR5 but not CXCR4 on their surface (Muciaccia, et al, 2005), and that genital epithelial cells preferentially sequester X4 virus (Berlier, et al, 2005). In patients infected with subtype B HIV-1, there is often a co-receptor switch in late-stage disease and T-tropic variants appear that can infect a variety of T cells

through CXCR4 (Clevestig, et al, 2005). These variants then replicate more aggressively with heightened virulence that causes rapid T cell depletion, immune system collapse, and opportunistic infections that mark the advent of AIDS (Moore 1997). Thus, during the course of infection, viral adaptation to the use of CXCR4 instead of CCR5 may be a key step in the progression to AIDS. A number of studies with subtype B-infected individuals have determined that between 40 and 50% of AIDS patients can harbour viruses of the SI, and presumably the X4, phenotype (karlson, et al, 1994; Koot et al, 1996).

SELF ASSESSMENT EXERCISE

- Identify why sexual intercourse is regarded as a major mode of HIV transmission

3.4 HIV Entry to the Cell



HIV enters macrophages and $CD4^+$ T cells by the adsorption of glycoproteins on its surface to receptors on the target cell followed by fusion of the viral envelope with the cell membrane and the release of the HIV capsid into the cell (Chan et al, 1998).

The interactions of the trimeric envelope complex (gp160 spike, discussed above) and both CD4 and a chemokine receptor (generally either CCR5 or CXCR4 but others are known to interact) on the cell surface. The gp160 spike contains binding domains for both CD4 and chemokine receptors. The first step in fusion involves the high-affinity attachment of the CD4 binding domains of gp120 to CD4. Once gp120 is bound with the CD4 protein, the envelope complex undergoes a

structural change, exposing the chemokine binding domains of gp120 and allowing them to interact with the target chemokine receptor. This allows for a more stable two-pronged attachment, which allows the N-terminal fusion peptide gp41 to penetrate the cell membrane. Repeat sequences in gp41, HR1 and HR2 then interact, causing the collapse of the extracellular portion of gp41 into a hairpin. This loop structure brings the virus and cell membranes close together, allowing fusion of the membranes and subsequent entry of the viral capsid (Chan et al, 1998; Wyatt, et al, 1998).

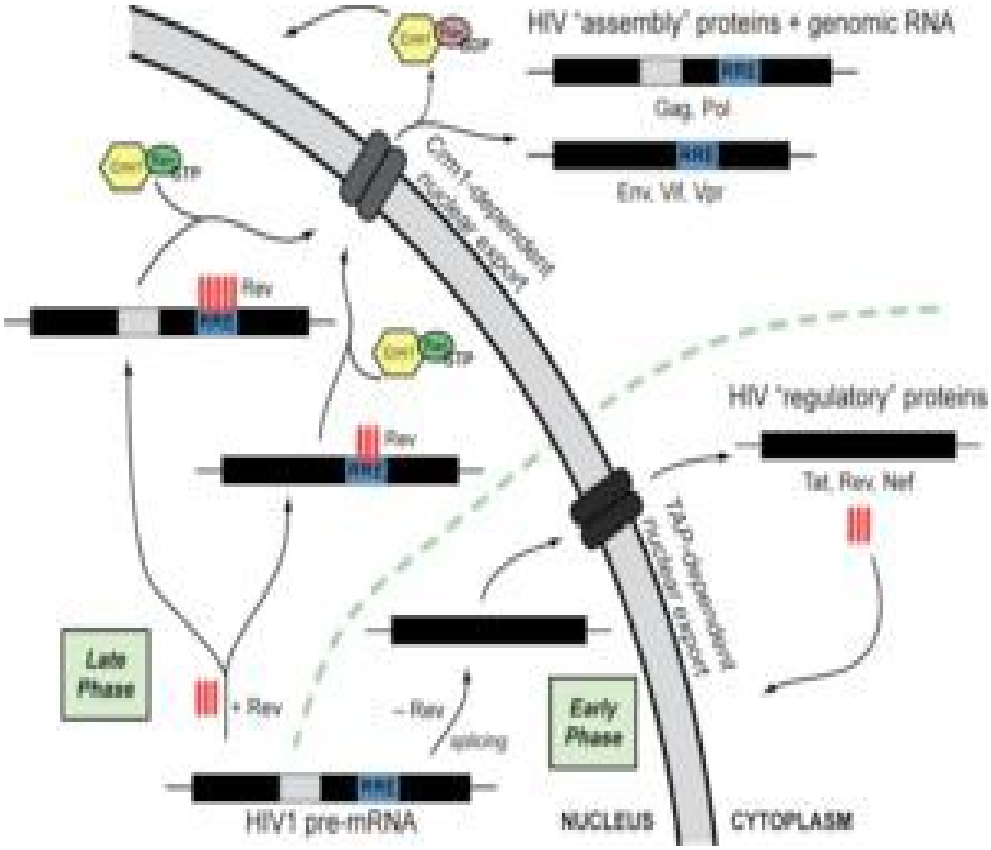
Once HIV has bound to the target cell, the HIV RNA and various enzymes, including reverse transcriptase, integrase, ribonuclease and protease, are injected into the cell (Chan, et al, 1998; Wyatt, et al, 1998).

HIV can infect dendritic cells (DCs) by this CD4-CCR5 route, but another route using mannose-specific C-type lectin receptors such as DC-SIGN can also be used. DCs are one of the first cells encountered by the virus during sexual transmission. They are currently thought to play an important role by transmitting HIV to T cells once the virus has been captured in the mucosa by DCs (Pope et al, 2003).

3.5 Replication and transcription

Once the viral capsid enters the cell, an enzyme called *reverse transcriptase* liberates the single-stranded (+)RNA from the attached viral proteins and copies it into a complementary DNA (Zheng, et al, 2005). This process of reverse transcription is extremely error-prone and it is during this step that mutations may occur. Such mutations may cause drug resistance. The reverse transcriptase then makes a complementary DNA strand to form a double-stranded viral DNA intermediate (vDNA). This vDNA is then transported into the cell nucleus. The integration of the viral DNA into the host cell's genome is carried out by another viral enzyme called *integrase* (Zheng et al, 2005).

This integrated viral DNA may then lie dormant, in the latent stage of HIV infection. To actively produce the virus, certain cellular transcription factors need to be present, the most important of which is NF-κB (NF kappa B), which is upregulated when T cells become activated (Hiscott, et al, 2001). This means that those cells most likely to be killed by HIV are in fact those currently fighting infection.



Rev-mediated HIV mRNA transport.

Rev (red) binds the Rev response element (RRE, blue) to mediate export of unspliced and singly spliced mRNA from the nucleus to the cytoplasm.

In this replication process, the integrated provirus is copied to mRNA which is then spliced into smaller pieces. These small pieces produce the regulatory proteins Tat (which encourages new virus production) and Rev. As Rev accumulates it gradually starts to inhibit mRNA splicing (Pollard, et al, 1998). At this stage, the structural proteins Gag and Env are produced from the full-length mRNA. The full-length RNA is actually the virus genome; it binds to the Gag protein and is packaged into new virus particles.

HIV-1 and HIV-2 appear to package their RNA differently; HIV-1 will bind to any appropriate RNA whereas HIV-2 will preferentially bind to the mRNA which was used to create the Gag protein itself. This may mean that HIV-1 is better able to mutate (HIV-1 infection progresses to AIDS faster than HIV-2 infection and is responsible for the majority of global infections).

3.6 Assembly and Release

The final step of the viral cycle, assembly of new HIV-1 virons, begins at the plasma membrane of the host cell. The Env polyprotein goes through the endoplasmic reticulum and is transported to the Golgi complex where it is cleaved by protease and processed into the two HIV envelope glycoproteins gp41 and gp120. These are transported to the plasma membrane of the host cell where gp41 anchors the gp120 to the membrane of the infected cell. The Gag and Gag-Pol polyproteins also associate with the inner surface of the plasma membrane along with the HIV genomic RNA as the forming virion begins to bud from the host cell. Maturation either occurs in the forming bud or in the immature virion after it buds from the host cell. During maturation, HIV proteases cleave the polyproteins into individual functional HIV proteins and enzymes. The various structural components then assemble to produce a mature HIV virion (Gelderbloon, 1997). This cleavage step can be inhibited by protease inhibitors. The mature virus is then able to infect another cell.

3.7 Classification of HIV

HIV was classified as a member of the genus Lentivirus (International Committee on Toxinomy of Virus, 2006), part of the family of Retroviridae. Lentiviruses have many common morphologies and biological properties. Many species are infected by lentiviruses, which are characteristically responsible for long-duration illnesses with a long incubation period (Levy, 2003). Lentiviruses are transmitted as single-stranded, positive-sense, enveloped RNA viruses. Upon entry of the target cell, the viral RNA genome is converted to double-stranded DNA by a virally encoded reverse transcriptase that is present in the virus particle. This viral DNA is then integrated into the cellular DNA by a virally encoded integrase so that the genome can be transcribed. Once the virus has infected the cell, two pathways are possible: either the virus becomes latent and the infected cell continues to function, or the virus becomes active and replicates, and a large number of virus particles are liberated that can then infect other cells.

Two species of HIV infect humans: HIV-1 and HIV-2. HIV-1 is thought to have originated in southern Cameroon after jumping from wild chimpanzees (*Pan troglodytes troglodytes*) to humans during the twentieth century (Gao et al, 1999; Keele et al, 2006). HIV-1 is the virus that was initially discovered and termed LAV.

HIV-2 may have originated from the Sooty Mangabey (*Cercocebus atys*), an Old World monkey of Guinea-Bissau, Gabon, and Cameroon (Reeves, et al, 2002).

HIV-1 is more virulent. It is easily transmitted and is the cause of the majority of HIV infections globally. HIV-2 is less transmittable and is largely confined to West Africa (Reeves, et al, 2002).

3.2 What about HIV-2? When did that get passed to humans?

Until recently, the origins of the HIV-2 virus had remained relatively unexplored. HIV-2 is thought to come from the SIV in Sooty Mangabeys rather than chimpanzees, but the crossover to humans is believed to have happened in a similar way (i.e. through the butchering and consumption of monkey meat). It is far rarer, significantly less infectious and progresses more slowly to AIDS than HIV-1. As a result, it infects far fewer people, and is mainly confined to a few countries in West Africa.

In May 2003, a group of Belgian researchers led by Dr. Anne-Mieke Vandamme, published a report (Keele et al, 2006), in Proceedings of the National Academy of Science. By analysing samples of the two different subtypes of HIV-2 (A and B) taken from infected individuals and SIV samples taken from sooty mangabeys, Dr Vannedamme concluded that subtype A had passed into humans around 1940 and subtype B in 1945 (plus or minus 16 years or so). Her team of researchers also discovered that the virus had originated in Guinea-Bissau and that its spread was most likely precipitated by the independence war that took place in the country between 1963 and 1974 (Guinea-Bissau is a former Portuguese colony). Her theory was backed up by the fact that the first European cases of HIV-2 were discovered among Portuguese veterans of the war, many of whom had received blood transfusions or unsterile injections following injury, or had possibly had relationships with local women.

4.0 CONCLUSION

The biology of HIV/AIDS gave us insight into the features and structure of the virus. It also equipped us with much needed and minute information on the physiological implications of HIV/AIDS virus. For example, we have seen that HIV reduces the CD4+ T cells and when $CD4^+$ T cell numbers decline below a critical level, cell-mediated immunity is lost, and the body becomes progressively more susceptible to opportunistic infections. If untreated, eventually most HIV-infected individuals develop AIDS (Acquired Immunodeficiency Syndrome).

5.0 SUMMARY

We hope you enjoyed your studies. This unit provided us with information on: the structure and genome of HIV/AIDS, viral tropism, entry into cell of HIV virus, replication and transcription of HIV and lastly, assembly and release of HIV. Ok

as usual, let us test our understanding of this unit by attempting the questions below.

6.0 TUTOR MARKED ASSIGNMENT

- HIV infection leads to low levels of $CD4^+$ T cells through three main mechanisms:----------and ----------
- Two species of HIV infect humans namely: ----------and ----------
- Draw and label the diagram of HIV. Explain briefly the structure and genome

ANSWER TO SELF ASSESSMENT EXERCISE

- Sexual intercourse is the major mode of HIV transmission. Both X4 and R5 HIV are present in the seminal fluid which is passed from partner to partner

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UNIT 2 KOCH POSTULATES - EVIDENCE THAT HIV CAUSES AIDS

CONTENT

1.0 INTRODUCTION

AIDS is caused by infection with a virus called human immunodeficiency virus (HIV). This virus is passed from one person to another through blood-to-blood and sexual contact (CDC, 2003). That is the standard explanation of what causes AIDS. But what evidence do scientists have to support this theory? This unit will shed more light on this.

2.0 OBJECTIVES

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

- Identify the proofs that HIV causes AIDS
- Identify the doubts that HIV causes AIDS
- Describe Koch 1 postulate of HIV/AIDS

3.0 MAIN CONTENT

3.1 Does HIV cause AIDS?

AVERT, an international HIV/AIDS based organization provided the following proofs:

- AIDS is a new epidemic disease

- AIDS does not occur without HIV
- HIV infection is the only factor that predicts who will develop AIDS
- Surveillance statistics support the HIV theory
- Modern antiretroviral treatment is highly beneficial.

3.2 Who doubts that HIV causes AIDS?

By far the most significant scientist to question the HIV/AIDS theory is Professor Peter Duesberg, a virologist at the University of California at Berkeley, who first wrote about this topic in 1987. Throughout the 1990s and into the new millennium, as HIV/AIDS researchers announced many new discoveries and amassed huge volumes of data, Dr Duesberg remained unconvinced. He admits that HIV exists, but he maintains that it is harmless, and that AIDS is caused by non-contagious factors including drug abuse, malnutrition, and even the very drugs used to combat HIV (Duesberg.com).

Other dissidents (often called "denialists" by their opponents) include the Perth Group of medical scientists and physicians from Australia. The Perth Group (led by Eleni Papadopulos) claims that nobody has conclusively proven the existence of HIV, so any proof that HIV causes AIDS has no foundation (PerthGroup.com).

Dissident arguments have received attention from the popular media, as well as from scientific journals. And with the rise of the Internet, alternative views have found a much wider audience, so that scarcely anyone interested in AIDS can have failed to hear of them.

Some of their followers are intrigued by conspiracy theories involving sinister drug companies or government persecution of minority groups. But alternative explanations can also appeal to those diagnosed with HIV or AIDS, who read that their condition might not be fatal, that they shouldn't take toxic drugs, and that unprotected sex poses no risks. Even a few AIDS service organisations have adopted non-HIV viewpoints. However, the proportion of scientists who doubt that HIV causes AIDS is tiny, and shows no sign of increasing.

3.3 How can we prove that HIV causes AIDS?

3.3.1 Koch's Postulates

In the nineteenth century, the German scientist Robert Koch developed a set of four "postulates" to guide people trying to prove that a germ causes a disease. Scientists agree that if HIV satisfies all of these conditions with regard to AIDS then it must be the cause of AIDS (Duesberg.com).

- Koch 1: The germ must be found in every person with the disease
- Koch 2: The germ must be isolated from someone who has the disease and grown in pure culture
- Koch 3: The germ must cause the disease when introduced into a healthy person
- Koch 4: The germ must be re-isolated from the infected person

Other evidence

Even Koch recognized that in some cases not all of his conditions could be met, so other evidence should also be considered. This is particularly true when the germ is a virus rather than a bacterium (Harden, 1992). Modern scientists are willing to consider a wide range of evidence. In particular, we can ask five key questions:

- Do surveillance statistics show a relationship between HIV and AIDS?
- How well does HIV infection predict illness and death?
- Do drugs designed to combat HIV benefit people with AIDS?
- Are there any credible causes besides HIV?
- What can we learn from Africa?

We will address these questions and more after looking at Koch's Postulates.

3.3.2 Koch 1: The germ must be found in every person with the disease

The US Centers for Disease Control and Prevention (CDC) defines a condition called idiopathic CD4+ T-lymphocytopenia, or ICL for short. Someone is diagnosed with ICL if they have a CD4+ cell count below 300 cells per cubic millimeter, or 20% of all T lymphocytes, on at least two occasions, but have no detectable HIV infection, nor any other known cause of immune deficiency (such as cancer therapy). As many dissidents have pointed out, this is essentially a definition of HIV-free AIDS. So just how common is this condition?

In 1993, a CDC task force published the results of an exhaustive survey of ICL in the USA. They had reviewed 230,179 AIDS-like cases reported since 1983 and identified 47 patients with ICL (plus 127 uncertain cases). All of the other people with AIDS who had received an HIV test produced a positive result. What's more, the team closely investigated the ICL cases and discovered that they didn't fit the usual AIDS profile. There were 29 male and 18 female patients, and 39 of them were white (4 others were of Asian descent). In 29 cases, the researchers could not fit the people into conventional risk groups for AIDS (homosexual men, haemophiliacs, injecting drug users, and the sexual partners of such groups). Whatever these 47 cases represent, they do not seem to be typical of the massive epidemic that we are interested in (Smith et al, 1993)

The findings of the ICL survey are backed up by large-scale monitoring studies, including the Multicenter AIDS Cohort Study (MACS). During the MACS, scientists monitored the health of 2,713 gay and bisexual men who tested negative for HIV antibodies. Over several years, only one of these men had persistently low CD4+ cell counts, and he was undergoing cancer therapy designed to weaken his immune system. Similar results have been found among blood donors, recipients of blood and blood products, injecting drug users and other groups: severe immune deficiency is virtually non-existent among those who test HIV-negative (NIH, 1995).

As Dr Duesberg has pointed out, quite a lot of people (mostly in the early 1980s) have been diagnosed with AIDS in the USA despite never taking an HIV test, and nobody knows whether these people were HIV-positive or not. However, based on the much larger sample of people who *have* been tested, Koch's first postulate has certainly been satisfied. The only way by which dissidents have been able to come up with significant numbers of HIV-free "AIDS" cases is by using much looser definitions of AIDS. Such definitions include many people with milder immune deficiency, which is generally not fatal (Cohen Science, 1994, UNAID/WHO, 2004).

SELF ASSESSMENT EXERCISE

AVERT, an international HIV/AIDS based organization identified some proofs that HIV causes AIDS. What are the proofs?

3.4 What about false positive test results?

Diagnosis of infection using antibody testing is one of the best-established concepts in medicine. The World Health Organisation and the US National Institutes of Health agree that modern HIV tests are extremely reliable, and are even more accurate than most other infectious disease tests (NIH Factsheet, 2004, UNAID/WHO, 2004).

Nevertheless, some dissidents have tried to dismiss the association between AIDS and HIV by claiming that many of those who test positive are not really infected with HIV. In particular, Christine Johnson has listed dozens of conditions reported to have produced false positive reactions on at least one occasion (under particular circumstances, using particular test kits) (Johnson, 1994).

It is true that no test is perfect. However, what the dissidents usually do not mention is how rare the reports of false positive results have been, especially in recent years. Nor do they mention that every person who uses a test kit is trained to spot the telltale signs of a suspicious result, and to keep testing by various

methods until no doubt remains. The conditions that cause false positive results are not only very uncommon, but are also typically short-lived, whereas HIV infection does not go away (CDC, 2001, Markin, 2001).

The dissident theory cannot satisfactorily explain why scientists have been able to use various techniques to detect the virus itself in virtually everyone with AIDS, as well as in most people with positive antibody test results, as explained in the next section. These methods (including DNA PCR, RNA PCR and viral culture) are not affected by any of the factors said to produce false positive results in antibody testing. Nor can the alternative theory fully explain why the association between AIDS and antibody test results is so exceptionally strong: virtually everyone with AIDS tests positive. And it cannot explain why the proportion of people testing HIV positive should have increased so dramatically over time. For example, the proportion of South African women testing HIV positive in annual antenatal surveys rose from 0.8% in 1990 to 10.4% in 1995, 24.5% in 2000 and 29.5% in 2004. The age distribution of these data is similar to that of other sexually transmitted infections.

4.0 CONCLUSION

To support the theory that AIDS is caused by HIV, this unit illustrated part 1 Koch postulates of HIV/AIDS. It also provided an insight on the false positive test results.

5.0 SUMMARY

In a bid to prove that HIV causes AIDS, this unit first highlighted existing proofs suggested by AVERT in this area. It further identified several contradictory views by renowned scientists. Unit also presented Koch 1 postulate on HIV/AIDS. Please note that the next unit acts as a continuation of the present unit, as it will at Koch 2,3 and 4 postulates. Hope you enjoyed your studies.

6.0 TUTOR MARKED ASSIGNMENT

- Explain Koch 1 Postulate of HIV/AIDS

ANSWER TO SELF ASSESSMENT EXERCISE

- AIDS is a new epidemic disease
- AIDS does not occur without HIV
- HIV infection is the only factor that predicts who will develop AIDS

- Surveillance statistics support the HIV theory
- Modern antiretroviral treatment is highly beneficial.

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UNIT 3 KOCH POSTULATES OF HIV/AIDS, CONT.

CONTENT

1.0 INTRUDUCTION

This unit is a follow up on the previous one. We discussed Koch 1 Postulates of HIV/AIDS in unit 2 and here we will discuss Koch Postulates 2, 3 and 4. This is a bid to clearly appreciate the assumption that HIV causes AIDS.

2.0 OBJECTIVES

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

- Explain Koch postulates of HIV/AIDS
- Identify the views of the 'Perth Group' on HIV/AIDS
- Illustrate how well HIV infection predict illness and death

3.0 MAIN CONTENT

3.1 Koch 2: The germ must be isolated from someone who has the disease and grown in pure culture

Koch required that the germ be isolated from all other material that could possibly cause disease, so that his third and fourth postulates could be properly tested.

In May 1983, Luc Montagnier and his colleagues in France reported the isolation of a virus they named LAV, which infected and killed CD4+ cells. A year later,

the American Robert Gallo announced he had isolated a virus called HTLV-III and found a way to grow it in culture. It was later discovered that the two viruses were genetically indistinguishable, and they were renamed HIV.

Researchers have been able to isolate and culture HIV from most AIDS patients whom they have examined (as well as from many other people with HIV antibodies) (O'brien and Goedert, 1996). They have isolated the virus from blood cells, blood plasma, lymph nodes, semen, vaginal fluids, amniotic fluids, bone marrow, brain, cerebrospinal fluid, intestines, breast milk, saliva, urine and tears, and cultured it in various cell types. Images taken using electron microscopy and other techniques have shown virus-like particles that have the size, shape, structure, density, proteins and behaviour expected of retroviruses (Salmine, et al, 1995).

Techniques developed in the mid-1990s have made it much easier to extract and sequence the complete genetic material (genome) of an isolated virus (Salmin, et al, 1995). The Los Alamos database now contains hundreds of full-length HIV genomes from around the world, each containing the same nine genes. Based on genetic similarities and differences, these sequences have been used to define family trees of HIV types, groups and subtypes as well as hybrids called recombinant forms.

Whole or partial HIV genomes have been detected in numerous AIDS patients, using a technique called PCR (the same technology is used to find DNA evidence with which to convict murderers or to settle paternity suits, as well as to detect the germs that cause hepatitis, tuberculosis and other diseases). Almost everyone who tests positive for HIV genetic material also tests positive for HIV antibodies, and vice versa, while those who test negative for one thing also lack the other. People who have been exposed to the same source of infection contain genetically very similar HIV strains – similar enough for court convictions.

Scientists have used a standard technique of genetic science called molecular cloning to obtain highly purified HIV. Genetic material extracted using PCR or other techniques has been introduced into bacteria or other cells (usually using phages or plasmids), which then produce many exact copies (clones) of the viral genes. If cloned viral genomes are inserted (transfected) into human cells then they produce a new generation of infectious HIV particles, which are free from contamination.

Virtually all experts agree that HIV has been isolated according to the most rigorous standards of modern virology, meaning that Koch's second postulate has without doubt been satisfied.

3.2 What about the Perth Group?

A small band of Australian scientists and physicians claims that HIV has never been properly isolated. The Perth Group has never said that HIV doesn't exist; rather they say that HIV has never been conclusively proven to exist. They don't trust any HIV tests, because they have not been verified using their "gold standard" of isolated virus. The Group uses the isolation argument to dismiss just about every type of evidence that HIV causes AIDS.

Virtually all virologists believe that the Perth Group's conditions are unnecessary. They say nobody has ever used such rules to isolate any type of virus, and that other techniques are much more effective. According to the Perth Group's rules, nobody has isolated or proven the existence of the viruses said to cause small pox, influenza, measles, mumps and yellow fever.

Experts argue that the Group's rules are unreasonably demanding and impossible to satisfy fully, even though their main requirements have already been met (Coon, 2000). Dr Duesberg is among those who have tried in vain to persuade the Perth Group that HIV definitely exists and has been isolated using the most rigorous methods available (Duesberg, 1996).

The Perth Group appears to have only two active members: a medical physicist called Eleni Papadopulos-Eleopulos and an emergency physician called Valendar Turner. In late 2006, Papadopulos-Eleopulos and Turner testified in the appeal trial of Andre Chad Parenzee, an HIV-positive man convicted of endangering life by having sex with three women without informing them of his infection. The two witnesses intended to demonstrate that HIV had not been proven to exist; that HIV tests were unreliable; and that there was no evidence of HIV transmission through sex.

The presiding judge concluded that the Perth Group members had no qualifications or practical experience in virology, immunology or epidemiology, and were not qualified to express opinions about the existence of HIV, or whether it had been shown to cause AIDS. The judge found that the pair relied entirely on the work of others, which they often took out of context and misrepresented. Their arguments were found to lack plausibility and cogency, and to have "minimal" probative value. "I am satisfied that no jury would conclude that there is any doubt that the virus HIV exists," said Justice Sulan. "I consider no jury would be left in any doubt that HIV is the cause of AIDS or that it is sexually transmissible (PARENZEE, 2007).

3.3 Koch 3 and 4: The germ must cause the disease when introduced into a healthy person, and the germ must be re-isolated from the infected person

The third and fourth postulates are much harder to prove. It's considered unethical to deliberately infect someone with pure HIV, so such an experiment has never taken place. However, there is no reason why the transmission has to be deliberate.

There have been three reports of lab workers developing immune deficiency after accidentally exposing themselves to purified, cloned HIV. As mentioned above, such cloned virus is free of all contamination from the original source. None of these people fitted conventional risk groups for the disease. In each case, HIV was isolated from the individual and, by genetic sequencing, was found to be the strain to which they'd been exposed. One of these workers developed PCP and had a CD4+ cell count below 50 cells before starting antiretroviral treatment.

Still, three examples don't make a totally conclusive proof, so it's worth looking for more evidence.

One line of argument can be based on animal experiments (NIH, 1995). In some studies, chimpanzees deliberately infected with HIV-1 have gone on to develop AIDS-like conditions (though this appears to be rare) (O'Niel et al, 2000), while HIV-2 has had the same effect on baboons (Locher et al, 2000). Macaque monkeys have developed AIDS after being infected with a hybrid virus called SHIV, which contains genes taken from HIV (Joag, et al, 1996). And in mice engineered to have a human immune system, HIV produces the same patterns of disease as in humans.

If we're prepared to bend the rules a bit further, we can look at people who've been infected with non-purified HIV. Such cases at least suggest that AIDS is infectious, though they don't rule out the possibility that more than one germ is involved.

Scientists have documented numerous cases of people developing AIDS after becoming infected with HIV as a result of blood transfusions, drug use, mother-to-child transmission, occupational exposure and sexual transmission. In such cases, they have recorded the development of HIV antibodies (seroconversion) using a series of blood tests, before progression to AIDS. Seroconversion is often accompanied by a mild flu-like illness or swollen glands (O'Brien et al, 1996).

Until the mid-1990s, nobody claimed that HIV had fulfilled Koch's last two postulates. Even today, the proof is not quite perfect. But most scientists believe the evidence is now strong enough to put the case beyond all reasonable doubt.

3.4 How well does HIV infection predict illness and death?

A mountain of evidence shows that much can be predicted from a positive test result. For example:

- At least half of people develop AIDS-defining conditions within 10 years of HIV infection, if they don't take antiretroviral drugs. Only a few do not develop AIDS within 20 years (Morgan et al, 2002).

- HIV-positive Americans and Canadians are over 1,000 times more likely to develop AIDS-defining diseases (such as PCP and Kaposi's sarcoma) than those who test negative (NIH, 2000; Schechter et al, 1993).

- A study in Uganda found that HIV-positive people were 16 times more likely to die over five years than those who tested negative. For those aged 25-34 years old, HIV infection raised the death rate by a factor of 27 (Nunn et al, 1997). Numerous other studies have found similar results in Tanzania, Malawi, Rwanda and other parts of Africa (Porter et al, 2004; Newell et al, 2004.

- A study of female sex workers in Thailand found the death rate to be over 50 times greater among those who tested positive. All of the positive women died of conditions associated with immune deficiency, compared with none of the negative women (The Lancet, 2000).

- During a 16-year, large-scale monitoring study of homosexual and bisexual men in the US, 60% of HIV-positives died compared with 2.3% of HIV-negatives (NIH factsheet, 2003).

- In the UK between 1979 and 1992, death rates increased massively among HIV-positive haemophiliacs, but remained unchanged among the rest. Similar research in the USA found that HIV-positive haemophiliacs were 11 times more likely to die over a ten-year period, compared with those who tested negative (The lancet, 1995).

- In a European study of babies born to HIV-positive women, none of those who tested negative developed AIDS, compared to 30% of those who tested positive. By their first birthday, 17% of the HIV-positive babies had died. A similar study in Uganda found that more than half of HIV-positive babies died before their second birthdays, compared to one sixth of those who were HIV-negative (Brahmbhatt, et al, 2006).

Alternative theories cannot explain why HIV tests should be so effective at predicting illness and death in so many diverse groups of people from all parts of the world.

It is even possible to predict the likelihood that someone will soon develop AIDS by measuring the amount of HIV in their blood, which is known as "viral load". Such measurements can be made using PCR, branched-DNA signal-amplification (bDNA) or quantitative microculture techniques. For example, the table below - based on a long term study of 1,604 patients - illustrates just how useful bDNA forecasts can be (Mellors et al, 1997).

Viral load (RNA copies per millilitre of blood plasma)	**Proportion of patients developing AIDS within six years**
less than 500	5.4%
501-3,000	16.6%
3,001-10,000	31.7%
10,001-30,000	55.2%
more than 30,000	80.0%

Dr Kary Mullis, who invented the PCR process, has questioned its ability to measure viral load. However, his arguments have been theoretical, and are not backed up by large-scale surveys, which have repeatedly shown a clear association between viral load and progression to AIDS (in all parts of the world). Dr Mullis' objections do not apply to the unrelated bDNA and quantitative microculture techniques. Modern bDNA tests produce very similar viral load counts to modern PCR tests (though this was less true of some earlier models). As with antibody tests, there is no convincing alternative explanation for why viral load counts should be such useful indicators.

4.0 CONCLUSION

As a follow up on the previous unit, we discussed Koch Postulates 2, 3 and 4. Koch required that the germ be isolated from all other material that could possibly cause disease, so that his third and fourth postulates could be properly tested. However the 3rd and 4th postulates state that the germ must cause the disease when introduced into a healthy person and the germ must be re-isolated from the infected person. The third and fourth postulates are much harder to prove. It is considered unethical to deliberately infect someone with pure HIV, so such an experiment has never taken place.

5.0 SUMMARY

We hope you enjoyed your studies. This unit further explained Koch postulates of HIV/AIDS, specifically, 2, 3 and 4. It also, identified the views of the 'Perth Group' on HIV/AIDS and illustrate, how well HIV infection predict illness and death.

6.0 TUTOR MARKED ASSIGNMENT

- How well does HIV infection predict illness and death?

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MODULE 4 HIV/AIDS – TRANSMISSION SYMPTOMS AND DIAGNOSIS

Unit 1 HIV Transmission
Unit 2 Symptoms and Diagnosis of HIV/AIDS
Unit 3 HIV Disease Progression Rates

UNIT 1 HIV/AIDS TRANSMISSION

CONTENT

1.0 INTRODUCTION

There is still misunderstanding about how HIV is transmitted from one person to another. Theories reviewed earlier were controversial and inconclusive. Knowing the basics helps you avoid getting the virus if you are HIV-, and avoid passing it on if you are HIV+. This unit provides HIV transmission facts.

2.0 OBJECTIVES

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

- Describe HIV transmission fact
- Identify body fluids that stimulates HIV/AIDS
- Explain how HIV is passed on
- Identify ways in which you can be infected with HIV
- Identify ways HIV is not transmitted

3.0 MAIN CONTENT

3.1 HIV Transmission Fact

There are various modes of transmission of HIV but first for infection to occur there are two important factors that facilitate HIV transmission, they are:

- Entry point: The virus must have a point of entry into the blood stream for it to cause infection
- Quantity: The virus must be present in large enough quantities to cause infection

3.2 HIV Transmission: Body Fluids

There are only three body fluids that have a large enough quantity of HIV to be infectious:

a. Blood: The blood of a person who is HIV infected has a very high level of HIV. This includes the monthly menstrual blood of women when having periods.
b. Breast milk: The breast milk of a woman who has HIV contains enough HIV to infect the child who is drinking that milk.
c. Sexual fluids (cum): During sex, men secrete two types of fluids from their penis, the first is the pre-cum which is a clear liquid that appears during initial sexual arousal and the second is the cum - a milky fluid that a man releases during ejaculation and also known as semen. In a man infected with HIV, both of these fluids contain enough HIV to infect another person. During sex a woman secretes vaginal fluid (cum) from her vagina. In a woman with HIV, this fluid contains enough of the virus to infect another person.

SELF ASSESSMENT EXERCISE

- Identify the transmission fact of HIV

3.3 How is HIV passed on?

HIV is found in the blood and the sexual fluids of an infected person, and in the breast milk of an infected woman. HIV transmission occurs when a sufficient quantity of these fluids get into someone else's bloodstream. There are various ways a person can become infected with HIV.

3.3.1 Ways in which you can be infected with HIV:

- *Unprotected sexual intercourse with an infected person* Sexual intercourse without a condom is risky, because the virus, which is present in an infected person's sexual fluids, can pass directly into the body of their partner. This is true for unprotected vaginal and anal sex. Oral sex carries a lower risk, but again HIV transmission can occur here if a condom is not used - for example, if one partner has bleeding gums or an open cut, however small, in their mouth.
- *Contact with an infected person's blood* If sufficient blood from an infected person enters someone else's body then it can pass on the virus.
- *From mother to child* HIV can be transmitted from an infected woman to her baby during pregnancy, delivery and breastfeeding. There are special drugs that can greatly reduce the chances of this happening, but they are unavailable in much of the developing world.
- *Use of infected blood products* Many people in the past have been infected with HIV by the use of blood transfusions and blood products which were contaminated with the virus - in hospitals, for example. In much of the world this is no longer a significant risk, as blood donations are routinely tested.
- *Injecting drugs* People who use injected drugs are also vulnerable to HIV infection. In many parts of the world, often because it is illegal to possess them, injecting equipment or works are shared. A tiny amount of blood can transmit HIV, and can be injected directly into the bloodstream with the drugs.

3.5 It is not possible to become infected with HIV through:

- sharing crockery and cutlery
- insect / animal bites
- touching, hugging or shaking hands
- eating food prepared by someone with HIV
- toilet seats

4.0 CONCLUSION

This unit identified HIV transmission facts and they are: entry point and quantity of the virus. We also recognized three body fluids that have a large enough quantity of HIV to be infectious as blood, breast milk and sexual fluid. Ways in which you can be infected with HIV were also highlighted.

5.0 SUMMARY

Hope you enjoyed your studies. In this unit, we provided information on HIV transmission fact, identified body fluids that stimulates HIV/AIDS, explained how

HIV is passed on, identified ways in which you can be infected with HIV and finally, identified ways HIV is not transmitted. Ok, let us attempt the questions below.

6.0 TUTOR MARKED ASSIGNMENT

- Identify and explain ways in which you can be infected with HIV/AIDS

ANSWER TO SELF ASSESSMENT EXERCISE

HIV transmission facts are:

- Entry point: The virus must have a point of entry into the blood stream for it to cause infection
- Quantity: The virus must be present in large enough quantities to cause infection

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UNIT 2 SYMPTOMS AND DIAGNOSIS OF HIV/AIDS

CONTENT

1.0 INTRODUCTION

People develop symptoms when they get infected with HIV and these are first signs that something is wrong with the body. Unfortunately most do not take it serious and are also unaware of the existence of such virus in their system until it is too late. In this unit, we will tackle this by identifying the early and later symptoms of HIV, as well as identify what happens in the body when infected. This unit will also explain how HIV infection is diagnosed; the type of blood test used and finally, identify the need for a follow-up test.

2.0 OBJECTIVES

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

- Identify the early symptoms of HIV infection
- Describe what happens inside the body when infected with HIV virus
- Identify the later symptoms of HIV/AIDS
- Explain how HIV infection is diagnosed
- Describe the kinds of blood tests used
- Explainwhy it is often necessary to repeat an HIV Test

3.0 MAIN CONTENT

3.1 What are the early symptoms of HIV infection?

Many people do not develop any symptoms when they first become infected with HIV. Some people, however, get a flu-like illness within three to six weeks after exposure to the virus. This illness, called Acute HIV Syndrome, may include fever, headache, tiredness, nausea, diarrhoea and enlarged lymph nodes (organs of the immune system that can be felt in the neck, armpits and groin). These symptoms usually disappear within a week to a month and are often mistaken for another viral infection.

During this period, the quantity of the virus in the body will be high and it spreads to different parts, particularly the lymphoid tissue. At this stage, the infected person is more likely to pass on the infection to others. The viral quantity then drops as the body's immune system launches an orchestrated fight.

More persistent or severe symptoms may not surface for several years, even a decade or more, after HIV first enters the body in adults, or within two years in children born with the virus. This period of "asymptomatic" infection varies from individual to individual. Some people may begin to have symptoms as soon as a few months, while others may be symptom-free for more than 10 years. However, during the "asymptomatic" period, the virus will be actively multiplying, infecting, and killing cells of the immune system.

3.2 What Happens Inside the Body?

Once HIV enters the human body, it attaches itself to a White Blood Cell (WBC) called CD4. Also, called T4 cells, they are the main disease fighters of the body. Whenever there is an infection, CD4 cells lead the infection-fighting army of the body to protect it from falling sick. Damage of these cells, hence can affect a person's disease-fighting capability and general health.

After making a foothold on the CD4 cell, the virus injects its RNA into the cell. The RNA then gets attached to the DNA of the host cell and thus becomes part of the cell's genetic material. It is a virtual takeover of the cell. Using the cell's division mechanism, the virus now replicates and churns out hundreds of thousands of its own copies. These cells then enter the blood stream, get attached to other CD4 cells and continue replicating. As a result, the number of the virus in the blood rises and that of the CD4 cells declines.

Because of this process, immediately after infection, the viral load of an infected individual will be very high and the number of CD4, low. But, after a while, the body's immune system responds vigorously by producing more and more CD4 cells to fight the virus. Much of the virus gets removed from the blood. To fight the fast-replicating virus, as many as a billion CD4 cells are produced every day, but the

virus too increases on a similar scale. The battle between the virus and the CD4 cells continues even as the infected person remains symptom-free.

But after a few years, which can last up to a decade or even more, when the number of the virus in the body rises to very high levels, the body's immune mechanism finds it difficult to carry on with the battle. The balance shifts in favour of the virus and the person becomes more susceptible to various infections. These infections are called Opportunistic Infections because they swarm the body using the opportunity of its low immunity. At this stage, the number of CD4 cells per millilitre of blood (called CD4 Count), which ranges between 500 to 1,500 in a healthy individual, falls below 200. The Viral Load, the quantity of the virus in the blood, will be very high at this stage.

Opportunistic infections are caused by bacteria, virus, fungi and parasites. Some of the common opportunistic infections that affect HIV positive persons are: Mycobacterium avium complex (MAC), Tuberculosis (TB), Salmonellosis, Bacillary Angiomatosis (all caused by bacteria); Cytomegalovirus (CMV), Viral hepatitis, Herpes, Human papillomavirus (HPV), Progressive multifocal leukoencephalopathy (PML) (caused by virus); Candidiasis, Cryptococcal meningitis (caused by fungus) and Pneumocystis Carinii pneumonia (PCP). Toxoplasmosis. Cryptosporidiosis (caused by parasites). HIV positive persons are also prone to cancers like Kaposi's sarcoma and lymphoma.

The Center for Disease Control (CDC), Atlanta has listed a series of diseases as AIDS-defining. When these diseases appear, it is a sign that the infected individual has entered the later stage of HIV infection and has started developing AIDS. The progression of HIV positive persons into the AIDS stage is highly individual. Some people can reach the AIDS stage in about five years, while some remain disease free for more than a decade. Measurement of the viral load and the CD4 count helps a doctor in assessing an infected person's health condition.

3.3 What are the later symptoms of HIV/AIDS?

- Lack of energy
- Weight loss
- Frequent fevers and sweats
- A thick, whitish coating of the tongue or mouth (thrush) that is caused by a yeast infection and sometimes accompanied by a sore throat
- Severe or recurring vaginal yeast infections
- Chronic pelvic inflammatory disease or severe and frequent infections like herpes zoster
- Periods of extreme and unexplained fatigue that may be combined with headaches, lightheadedness, and/or dizziness

- Rapid loss of more than 10 pounds of weight that is not due to increased physical exercise or dieting
- Bruising more easily than normal
- Long-lasting bouts of diarrhoea
- Swelling or hardening of glands located in the throat, armpit, or groin
- Periods of continued, deep, dry coughing
- Increasing shortness of breath
- The appearance of discoloured or purplish growths on the skin or inside the mouth
- Unexplained bleeding from growths on the skin, from mucous membranes, or from any opening in the body
- Recurring or unusual skin rashes
- Severe numbness or pain in the hands or feet, the loss of muscle control and reflex, paralysis or loss of muscular strength
- An altered state of consciousness, personality change, or mental deterioration
- Children may grow slowly or fall sick frequently. HIV positive persons are also found to be more vulnerable to some cancers.

SELF ASSESSMENT EXERCISE

What happens once HIV virus enters inside the human body?

3.4 How Is HIV Infection Diagnosed?

A blood test is used to confirm whether a person has been infected with HIV. Anyone who has engaged in risky behavior - such as sharing drug - injecting equipment or having unprotected sexual contact with an infected person or with someone whose HIV status is unknown - should consider being tested.

A positive HIV test result does not mean that a person has AIDS. Not everyone who has HIV infection develops AIDS. Experts estimate that about half the people with HIV will develop AIDS within 10 years after becoming infected.

Early diagnosis of HIV infection is important because:

- It allows people to seek treatment that will help suppress HIV's attack on the immune system and prevent opportunistic infections.
- It helps women at risk for HIV infection who are planning a pregnancy or who are already pregnant take steps to reduce the risk of transmitting the infection to the baby.
- It alerts those who are infected that they could infect others

3.5 What Kinds Of Blood Tests Are Used?

The blood tests most commonly used to diagnose HIV infection work by measuring the levels of antibodies produced by the body against HIV. Antibody-detecting assays, or tests, include the:

- Enzyme immunoassay (EIA)
- Enzyme-linked immunosorbent assay (ELISA)
- Western blot test

Usually, the first test that laboratories use to detect the presence of HIV antibodies is an EIA or the ELISA. If the first test produces a positive result (HIV antibodies appear to be present), then the more sensitive Western Blot test is used to confirm it.

EIA or ELISA tests take from one to two weeks to complete, depending on where the test is performed.

3.6 Why Is It Often Necessary To Repeat An HIV Test?

Although a negative result on an HIV blood test usually means that the person is not infected with the virus that is not always the case. The body may take three to six months after exposure to the virus to produce enough antibodies to be detectable in the bloodstream.

Because of this delay between infection and the appearance of HIV antibodies, a person should be retested six months after the last possible exposure to HIV. It is also important to remember that a person who has been exposed to HIV can pass the virus to others even before HIV antibodies appear in the bloodstream.

4.0 CONCLUSION

In conclusion, this unit recognized HIV early symptoms to include fever, headache, tiredness, nausea, diarrhoea and enlarged lymph nodes (organs of the immune system that can be felt in the neck, armpits and groin). These symptoms usually disappear within a week to a month and are often mistaken for another viral infection. Later symptoms include: lack of energy, frequent fever and sweats, a thick, whitish coating of the tongue or mouth, severe or recurring of vaginal infection, etc. This unit also stressed the importance of early HIV diagnosis and also the need for a repeat on HIV test.

5.0 SUMMARY

We hope you found this unit interesting. In this unit, we identified early as well as later symptoms of HIV infection. We also described what happens inside the body when infected with HIV virus. We further explained how HIV infection diagnosed, described what kinds of blood tests are used for HIV testing and finally, explained why it is often necessary to repeat of HIV Test

6.0 TUTOR MARKED ASSIGNMENT

- What are the early signs of HIV infection?
- What are the later signs of HIV infection?
- Why is early diagnosis of HIV very important?

ANSWER TO SELF ASSESSMENT EXERCISE

Once HIV enters the human body, it attaches itself to a White Blood Cell (WBC) called CD4. Also, called T4 cells, they are the main disease fighters of the body. Whenever there is an infection, CD4 cells lead the infection-fighting army of the body to protect it from falling sick. Damage of these cells, hence can affect a person's disease-fighting capability and general health.

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UNIT 3 HIV DISEASE PROGRESSION RATES

CONTENT

1.0 INTRODUCTION

Following infection with HIV-1, the rate of clinical disease progression varies between individuals. Factors such as host susceptibility, genetics and immune function (Morgan *et al.*, 2002b), health care and co-infections (Morgan *et al.*, 2002a) as well as viral genetic variability (Campbell *et al.*, 2004) may affect the rate of progression to AIDS.

2.0 OBJECTIVES

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

- Explain the term ‘rapid progressor
- Identify causes of long term non progressors
- Describe highly expose persistently seronegative
- Identify prediction of progressor rate
- Explain HIV sub-type variation and effect on progression rate
- Explain host genetic susceptibility
- Identify the effect of co-infection on progression rate

3.0 MAIN CONTENT

3.1 Rapid Progressors

A small percentage of HIV-infected individuals rapidly progress to AIDS within four years after primary HIV-infection and are termed Rapid Progressors (RP) (Anzala *et al.*, 1995). Indeed some individuals have been known to progress to AIDS and death within a year after primo-infection. Rapid progression was originally thought to be continent specific, as some studies reported that disease progression is more rapid in Africa (N'Galy *et al.*, 1988; Anzala *et al.*, 1995; Whittle *et al.*, 1992), but others have contested this view (Marlink et a., 1994; French *et al.*, 1999; Morgan *et al.*, 2002).

3.2 Long term non-progressors

Another subset of individuals who are persistently infected with HIV-1, but show no signs of disease progression for over 12 years and remain asymptomatic are classified as Long Term Non-Progressors (LTNP). In these individuals, it seems that HIV-infection has been halted with regard to disease progression over an extended period of time (Buchbinder *et al.*, 1994; Cao *et al.*, 1995; Easterbrook, 1994; Levy, 1993). However, the term LTNP is a misnomer, as it must be noted that progression towards AIDS can occur even after 15 years of stable infection (Harrer *et al.*, 1996). LTNP are not a homogeneous group regarding both viral load and specific immune responses against HIV-1. Some LTNPs are infected with HIV that inefficiently replicates (Deacon *et al.*, 1995; Kirchhoff *et al.*, 1995) whilst others are infected with HIV that is virally fit and replicates normally, but the infected individual has had a strong and broad set of HIV-specific humoral and cell-mediated responses that seems to delay the progression to AIDS. In some cohorts, individuals who experience signs of progression, but whose clinical and laboratory parameters remain stable over long periods of time, are classified as Long Term Survivors (LTS) (Schrager *et al.*, 1994; Campbell *et al.*, 2004).

3.3 Highly Exposed Persistently Seronegative

There is another, smaller percentage of individuals who have been recently identified. These are called Highly Exposed Persistently Seronegative (HEPS). This is a small group of individuals and has been observed only in a group of uninfected HIV-negative prostitutes in Kenya and in The Gambia. When these individuals' PBMCs are stimulated with HIV-1 peptides, they have lymphoproliferative activity and have HIV-1 specific CD8+ CTL activity suggesting that transient infection may have occurred (Clerici *et al.*, 1994; Pinto *et al.*, 1995; Rowland-Jones *et al.*, 1995; Fowke *et al.*, 1996). This does not occur in unexposed individuals. What is interesting, is that the CTL epitope specificity differs between HEPS and HIV positive individuals, and in HEPS, the maintenance of responses appears to be dependent upon persistent exposure to HIV (Kaul *et al.*, 2001).

SELF ASSESSMENT EXERCISE

- Individuals who experience signs of progression, but whose clinical and laboratory parameters remain stable over long periods of time, are classified as----------

3.4 Prediction of Progression Rates

During the initial weeks after HIV infection, qualitative differences in the cell-mediated immune response are observed that correlate with different disease progression rates (i.e., rapid progression to WHO stage 4 and the rapid loss of CD4+ T cell levels versus normal to slow progression to WHO stage 4 and the maintenance of CD4+ T cell counts above 500/µl). The appearance of HIV-1-specific CD8+ cytotoxic T cells (CTLs) early after primo-infection has been correlated with the control of HIV-1 viremia (Koup *et al.*, 1994; Borrow *et al.*, 1994). The virus which escapes this CTL response have been found to have mutations in specific CTL epitopes (Philips *et al.*, 1991; Borrow *et al.*, 1997; Price *et al.*, 1997; Rowland-Jones *et al.*, 1992). Individuals with a broad expansion of the V-beta chain of the T cell receptor of CD8+ T cells during primo-infection appear seem to have low levels of virus six to twelve months later, which is predictive of relatively slow disease progression. In contrast, individuals with an expansion of only a single subset of the V-beta chain of the CD8+ T cells are not able to control HIV levels over time, and thus have high levels of virus six to twelve months later (Pantaleo *et al.*, 1997). LTNP's have also been shown to have a vigorous proliferation of circulating activated HIV-1-specific CD4+ T cell (Rosenberg *et al.*, 1997) and CTL response (Rowland–Jones *et al.*, 1999; Dyer *et al.*, 1999) against multiple epitopes with no detectable broadly cross-reactive neutralizing antibodies in the setting of an extremely low viral load (Harrer *et al.*, 1996). However, a few reports have correlated the presence of antibodies against Tat in LTNP status.

3.5 HIV Subtype Variation and Effect on Progression Rates

The HIV-1 subtype that an individual becomes infected with can be a major factor in the rate of progression from sero-conversion to AIDS. Individuals infected with subtypes C, D and G are 8 times more likely to develop AIDS than individuals infected with subtype A (Kanki *et al.*, 1999). In Uganda, where subtypes A and D are most prevalent (Kaleebu *et al.*, 2000), subtype D is associated with faster disease progression compared with subtype A (Kaleebu *et al.*, 2002).

Age has also been shown to be a major factor in determining survival and the rate of disease progression, with individuals over 40 years of age at sero-conversion

being associated with rapid progression (Koblin *et al.*, 1999; Pezotti *et al.*, 1999; Collaborative Group, 2000; Morgan *et al.*, 2002b).

3.6 Host Genetic Susceptibility

The Centers for Disease Control and Prevention (CDC) has released findings that genes influence susceptibility to HIV infection and progression to AIDS. HIV enters cells through an interaction with both CD4 and a chemokine receptor of the 7 Tm family. They first reviewed the role of genes in encoding chemokine receptors (CCR5 and CCR2) and chemokines (SDF-1). While CCR5 has multiple variants in its coding region, the deletion of a 32-bp segment results in a nonfunctional receptor, thus preventing HIV entry; two copies of this gene provide strong protection against HIV infection, although the protection is not absolute. This gene is found in up to 20% of Europeans but is rare in Africans and Asians. Multiple studies of HIV-infected persons have shown that presence of one copy of this gene delays progression to the condition of AIDS by about 2 years. And it is possible that a person with the CCR5-Δ32 (CCR5 delta 32) receptor gene will not be infected with HIV.

The National Institute of Health (NIH) has funded research studies to learn more about this genetic mutation. In such research, NIH has found that there exist genetic tests that can determine if a person has this mutation. Implications of a genetic test may in the future allow clinicians to change treatment for the HIV infection according to the genetic makeup of an individual *(Gonzalez et al)*. Currently there exists several at-home tests for the CCR5 mutation in individuals; however, they are not diagnostic tests.

A relatively new class of drugs for HIV treatment relies on the genetic makeup of the individual. Entry inhibitors bind to the CCR5 protein to block HIV from binding to the CD4 cell.

3.7 The effect of co-infections on progression rates

Coinfections or immunizations may enhance viral replication by inducing a response and activation of the immune system. This activation facilitates the three key stages of the viral life cycle: entry to the cell; reverse transcription and proviral transcription (Lawn *et al.*, 2001). Chemokine receptors are vital for the entry of HIV into cells. The expression of these receptors is inducible by immune activation caused through infection or immunization, thus augmenting the number of cells that are able to be infected by HIV-1 (Wahl *et al.*, 1998; Juffermans *et al.*, 2001). Both reverse transcription of the HIV-1 genome and the rate of transcription of proviral DNA rely upon the activation state of the cell and are less likely to be successful in quiescent cells. In activated cells there is an increase in

the cytoplasmic concentrations of mediators required for reverse transcription of the HIV genome (Zack *et al.*, 1990; Kinoshita *et al.*, 1998). Activated cells also release IFN-alpha which acts on an autocrine and paracrine loop that up-regulates the levels of physiologically active NF-kappa B which activates host cell genes as well as the HIV-1 LTR (Gaynor, 1992; Baeuerle, 1991). The impact of co-infections by micro-organisms such as Mycobacterium tuberculosis can be important in disease progression, particularly for those who have a high prevalence of chronic and recurrent acute infections and poor access to medical care (Blanchard *et al.*, 1997). Often, survival depends upon the initial AIDS-defining illness (Morgan *et al.*, 1997). Co-infection with DNA viruses such as HTLV-1, herpes simplex virus-2, varicella zoster virus and cytomegalovirus may enhance proviral DNA transcription and thus viral load as they may encode proteins that are able to trans-activate the expression of the HIV-1 pro-viral DNA (Gendelman *et al.*, 1986). Frequent exposure to helminth infections, which are endemic in Africa, activates individual immune systems, thereby shifting the cytokine balance away from an initial Th1 cell response against viruses and bacteria which would occur in the uninfected person to a less protective T helper 0/2-type response (Bentwich *et al.*, 1995). HIV-1 also promotes a Th1 to Th0 shift and replicates preferentially in Th2 and Th0 cells (Maggi *et al.*, 1994). This makes the host more susceptible to and less able to cope with infection with HIV-1,viruses and some types of bacteria

4.0 CONCLUSION

We have seen that following infection with HIV-1, the rate of clinical disease progression varies between individuals. Contributory factors include: host susceptibility, genetics and immune function, health care and co-infections, as well as viral genetic variability.

5.0 SUMMARY

In summary, this unit explained the term ‘rapid progressosr, identified the causes of long term non pregressors, described highly expose persistently seronegative and prediction of progressor rate. It further explained HIV sub-type variation and effect on progression rate, host genetic susceptibility and finally identified the effect of co-infection on progression rate

6.0 TUTOR MARKED ASSIGNMENT

- Identify what happens in the case of highly exposed persistent seronegative individuals
- Describe HIV sub type variation and effect on progression rate.

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MODULE 5 COMPREHENSIVE HIV PREVENTION

UNIT 1 HOW CAN HIV TRANSMISSION BE PREVENTED?

CONTENT

1.0 INTRODUCTION

HIV can be transmitted in three main ways:

- Sexual transmission
- Transmission through blood
- Mother-to-child transmission

Wherever there is HIV, all three routes of transmission will take place. However the number of infections resulting from each route will vary greatly between countries and population groups.

For each route of transmission there are things that an individual can do to reduce or eliminate risk. There are also interventions that have been proven to work at the community, local and national level.

To be successful, an HIV prevention programme must make use of *all* approaches known to be effective, rather than just implementing one or a few select actions in isolation. The share of resources allocated to each area should reflect the nature of the local epidemic - for example, if most infections occur among men who have sex with men then this group should be a primary target for prevention efforts.

Although most preventive measures are described in this module/unit it should be noted that many people do not fit into only one "risk category". For example, injecting drug users need access to condoms and safer sex counselling, while teenagers and school children needs sex education.

2.0 OBJECTIVES

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

- Identify requirements for HIV prevention
- Identify and explain prevention of HIV via sexual transmission, blood transmission and mother-to-child transmission
- Discuss the obstacles to HIV prevention
- Identify policy measures of HIV/AIDS

3.0 MAIN CONTENT

3.1 Requirements for HIV Prevention

There are three key things that can be done to help prevent all forms of HIV transmission.

- First among these is promoting widespread awareness of HIV and how it can be spread. Media campaigns and education in schools are among the best ways to do this.
- Another essential part of a prevention programme is HIV counselling and testing. People living with HIV are less likely to transmit the virus to others if they know they are infected and if they have received counselling about safer behaviour. In particular, a pregnant woman who has HIV will not be able to benefit from interventions to protect her child unless her infection is diagnosed. Those who discover they are uninfected can also benefit, by receiving counselling on how to remain that way (The Lancet, 2000; UNAIDS, 2001).
- The third key factor is providing antiretroviral treatment. This treatment enables people living with HIV to enjoy longer, healthier lives, and as such it acts as an incentive for HIV testing. It also brings HIV-positive people into contact with health care workers who can deliver prevention messages and interventions. Studies suggest that HIV-positive people may be less

likely to engage in risky behaviour if they are enrolled in treatment programmes. Nevertheless, it is also possible that widespread availability of treatment may make some members of the wider population less fearful of HIV infection, and hence less willing to take precautions (Kennedy, et al., 2007; Crepaz, et al. 2004).

3.2 HIV Prevention: Sexual Transmission

Someone can eliminate or reduce their risk of becoming infected with HIV during sex by choosing to:

- Abstain from sex or delay first sex
- Be faithful to one partner or have fewer partners
- Condomise, which means using male or female condoms consistently and correctly

There are a number of effective ways to encourage people to adopt safer sexual behaviour, including media campaigns, social marketing, peer education and small group counselling. These activities should be carefully tailored to the needs and circumstances of the people they intend to help. Specific programmes should target key groups such as young people, women, men who have sex with men, injecting drug users and sex workers (Lamptey and Price, 1998; Pressman and Levy, 1998; UNAIDS, 1999).

Comprehensive sex education for young people is an essential part of HIV prevention. This should include training in life skills such as negotiating healthy sexual relationships, as well as accurate and explicit information about how to practise safer sex. Studies have shown that this kind of comprehensive sex education is more effective at preventing sexually transmitted infections than education that focuses solely on teaching abstinence until marriage (Santelli, et al., 2006).

Numerous studies have shown that condoms, if used consistently and correctly, are highly effective at preventing HIV infection. Also there is no evidence that promoting condoms leads to increased sexual activity among young people. Therefore condoms should be made readily and consistently available to all those who need them (UNAIDS, 2004).

There is now very strong evidence that male circumcision reduces the risk of HIV transmission from woman to man by around 50%, which is enough to justify its promotion as an HIV prevention measure in some high-prevalence areas. It is not known whether circumcision also affects the likelihood of male-to-female or male-

to-male sexual transmission; further research on this issue is ongoing (NIAID, 2006).

Another significant intervention is providing treatment for sexually transmitted infections, such as chlamydia and gonorrhoea. This is because such infections, if left untreated, have been found to facilitate HIV transmission during sex (WHO. 2006, Hitchcock and Francen, 1999).

One group that should not be overlooked by HIV prevention programmes is those who are already living with the virus. Regular counselling can help HIV positive people to sustain safer sexual behaviour, and so avoid onward transmission (Richardson, et al, 2004; International HIV/AIDS Alliance, 2003).

SELF ASSESSMENT EXECRICE

What are the requirements for HIV prevention?

3.2.1 Sexual Transmission and HIV Prevention: What are the obstacles?

It is usually not easy for people to sustain changes in sexual behaviour. In particular, young people often have difficulty remaining abstinent, and women in male-dominated societies are frequently unable to negotiate condom use, let alone abstinence. Many couples are compelled to have unprotected sex in order to have children. Others associate condoms with promiscuity or lack of trust (Marston and King, 2006).

Some societies find it difficult to discuss sex openly, and some authorities restrict what subjects can be discussed in the classroom, or in public information campaigns, for moral or religious reasons. Particularly contentious issues include premarital sex, condom use and homosexuality, the last of which is illegal or taboo in much of the world. Marginalisation of groups at high risk - such as sex workers and men who have sex with men - can be a major hindrance to HIV prevention efforts; authorities are often unwilling to allocate adequate resources to programmes targeting these groups.

Safe male circumcision demands considerable medical resources and some cultures are strongly opposed to the procedure.

3.3 HIV Prevention: Transmission through Blood

People who share equipment to inject recreational drugs risk becoming infected with HIV from other drug users. Methadone maintenance and other drug treatment programmes are effective ways to help people eliminate this risk by giving up

injected drugs altogether. However, there will always be some injecting drug users who are unwilling or unable to end their habit, and these people should be encouraged to minimise the risk of infection by not sharing equipment (WHO, 2005).

Needle exchange programmes have been shown to reduce the number of new HIV infections without encouraging drug use. These programmes distribute clean needles and safely dispose of used ones, and also offer related services such as referrals to drug treatment centres and HIV counselling and testing. Needle exchanges are a necessary part of HIV prevention in any community that contains injecting drug users (WHO, 2005).

Also important for injecting drug users are community outreach, small group counselling and other activities that encourage safer behaviour and access to available prevention options (WHO, 2004).

Transfusion of infected blood or blood products is the most efficient of all ways to transmit HIV. However, the chances of this happening can be greatly reduced by screening all blood supplies for the virus, and by heat-treating blood products where possible. In addition, because screening is not quite 100% accurate, it is sensible to place some restrictions on who is eligible to donate, provided that these are justified by epidemiological evidence, and don't unnecessarily limit supply or fuel prejudice. Reducing the number of unnecessary transfusions also helps to minimise risk (UNAIDS, 1997; WHO 2002).

The safety of medical procedures and other activities that involve contact with blood, such as tattooing and circumcision, can be improved by routinely sterilising equipment. An even better option is to dispose of equipment after each use, and this is highly recommended if at all possible.

Health care workers themselves run a risk of HIV infection through contact with infected blood. The most effective way for staff to limit this risk is to practise universal precautions, which means acting as though every patient is potentially infected. Universal precautions include washing hands and using protective barriers for direct contact with blood and other body fluids.

3.3.1 Blood Transmission and HIV Prevention: What are the obstacles?

Despite the evidence that they do not encourage drug use, some authorities still refuse to support needle exchanges and other programmes to help injecting drug users. Restrictions on pharmacies selling syringes without prescriptions, and on possession of drug paraphernalia, can also hamper HIV prevention programmes by making it harder for drug users to avoid sharing equipment.

Many resource-poor countries lack facilities for rigorously screening blood supplies. In addition a lot of countries have difficulty recruiting enough donors, and so have to resort to importing blood or paying their citizens to donate, which is not the best way to ensure safety.

In much of the world the safety of medical procedures in general is compromised by lack of resources, and this may put both patients and staff at greater risk of HIV infection.

3.4 HIV Prevention: Mother-to-child transmission

HIV can be transmitted from a mother to her baby during pregnancy, labour and delivery, and later through breastfeeding. The first step towards reducing the number of babies infected in this way is to prevent HIV infection in women, and to prevent unwanted pregnancies.

There are a number of things that can be done to help a pregnant woman with HIV to avoid passing her infection to her child. A course of antiretroviral drugs given to her during pregnancy and labour as well as to her newborn baby can greatly reduce the chances of the child becoming infected. Although the most effective treatment involves a combination of drugs taken over a long period, even a single dose of treatment can cut the transmission rate by half.

A caesarean section is an operation to deliver a baby through its mother's abdominal wall, which reduces the baby's exposure to its mother's body fluids. This procedure lowers the risk of HIV transmission, but is likely to be recommended only if the mother has a high level of HIV in her blood, and if the benefit to her baby outweighs the risk of the intervention.

Weighing risks against benefits is also critical when selecting the best feeding option. The World Health Organisation advises mothers with HIV not to breastfeed whenever the use of replacements is acceptable, feasible, affordable, sustainable and safe. However, if safe water is not available then the risk of life-threatening conditions from replacement feeding may be greater than the risk from breastfeeding. An HIV positive mother should be counselled on the risks and benefits of different infant feeding options and should be helped to select the most suitable option for her situation (WHO/UNICEF/UNAIDS/UNFPA, 2003).

3.4.1 Mother-to-Child Prevention: What are the obstacles?

In much of the world a lack of drugs and medical facilities limits what can be done to prevent mother-to-child transmission of HIV. Antiretroviral drugs are not widely available in many resource-poor countries, caesarean section is often

impractical, and many women lack the resources needed to avoid breastfeeding their babies.

HIV-related stigma is another obstacle to preventing mother-to-child transmission. Some women are afraid to attend clinics that distribute antiretroviral drugs, or to feed their babies formula, in case by doing so they reveal their HIV status.

3.5 Policy measures

To be successful, a comprehensive HIV prevention programme needs strong political leadership. This means politicians and leaders in all sectors must speak out openly about AIDS and not shy away from difficult issues like sex, sexuality and drug use.

An effective response requires strategic planning based on good quality science and surveillance, as well as consideration of local society and culture. All sectors of the population should be actively involved in the response, including employers, religious groups, non-governmental organisations and HIV-positive people. Many of the world's most successful HIV prevention efforts have been led by the affected communities themselves.

HIV epidemics thrive on stigma and discrimination related to people living with the virus and to marginalised groups such as sex workers. Their spread is also fuelled by gender inequality, which restricts what women can do to protect themselves from infection. Protecting and promoting human rights should be an essential part of any comprehensive HIV prevention strategy. This includes legislating against the many forms of stigma and discrimination that increase vulnerability.

4.0 CONCLUSION

This unit identified three main routes if HIV transmission namely, Sexual transmission, transmission through blood and mother-to-child transmission We also observed that for each route of transmission there are things that an individual can do to reduce or eliminate risk. There are also interventions that have been proven to work at the community, local and national level. These and much more were presented in this unit.

5.0 SUMMARY

We hope you enjoyed your studies. Specifically, this unit identified requirements for HIV prevention and discussed various obstacles to HIV prevention as well

relevant policy measures to curb the HIV scourge. Now let us attempt the questions below.

6.0 TUTOR MARKED ASSIGNMENT

HIV can be transmitted in three main ways namely----------

Identify obstacles to HIV prevention in the following areas: Sexual transmission, blood transfusion, mother-to-child transmission.

ANSWER TO SELF ASSESSMENT EXERCISE

- There are three key things that can be done to help prevent all forms of HIV transmission. First among these is promoting widespread awareness of HIV and how it can be spread. Media campaigns and education in schools are among the best ways to do this. Another essential part of a prevention programme is HIV counselling and testing. The third key factor is providing antiretroviral treatment.

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UNIT 2 THE ABC OF HIV PREVENTION

CONTENT

1.0 INTRODUCTION

HIV can be transmitted in the sexual fluids, blood or breast milk of an infected person. HIV prevention therefore involves a wide range of activities including prevention of mother-to-child transmission, harm reduction for injecting drug users, and precautions for health care workers.

This unit looks at strategies for preventing sexual transmission of HIV, and in particular the much-discussed "ABC" approach. So what exactly is the ABC approach, why does it cause such controversy, and does it work?

2.0 OBJECTIVES

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

- Explain ABC approach of HIV prevention?
- Identify PEPFAR and UNAIDS Definition of ABC Approach of HIV Prevention?
- Identify why Uganda is so often mentioned in relation to the ABC of HIV Prevention

3.0 MAIN CONTENT

3.1 What is ABC approach of HIV Prevention?

One of the difficulties with the ABC approach is the lack of a clear definition. The slogan seems to have first been adopted by the Botswana government in the late 1990s. Seen on billboards around the country it exalted the fact that:

Avoiding AIDS as easy as...

- A bstain
- B e faithful
- C ondomise

3.1.1 Is ABC Approach saying something new?

Since the late 1980s it had been known that individuals could take action to either reduce or avoid altogether the risk of becoming infected with HIV through sexual transmission.

The risk could be avoided altogether by avoiding any sexual activities that could cause transmission of HIV (i.e. Abstain).

The risk could also be reduced, through avoiding sexual intercourse other than with a mutually faithful uninfected partner (i.e. Be faithful) or through the correct and consistent use of condoms (i.e. Condomise).

So Botswana had not really developed a new approach to HIV prevention, but rather a new way of putting across known risk reduction and risk avoidance strategies.

3.1.2 Was ABC Approach Controversial?

The ABC definition adopted by Botswana was not particularly controversial. It was primarily a slogan used as part of a general public AIDS awareness campaign, and it did not attempt to define the circumstances under which the component parts of A, B and C would be promoted and whom they would be promoted to.

But since the use of this slogan in Botswana there have been other variations which have more specific definitions, most notably those adopted by the US-funded PEPFAR initiative, and others adopted by UNAIDS.

SELF ASSESSMENT EXERCISE

- What is ABC approach to HIV prevention?
- Is the ABC approach saying something new?

3.2 What is the President Emergency Plan for AIDS Relief (PEPFAR) Definition of ABC Approach of HIV Prevention?

PEPFAR follows an ABC strategy through "population-specific interventions" that emphasise:

- A bstinence for youth, including the delay of sexual debut and abstinence until marriage
- B eing tested for HIV and being faithful in marriage and monogamous relationships
- C orrect and consistent use of condoms for those who practice high-risk behaviours.

Those who practice high-risk behaviours include "prostitutes, sexually active discordant couples [in which one partner is known to have HIV], substance abusers, and others". The PEPFAR definition does not include the promotion of condoms to young people in general (PEPFAR, 2004). However, PEPFAR does say that its funds may be used to support programmes that deliver age-appropriate "ABC information" for young people, provided they are informed about failure rates of condoms, and provided the programmes do not appear to present abstinence and condom use as equally viable, alternative choices.

3.3 What is the UNAIDS Definition of ABC?

For UNAIDS, ABC means:

- A bstinence or delaying first sex
- B eing safer by being faithful to one partner or by reducing the number of sexual partners
- C orrect and consistent use of condoms for sexually active young people, couples in which one partner is HIV-positive, sex workers and their clients, and anyone engaging in sexual activity with partners who may have been at risk of HIV exposure (UNAIDS, 2004).

3.4 So what is the Controversy About?

The controversy arises from the differences between these two definitions of ABC, and in particular the fact that with the PEPFAR definition there is no promotion of

condoms for young people (or anyone else outside the "high risk groups"), and that with abstinence the emphasis is on abstaining until marriage.

If, in countries where there is a high prevalence of HIV infection resulting from sexual transmission, young people delay having sex for the first time, then this risk avoidance will indeed result in them avoiding infection whilst they are adopting this approach.

However, abstinence until marriage does not always ensure safety, because marriage in itself provides no protection from infection. Many people are unsure of the HIV status of their partners, and those who are faithful cannot be certain that their partner is maintaining the same commitment.

Abstinence is not a realistic option for the millions of women and girls who are in abusive relationships, or those who have been taught always to obey men. People who do not abstain should do everything possible to reduce risk, including using condoms.

“condoms, when distributed with educational materials as part of a comprehensive prevention package, have been shown to significantly lower sexual risk and activity, both among those already sexually active and those who are not." – (UNAIDS, October 2004).

A large number of AIDS organisations and experts have voiced concern that PEPFAR is putting too much emphasis on abstinence until marriage, and is not doing enough to make young people aware that condoms, if used correctly and consistently, are highly effective at preventing HIV infection.

3.5 Why is Uganda so Often Mentioned in Relation to the ABC of HIV Prevention?

The ABC approach to HIV prevention is often said to have started in Uganda, and it is said by some people to have been the reason for Uganda's unique success in reducing its HIV prevalence (defined as the proportion of adults living with HIV).

3.5.1 What Happened in Uganda?

When HIV was rapidly spreading through the population of Uganda in the late 1980s, President Yoweri Museveni, unlike most other African leaders at the time, recognised the danger and took swift action showing forceful leadership. Uganda's response was powerful and wide-ranging. The government launched an aggressive media campaign involving posters, radio messages and rallies; they trained

teachers to begin effective HIV and AIDS education; and - most importantly - they mobilised community leaders, churches and indeed the public in general.

The government worked alongside many independent organisations, using different messages to address different groups of people according to their needs as well as their ability to respond. Young people were encouraged to wait before first having sex, or to return to abstinence if they were not virgins. All sexually active people were given the message of "zero grazing", which meant staying with regular partners and not having casual sex. Those who did not abstain were encouraged to use condoms, which were promoted to the population as a whole.

In order to encourage people to take up such strategies - and to make them effective - action was taken to encourage candid discussion of HIV and AIDS, to reduce stigma, to better the status of women, to improve testing facilities, to treat other sexually transmitted infections and to provide better care for those already infected. Fear was also a part of the strategy, but the campaigns explained how to avoid or reduce risk and so not be overtaken by fear.

There is no evidence of the term "ABC" being used in Uganda's campaigns at this time, although they did incorporate some elements of abstinence, being faithful and using condoms.

3.5.2 What worked in Uganda?

What appears to have worked in Uganda was a combination of risk avoidance and risk reduction approaches. These resulted in a fall in the annual number of new infections between the late 1980s and mid 1990s, which in turn led to a reduction in HIV prevalence. In later years, an increase in the death rate probably made a contribution to further declines in prevalence, while the number of new infections remained more or less unchanged.

What has been particularly important in Uganda has been the combination of messages and approaches that have been used, including the widespread promotion and distribution of condoms. During the 1990s, schemes funded by USAID and other donors greatly increased condom use (WHO, 2004).

“The ABC approach in Uganda was and still is more than just abstinence and needs to be balanced without any emphasis on one aspect. Neither 'A' nor 'B' nor 'C' on its own can provide the answer to reducing risk of infection that is practical for every member of the population.” (Talisuna, 2005).

4.0 CONCLUSION

This unit looked at strategies for preventing sexual transmission of HIV, and in particular the much-discussed "ABC" approach. The ABC approach was viewed as A – Abstain, B – Be faithful and C – Condomise. The ABC approach to HIV prevention is often said to have started in Uganda, and it is said by some people to have been the reason for Uganda's unique success in reducing its HIV prevalence (defined as the proportion of adults living with HIV).

5.0 SUMMARY

Hope you found this unit interesting. This unit explained ABC approach of HIV prevention, identified PEPFAR and UNAIDS Definition of ABC Approach of HIV Prevention and provided insight on why Uganda so often mentioned in relation to the ABC of HIV Prevention

6.0 TUTOR MARKED ASSIGNMENT

- Why is Uganda so Often Mentioned in Relation to the ABC of HIV Prevention?
- What happened in Uganda?
- What worked in Uganda?

ANSWER TO SELF ASSESSMENT EXERCISE

The ABC approach is:

- A bstain
- B e faithful
- C ondomise

The ABC approach is not saying something new, rather it indicates a new way of putting across known risk reduction and risk avoidance strategies.

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UNIT 3 USING CONDOMS

CONTENT

1.0 INTRODUCTION

Condom, a much publicized and marketed method of HIV/AIDS prevention is indeed a very controversial one. While it has formed part of sex education, especially for HIV prevention, it has not gone down well with religious bodies and other related organization. Reason being that it promotes promiscuity. However, in this unit, we will equip you with basic information about condom, its usage, its effectiveness, modes of disposal, etc. This unit will also provide tips on how to persuade a partner to use a condom as well as other confidence tips.

2.0 OBJECTIVES

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

- Identify the need for Condom, and ways of choosing the right Condom
- Identify what Condoms are made of
- Describe Spermicides and Nonoxynol 9
- Describe how to check if a condom is safe to use
- Identify what to do in the case of Condom breakage
- Illustrate the effectiveness of condom
- Describe way of persuading a partner to use a condom

3.0 MAIN CONTEN

3.1 Why do I need to use a condom?

A Condom

Condoms are the only form of protection that can both help to stop the transmission of sexually transmitted diseases (STDs) such as HIV and prevent pregnancy.

3.2 How do I choose the right condom?

Condoms

A number of different types of condom are now available. What is generally called a condom is the 'male' condom, a sheath or covering which fits over a man's penis, and which is closed at one end.

There is also now a female condom, or vaginal sheath, which is used by a woman and which fits inside her vagina. The rest of this unit is about the male condom.

3.3 What are condoms made of?

Condoms are usually made of latex or polyurethane. If possible you should use a latex condom, as they are slightly more reliable, and in most countries they are most readily available.

Latex condoms can only be used with water based lubricants, not oil based lubricants such as Vaseline or cold cream as they break down the latex. A small number of people have an allergic reaction to latex and can use polyurethane condoms instead.

Polyurethane condoms are made of a type of plastic. They are thinner than latex condoms, and so they increase sensitivity and are more agreeable in feel and appearance to some users. They are more expensive than latex condoms and slightly less flexible so more lubrication may be needed. However both oil and water based lubricants can be used with them.

It is not clear whether latex or polyurethane condoms are stronger – there are studies suggesting that either is less likely to break. With both types however, the likelihood of breakages is very small if used correctly.

The lubrication on condoms also varies. Some condoms are not lubricated at all, some are lubricated with a silicone substance, and some condoms have a water-based lubricant. The lubrication on condoms aims to make the condom easier to put on and more comfortable to use. It can also help prevent condom breakage.

3.4 Spermicides and Nonoxynol 9

Condoms and lubricants sometimes contain a spermicide called Nonoxynol 9. Adding Nonoxynol 9 to condoms was thought in the past to help to prevent pregnancy and the transmission of HIV and other STDs, but it is now known to be ineffective.

Some people have an allergic reaction to Nonoxynol 9 that can result in little sores, which can actually make the transmission of HIV more likely. Because of this, you should only use condoms and lubricants containing Nonoxynol 9 if you are HIV negative and know that your partner is too. However, using a condom (even if it contains Nonoxynol 9) is much safer than having unprotected sex.

3.5 What shapes are there and which should I choose? What about flavoured condoms?

A Condom

Condoms come in a variety of shapes. Most have a reservoir tip although some do have a plain tip. Condoms may be regular shaped (with straight sides), form fit (indented below the head of the penis), or they may be flared (wider over the head of the penis).

Ribbed condoms are textured with ribs or bumps, which can increase sensation for both partners. Condoms also come in a variety of colours.

It's up to you which shape you choose. All of the differences in shape are designed to suit different personal preferences and enhance pleasure. It is important to communicate with your partner to be sure that you are using condoms that satisfy both of you.

Some condoms are flavoured to make oral sex more enjoyable. They are also safe to use for penetrative sex as long as they have been tested and approved.

3.6 What about the condom size?

Condoms are made in different lengths and widths, and different manufacturers produce varying sizes.

There is no standard length for condoms, though those made from natural rubber will in addition always stretch if necessary to fit the length of the man's erect penis.

The width of a condom can also vary. Some condoms have a slightly smaller width to give a "closer" fit, whereas others will be slightly larger. Condom makers have realised that different lengths and widths are needed and are increasingly broadening their range of sizes.

The brand names will be different in each country, so you will need to do your own investigation of different names. There is no particular best brand of condom.

3.7 When do you use a condom?

You need to use a new condom every time you have sexual intercourse. Never use the same condom twice. Put the condom on after the penis is erect and before any contact is made between the penis and any part of the partner's body. If you go from anal intercourse to vaginal intercourse, you should consider changing the condom.

3.8 Where can I get condoms?

There are no age limitations on buying condoms. Buying a condom no matter how old you are shows that you are taking responsibility for your actions. Family planning and sexual health clinics provide condoms free of charge. Condoms are available to buy from supermarkets, convenience stores, hotels, etc.

3.9 How can I check a condom is safe to use?

Condoms that have been properly tested and approved carry the British Standard Kite Mark or the EEC Standard Mark (CE). In the USA, condoms should be FDA approved, and elsewhere in the world, they should be ISO approved.

Condoms have an expiration (Exp) or manufacture (MFG) date on the box or individual package that tells you when it is safe to use the condom until. It's important to check this when you use a condom. You should also make sure the package and the condom appear to be in good condition.

Condoms can deteriorate if not stored properly as they are affected by both heat and light. So it is best not to use a condom that has been stored in your back pocket, your wallet, or the glove compartment of your car. If a condom feels sticky or very dry you should not use it as the packaging has probably been damaged.

3.10 How do you use a condom?

Condom

Open the condom package at one corner being careful not to tear the condom with your fingernails, your teeth, or through being too rough. Make sure the package and condom appear to be in good condition, and check that if there is an expiry date that the date has not passed.

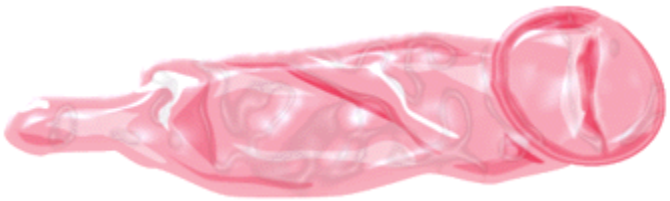
Place the rolled condom over the tip of the hard penis, and if the condom does not have a reservoir top, pinch the tip of the condom enough to leave a half inch space for semen to collect. If the man is not circumcised, then pull back the foreskin before rolling on the condom.

Pinch the air out of the condom tip with one hand and unroll the condom over the penis with the other hand. Roll the condom all the way down to the base of the penis, and smooth out any air bubbles. (Air bubbles can cause a condom to break.)

If you want to use some extra lubrication, put it on the outside of the condom. But always use a water-based lubricant (such as KY Jelly or Liquid Silk) with latex condoms, as an oil-based lubricant will cause the latex to break.

The man wearing the condom doesn't always have to be the one putting it on - it can be quite a nice thing for his partner to do.

3.11 What do you do if the condom would not unroll?



A Condom

The condom should unroll smoothly and easily from the rim on the outside. If you have to struggle or if it takes more than a few seconds, it probably means that you are trying to put the condom on upside down. To take off the condom, do not try to roll it back up. Hold it near the rim and slide it off. Then start again with a new condom.

SELF ASSESSMENT EXERCISE

- How can you check if a condom is safe to use?
- When do you use a condom?

3.12 When do you take off the condom?

Pull out before the penis softens, and hold the condom against the base of the penis while you pull out, so that the semen does not spill. Condom should be disposed properly for example wrapping it in a tissue and throwing it away. It is not good to flush condoms down the toilet - they're bad for the environment.

3.13 What do you do if a condom breaks?

If a condom breaks during sexual intercourse, then pull out quickly and replace the condom. Whilst you are having sex, check the condom from time to time, to make sure it has not split or slipped off. If the condom has broken and you feel that semen has come out of the condom during sex, you should consider getting emergency contraception such as the morning after pill.

3.14 What condoms should you use for anal intercourse?

With anal intercourse more strain is placed on the condom. You can use stronger condoms (which are thicker) but standard condoms are just as effective as long as they are used correctly with plenty of lubricant. Condoms with a lubricant containing Nonoxynol 9 should NOT be used for anal sex as Nonoxynol 9 damages the lining of the rectum increasing the risk of HIV and other STD transmission.

3.15 Is using a condom effective?

If used properly, a condom is very effective at reducing the risk of being infected with HIV during sexual intercourse. Using a condom also provides protection against other sexually transmitted diseases, and protection against pregnancy. In the laboratory, latex condoms are very effective at blocking transmission of HIV because the pores in latex condoms are too small to allow the virus to pass through. However, outside of the laboratory condoms are less effective because people do not always use condoms properly.

3.16 How do you dispose of a used condom?

All condoms should be disposed of by wrapping in tissue or toilet paper and throwing them in the bin. Condoms should not be flushed down the toilet as they may cause blockages in the sewage system and pollution.

Latex condoms are made mainly from latex with added stabilizers, preservatives and vulcanizing (hardening) agents. Latex is a natural substance made from rubber trees, but because of the added ingredients most latex condoms are not biodegradable. Polyurethane condoms are made from plastic and are not

biodegradable. Biodegradable latex condoms are available from some manufacturers.

3.17 How can I persuade my partner that we should use a condom?

It can be difficult to talk about using condoms. But you shouldn't let embarrassment become a health risk. The person you are thinking about having sex with may not agree at first when you say that you want to use a condom when you have sex. These are some comments that might be made and some answers that you could try...

EXCUSE	ANSWER
Don't you trust me?	Trust isn't the point, people can have infections without realising it
It does not feel as good with a condom	I'll feel more relaxed, If I am more relaxed, I can make it feel better for you.
I don't stay hard when I put on a condom	I'll help you put it on, that will help you keep it hard.
I am afraid to ask him to use a condom. He'll think I don't trust him.	If you can't ask him, you probably don't trust him.
I can't feel a thing when I wear a condom	Maybe that way you'll last even longer and that will make up for it
I don't have a condom with me	I do
It's up to him... it's his decision	It's your health. It should be your decision too!
I'm on the pill, you don't need a condom	I'd like to use it anyway. It will help to protect us from infections we may not realise we have.
It just isn't as sensitive and I can't feel a thing	Maybe that way you will last even longer and that will make up for it
Putting it on interrupts everything	Not if I help put it on
I guess you don't really love me	I do, but I am not risking my future to prove it
I will pull out in time	Women can get pregnant and get STDs from pre-ejaculate
But I love you	Then you'll help us to protect ourselves.
Just this once	Once is all it takes

There are many reasons to use condoms when having sex. You could go through these reasons with your partner and see what she/he thinks.

3.18 Reasons to use condoms

1. Condoms are the only contraceptive that help prevent both pregnancy and the spread of sexually transmitted diseases (including HIV) when used properly and consistently.
2. Condoms are one of the most reliable methods of birth control when use properly and consistently.
3. Condoms have none of the medical side-effects of some other birth control methods may have.
4. Condoms are available in various shapes, colours, flavours, textures and sizes - to increase the fun of making love with condoms.
5. Condoms are widely available in pharmacies, supermarkets and convenience stores. You don't need a prescription or have to visit a doctor.
6. Condoms make sex less messy.
7. Condoms are user friendly. With a little practice, they can also add confidence to the enjoyment of sex.
8. Condoms are only needed when you are having sex unlike some other contraceptives which require you to take or have them all of the time.

Here are also some tips that can help you to feel more confident and relaxed about using condoms.

3.19 Confidence tips

- Keep condoms handy at all times. If things start getting steamy - you'll be ready. It's not a good idea to find yourself having to rush out at the crucial moment to buy condoms - at the height of the passion you may not want to.
- When you buy condoms, don't get embarrassed. If anything, be proud. It shows that you are responsible and confident and when the time comes it will all be worthwhile. It can be more fun to go shopping for condoms with your partner or friend. Nowadays, it is also easy to buy condoms discreetly on the internet.
- Talk with your partner about using a condom before having sex. It removes anxiety and embarrassment. Knowing where you both stand before the passion stands will make you lot more confident that you both agree and are happy about using a condom.
- If you are new to condoms, the best way to learn how to use them is to practice putting them on by yourself or your partner. It does not take long to become a master.
- If you feel that condoms interrupt you passion then try introducing condoms into your lovemaking. It can be really sexy if your partner helps you put it on or you do it together.

4.0 CONCLUSION

Condoms are regarded as the only form of protection that can both help to stop the transmission of sexually transmitted diseases (STDs) such as HIV and prevent pregnancy. A number of different types of condom are now available. What is generally called a condom is the 'male' condom, a sheath or covering which fits over a man's penis, and which is closed at one end. There is also now a female condom, or vaginal sheath, which is used by a woman and which fits inside her vagina. Condoms are usually made of latex or polyurethane. If possible you should use a latex condom, as they are slightly more reliable, and in most countries they are most readily available. However, we also observed that it can be difficult to talk about using condoms. But you should not let embarrassment become a health risk. The person you are thinking about having sex with may not agree at first when you say that you want to use a condom when you have sex, but persistent pays.

5.0 SUMMARY

In this unit, we provided in-depth information on condom, its usage, how to choose the right one, what condoms are made of and the role of Spermicides and Nonoxynol 9. We also describe how to check if a condom is safe to use, identified what to do in the case of Condom breakage and finally describe way of persuading a partner to use a condom.

6.0 TUTOR MARKED ASSIGNMENT

- How can you persuade your partner to use a condom?
- Identify reasons for use of Condom

ANSWER TO SELF ASSESSMENT EXERCISE

- When do you use a condom? You need to use a new condom every time you have sexual intercourse. Never use the same condom twice. Put the condom on after the penis is erect and before any contact is made between the penis and any part of the partner's body. If you go from anal intercourse to vaginal intercourse, you should consider changing the condom.
- How can I check a condom is safe to use? Condoms that have been properly tested and approved carry the British Standard Kite Mark or the EEC Standard Mark (CE). In the USA, condoms should be FDA approved, and elsewhere in the world, they should be ISO approved. Condoms have an expiration (Exp) or manufacture (MFG) date on the box or individual package that tells you when it is safe to use the condom until. It's important to check this when you use a condom. You should also make sure the

package and the condom appear to be in good condition. Condoms can deteriorate if not stored properly as they are affected by both heat and light. So it's best not to use a condom that has been stored in your back pocket, your wallet, or the glove compartment of your car. If a condom feels sticky or very dry you should not use it as the packaging has probably been damaged.

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UNIT 4 ABSTINENCE, SEX EDUCATION AND HIV PREVENTION

CONTENT

1.0 INTRODUCTION

Sex education is an important part of effective HIV prevention. It is generally accepted that it enables people to acquire knowledge and develop skills that they can use to protect and promote their sexual health through minimising the risks that they might face in the course of their sexual experiences. In recent years there has been discussion about what form sex education should take and the advantages and disadvantages of adopting an abstinence-based approach as an alternative to a more comprehensive approach. This discussion has assumed added significance with an increasing emphasis on the provision of funding for abstinence-based approaches to sex education in some of the parts of the world worst affected by HIV and AIDS.

2.0 OBJECTIVES

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

- Discuss abstinence-based approach to sex education?

- Identify how abstinence-based approach to sex education differs from comprehensive sex education.
- Determine whether abstinence-based and comprehensive approaches to sex education can be combined.
- Identify disagreements trailing these methods.
- Determine which method is best.
- Identify what research evidence show about the effects of abstinence-based approaches.
- Identify the difference in the content of abstinence-based and comprehensive programmes of sex education.
- Determine if it is realistic to encourage abstinence until marriage.

3.0 MAIN CONTENT

3.1 What is an abstinence-based approach to sex education?

An abstinence-based approach to sex education focuses on teaching young people that abstaining from sex until marriage is the best means of ensuring that they avoid infection with HIV, other sexually transmitted infections and unintended pregnancy. As well as seeing abstinence from sex as the best option for maintaining sexual health, many supporters of abstinence-based approaches to sex education also believe that it is morally wrong for people to have sex before they are married.

Abstinence approaches are represented in programmes such as *Project Reality* and *True Loves Waits* (both developed in the US), which aim to teach young people that they should commit to abstain from sex until marriage.

3.2 How does this differ from comprehensive sex education?

The main difference between abstinence-based and comprehensive approaches to sex education is that:

- Comprehensive approaches do not focus either solely or so closely on teaching young people that they should abstain from sex until they are married. And although they do explain to young people the potential benefits of delaying having sex until they are emotionally and physically ready, they also make sure that they are taught how to protect themselves from infections and pregnancy when they do decide to have sex.

Examples of what programmes of comprehensive sex education comprise include SHARE (Sexual Health and Relationships: Safe, Happy and Responsible) in the

UK and the guidelines produced by SIECUS (Sexuality Information and Education Council of the United States) in the US (SHARE, 2004).

3.3 Can abstinence-based and comprehensive approaches to sex education be combined?

Some people have argued that is it possible to combine the main elements of both comprehensive and abstinence-based approaches to sex education in one approach. These people point out that supporters of both abstinence-based and comprehensive approaches share the view that sex education plays an important role in HIV prevention and both approaches emphasise the potential benefits of delaying having sexual intercourse in terms of helping young people avoid HIV, other STIs and unintended pregnancies. On the basis of this it has been argued that abstinence-based and comprehensive approaches can be reconciled into one inclusive approach which is sometimes called *abstinence-plus* (Halperin, 2004).

In abstinence-plus sex education, although the main emphasis is on abstaining from sex as the preferred choice of protection, young people are also provided with information about contraception and disease prevention so that they can protect themselves when they do become sexually active (Kaiser, 2003). Abstinence-plus approaches include programmes such as the *Reducing the Risk* and *APAUSE (Added Power And Understanding in Sex Education)* which have been developed in the US and UK, respectively (Barth, 2004; Card, 1999). Both these programmes comprise school-based curricula which explicitly emphasise that students should avoid unprotected intercourse, either by not having sex or (for students who choose to have sex) by using contraceptives.

SELF ASSESSMENT EXERCISE

- What is sex education?
- What is abstinence-based sex education
- What is abstinence-plus sex education

3.4 Why is there so much disagreement?

Despite the similarities in some of the things that supporters of abstinence-based and comprehensive approaches believe about sex education and what it can achieve in terms of young people's sexual health, it is probably overly optimistic to think that it is possible to build consensus on a single approach. This is because these superficial similarities mask profound differences in the values and attitudes which inform the views of supporters of abstinence-based and comprehensive sex education.

Many supporters of abstinence-based sex education have a background in or connection to Christian organisations that have strong views about sex and sexuality. Not only do they often believe that sex should only take place in the context of marriage, but some are also opposed to same-sex relationships and abortion (Maher, 2005). As a result of the strong faith basis for their beliefs about sex, supporters of abstinence education see the main objective as being to equip (and encourage) young people to refuse or avoid sex altogether, and they may exclude from their programmes any other information that they believe conflicts with this view. This may result in an abstinence-only course failing to include basic information about what activities transmit HIV and how such transmission can be avoided.

As a result of the strong faith basis for their beliefs about sex, supporters of abstinence education see the main objective as being to equip (and encourage) young people to refuse or avoid sex.

Even where supporters of abstinence-based sex education disavow a strong religious basis for their beliefs about what young people should be taught, they often highlight issues about fidelity to one partner, and reject provision of information about steps young people can take to protect themselves against disease and unintended pregnancy because they argue that to do so sends a mixed message.

In contrast, most supporters of comprehensive sex education regard having sex and issues to do with sexuality as matters of personal choice that should not be dictated by religious or political dogmas. Working from an understanding of human rights, which means that people are entitled to access information about matters that affect them and the decisions that they make, they see sex education as being about providing young people with the means by which they can protect themselves against abuse and exploitation as well as unintended pregnancies, sexually transmitted diseases and HIV/AIDS. They argue that without access to information about all aspects of sex and sexuality making these decisions freely is impossible. While they think that is important that sex education is sensitive to faith issues, they assert that it should not be based on any set of specific religious values (Blake, 2002; Whiteheat et al. 2001).

These fundamentally different views about sex and sexuality mean that supporters of abstinence-based and comprehensive approaches to sex education see the 'problem' of what to do about young people and sex quite differently and therefore reach quite different conclusions about the 'solution'. If, as supporters of comprehensive sex education tend to believe, the underlying premise of sexual health interventions is to meet social and utilitarian ideals then the solutions that are proposed are more likely to include earlier and more comprehensive sex

education, more liberal abortion laws and freely available contraception. By contrast if, as supporters of abstinence-based approaches feel, the underlying motive has a strong religious dimension then the solutions are more likely to revolve around abstinence campaigns and be characterised by reluctance to promote contraception UNICEF, 2001).

3.5 Which method is best?

One of the ways in which the debate between supporters of abstinence and comprehensive approaches to sex education has been framed is in terms of which is the most effective.

Although at first glance the evidence can seem confusing, with claims coming from both groups about the proven effectiveness of programmes embodying their values, when only the most reliable studies are taken into account the position is clear (Oakley, et al, 1995). There is good evidence, from studies of programmes implemented in the US, UK and other European countries and countries in Africa and Asia(Collin et al, 2002; Kirby et al, 1994), that comprehensive sex education can reduce behaviours that put young people at risk of HIV, STIs and unintended pregnancy. Studies have repeatedly shown too that this kind of sex education does not lead to the earlier onset of sexual activity among young people and, in some cases, will even lead to it happening later.

In contrast there is no such robust evidence for the effectiveness of abstinence education. Almost all the studies that have claimed to show any positive outcomes are not well-enough designed to sustain these claims (Kirby, 2002; Collin et al, 2002; Underhill, 2007), so it is not possible to infer whether they work or not from the research reports.

3.6 What does the research evidence show about the effects of abstinence-based approaches?

The research that is available currently shows at best mixed outcomes for abstinence-based approaches to sex education, benefiting some young people in the short term but placing them at greater risks later. For example, two studies suggest that for some young people making pledges to abstain from sexual intercourse until marriage does lead to delay in the timing of their first sexual intercourse. But these young people tend to hold strong religious beliefs and enjoy being an exclusive group among peers who do not take abstinence pledges. As the researchers note this means that pledging abstinence is not appropriate for young people who do not hold strong religious views and, moreover, if lots of young people are involved in making pledges (as using abstinence education as a method of sex education requires) the sense of being special will be dissipated (Bearman,

2001). In addition, while making an abstinence pledge may work for some groups of young people as a way of delaying when they have sexual intercourse, the majority still have sex before they are married and when they do they report using condoms less often than 'non-pledgers' and are more likely to substitute anal or oral sex for vaginal sexual intercourse (Brucker, et al, 2005).

In April 2007 a company called Mathematica Policy Research published the results of a Congressionally mandated evaluation of federally funded abstinence-based programmes in American schools. The investigation, which looked at four programmes offering a range of settings and strategies, found that rates of abstinence and unprotected sex in students who took part in the programmes were virtually identical to rates among students who had been randomly assigned to not take part. The ages at first sexual intercourse were also nearly identical, as were the numbers of sexual partners. It appears that the programmes had no impact on how the students behaved.

With regards to HIV prevention, a systematic review of all relevant studies, published in October 2007, concluded, "Evidence does not indicate that abstinence-only interventions effectively decrease or exacerbate HIV risk among participants in high-income countries; trials suggest that the programs are ineffective." Nevertheless the authors stressed the paucity of robust data and the need for more rigorous trials. They noted that most studies have been conducted among American youth, which may limit the generalisability of their findings.

3.7 What is the difference in the content of abstinence-based and comprehensive programmes of sex education?

Another way in which the debate gets framed is in relation to differences in beliefs about what the 'real facts' are that young people should be presented with in the context of sex education. Many supporters of abstinence-based sex education say that comprehensive programmes are too positive about the protective potential of contraceptives and understate their failure rate and the risks of contracting HIV or another STI (Stammers, 2003). In addition, they criticise programmes of comprehensive sex education for placing too little emphasis on abstinence and sending young people a mixed message by referring both to abstaining from or delaying when they first have sexual intercourse, and the benefits of using contraception (The Physicians Consortium, 2002). Some reviews of abstinence - based programmes suggest factual inaccuracies.

For their part critics of abstinence-based programmes have said that they are too negative about the effectiveness of contraception and sometimes include inaccurate information about failure rates. Proponents of abstinence-based approaches have been accused of overstating condom failure rates, exaggerating

the risks of infection with HIV and other STIs, reinforcing gender and sexuality stereotypes, and presenting sex and sexuality in an overly negative way (Blake et al, 2001; The Seattle Times, 2007).

The criticisms levelled against comprehensive programmes of sex education are difficult to sustain because research suggests that in practice many sex educators are very concerned not to present sex in too positive a light and tend to avoid coverage of sensitive and potentially embarrassing subjects like homosexuality and abortion. Young people consistently report that the underlying message is that they should not have sex (Buster, et al, 2002; Forrest, 2004). Moreover, much of the evidence for the ineffectiveness of condoms and other contraceptives cited by critics of comprehensive programmes is highly suspect, being based on poor quality research or the outcome of a partial reading of its results (Pater, 2002).

In contrast, those criticisms levelled at abstinence-based approaches do seem to have a firmer foundation. Some reviews of programme materials suggest factual inaccuracies - such as massively overestimating the prevalence of HIV and STIs and the failure rates of condoms when properly used - are common (Batcher 2004). These reviews have also shown that these programmes tend to project stereotypes about gender, repress information about positive aspects of sexual relationships, and overstate the emotional risks and dangers associated with sex (Blake et al, 2001).

3.8 Is it realistic to encourage abstinence until marriage?

The premise on which abstinence education is founded - that it is reasonable to wait until marriage before having sex for the first time and then be faithful to that one partner for life - may well be unrealistic for many young people because it fails to reflect the nature of modern, industrial societies in which people marry later in life, if at all. And with the high frequency of breakdown in marriage, people are very likely to have several sexual partners over their lifetime. Across the US, the UK and the rest of Europe data on sexual lifestyles consistently show that the age at which people first marry has risen to around 30 years old and that about a fifth of marriages end in divorce or separation within five years (National Statistics, 2005). Yet while the age at which people marry has risen, the age at which they first have sexual intercourse has been falling to around 16 years old, and a diminishing minority of people report that their first sexual partner was also their marriage partner (Wellington, et al, 2001).

3.9 So can we decide whether one approach is better than the other?

It is very important to note that debates about research into the effectiveness of different types of sex education, and criticisms of the extent to which programmes

contain factual inaccuracies and are guilty of stereotyping, do not always represent objective attempts to weigh the evidence that these studies have produced. While the debate between supporters of both approaches has populated these areas of difference it is not in pursuit of a resolution of their differences but rather a definitive answer that suits their moral agenda. There is no doubt that, whatever evidence is assembled, people who hold particular strong moral views are unlikely to give up supporting their preferred approach regardless of whether it works or whether someone else thinks it presents a distorted picture of the facts.

4.0 CONCLUSION

Prevention is better than cure, so goes the saying. Sex education is indeed an important part of effective HIV prevention. It is generally accepted that it enables people to acquire knowledge and develop skills that they can use to protect and promote their sexual health through minimizing the risks that they might face in the course of their sexual experiences. An abstinence-based approach to sex education focuses on teaching young people that abstaining from sex until marriage is the best means of ensuring that they avoid infection with HIV, other sexually transmitted infections and unintended pregnancy. However, the main difference between abstinence-based and comprehensive approaches to sex education is that comprehensive approaches do not focus either solely or so closely on teaching young people that they should abstain from sex until they are married. And although they do explain to young people the potential benefits of delaying having sex until they are emotionally and physically ready, they also make sure that they are taught how to protect themselves from infections and pregnancy when they do decide to have sex.

5.0 SUMMARY

Hope you found this unit stimulating. This unit illustrated abstinence-based approach to sex education as well as identified how abstinence-based approach to sex education differs from comprehensive sex education. This unit determined whether abstinence-based and comprehensive approaches to sex education can be combined, identified disagreements trailing these methods. It also explained what research evidence show about the effects of abstinence-based approaches, identified the difference in the content of abstinence-based and comprehensive programmes of sex education and further determined if it is realistic to encourage abstinence until marriage.

6.0 TUTOR MARKED ASSIGNMENT

- Identify the differences between comprehensive sex education and abstinence-based sex education.
- Why is there so much disagreement between sex education and abstinence-based sex education?

ANSWER TO SELF ASSESSMENT EXERCISE

- Sex education are techniques and principles geared towards enabling people to acquire knowledge and develop skills that they can use to protect and promote their sexual health through minimizing the risks that they might face in the course of their sexual experiences
- An abstinence-based approach to sex education focuses on teaching young people that abstaining from sex until marriage is the best means of ensuring that they avoid infection with HIV, other sexually transmitted infections and unintended pregnancy. As well as seeing abstinence from sex as the best option for maintaining sexual health, many supporters of abstinence-based approaches to sex education also believe that it is morally wrong for people to have sex before they are married.
- In abstinence-plus sex education, although the main emphasis is on abstaining from sex as the preferred choice of protection, young people are also provided with information about contraception and disease prevention so that they can protect themselves when they do become sexually active.

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UNIT 5 AIDS VACCINES AND MICROBICIDES

CONTENT

1.0 INTRODUCTION

AIDS vaccines and microbicides do not yet exist but they are undergoing research and development. Vaccines and microbicides have a number of key advantages over today's HIV prevention options.

Even a partially effective vaccine or microbicide could save many millions of lives. Experts have calculated that a vaccine that is 50% effective, given to just 30% of the population could reduce the number of HIV infections in the developing world by more than half over 15 years. More effective vaccines could cut the infection rate by more than 80% (AIVI, 2006).

"Developing a safe and effective vaccine against HIV is critical to our efforts to control the devastating pandemic of HIV and AIDS" (NIAID 1996).

2.0 OBJECTIVES

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

- Define vaccine
- Define Microbicide
- Identify the advantages of AIDS vaccine and Microbicide
- Identify how AIDS vaccine could work
- Identify why it is difficult to develop an AIDS vaccine
- Identify the challenges in developing HIV microbicides

3.0 MAIN CONTENT

3.1 What is a vaccine?

A vaccine is something that teaches the body to recognise and defend itself against viruses or bacteria that cause disease. Vaccines either help to prevent infection, or help to prevent or delay illness in people who are already infected. A vaccine is not the same thing as a cure for AIDS.

Effective vaccines have already been developed for some diseases, such as smallpox, polio and tetanus, and these have saved millions of lives. But there is still no vaccine against HIV, the virus that causes AIDS.

3.2 What is a microbicide?

A microbicide is something designed to destroy microbes (bacteria and viruses) or to reduce their ability to establish an infection. A microbicide for preventing HIV infection would be applied to the vagina or rectum to prevent the virus being passed on during sex.

It is quite possible that an HIV microbicide could be available sooner than an AIDS vaccine – perhaps even as early as 2010.

3.3 What are the advantages of AIDS vaccines and microbicides?

An AIDS vaccine would have many advantages over current options for preventing HIV infection. In particular, the protection offered by a vaccine during sex would not depend on the consent of both partners (unlike condom use), and would not require behaviour change (unlike abstinence). A vaccine would be invaluable for couples wishing to conceive a child while minimising the risk of HIV transmission.

Children could be given a vaccine before ever being exposed to the virus, and ideally this would protect them from all routes of HIV transmission, including unsafe injections. Vaccinating large numbers of people would probably require relatively little equipment and expertise, and would be much simpler and cheaper than providing antiretroviral treatment for those already infected.

A microbicide would share many of the advantages of a vaccine. It would be especially useful for women unable to insist on using condoms, who might be able to use a microbicide without their partners knowing. However, a microbicide would not be able to prevent all forms of HIV transmission, and would require regular reapplication. Unlike vaccines, an effective microbicide must be made into a commodity that people will want to use regularly, such as a cream, gel or vaginal ring.

SELF ASSESSMEMT EXERCISE

- What is a vaccine?
- What is a Microbicide?

3.4 How would an AIDS vaccine work?

An AIDS vaccine could be effective in either of two ways. A “preventive” vaccine would stop HIV infection occurring altogether, whereas a “therapeutic” vaccine would not stop infection, but would prevent or delay illness in people who do become infected, and might also reduce the risk of them transmitting the virus to other people. Although a preventive vaccine would be ideal, a therapeutic vaccine would also be highly beneficial.

The basic idea behind all AIDS vaccines is to encourage the human immune system to fight HIV. The immune system works using a combination of cells and chemicals called antibodies. Early vaccine research focused on teaching the immune system to produce antibodies that would block HIV entering human cells. However, products designed to work this way failed in clinical trials because the antibodies worked only against lab-cultured HIV, not against the wild strains of virus.

Today most research focuses on encouraging the immune system to produce cells to fight HIV. Nevertheless, many scientists believe such “cell-mediated” approaches will not be very effective on their own, even as therapeutic vaccines. It seems likely that a really effective vaccine will have to take a two-pronged approach involving both cells and antibodies.

A vaccine could consist of whole HIV that has been modified to make it harmless (though this raises safety issues), or it could be based on parts of the virus, such as proteins or genes. One way to deliver parts of HIV is to put them inside a "vector" that is harmless to humans, such as the canarypox virus.

3.5 Why is it difficult to develop an AIDS vaccine?

Developing a vaccine against HIV is a very difficult challenge for scientists. There are many reasons for this, including:

- Nobody has ever recovered from HIV infection, so there is no natural mechanism to imitate
- HIV destroys the immune system cells that are meant to fight against it
- Soon after infection, HIV inserts its genetic material into human cells, where it remains hidden from the immune system
- HIV occurs in several subtypes, each of which is very different from the others
- Even within each subtype, HIV is highly variable and constantly changing
- There are no good animal models to use in experiments

3.6 Can these difficulties be overcome?

There are reasons to be optimistic about the search for an AIDS vaccine, despite the slow progress so far. Vaccines against other diseases took many decades to develop, whereas HIV was only discovered in the mid 1980s. It is therefore much too early to give up hope, especially given the current speed of scientific progress. In the past, some experts doubted the possibility of an effective polio vaccine, yet today polio is close to being eradicated.

One particular reason for remaining hopeful is that most people remain healthy for several years after becoming infected with HIV, and a small minority have survived as long as 20 years without developing AIDS, even though they never entirely rid themselves of the virus. Also it appears that a few people have some kind of natural resistance to HIV infection, meaning they never become infected despite repeated exposure to the virus. These facts suggest that the immune system can be quite effective at controlling HIV.

3.7 How would an HIV microbicide work?

A microbicide could work in at least four different ways:

- Kill or inactivate HIV
- Stop the virus entering human cells

- Enhance the body's normal defence mechanisms against HIV
- Inhibit HIV replication

It is possible that a microbicide could work in much the same way as a vaccine, so research in one area could benefit the other. Alternatively, a microbicide could work in a similar way to today's antiretroviral drugs, or it could act like a detergent.

3.8 What are the challenges in developing HIV microbicides?

There are many chemicals that kill HIV, including undiluted household bleach. But what is needed for a microbicide is something that works against HIV without causing discomfort or irritation. For example, when researchers investigated using the spermicide Nonoxynol-9 as an HIV microbicide they were surprised to find it actually increased the rate of transmission, probably because it caused vaginal lesions and inflammation, which made it easier for HIV to establish an infection, even though Nonoxynol-9 killed the virus in lab tests (Microbicide, WHO, 2004).

For a microbicide to become popular, researchers must develop not only the active ingredient but also a delivery method that is safe, effective, acceptable and affordable. Ideally this would provide protection for several days or even weeks at a time.

Another major issue is how a microbicide affects sperm. To reach all those in need, scientists will have to develop both contraceptive and non-contraceptive microbicides.

3.9 How are the candidates tested?

Any potential vaccine or microbicide must pass through three phases of clinical trials before being judged safe and effective. The first phase usually lasts from twelve to eighteen months, whereas the last phase can take three or four years to complete. In most cases volunteers must be HIV-negative at the start of the trial, though it is important also to test safety in those who are already infected. Some therapeutic vaccine candidates may be tested on HIV-positive people to see if they can delay disease progression.

- Phase I involves a small number of volunteers to test the safety of various doses
- Phase II involves hundreds of volunteers to further assess safety and, in some cases, positive responses
- Phase III involves thousands of volunteers to test safety and effectiveness

A recent innovation is the Phase IIb trial, a larger variant of the Phase II trial that provides some indication of effectiveness.

Trials of AIDS vaccines and microbicides are made more difficult by the ethical obligation to provide condoms and prevention counselling to all those who take part. Providing such services lowers the overall rate of HIV transmission, which increases the number of volunteers required to produce a significant result (HIV Vaccine and Microbicide Work Group, 2006).

3.11 What vaccine trials have already taken place?

The first AIDS vaccine candidate to undergo Phase III trials was called AIDSVAX. Two separate studies were conducted. One had around 5,400 participants - mostly gay American men - while the other involved around 2,500 injecting drug users in Thailand. The vaccine, known as AIDSVAX, was made from a single HIV protein and was meant to stimulate a protective antibody response. The trials began in 1998 and 1999 respectively, and ended in 2003. No beneficial effect was found in either population group (The Guardian, 2004).

Two Phase IIb trials of a vaccine candidate created by the pharmaceutical company Merck were halted in September 2007. The studies - known as STEP and Phambili - had been expected to produce their first results by 2010. They were stopped when researchers found that people receiving the vaccine were no less likely to become infected with HIV than those taking a placebo. The STEP trial had started in 2004 in the USA, Canada, Australia, Peru and the Caribbean; the Phambili trial had begun in January 2007 in South Africa (BBC, 2007).

There is some concern that slightly more HIV infections occurred among people who received the Merck vaccine than among those who took a placebo. The vaccine was delivered using adenovirus type 5, which causes the common cold. It has been suggested that the vaccine may have provoked a different immune response among people who already had some immunity to the adenovirus strain, and that this may have made them more susceptible to HIV infection. This hypothesis - which has yet to be fully investigated - raises questions about the use of adenovirus in future vaccines (New York Times, 2007).

Leading vaccine researcher Dr. Gary Nabel has described the results of the Merck vaccine trial as “a big blow to the field”. Several other vaccine studies have been delayed to ensure that mistakes are not repeated (Baltimore Sun, 2007). Nevertheless Dr. Seth Berkley, President and CEO of the International AIDS Vaccine Initiative, has stressed that the outcomes are not all negative:

"Though the Merck candidate failed, the trial did not. The contribution of the volunteers was not in vain. As a result of their dedication, the field will have new data that will inform future vaccine design, help with the prioritization of candidates in the pipeline and guide decisions on how to best proceed with ongoing and upcoming trials (AVI, 2007).

3.12 What vaccine trials are now underway?

As of August 2007, thirty-six human trials of AIDS vaccine candidates were taking place around the world. This number included twenty-seven Phase I trials; eight Phase I/II, Phase II or Phase IIb trials; and one Phase III trial (AIDS vaccine advocate coalition, 2007). Many more products were still at the development stage.

The Phase III trial, which has recruited 16,000 young adults in Thailand, is of a canarypox-based vaccine called ALVAC combined with an AIDSVAX booster. ALVAC is designed to stimulate a cellular response to HIV, while AIDSVAX promotes the production of antibodies.

AIDSVAX failed in previous Phase III trials. The hope is that combining it with ALVAC will produce better results. The study is due to end in late 2009, but the first preliminary results may be published in 2008 (Clinical trials coalition, 2006).

3.13 How many microbicide trials are under way?

Nine HIV microbicide trials were in progress at the start of February 2007.

Three microbicide candidates are undergoing Phase III trials to test their effectiveness:

- BufferGel, which maintains acidity in the vagina
- Carraguard, an entry inhibitor based on carrageenan, which is derived from seaweed
- PRO 2000, another entry inhibitor

In August 2006, Family Health International decided to halt a Phase III trial of a surfactant called SAVVY after preliminary results showed no evidence of a protective effect. The organisation has no plans to further investigate this product (Family Health International, 2006).

Two Phase III trials of an entry inhibitor called cellulose sulphate (also known as Ushercell) were halted in January 2007 after some sites recorded a higher HIV infection rate among women who used the gel, compared to those in the placebo

group. It is not yet known why cellulose sulphate was associated with a increased risk of infection; this result was entirely unexpected (Polydex Pharmaceutical Rep. 2007).

3.14 How soon could we have an effective vaccine?

In 1984, at the press conference arranged to announce the discovery of HIV, the US Health and Human Services Secretary Margaret Heckler said she hoped a vaccine against AIDS would be ready for testing in about two years (Public health response to AIDS, 1985).

Unfortunately, the problem has turned out to be much more challenging than Secretary Heckler expected. Today's researchers agree that the quest for an AIDS vaccine still has a long way to go. It is unlikely that any effective product will be available before 2015 at the earliest. It's even possible that the search could last decades.

"If you took a poll of reputable scientists they would say that [an AIDS vaccine] is further away today than they would have said even ten years ago. It is not a question that we have not made progress in that time. It's just that all the simple things didn't work and even the quite non-simple things didn't work." (The Times, 2005).

The news media regularly announce a new "breakthrough" in AIDS vaccine research. However, most of these stories refer to products in Phase I or Phase II trials, where there has been no evidence of the product actually working in humans. Such stories are realistically talking only about potential breakthroughs.

Few if any vaccines are 100% effective. Most probably the first AIDS vaccines to succeed in trials will offer only partial protection, and these may need to be improved or combined with other products before being suitable for widespread use. Vaccine development is likely to proceed by small, incremental steps; we are unlikely to see any immediate "miracle breakthrough".

4.0 CONCLUSION

It is very unlikely that HIV and AIDS will ever be eradicated without new scientific developments. Eventually, unless great progress is made in prevention, the number of people living with HIV will outstrip the resources available for treatment. The search for effective vaccines and microbicides must therefore be one of the very highest priorities for scientific research.

However, it is not realistic to expect such research to produce a major breakthrough for some time yet, and we should be wary of news stories suggesting otherwise. Any new discovery needs to undergo trials lasting years, and must then be distributed around the world before we will see its full benefits.

In the mean time, the world must continue to scale-up existing prevention and HIV treatment programmes. Millions of lives can be saved using the knowledge and tools already at our disposal, provided the world commits itself wholeheartedly to the cause.

5.0 SUMMARY

We hope you enjoyed your studies. Here we defined AIDS vaccine and midribicide, as well as illustrated their advantages and disadvantages. We also identified how AIDS vaccine could work and the difficulties experienced in developing the ADS vaccine. Ok, let us attempt the questions below.

6.0 TUTOR MARKED ASSIGNMENT

- What are the advantages of AIDS vaccine and mirobicides?
- Why is it difficult to develop an AIDS vaccine?
- Can these difficulties be overcome?

ANSWER TO SELF ASSESSMENT EXERCISE

- A vaccine is something that teaches the body to recognise and defend itself against viruses or bacteria that cause disease. Vaccines either help to prevent infection, or help to prevent or delay illness in people who are already infected. A vaccine is not the same thing as a cure for AIDS.
- A microbicide is something designed to destroy microbes (bacteria and viruses) or to reduce their ability to establish an infection. A microbicide for preventing HIV infection would be applied to the vagina or rectum to prevent the virus being passed on during sex.

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MODULE 6 MANAGEMENT OF HIV/AIDS

UNIT 1 HIV/AIDS MANAGEMENT CHECKLIST

CONTENT

1.0 INTRODUCTION

HIV counseling is a very sensitive and professional practice which requires rules and checklist geared towards protecting the client and making a positive test result as less traumatic as possible. This unit thus provides checklists for HIV pre-test and post-test counseling.

2.0 OBJECTIVES

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

- Identify and describe checklists for HIV pre and post test counseling
- Describe the revised guideline for HIV counseling, testing and referral
- Identify ANCARD HIV testing policy

3.0 MAIN CONTENT

3.1 Checklist for HIV testing and pre-test discussion

Specific consent to be tested for HIV must always be obtained. It is recommended to consider addressing the following issues in the course of pre-test discussion, depending on their applicability or relevance to the individual. If not addressed in the pre-test discussion, they should be addressed during post-test counselling.

1 The test is for HIV infection, not a test for AIDS.
2 Significance of the 'window period' in relation to recent risk behaviour and the resulting accuracy of the test result.
3 Significance of a positive test with respect to:

- medical implications (prognosis, treatment)
- psychological issues (coping, support, relationships)
- social implications (who needs to know, employment, discrimination implications for insurance)
- HIV status not being notifiable.

4. Explain safeguards regarding preservation of confidentiality ie, anonymous encryption of test form.

5. Discuss future prevention measures.

6. Safer sexual behaviour.

7. Safe drug injecting behaviour.

8. How results of test are to be obtained (in person, face to face).

9. Any costs that may be involved.

3.2 Checklist for Post-test counselling

1. Providing the test result:

- in person, face to face.
- not by telephone, message or mail.

2. Explain the test result:

- Discuss need for further testing (repeat/confirmatory test,

viral load, CD4 count).

3. If negative:

- Discuss possible significance of 'window period' if recent high risk behaviour and need for repeat test for final confirmation.
- Reinforce behavioural changes needed to prevent HIV infection in future eg, prescription for condoms, information on needle exchange outlets/services.
- Refer for specialist sexual health counselling, if required.

4. If positive:

- Schedule adequate time to give positive results.
- Arrange initial psychological support arrangements and follow-up appointment.
- Discuss need for further testing (repeat/confirmatory test, viral load, CD4 count).
- Discuss with an infectious disease consultant including process for partner notification.
- Referral for specialist counselling and support.
- Provide information on HIV and community resources.
- Reinforce safe sex and needle-using behaviours.
- Explain partner notification and other implications of positive diagnosis.

3.3 Revised guidelines for HIV counselling, testing and referral.

This contains the most recent set of guidelines on HIV testing produced by the US Centers for Disease Control (CDC). It provides an excellent, comprehensive, evidence-based summary of best practice with regard to HIV counselling, testing and referral.

The CDC guideline states 'the goals of HIV counselling testing and referral are to:

1. ensure that HIV infected persons and persons at increased risk for HIV

- have access to HIV testing to promote early knowledge of their HIV status
- receive high-quality HIV prevention counselling to reduce their risk for transmission or acquiring HIV
- have access to appropriate medical, preventive and

psychosocial support services

2. promote early knowledge of HIV status through HIV testing and ensure that all persons either recommended or receiving HIV testing are provided information regarding transmission, prevention and the meaning of HIV test results.'

The document provides guidance about the nature of pre-test counselling which should consist of both provision of information regarding HIV transmission and prevention and the meaning of HIV test results. HIV prevention counselling should also be given to help identify the specific behaviours putting clients at risk for acquiring or transmitting HIV and to commit to steps to reduce this risk.

The document states that information can be provided in a pamphlet, brochure or video rather than in a face-to-face encounter and that prevention counselling could take only a few minutes for those at lower risk for acquiring HIV.

SELF ASSESSMENT EXERCISE

The CDC guidelines state: 'the goals of HIV testing and referral are to: ----------

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3.4 HIV testing policy. Australian National Council on AIDS and Related Diseases (ANCARD).

ANCARD provides the most recent Australian guidelines on HIV testing policy and related issues.

It suggests that *HIV test discussion* rather than *pre-test counselling* should precede HIV testing and that post-test counselling should be provided with the results of the HIV test.

The ANCARD document suggests that HIV test discussion should include:

- an assessment of risk of HIV infection and reasons for testing
- information about confidentiality and privacy
- obtaining informed consent
- discussion of the test's implications and consequences
- arranging follow-up

- an assessment of support mechanisms while waiting for the test result and/or if the result is positive.

Post-test counselling should include:

- giving the result in person in a sensitive and supportive manner and re-assessing support mechanisms and requirements of the client

and, if the result is negative:

- reinforcing prevention of infection

and, if the result is positive:

- discussing immediate needs and support, safe behaviours, informing others, managing emotions, options in treatment, ongoing counselling, etc.

4.0 CONCLUSION

Specific consent to be tested for HIV is very important and must always be obtained. We also must bear in mind that the test is for HIV infection, not for AIDS.

5.0 SUMMARY

We hope you enjoyed your studies. In this unit, we identified checklists for HIV pre and post test counseling, describe the revised guideline for HIV counseling, testing and referral and lastly identified ANCARD HIV testing policy

6.0 TUTOR MARKED ASSIGNMENT

- Enumerate and briefly explain the Checklist for HIV pre and post test Counselling.

7.0 REFERENCES/FURTHER READINGS

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UNIT 2 HIV COUNSELLING AND THE PSYCHOSOCIAL MANAGEMENT OF PATIENTS WITH HIV OR AIDS

CONTENT

1.0 INTRODUCTION

In the previous unit, we identified checklist for HIV pre and post-test counseling. We will also provide similar information here but in a broader and more comprehensive sense, which also includes psychosocial management of HIV. We will start with a brief defining of HIV counseling then identify aims of counseling in infection prevention.

2.0 OBJECTIVES

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

- Define HIV Counselling
- Explain aims of counselling 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 councelling
- Illustrate points in Post-test discussion
- Identify and explain causes of uncertainty in the case of HIV positive result
- Describe counselling during combination antiretroviral therapy

3.0 MAIN CONTENT

3.1 What is HIV counseling?

Counselling in HIV and AIDS has become a core element in a holistic model of health care, in which psychological issues are recognised as integral to patient management. HIV and AIDS counselling 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 counselling should have these dual aims because the spread of HIV can be prevented by changes in behaviour. One to one prevention counselling 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 counselling can both minimise morbidity and reduce its occurrence. All counsellors in this field should have formal counselling training and receive regular clinical supervision as part of adherence to good standards of clinical practice.

3.2 Aims of Counselling in HIV Infection Prevention

- Determining whether the lifestyle of an individual places him or her at risk
- Working with an individual so that he or she understands the risks
- Helping to identify the meanings of high risk behaviour
- Determining whether the lifestyle of an individual places him or her at risk

- Working with an individual so that he or she understands the risks
- Helping to identify the meanings of high risk behaviour
- Helping to define the true potential for behaviour change
- Working with the individual to achieve and sustain behaviour change

Support

- Individual, relationship, and family counselling to prevent and reduce psychological morbidity associated with HIV infection and disease.

3.3 Different HIV counselling programmes and services

- Counselling before the test is done
- Counselling after the test for those who are HIV positive and HIV negative
- Risk reduction assessment to help and prevent transmission
- Counselling after a diagnosis of HIV disease has been made
- Family and relationship counselling
- Bereavement counselling
- Telephone "hotline" counselling
- Outreach counselling
- Crisis intervention
- Structured psychological support for those affected by HIV
- Support groups

SELF ASSESSMENT EXERCISE

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

3.4 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 HIV antibody positive. 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 counselling and referral to counsellor

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

3.4.2 Points for Counsellor and/or Physician to Cover for Pre-test Councelling

- What is the HIV antibody test (including seroconversion)
- The difference between HIV and AIDS
- The window period for HIV testing
- Medical advantages of knowing HIV status and treatment options
- Transmission of HIV
- Safer sex and risk reduction
- Safer injecting drug use
- If the client were positive how would the client cope: personal resources, support network of friends/partner/family
- Who to tell about the test and the result
- Partner notification issues
- HIV status of regular partner: is partner aware of patient testing?
- Confidentiality
- Does client need more time to consider?
- Is further counselling indicated?
- How the results of the test are obtained (in person from the physician or counsellor)

Patients may present for testing for any number of reasons, ranging from a generalised 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 counselling expertise. In genitourinary medicine clinics where HIV antibody testing is routinely offered as a part of sexual health screening, health advisers provide counselling 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 counsellor then the risk assessment becomes meaningless; patients may be inappropriately reassured, for example, and be unable to disclose the real reason for testing. Counselling 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 counsellor 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. Counselling support

should be available to the patient in the weeks and months following the positive test results.

3.7 Counselling 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.

Counselling 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

Counselling 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 therefore includes pre-test and post-test counseling, bereavement counseling, crisis intervention, Telephone 'hotline' counselling, outreach counseling, etc.

5.0 SUMMARY

This unit provided information on HIV Counselling, aims of counselling in HIV infection prevention, different HIV counseling programmes and services, pre and post-test discussion, causes of uncertainty in the case of HIV positive result and counselling during combination antiretroviral therapy

6.0 TUTOR MARKED ASSIGNMENT

- Identify points for counsellor and/or physician to cover for pre-test councelling
- Identify and briefly explain causes of uncertainty in the case of HIV positive result

ANSWER TO SELF ASSESSMENT EXERCISE

- Counselling in HIV and AIDS has become a core element in a holistic model of health care, in which psychological issues are recognised as integral to patient management. HIV and AIDS counselling 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 counselling should have these dual aims because the spread of HIV can be prevented by changes in behaviour.

- Different HIV counselling programmes and services

  Counselling before the test is done
  Counselling after the test for those who are HIV positive and HIV negative
  Risk reduction assessment to help and prevent transmission
  Counselling after a diagnosis of HIV disease has been made
  Family and relationship counselling
  Bereavement counselling
  Telephone "hotline" counselling
  Outreach counselling
  Crisis intervention
  Structured psychological support for those affected by HIV
  Support groups

7.0 REFERENCES/FURTHER READINGS

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UNIT 3 PSYCHOLOGICAL RESPONSES TO AN HIV POSITIVE RESULT

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 counsellor 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 counselling follow up should be given. Counselling 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 counselling

- Discuss counselling patients and partners together
- Explain the concept of 'the worried well'
- Identify characteristics of the worried well
- Identify HIV/AIDS related problems with the body image

3.0 MAIN CONTENT

3.1 Psychological issues in HIV/AIDS counselling

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 counselling 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 counselling 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 counselling sessions required during any of these periods largely depends on the individual presentation of the patient and the clinical judgment of the counsellor.

SELF ASSESSMENT EXERCISE

- Identify the importance of counseling patients and spouses together

3.3 The Worried Well

Patients known as the "worried well" present with multiple physical complaints which they interpret as sure evidence of their HIV infection. Typically, fears of infection reach obsessive proportions and frank obsessive and hypochondriacal states are often seen. This group shows a variety of characteristic features, and they are rarely reassured for more than a brief period after clinical or laboratory confirmation of the absence of HIV infection. A further referral for behavioural psychotherapy or psychiatric intervention may be indicated, rather than frequent repetition of HIV testing.

3.4 Characteristics of the worried well

- Repeated negative HIV tests
- Low risk sexual history, including covert and guilt inducing sexual activity
- Poor post adolescence sexual adjustment
- Social isolation
- Dependence in close relationships (if any)
- Multiple misinterpreted somatic features usually associated with undiagnosed viral or postviral states (not HIV) or anxiety or depression
- Psychiatric history and repeated consultation with general practitioners or physicians
- High levels of anxiety, depression, and obsessional disturbance
- Increased potential for suicidal gestures

3.4 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 emphasising 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, counselling remains an integral part of the management of patients with HIV, and their partners and family.

3.5 HIV/AIDS Related Problems with Body Image

- Wasting
- Severe facial dermatitis
- Permanent indwelling line
- Weakness and dependency
- Slowing of mental functions
- Loss of libido
- Premature greying and loss of hair
- Facial molluscum contagiosum
- Kaposi's sarcoma—common on face
- Progressive visual loss from retinitis
- Incontinence (especially faecal)

4.0 CONCLUSION

We have seen that 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. 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 counselling may be of help for the patient's partner and other family members.

5.0 SUMMARY

This unit looked at the psychological issues in HIV/AIDS counseling as well counselling patients and partners together. We also looked at the concept of the worried well.

6.0 TUTOR MARKED ASSIGNMENT

- Who is ‘the worried well’?

- What are the characteristics of the worried well?

ANSWER TO SELF ASSESSMENT EXERCISE

- 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 counselling 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.

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UNIT 4 MEDICAL MANAGEMENT OF HIV/AIDS

CONTENT

1.0 INTRODUCTION

Medical management of patients with AIDS is a balance between acute treatment and attempting to control chronic symptoms and conditions. As patients approach the end stage of the illness, they may decline certain investigations and treatments if these seem unlikely to be of much long term benefit.

Most people who are infected with HIV obtain their care from a specialist hospital clinic. These clinics usually provide a safe environment and a variety of services, such as access to specialised drugs, walk-in and day care facilities, counselling, complementary treatments, peer support, and advice about services and benefits.

2.0 OBJECTIVES

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

- Identify common symptoms in AIDS and its management
- Identify common causes of pain in AIDS
- Describe common Conditions in AIDS that require active management until death

3.0 MAIN CONTENT

3.1 Common symptoms in AIDS and Management

Causes	Management
Cough;	
Sinus disease with postnasal drip, bacterial chest infection, pneumocystis carinii pneumonia, pulmonary Kaposi's sarcoma, tuberculosis	Sputum for diagnosis, treat specific conditions when appropriate, consider decongestants
Diarrhoea	
Treatable—Salmonella, giardia, campylobacter, clostridium difficile, cytomegalovirus	Stool samples may be appropriate, specific antibacterial treatment
Unresponsive—Cryptosporidium, microsporidium, and "pathogen negative" diarrhea	Symptomatic control can be difficult, subcutaneous diamorphine or octreotide may be tried
Anorexia, nausea, vomiting	
Candida, malignancy (early satiety, acute abdomen), drugs, constipation	Review candida treatment, re-evaluate medication, consider antiemetics, dietary advice
Pruritus	
Dry skin, drug reactions, scabies, folliculitis	Emollients, antipruritics, anti-scabies treatment, topical corticosteroids
Malaise, weakness, pyrexia	
May be no detectable cause, drug reactions	Investigate, consider corticosteroids

3.2 Common Causes of Pain in AIDS

Oropharyngeal—Candida, herpes viruses (herpes simplex virus, cytomegalovirus, varicella zoster virus), apthous-type ulcers, malignancy, gingivitis, tooth abscesses

Retrosternal—Oesophageal candida, infection with cytomegalovirus or herpes simplex virus, giant oesophageal ulcers, reflux oesophagitis, pneumocystis carinii pneumonia

Headache—Toxoplasmosis, cryptococcal meningitis, cerebral lymphoma

Abdominal—Diarrhoea with or without infection, AIDS related sclerosing cholangitis, malignancy (such as Kaposi's sarcoma, lymphoma), drugs (such as clarithromycin), constipation

Perianal and perineal—Herpes simplex virus (very common, needs high index of suspicion), candida, excoriation of skin due to diarrhea

New symptoms, however, may still warrant invasive investigations because atypical presentations and extensive differential diagnoses can make easily treatable conditions unrecognisable—for example, endoscopy should be considered when investigating retrosternal pain and nausea not responding to antifungal treatment. There are also some distressing symptoms that can be controlled effectively only by specific treatment of the underlying condition (such as perianal infection with herpes simplex virus and oro-oesophageal candida).

While the underlying cause of pain and other symptoms in AIDS is often susceptible to specific treatment, symptomatic treatment should not be delayed.

Patients entering the terminal phases of disease are often receiving several drugs because many symptoms and conditions need continued prophylaxis or chronic suppressive treatment. Uncontrolled cytomegalovirus retinitis, for example, can cause blindness, so usually needs to be actively managed until death. Systemic infection with mycobacterium avium complex, may cause general malaise with fevers, anaemia, and debilitating fatigue. In this situation specific treatment of the infection, which usually involves two or three drugs, may be appropriate, or use of corticosteroids for further palliation of symptoms may be considered.

Drug treatments should be reviewed regularly so that clinical problems are controlled with a minimum of polypharmacy. Patients with AIDS and clinical or subclinical dementia can be susceptible to cognitive impairment with various drugs—sedatives, anxiolytics, strong opioids, and antidepressants.

Management of nutritional and dietary intake is also important for both medical and psychological reasons. In the final stages of AIDS, combination antiretroviral treatment may be helpful, but it is less effective than earlier in the disease and decisions about continuation should be taken with the patient.

3.3 Common Conditions in AIDS that Require Active Management until death

	Presentation and symptoms	Treatment
Cytomegalovirus retinitis;	Potential blindness, scotomas	Intravenous ganciclovir, foscarnet (needs indwelling catheter such as PortaCath or Hickman line); oral ganciclovir may be appropriate in some cases. These drugs are expensive and must be prescribed by hospital doctor
Candidiasis	*Oropharyngeal or oesophageal*—Painful oropharynx, retrosternal pain, dysphagia, anorexia, nausea and vomiting. Even mild oral candidiasis can be symptomatic and associated with oesophageal candida	Fluconazole or itraconazole (may need up to four times recommended dose as resistance common in advanced AIDS). Intravenous amphotericin (intermittent treatment may be needed in resistant cases)
Herpes simplex virus	*Oropharyngeal or anogenital*—Pain, paraesthesia, ulcers. Patients may complain of "piles" or bleeding from rectum	Aciclovir for acute episode (may need maintenance and up to four times recommended dose)
Mycobacterium avium complex	Fevers, night sweats, malaise, fatigue, anorexia, weight loss, diarrhoea, anaemia; symptoms may occur singly or in combination	Combination therapy (such as rifabutin, clarithromycin, and ethambutol) may give good palliation of symptoms. Single drug for short periods if compliance or polypharmacy a problem
Kaposi's sarcoma	*Pulmonary*—Cough, dry or productive and may be paroxysms; progressive breathlessness; haemoptysis; effusions	Symptomatic control (such as anxiolytics, opioids, oxygen). Chemotherapy and radiotherapy of limited value. Poor prognosis

	Cutaneous, lymphatic, or other viscera—Local symptoms such as lymphadenopathy, oedema, loss of function, breakdown or necrosis of skin, pain, possible severe disfigurement and distortion of tissues	Variable natural course (minor or considerable morbidity). Chemotherapy and radiotherapy may help palliation of problems. Corticosteroids can help oedema but may exacerbate infections
AIDS dementia complex	Variable neurological presentations, including dementia and psychiatric illness	Difficult and distressing to manage, requiring multidisciplinary approach. Combination therapy, which must include high dose zidovudine, is worth trying
Pneumocystis carinii pneumonia	Dry cough, possibly paroxysmal and distressing; chest pain; breathlessness; fevers; sweats; malaise; anorexia	Prophylaxis (primary or secondary) usually continued (co-trimoxazole, dapsone, pentamidine)
Co-infection with hepatitis B or C	Usually incidental finding with liver function tests	Poor response to treatment. Complicates prescribing of hepatotoxic drugs

4.0 CONCLUSION

Adequate management of HIV/AIDS is of utmost importance. Specifically, this unit highlighted common medical condition of HIV/AIDS and how to tackle them. Many patients do not have a general practitioner and are often reluctant to register with one. Even if they are registered they may still not disclose their HIV status. The reasons for this include their concerns over confidentiality, the fear of discrimination, and the risk of rejection because of the belief of potential financial costs to the practice of looking after a patient with AIDS. This means that general practitioners often become involved with AIDS patients only at a late stage in their disease. It is important to encourage patients to register early with a general practitioner in whom they can build trust so that the transition to shared care can be made as smoothly as possible.

5.0 SUMMARY

We hope you found this unit interesting and insightful too. Here we identified common symptoms in AIDS and its management**,** common causes of pain in AIDS and also describe common conditions in AIDS that require active management until death

6.0 TUTOR MARKED ASSIGNMENT

- Identify and briefly explain common symptoms in AIDS as well as the medical management techniques

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UNIT 5 NUTRITIONAL MANAGEMENT OF HIV/AIDS

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

3.0 MAIN CONTENT

3.1 Nutritional Problems Associated with HIV Disease

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 Nutritional 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

We hope this unit was helpful. In this unit, we discussed the primary nutritional problems caused by HIV, described the nutritional problems associated with combination anti-retroviral therapy and listed practical suggestions for improving nutritional status in symptomatic AIDS.

6.0 TUTOR MARKED ASSIGNMENT

- Give practical suggestions for food intake in symptomatic AIDS

ANSWER TO SELF ASSESSMENT EXERCISE

Side effects possibly associated with anti-retroviral therapy such as a fat redistribution syndrome and metabolic complications 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

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