18

SEXUALLY TRANSMITTED DISEASES IN SIKKIM

DONE BY:

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TABLE OF CONTENTS

LIST OF TABLES AND GRAPHS

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CERTIFICATE

Certified that Shri. HEMANT POUDYAL has carried out this work under the supervision of the undersigned, supported by the State Council of Science & Technology for Sikkim, Gangtok. The work has been based on the original survey carried out by Shri Hemant in Gangtok and its surroundings. This project has not formed a basis for the award of any other Degree/ Diploma of any University or Institute.

20/10/05.

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OUR PRIORITIES : 2001 - 2002

RURAL TECHNOLOGY AND ENTREPRENEURSHIP DEVELOPMENT PROGRAMME FOR THE UNEMPLOYED
PROGRAMMES FOR GENERATING SUPPLEMENTARY INCOME FOR THE WOMEN
INFORMATION TECHNOLOGY FOR SCIENCE EDUCATION AND POPULARIZATION ESPECIALLY AMONGST STUDENT COMMUNITY.

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HEMANT POUDYAL

INTRODUCTION

Sexually Transmitted Diseases (STD) are common in both developing as well as the developed countries. The origin of these diseases dates back to antiquity. History reveals many famous people who fallen preys to these diseases to mention a few are Caesar, Cleopatra, Napoleon, Oscar Wilde and Hitler. These overlooked diseases have already become a menace in the health scenario. There has been a lot of social dilemma associated with STDs and sex thus inhibiting an open discussion about the matter in the society.

These diseases were earlier also called Venereal Diseases (VD) until the fourth world health assembly in 1975 session chose the name Sexually Transmitted Diseases (STD).

STDs are among the most common infectious diseases in the world today. More than 20 STDs have now been identified, Understanding the basic facts about STDs – the ways in which they are spreading, their common symptoms, and their treatments are the first step toward prevention. Biologists and veneralogists are looking for better methods of diagnosis and treatments, as well as for vaccines and topical microbicides to prevent STDs.

STDs affect men and women of all backgrounds and economic levels. It has been observed that STDs are most prevalent among teenagers and young adults. Nearly two-thirds of all STDs occur in people younger than 25 years of age. The reported cases of STDs continues to rise probably because sexually active people today are more likely to have multiple sex partners during their lives and are potentially at risk for developing STDs. Most of the time, STDs cause no symptoms, particularly in women. When and if symptoms develop, they are often confused with those of other diseases not transmitted through sexual contact. Even when an STD causes no symptoms, however, a person who is infected may be able to pass the disease on to a sex partner

When diagnosed and treated early, many STDs can be treated effectively. Some infections have become resistant to the drugs used to treat them and now require newer types of antibiotics. Experts believe that having STDs other than AIDS increases one's risk for becoming infected with the AIDS virus.

The best way to prevent STDs is to avoid multiple sexual contacts. Practicing safe sex is the only way to escape the diseases. The following points are important in the prevention of venereal diseases.

- Have a mutually monogamous sexual relationship with an uninfected partner.
- Correctly and consistently use of condoms.
- Use sterile needles if injecting intravenous drugs. Sharing of needles can be dangerous.
- Prevent and control other STDs to decrease susceptibility to HIV infection and to reduce your infectiousness if you are HIV-infected.
- Delay having sexual relations as long as possible. The younger people are when having sex for the first time, the more susceptible they become to developing an STD. The risk of acquiring an STD also increases with the number of partners over a lifetime.

- Have regular checkups for STDs even in the absence of symptoms, and especially if having sex with a new partner if one is sexually active.

Many a time people are too embarrassed or frightened to ask for any assistance or information pertaining to STDs. Most STDs are readily treated, and the earlier a person seeks treatment and warns sex partners about the disease, the less likely the disease will do irreparable physical damage, be spread to others or, in the case of a woman, be passed on to a newborn baby.

The following table shows almost all the sexually transmitted diseases and their causative organisms.

Srl no.	Disease	Organism(s)
1.	Acquired Immunodeficiency Syndrome (AIDS)	Human immunodeficiency virus (HIV-1 and HIV-2
2.	Bacterial vaginosis	*Bacteroides* spp. *Gardnerella vaginalis* *Mobiluncus* spp. *Mycoplasma hominis* *Ureaplasma urealyticum*
3.	Chancroid	*Haemophilus ducreyi*
4.	Chlamydial infections (PID, NGU, and LGV), Lymphogranuloma venereum (LGV)	*Chlamydia trachomatis*
5.	Cytomegalovirus infections	Cytomegalovirus
6.	Enteric infections	Various Gram negative bacteria, *Campylobacter fetus*, *Shigella*sp., *Escherichia coli*, *Salmonella sp.*
7.	Amebiasis	*Entamoeba histolytica* (protozoan)
8.	Giardiasis	*Giardia lamblia* (protozoan)
9.	Genital herpes	Herpes simplex virus
10.	Genital (venereal) warts Venereal warts or condyloma acuminata	Human papillomavirus
11.	Gonorrhea	*Neisseria gonorrhoeae*
12.	Granuloma inguinale (donovanosis)	*Calymmatobacterium granulomatis*
13.	Group B streptococcal infections	*Streptococcus agalactiae*
14.	Leukemia/Lymphoma/Myelopathy	HTLV-I and II

15.	Molluscum contagiosum	Molluscum contagiosum virus
16.	NGU or Nongonococcal urethritis	*Chlamydia trachomatis, Gardnerella vaginalis, Ureaplasma urealyticum*
17.	Pelvic Inflammatory Disease (PID)	*N. gonorrhoeae*- most common , *Chlamydia trachomatis*- most common, Anaerobic bacteria (ex. *Bacteroides*), Facultative Gram negative rods (ex. *Escherichia coli*), *Mycoplasma hominis, Actinomyces israelii*
18.	Pubic lice, Pediculosis	*Pediculus humanus* and *Phthirius pubic*
19.	Scabies	*Sarcoptes scabiei* var. hominis
20.	Syphilis	*Treponema pallidum*
21.	Trichomoniasis	*Trichomonas vaginalis*
22.	Vaginal yeast infections, Mycotic vulvovaginitis	*Candida albicans*
23.	Viral hepatitis	Hepatitis A, B, C, D viruses

Out of these twenty-three sexually transmitted diseases the most common and the most serious ones in the state are HIV, Chlamydia, Gonorrhoea, Syphilis, Genital warts and Genital herpes. These six diseases have been discussed in details in section A of this project.

For the better understanding of the diseases the project has been divided into two sections, section A and B. The disease and their organisms have been discussed in details in the first section and the second section basically involves a statistical study on the present STD scenario in the state.

Section A: Details of the disease and their causative organism, their diagnosis and treatment.

ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS)

INTRODUCTION

AIDS is a potentially lethal sexually transmitted disease and is caused by the HIV virus. The Human Immuno Deficiency Virus (HIV) is a member of the *retroviridae family* and the *Lent virus* group. Infections with HIV are manifest in immuno deficiency with associated opportunist infections and are called the Acquired Immuno Deficiency Syndrome (AIDS). The term AIDS implies to the most advance stage of an HIV infection. HIV invades and destroys the immune system, which protects the body from infection. This means that a person who carries the HIV virus is prone to many different illnesses and may die from diseases that are harmless to healthy people.

AIDS is still most widespread south of the Sahara in Africa, Asia, and the Caribbean islands, and is more common among homosexual and bisexual men. However, in more developed countries the disease is becoming more frequent among heterosexuals, especially young people.

Intravenous drug users and people with many different partners are particularly at risk from HIV. The virus is found in bodily fluids such as blood, sperm and vaginal secretions, and can pass through little scratches that may occur during sexual intercourse.

Characteristic of many retro viruses is their ability to cause malignant transformation of host cells as demonstrated with the human T-cells leukaemia virus (type 1). The syndrome first appeared in the early 1980s in certain homosexual men in the USA.A new strain appeared in mid 1980s in West Africa and the two types was classified as HIV-1 and HIV-2.

THE BIOLOGY OF HIV-1 AND HIV-2

(A) STRUCTURE:

The retro viruses are RNA containing eukaryotic viruses and HIV is one of its kinds. It is medium sized, about 100-150 nm in diameter. They contain an RNA directed DNA polymerase enzyme known as *reverse transcriptase*. Howard Temin and David Baltimore independently discovered this enzyme in 1970. *Reverse transcriptase* synthesizes DNA in the 5' to 3' direction from a RNA template hence the name '*reverse transcriptase*'.

The two strains HIV-1 & HIV-2 are structurally similar. The virus generally consists of the following two parts.

The virus consists of a **core (i.e. caspid)** composed of p24 or p25 capsid protein containing two identical RNA strands that are associated with the *reverse transcriptase* (Pol p55, p61) and the nucleocaspid RNA-binding proteins. Two other enzymes are also located in the core caspid, namely 12kDa protease and a 12kDa integrase.

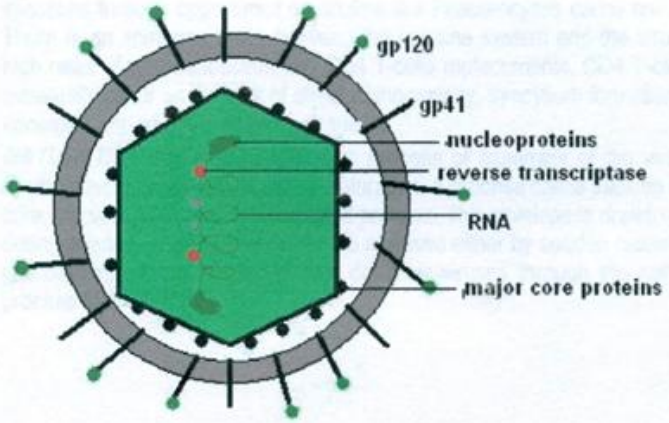
The outer membrane, usually called as the **envelope** is a complex structure consisting of a matrix protein p17, surrounded by a lipid bilayer around which is a glycoprotein coat containing gp120 'spikes' with a Tran membranous glycoprotein gp41 (gp stands for glycoprotein). The

envelope is partly derived from the host cell membrane as the virus buds through the last stages of replication. The virus expresses a number of accessory proteins products involved in up regulation of replication or infectivity. The gene product associated with Tran activation and the major proteins involved in up regulation of replication are Tat and p14.

The HIV genome is a single stranded dimeric RNA with three principal genes and long terminal repeat (LTR) regions that enable integration into the host DNA to form a provirus. The long terminal repeats contain promoter sequences. The three principal genes are

1. **Gag**: These codes for core proteins like p24 and p25.
2. **Pol**: These codes for polymerase, i.e., reverse transcriptase.
3. **Env**: These codes for envelop proteins.

The Tran activating sequence, Tat, binds to a portion on the long terminal repeats (LTR), called the Tat responsive region (TAR) to facilitate viral replication. The Rev (p19) gene enhances the production of HIV structural proteins post transcriptionally. There are two open reading frames in the HIV genome, one of which is *vif p23* (a 23 KDa cystein protein), the factor for viral infectivity and the other is nef or negative factor,a myristotlated protein which is membrane associated and is now known to deregulate the replication processes. Two other genes namely, *vpr* (p18) and *vpu* (p15, exclusively found in the genome of HIV-1) have been identified but their role is unclear. The HIV-2 type has *vpx* (p15) instead of *vpu* in their genome.



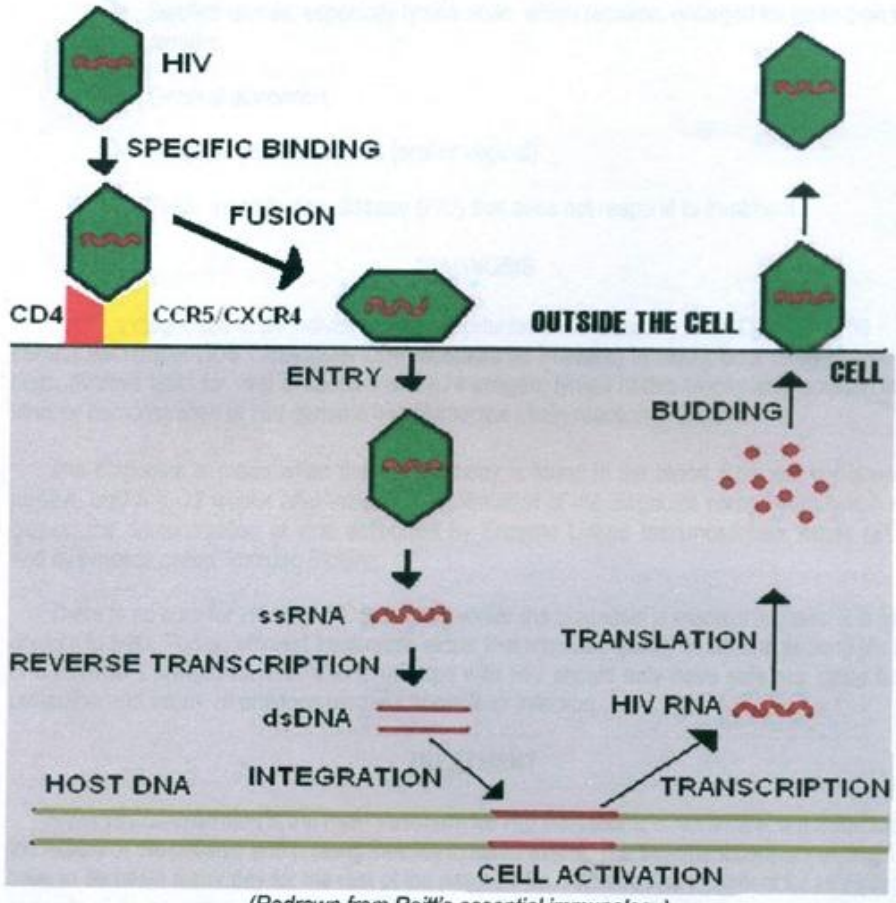
STRUCTURE OF HIV

(B)INTRACELLULAR LIFE CYCLE OF HIV (REPLICATION OF THE VIRUS)

1. ***ADSORPTION:*** The CD4 helper T-cells (thymus dependent lymphocytes) carries CD4 receptors as markers on their surface. HIV infects T-helper cells through binding of is envelope gp120 to CD4 and either CCR5 or CXCR4 chemokine receptor cofactors on the lyphocytes. The attachment process causes structural changes to occur in gp120 'spikes' that exposes cleavage sites, which can then be attacked by enzymes, causing it to split open to expose gp41. Gp41 causes fusion to take place between the viral envelop and the host cell membrane, thus allowing the core of the virus to enter the host cell. This

process is called Adsorption. It also infects macrophages, microglia, T-cells stimulating dendritic cells and FDCs, the latter through a CD4-independent pathway.

2. ***PENETRATION:*** Now the virus enters the cell either by invaginal of the cell membrane round the virus particle (PHAGOCYTOSIS) or by the fusion of the viral envelop and the cell membrane as described above.
3. ***UNCOATING:*** The viral particle entres the hose cell along with the core. Thus to make the genetic material material free from the core and functional, uncoating takes place. The uncoating of the viral genome takes place and the viral genome lies exposed in the cytoplasm.
4. ***VIRAL SYNTHESIS:*** In the retro viruses the RNA do not enter the translation stage directly but within the cell, the viral RNA is converted by *reverse transcriptase* to DNA, which in turn generates a messenger RNA that enters in translation. This process is called **reverse transcription**. The DNA produced in the reverse transcription stage can be incorporated into the host genomes and is called as the provirus, where it lies dormant until the cell is activated by stimulators such as TNF (tumor necrosis factor). There is usually a long asymptomatic phase after the early acute viral infection has been curtailed by a CD8 CTL immune response and the virus is sequestered to the FDC in the lymphoid follicles where it is progressively destroys the dendritic cell meshwork. The disastrous fall in CD4 T-helpers destroys cell defenses and leaves the patients open to life threatening infections through opportunist organisms like *Pneumocystis carinii* and cytomegalovirus. There is an immense battle between the immune system and the virus, with extremely high rates of viral destruction and CD4 T-cells replacements. CD4 T-cells depletion may eventually occur as a result of direct pathogenicity, syncytium formation, susceptibility to apoptosis and possibly other mechanisms.
5. ***MATURATION AND RELEASE:*** The process of assembly of the virus from its newly synthesised components id called maturation. A process called packing assembles all the core, the viral RNA and its associated proteins. The envelope is drawn out of the host cell during release. The daughter cells are released either by sudden rupture of the cell or by gradual extrusion of the enveloped daughter viruses through the cell membrane by a process called budding.



(Redrawn from Roitt's essential immunology)

Highly active antiretroviral drug therapy (HAART), combining inhibitors of *reverse transcriptase* and protease, can eliminate detectable virus early in disease, although latent virus remains. Vaccines (AIDSVAX) are being targeted to Th1 responses but it is a very difficult virus to control.

SYMPTOMS

The symptoms of HIV/AIDS can be summarised as follows:

- Fever
- Diarrhoea
- Sweating at night
- Loss of weight

- Swollen glands, especially lymph node, which remains, enlarged for more than three months.
- General discomfort.
- Frequent yeast infections (oral or vaginal).
- Pelvic inflammatory disease (PID) that does not respond to treatment.

DIAGNOSIS

AIDS is diagnosed in an individual with opportunistic infections, by low CD4(below 200 × 10^6 cells/L) but normal CD8 T-cells(with CD8 receptors as markers) in blood, poor delayed-type skin tests, positive tests for viral antibodies and p24 antigen, lymph nodes biopsy and isolation of live virus or demonstration of HIV genome by polymerase chain reaction (PCR).

The diagnosis is made when the HIV antibody is found in the blood. The test is not usually positive until 6 to 12 weeks after infection. Confirmation of the diagnosis comes from lymph node biopsy, the demonstration of viral antibodies by Enzyme Linked Immunosorbent Assay (ELISA) and by process called 'Immuno Blotting'.

There is no cure for HIV and AIDS, but the earlier the diagnosis is made, the easier it is for the doctors to help. Today, efficient treatments exists that increase quality of life and prolong life itself of a HIV/AIDS patient. Anyone who is infected with HIV should only have safe sex using barrier protection and inform all previous partners about their infection.

TREATMENT

Antiretroviral treatment is the main treatment for HIV infection. It is not a cure, but it can reduce the misery of the patients and prolong their life to some extent. The treatment consists of drugs that have to be taken every day for the rest of the patient's life. Antiretroviral treatment for HIV infection consists of drugs, which work against HIV infection itself by slowing down the reproduction of HIV in the body. The drugs are often also referred as:

- **Antiretroviral**
- **Anti-HIV drugs**
- **HIV antiviral drugs**

For antiretroviral treatment to be effective for a long time, it has been found that more than one antiretroviral drug must be used at a time so that the virus does not develop resistance for one particular drug. This is what is known as Combination Therapy. The term Highly Active Antiretroviral Therapy (HAART) is used to describe a combination of three or more anti-HIV drugs. Since HIV infection weakens the body defence system treatment must also be provided for Opportunistic Infections.

Twenty drugs have been approved by the Food and Drug Administration (FDA) to treat individuals infected with HIV. After infection HIV requires specific enzymes to grow and multiply. These drugs fall into three categories.

1. **Reverse transcriptase (RT) inhibitors**: HIV needs an enzyme called reverse transcriptase to make copies of it. Reverse transcriptase inhibitors interfere with the production of the enzyme and hence restrict the multiplication of the virus. There are two main types of RT inhibitors, and they each work differently.

   - **Nucleoside/nucleotide drugs** provide faulty DNA building blocks, halting the DNA chain that the virus uses to make copies of it. These drugs include abacavir, zalcitabine[2] (ddC), tenofovir, stavudine[1] (d4T), zidovudine (ZDV), lamivudine (3TC) and didanosine (ddI).

   - **Non-nucleoside RT inhibitors** bind to reverse transcriptase so the virus cannot carry out its copying function. Drugs like Delavirdine, Nevirapine and Efavirenz fall under this category.

2. **Protease inhibitors (PI):** Protease inhibitors interfere with the protease enzyme that HIV uses to produce infectious viral particles. Protease inhibitor drugs includes drugs like Ritonavir, Saquinavir, Indinavir, Amprenavir, Fosamprenavir Calcium, Lopinavir, Atazanavir, Entricitabin and Nelfinavir

Fusion inhibitors: This class of antiretroviral drugs works by changing the shape of the viral envelope. Fusion inhibitors interfere with the virus' ability to fuse with the cellular membrane, thereby blocking entry into the host cell. This is the latest class of antiretroviral drugs. Example: Enfuvirtide. One of these drugs - commonly called T-20 - has been licensed both in the US and in Europe since 2003, but only for use by people who have already tried other treatments.

There is no cure for AIDS the only way is prevention. The use of condoms is the most appropriate way to escape the disease. NGOs and professional social can play a major role in the control of the disease by raising awareness amongst the masses.

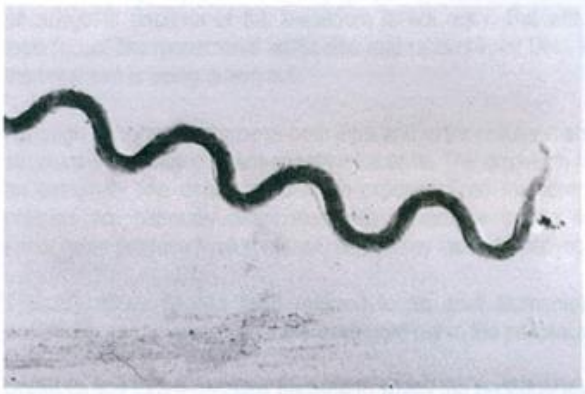
SYPHILIS

INTRODUCTION

Syphilis is caused by a spirochete called *Treponema pallidum*. *Treponema* is a Gram-negative like, thin, motile, spiral shaped bacterium belonging to the order *Spirochaetales*. *Schaudinn discovered T. Pallidium* in 1905. The name spirochete is derived from the Greek words meaning, "coiled hair." Syphilis was an epidemic in late Fifteenth century Europe. In India the disease is believed to be

brought in by the people of Portuguese expedition to India and is locally called as the '*feranga roga*'. Before the advancement of AIDS syphilis was considered to be the most serious sexually transmitted diseases.

Until the late nineties syphilis and gonorrhoea was considered to be the manifestation of the same disease. Only in 1837 an eminent French Venereologist Phillip Ricord discovered the specificity of the two diseases through a series of experimental inoculations from syphilitic chancres. Ricord was also among the first physicians to differentiate primary, secondary, and tertiary syphilis, the three stages of infection.



The spirochete *Treponema pallidum*

Syphilis results in the formation of lesions throughout the body. *Treponema pallidum* may also pass from an infected pregnant woman across the placenta to the developing foetus resulting in congenital syphilis. Hence, the disease is passed on through infectious blood, mucosal or sexual contact. *T. pallidum* attacks the interstitial spaces of tissue at the site of infection and moves rapidly to other locations. Syphilis has three distinct stages and a latency stage between the second and third stages, during all of which the principal pathogen is *T. pallidum*.

THE BIOLOGY OF SPIROCHETE *Treponema pallidum*

(A)STRUCTURE

Treponemes are helically coiled, corkscrew-shaped cells, 6 to 15 µm long and 0.1 to 0.2 µm wide. They have an outer membrane, which surrounds the periplasmic flagella, a peptidoglycan-cytoplasmic membrane complex and a protoplasmic cylinder. The presence of peptidoglycan in the cell wall has been confirmed by biochemical analysis Multiplication is usually by binary transverse fission. Treponemes have not yet been cultured in vitro. Owing to is extremely small size a dark

field microscope is used to observe the bacterium. The spirochetes are able to swim in viscous environments (e.g. oral cavity, intestinal tract). *T. Palladium* has a "cork-screw" mechanism for its motility, which allows it to carve its way through the tissues. It exhibits characteristic motility that consists of rapid rotation about its longitudinal axis and bending, flexing, and snapping about its full length

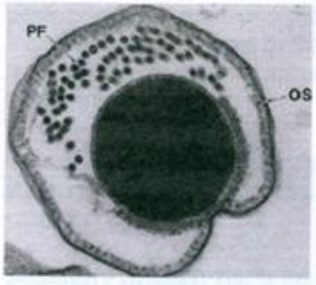
The dry weight composition of *T pallidum* is approximately 70 percent proteins, 20 percent lipids, and 5 percent carbohydrates. The lipid composition of *T pallidum* is complex, consisting of several phospholipids, including cardiolipin, and a poorly characterized glycolipid which is bio chemically and immunologically distinct from lipopolysaccharide. Due to the inability to grow *T pallidum* in vitro an antigenic analysis of the bacterium is not easy. But with the accent of modern molecular techniques, like monoclonal antibodies and recombinant DNA technology much information about the organism is being drawn out.

Although treponemes possess both intra and extra cellular membranes, they differ considerably in structure from enteric Gram-negative bacteria. The organism has an outer membrane containing an extremely low density of surface-exposed Tran membrane proteins. The outer membrane proteins are generally associated with adherence to the surface of host cells and virulent spirochetes produce hyaluronidase, which may facilitate perivascular infiltration.

Typically, three flagella (also referred to as axial filaments) originate from each end of the bacterium, and, winding about the bacterium within the periplasmic space, overlap at the midpoint.

Unlike typical Gram-negative bacteria in which the peptidoglycan layer is present under the outer membrane, in treponemes the murein (type of peptidoglycan unique to bacteria) layer overlies the cytoplasmic membrane. The cytoplasmic membrane covers the protoplasmic cylinder and contains the majority of the bacterium's integral membrane proteins and possesses higher amounts of lipid-modified polypeptides (lipoproteins).

T pallidum can survive in the host tissue. The basic reason is that virulent spirochetes are coated with host cell fibronectin, which protects it against phagocytosis. During the course of infection, antibodies develop to a number of treponemal proteins especially the lipoproteins and flagella. Several gene products are known to be associated with virulent strains of *T.pallidum* although their role in their virulence is still not clearly understood.



Cross section of a spirochete PF=periplasmic flagella OS=outer sheath

(B)STAGES OF INFECTION OF SYPHILIS

T.pallidum enters the body during sexual intercourse through mucous membranes possibly through microscopic abrasions and quickly spreads via blood and the lymphatic system of the body. Moisture is essential for this organism to survive and thus is usually found in the genitals, mouth of the anal regions. Multiplications of the organism occur at the site of entry resulting in a single, painless papule, the primary chancre. *T. pallidum* attacks the interstitial spaces of tissue at the site of infection and moves rapidly to other locations. Following inoculation, *T. pallidium* penetrates intact mucous membranes or broken skin, begins to divide slowly and disseminates. The spirochete basically divides by the process of binary transverse fission.

The primary chancre consists of an inflammatory response characterised by the presence of lymphocytes, monocytes, plasma cells and some macrophages. Meta chromatic acid mucopolysaccharides accumulate at the site of the chancre, which gives the lesions their characteristic firmness. The mucopolysaccharides are in parts derived from the capsules of the organism and in parts from the host tissue constituents. The mucopolysaccharides (like fibronectin) readily adhere to the surface of the organism thus enabling the organism to avoid antibody recognition. *T. palladium* also produces an enzyme called mucopolysaccharidade that can degrade cell surface mucopolysaccharides. By the action of this enzyme the mucopolysaccharides that join capillary epithelial cells are degraded thus paving the way for the organism to the perivascular area. Further degradation of the mucopolysaccharides in the blood vessels may result for the obliterative endarteritis characteristic of syphilis.

As already stated earlier the disease basically has three stages: the primary stage, the secondary stage and the tertiary stage. There is a latency stage between the secondary and the tertiary stage when the spirochete is "inactive". These stages are described below in details.

Primary Syphilis: This is the first stage of syphilis. The first symptoms appears after 10-60 days of exposure to *T. palladium*. The area of infection is marked by the appearance of a hard, prominent, round, ulcerous development called a "chancre" which in French means broken sore. The chancre usually appears on the penis, labia, cervix, anorectal region or around the mouth and is the site of primary infections. It is also possible that chancre may develop anywhere on the skin exposed to *T. palladium*. The chancre may heal within 4-6 weeks, even without treatment, but may leave a scar. The healing probably depends on the hormonal and the cell-mediated immunity. The lymph nodes also become inflamed. Histological examination of the lesions reveals an inflammation of the inner wall of an artery (endarteritis) and membranous sac surrounding the heart (periarteritis), characteristic of syphilitic lesions at all stages. The spirochete is susceptible to antibiotics and to phagocytosis by the polymorphonuclear leukocytes, macrophages and activated T-lymphocytes. Ingestion of the spirochete by the phagocytic cells is often seen, but the organisms survive due to the fibronectin coat on the spirochetes, derived from the host cells, which protects the organism from phagocytosis.

A small number of the organisms are sequestered intercellularly and this may explain the chronic and relapsing nature of syphilis. If syphilis is not treated early in this stage, it will progress into secondary syphilis.

Secondary Syphilis: This is the second stage of the disease characterised by appearance of red rashes on the skin. It is remarkable that these rashes appear soon after the host defence system

has controlled the primary lesions. After about 1-6 weeks of healing of the chancre pale red rashes appears usually on the palms or soles of feet, but may occur over the entire body especially on the penis, labia and anus and sometimes on the mouth and lips also. A fever, sore throat, headaches, joint pains, poor appetite, weight loss, muscular fatigue and hair loss may accompany this rash. Sores around the genitals or anus secrete extremely infectious fluids. These symptoms usually last for 3-6 months and then may re-appear at any time.

The Latency Stage: In this stage the spirochete is said to be "inactive" or "resting" and shows no obvious symptoms of the disease, yet *T. pallidium* is still present in the host's body, lodging itself into the host's tissue. The latency stage can last for a few months or even for the lifetime of the host. If tested the infected person tests positive for the disease. The infected individual is not infectious in this stage but there is a possibility of spreading the disease to those in sexual contact with him/her. Approximately 50-70% of carriers in this stage will not progress to the next stage i.e. tertiary syphilis.

Tertiary Syphilis: This is the most serious stage of syphilis and is often fatal as the spirochete invades the host nervous system, characteristic to this stage. Death in adults is due to the variable occurrence of later complications of syphilis in the skin, bones, central nervous system, heart and blood vessels. An unusual feature of this stage is the occurrence of Gummata lesions in the spleen, skin, liver or bones and they frequently lead to destruction of soft tissue or bone if not treated on time. Lesions of late syphilis sometimes occur for an extended period from as few as 2 to over 40 years from onset of infection. Gummas of critical organs, like the heart, brain and liver, can be fatal. This stage of syphilis can lead to cardiovascular syphilis, neurosyphilis or death.

Congenital Syphilis: It is generally rare for bacteria to cross the placental barrier and infect the foetus, *T. palladium* being an exception. Transmission of *T. palladium* from the infected mother to the foetus can take place from the tenth week to late pregnancy. Foetal infection is usually seen if the mother is suffering from the primary or the secondary stage of the disease. Approximately 50 percent of foetuses are aborted or stillborn; the rest exhibit diverse syphilitic stigmata.

SYMPTOMS

The symptoms of syphilis are divided into three stages keeping in view the three stages of the disease. Up to 12 weeks after the time of infection (primary syphilis):

- One or more red lesions (chancre) will develop on the penis, labia (lips of the vagina), and anus and sometimes on the mouth and lips. These lesions disappear after a week.

Up to six months after the time of infection (secondary syphilis):

- A red rash appears on the chest, back, arms, legs, hands and soles of the feet
- High fever

- Sore throat
- Muscular fatigue
- General feeling of discomfort.

If the illness is not treated by the second stage, it will disappear for a while. However, the disease can lie dormant in the body and return up to 20 years or more. At this more advanced stage of the disease (tertiary syphilis) and the symptoms will be:

- Heart failure
- Neurovascular diseases
- Paralysis
- Insanity
- Possible death.

DIAGNOSIS

Serological testing makes the diagnosis of syphilis in most patients. The two general types of tests are biologically non-specific (nontreponemal) tests and the specific treponemal tests.

- **Nontreponemal tests** measure IgG and IgM antibodies (reagin antibodies) developed against lipids from damaged cells during the early stage of the disease. The antigen used for the nontreponemal tests is **Cardiolipin-Lecithin-Cholesterol antigen** which is not derived from spirochaetes but from beef's heart. The two tests used commonly are the Venereal Disease Research Laboratory (VDRL) test and the Rapid Plasma Reagin (RPR) test. Both tests measure coagulation of cardiolipin-Lecithin-cholesterol antigen by the patient's serum. Both tests are rapid, although complement in serum must be inactivated for 30 minutes before the VDRL test can be performed. Only the VDRL test can be used to test cerebrospinal fluid from patients with suspected neurosyphilis. Another nontreponemal test called the automated resin test (ART) can also be used for diagnosis

- **Treponemal tests** are specific antibody tests used to confirm positive reactions with the VDRL or RPR tests. The treponemal tests can be positive before the non treponemal tests become positive in early syphilis. Treponemal test may also remain positive when the non-specific tests revert to negative in some patients who have late syphilis. The tests most commonly used are Fluorescent Treponemal Antibody Absorption (FTA-ABS) test and the Microhemagglutination Test for *T. pallidum* (MHA-TP). The MHA-TP is technically easier to perform and interpret than the FTA-ABS tests.

TREATMENT

Long doses of Penicillin are used for untreated infections with *T. pallidium* for about 21 to 30 days. Long-acting benzathine penicillin is used for the early stages of syphilis, and penicillin G is recommended for congenital and late syphilis. Tetracycline, erythromycin, and chloramphenicol can be used as alternative antibiotics for patients allergic to penicillin. Only penicillin or chloramphenicol can be used for patients with neurosyphilis.

GONORRHOEA

INTRODUCTION

The disease gonorrhoea is a specific type of urethritis caused by gonococci called *Neisseria gonorrhoea* and practically always involves mucous membranes of the urethra, resulting in a copious discharge of pus, more apparent in the male than in the female.

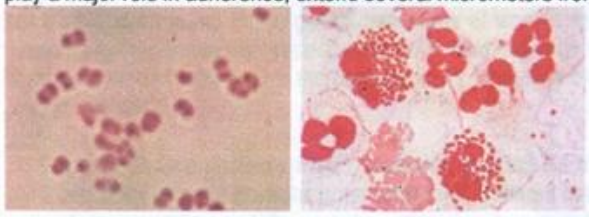
Galen in the second century implied a "flow of seed" first used the term "gonorrhoea". For centuries thereafter, gonorrhoea and syphilis were often confused to be the same, resulting from the fact that the two diseases were often present together in infected individuals. Paracelsus (1530) thought that gonorrhoea was an early symptom of syphilis.

A. Neisser first described the causative agent of gonorrhoea, Neisseria gonorrhoea, in 1879 in the pustular exudates of a case of gonorrhoea. The organism was grown in pure culture in 1885, and its etiological relationship to human disease was later established to fulfil the experimental requirements of Koch's postulates. In the vocabulary of the public health and medical microbiologist, N. *gonorrhoea* is often referred to as the "gonococcus" and the infection as gonococcal infection.

BIOLOGY OF *Neisseria gonorrhoea*

(A)STRUCTURE

Neisseria gonorrhoea is a Gram-negative coccus, about 0.6 to 1.0 µm in diameter. It usually seen in pair with adjacent flattened sides. The organism is frequently found intracellularly in polymorphonuclear leukocytes (neutrophils) of the gonorrhoea postural exudates. Fimbriae, which play a major role in adherence, extend several micrometers from the cell surface.



Left: *Neisseria gonorrhoeae* Gram stain of pure culture; **Right: *Neisseria gonorrhoeae*** Gram stain of pustular exudates. **SOURCE**: Todar's Online Textbook of Bacteriology.

A typical gram-negative bacterial cell wall consists of two layers: the outer membranes comprising of proteins, lipids and carbohydrates and the inner ridged peptidoglycan layer. The peptidoglycan layer is thinner in gram-negative bacteria and the outer layer has high density lipopolysaccharides (LSP).
The LPS are important for a number of regions. Their lipid portions lipid A is a highly reactive molecule and is responsible for the endotoxic shocks if humans are exposed to it. Also every lipopolysaccharide has a side chain, the O side chain that is important for antigen identification by the immune system.
Neisseria gonorrhoea possesses a typical Gram-negative outer membrane composed of proteins, phospholipids, and lipopolysaccharide (LPS). However, neisserial lipopolysaccharide is distinguished from enteric lipopolysaccharide by its highly-branched basal oligosaccharide structure and the absence of repeating O side chain. For these reasons, neisserial LPS is referred to as lipooligosaccharide (LOS). The bacterium characteristically releases outer membrane fragments called "blebs" during their growth period. These blebs contain lipooligosaccharide and probably have a role in pathogenesis if they are disseminated during the course of an infection.

A major porin protein, **P.I (Por),** in the outer membrane of the bacterium is thought to be the mediator for the penetration of a host cell. Each *N. gonorrhoea* strain expresses only one type of Por; however, there are several variations of Por that partly account for different antigenic types of the bacterium.

An Opa (P.II) protein is another outer membrane proteins found in *N. gonorrhoea*. These proteins are subject to phase variation and are usually found on cells from colonies possessing a unique opaque phenotype called **O+**. At any particular time, the bacterium may express zero, one, or several different Opa proteins, and each strain has 10 or more genes for different Opa proteins.

Rmp (P.III) is another outer membrane protein characteristic to all strains of the bacterium. It does not undergo antigenic variation and is found in a complex with Por and LOS. It shares partial homology with the OmpA protein of *Escherichia coli.* Antibodies to Rmp, induced either by a neisserial infection or by colonization with *E. coli,* tend to block bactericidal antibodies directed against Por and LOS. In fact, anti-Rmp antibodies may increase susceptibility to infection by *N. gonorrhoea.*

Strains of *N. gonorrhoea* produce two distinct extra cellular IgA1 proteases, which cleave the heavy chain of the human immunoglobulin at different points within the hinge region. Split products of IgA1 have been found in the genital secretions of women with gonorrhoea, suggesting that the bacterial IgA1 protease is present and active during genital infection. It is thought that the Fab fragments of IgA1 may bind to the bacterial cell surface and block the Fc-mediated functions other immunoglobulins.

N. Gonorrhoea is a relatively fragile organism, susceptible to temperature changes, drying, UV light, and other extreme environmental conditions. Different strains of *N. gonorrhoea* have different cultural requirements and thus media containing haemoglobin, NAD, yeast extract and other supplements are needed for isolation and growth of the organism. Cultures are grown at 35-36 degrees in an atmosphere of 3-10% added CO_2.

(B)MODE OF INFECTION

Gonorrhoea in adults is almost completely transmitted by sexual intercourse. Gonorrhoeal infection is generally limited to superficial mucosal surfaces lined with columnar epithelium. The areas most frequently involved are the urethra, cervix, rectum, pharynx, and conjunctiva. Squamous epithelium, which lines the adult vagina, is not susceptible to infection by the *N. gonorrhoea.* However, the pre pubescent vaginal epithelium, which has not been keratinized under the influence of estrogen, may be infected. Hence, gonorrhoea in young girls may present as vulvovaginitis. Mucosal infections are usually characterized by a purulent discharge.

The pathogenic mechanism basically involves the attachment of the bacterium to nonciliated epithelial cells via Pili (fimbriae) and the production of lipopolysaccharide endotoxin. The bacteria attaches to columnar epithelial cells, penetrate them, and multiply on the basement membrane. Attachment to the epithelial cells is brought about by the action of fimbriae and an outer membrane protein called Opa (P.II) proteins, although non-specific factors such as surface charge and hydrophobicity may play a role. *Neisseria gonorrhoea* can produce one or more Opa (P.II) proteins.

The bacteria attach specifically to microvilli of nonciliated columnar epithelial cells and attachment to ciliated cells does not occur.

After the bacteria attach to the nonciliated epithelial cells of the fallopian tube, they are surrounded by the microvilli, which draw them to the surface of the mucosal cell. The bacteria enter the epithelial cells by a process called Parasite-Directed Endocytosis (PDE). During endocytosis the membrane of the mucosal cell retracts and pinches off a membrane-bound vacuole that contains the bacteria. The vacuole is transported to the base of the cell where the bacteria are released by exocytosis into the sub epithelial tissue. The *Neisseria* are not destroyed within the endocytic vacuole, but it is not yet clear if they actually replicate in the vacuoles as intracellular parasites or not.

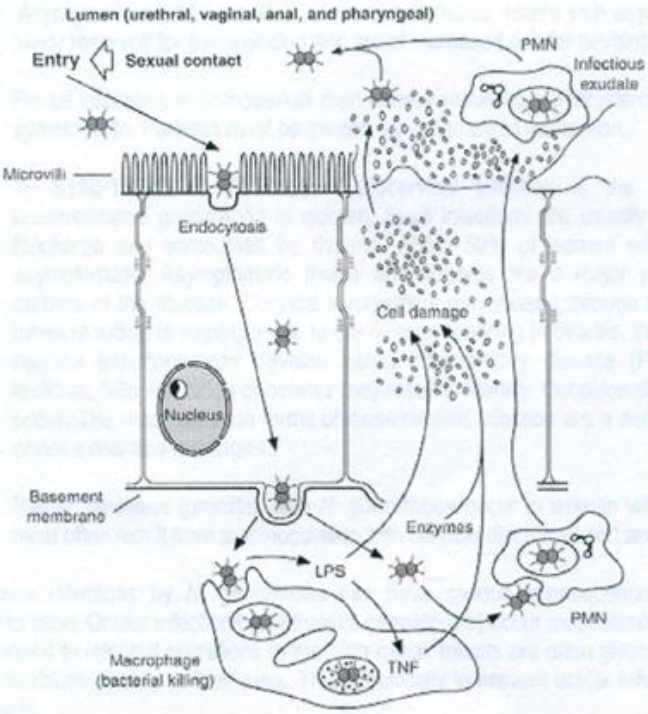
During infection, bacterial lipooligosaccharide (LOS) and peptidoglycan are released by the autolysis of cells. Both bacterial polysaccharides activate the host alternative complement pathway, while LOS also stimulates the production of tumour necrosis factor (TNF) that causes cell damage. Neutrophils are immediately attracted to the site and feed on the bacteria. For unknown reasons, many gonococci are able to survive inside of the phagocytes, at least until the neutrophils themselves die and release the ingested bacteria.

Neisserial LOS has an intense effect on the virulence and pathogenesis of *N. gonorrhoea.* The bacteria can express several antigenic types of LOS and can alter the type of LOS they express by an unknown mechanism. Gonococcal LOS produces mucosal damage in fallopian tube organ cultures and brings about the release of enzymes, such as proteases and phospholipases, which may be important in pathogenesis. Thus, gonococcal LOS appears to have an indirect role in mediating tissue damage. Gonococcal LOS is also involved in the resistance of *N. gonorrhoea* to the bactericidal activity of normal human serum. Specific LOS oligosaccharide types are known to be associated with serum-resistant phenotypes of *N. gonorrhoea.*

N. Gonorrhoea can utilize host-derived N-acetylneuraminic acid (sialic acid) to sialylate the oligosaccharide component of its LOS, converting a serum-sensitive organism to a serum-resistant one. Organisms with nonsialylated LOS are more infective than those with sialylated LOS but the latter are more resistant to bactericidal effects of serum. There is also antigenic similarity between neisserial LOS and antigens present on human erthyrocytes. This similarity may preclude an effective immune response to these LOS antigens by maintaining the immunotolerance of the host.

N. Gonorrhoea is highly efficient at utilizing transferrin-bound iron for in vitro growth; many strains can also utilize lactoferrin-bound iron. The bacteria bind only human transferrin and lactoferrin. This specificity is thought to be a reason for *N. gonorrhoea* being an exclusively human pathogen.

Occasionally *Neisseria gonorrhoea* enters the bloodstream causing a Gram-negative bacteraemia, which may lead to a disseminated bacterial infection. Asymptomatic infections of the urethra or cervix usually serve as focal sources for bacteraemia. Strains of *N. gonorrhoea* that causes disseminated infections are usually resistant to complement and the serum bactericidal reaction. This accounts for their ability to persist in the bacteraemia. In Gram-negative bacteraemia of this sort, the effect of bacterial endotoxin can be exacerbated by the lyis of bacterial cells that may simply liberate soluble LPS.



Pathogenesis of uncomplicated gonorrhea according to Morse in Baron, Chapter 14, Neisseria, Branhamella, Moraxella and Eikenella. SOURCE: Todar's Online Textbook of Bacteriology

SYMPTOMS

The early symptoms of gonorrhoea are often mild, and many women who are infected have no visible symptoms of the disease. If symptoms of gonorrhoea develop, they usually appear within 2 to 10 days after sexual contact with an infected partner, although a small percentage of patients may be infected for several months without showing symptoms.

- **SYMPTOMS IN MALE:** Uncomplicated gonorrhoea in the adult male is an inflammatory and pyogenic infection of the mucous membranes of the anterior urethra. The most common symptom is a discharge that may range from a scanty, clear or cloudy fluid to one that is copious and purulent. Dysuria (difficulty in urination) is often present. Inflammation of the urethral tissues results in the characteristic redness, swelling, heat, and pain in the region. There is intense burning and pain during urination. The organism may also invade the prostate resulting in prostatitis, or extend to the testicles resulting in orchitis

Asymptomatic infections also may occur in males. Males with asymptomatic urethritis are a major reservoir for transmission and are at increased risk for developing complications.

Rectal infections in homosexual men usually result from anal intercourse and are more often symptomatic. Partners must be treated as well to avoid reinfection.

- **SYMPTOMS IN FEMALE:** Endocervical infection is the most common form of uncomplicated gonorrhoea in women. Such infections are usually characterized by vaginal discharge and sometimes by dysuria. About 50% of women with cervical infections are asymptomatic. Asymptomatic males and females are a major problem as unrecognized carriers of the disease. Cervical involvement may extend through the uterus to the fallopian tubes resulting in salpingitis, or to the ovaries resulting in ovaritis. Women with uncomplicated cervical infections may develop pelvic inflammatory disease (PID). The involvement of testicles, fallopian tubes or ovaries may result in sterility. Occasionally, disseminated infections occur. The most common forms of disseminated infection are a dermatitis-arthritis syndrome, endocarditis and meningitis.

Rectal infections (proctitis) with *N. gonorrhoea* occur in women with cervical infection. They most often result from autoinoculation with cervical discharge and are rarely symptomatic.

Ocular infections by *N. gonorrhoea* can have serious consequences of corneal scarring or perforation. Ocular infections (ophthalmia neonatorum) occur most commonly in newborns that are exposed to infected secretions in the birth canal. Infants are often given a dose of silver nitrate or an antibiotic directly to their eyes. This is basically to prevent ocular infection by *N. gonorrhoea* in infants.

Other symptoms affecting the rectal area include itching, discharge and sometimes-painful bowel movements.

DIAGNOSIS

A diagnosis is made through detection of bacteria in samples taken from the urethra, cervix, throat or rectum. The sample is examined for typical gram-negative diplococci .As with Chlamydia, further testing is recommended once treatment has ended to check whether the infection has cleared.

TREATMENT

The recommended treatment for uncomplicated infections is a third-generation cephalosporin or a fluoroquinolone plus an antibiotic (e.g., doxycycline or erythromycin) effective against possible coinfection with *Chlamydia trachomatis*. Sex partners should also be referred and treated. The current CDC Treatment Guidelines recommend treatment of all gonococcal infections with antibiotic regimens effective against resistant strains like penicillinase-producing N. gonorrhoea (PPGN). The recommended antimicrobial agents are ceftriaxone, cefixime, ciprofloxacin, or oflaxacin. There is no effective vaccine to prevent gonorrhoea. Candidate vaccines consisting of PilE protein or Por are of little benefit. The development of an effective vaccine has been hampered by the lack of a suitable animal model and the fact that an effective immune response has never been demonstrated. The discovery of various antibiotics (penicillin, tetracycline, erythromycin, and cefoxitin) resistant strains of the cocci has made the development of a universal vaccine more difficult. Condoms are effective in preventing the transmission of gonorrhoea.

HERPES GENITALIS (GENITAL HERPES)

INTRODUCTION

Genital herpes is a highly contagious viral condition caused by the Herpes simplex virus (HSV). Herpes is transmitted through direct skin-to-skin contact. This occurs when a contagious area comes into contact with a mucous membrane, primarily the mouth and genitals. It principally infects the skin and mucous membranes of the genitals and rectum, but can also appear in areas such as the mouth. It is transmitted primarily through physical and sexual contact. During birth, the presence of herpes simplex virus on the genitalia or in the birth canal is a threat to the infant. Infection in the newborn infant can lead to herpetic meningitis, herpetic viremia (herpes virus particles present in the blood) and chronic skin infection.

BIOLOGY OF Herpes Simplex Virus (HSV)

(A)STRUCTURE

There are two serotypes of HSV namely HSV-1 and HSV-2, which are typically associated with facial and genital lesions respectively. Both the types of HSV share group specific antigen but can

be differentiated by testing for type specific antigen and by DNA restrictions enzyme analysis. The virus structurally can be divided into three parts as follows.

THE CASPID: The HSV virion has a characteristic icosahedral caspid containing the genetic material, i.e. DNA. The viral genome is a double stranded DNA molecule the sequence of which has been determined. The genome contains a total of 73 genes out of which 38 are known to be non-essential for the virus growth in tissue cultures although some are likely to be important in the viral life cycle in humans. The sizable number of non-essential genes provides many potential target sites for the insertion of foreign genes thus facilitating recombinants.

THE ENVELOPE: The virion possesses a glycoprotein containing lipid envelope. The HSV envelope contains at least 8 glycoproteins.

THE TEGUMENT: An amorphous proteineaceous layer connects the viral envelope and the caspid and is called the matrix or tegument. The tegument contains at least 15-20 proteins.

(B) LIFE CYCLE OF THE VIRUS

Virus entry requires sequential interaction between specific viral membrane glycoproteins and cellular receptors. Upon penetration the nucleocapsid is transported to the nuclear pores and the viral DNA is released into the nucleus where the uncoating of the viral genome takes place. The α-TIF protein that functions in enhancing immediate early viral transcription via cellular transcription factors accompanies the viral genome. The earliest changes the host cell nucleus includes the peripheral clumping of the chromatin and a homogeneous ground glass appearance combined with an inflation of the nucleus.

The viral genome replication basically is of two phase's namely lytic and latent phase. In the lytic phase the replication of the virus will start immediately after infection where as in latent phage the viral genome gets integrated with the host genome to form a prophage. The prophage can stay unexpressed for many generations but can suddenly be active and will go into the replication phage.

Viral transcription takes place in three board temporal classes; immediate early, early and late. Immediate early transcription takes place in the absence of *de novo* protein synthesis and several immediate early proteins have been shown to act as activators for early and late transcription. Early mRNAs are generally considered to be those, which do not require viral DNA replication for their expression while replication of the DNA is must for the regulation of late mRNA synthesis. The early functions are basically for viral replication and the late functions are predominantly structural.

Caspid assembly and DNA packing takes place in the host cell nucleus and the virion acquires tegument and envelop as it passes out of the nucleus and through the nucleus. The process of exocytosis, which does not involve the lysis of the host cell, releases the virus.

The assembly and the maturation processes of the herpes viruses are complex processes. Mature capsids bud through the inner nuclear membrane that contains viral glycoproteins. In the early maturation process in the nucleus, capsids appear to be surrounded by the primary tegument protein, U_L 31 and this directs the budding through the inner nuclear membrane into which the U_L31 and U_L34 phosphorylated membrane protein has been inserted. These primarily enveloped capsids then bud through the outer nuclear membrane where the primary envelope is lost. The cytoplasmic capsids then associate with the numerous tegument proteins of the mature virion, including a-TIF and *vhs*, which appear to functionally interact to help final envelopment. Final envelopment takes place as the mature capsids and associated tegument proteins bud into

exocytotic vesicles, the membranes of which contain all the glycoproteins associated with the mature virions. Infectious virions can either remains cell associated within these vesicles, and spread to uninfected cells via virus-induced fusion, or can be released from the cell in exocytotic vesicles.

Latent infections are the distinguishing features of the herpes virus and various cell types can harbour latent viral genomes. The site of HSV primarily is the neurons of sensory ganglia although cases are seen where the site of HSV latency is the central nervous system (CNS) and certain non-neuronal tissues. Following infections the virus travels up the nerve to the ganglia where a limited lytic infection may occur before latency is established. During latency lytic genes transcription stops and only one viral promoter remains active, generating a number of RNAs which has been termed as the latency-associated transcripts (LATs) and its function remains unknown though the use of LAT promoter is considered important for the potential use of HSV-1 vectors in neurons. HSV can remain latent within the sensory ganglia for the lifetime of the host cell. The initial and final stages of latent infections resemble those of lytic infections and the latent phase can be thought of as the prolonged and sometimes indefinite suspension of the infection process.

STMPTOMS

The symptoms of herpes simplex virus usually occur a week after infection, but sometimes take longer to appear. Initially, the skin becomes reddened and multiple small blisters filled with a clear, straw-coloured fluid appear. Prior to the presence of blisters, the infected individual may also experience increased skin sensitivity, tingling, burning or pain at the site where blisters will appear. Later, the blisters burst leaving shallow, painful ulcers, which eventually scab and heal over a period of 7 to 14 days. The symptoms of the disease can be summed up as follows:

- Swelling and tenderness of the lymph nodes in the groin area.
- In women, vaginal discharge and painful urination.
- In men, possibilities of painful urination if the lesion is near the opening of the urethra.
- Flu-like symptoms (headache, fever)

DIAGNOSIS

Herpes can be diagnosed by examination and appearance of the lesions in most cases. There are two main laboratory methods to diagnose the virus: a culture and a blood test.

A swab of an open lesion is needed for the culture. The sample is sent to a lab to be grown in a Petri dish. Typing of HSV-1 or HSV-2 may be done with the culture. This sample must contain active herpes virus or it will produce a false negative test result; therefore the specimen must be obtained while the lesion is in the early stage of development.

When a person is exposed to a virus, the body responds by developing antibodies against it. These antibodies remain in the body and help lessen or prevent the severity of reoccurrences. A

blood test checks for these antibodies to the virus, not the virus itself. It can take two weeks to three months after exposure to HSV for antibodies to be detected in the blood. Since many adults have antibodies to HSV-1, testing for this virus may not be helpful in establishing a clinical diagnosis. A positive blood test specific for HSV-2 often implies past exposure to the anogenital area. Blood tests for HSV-2 can be helpful to the clinician if a patient presents with an ulcer on the genitals, when the culture test is negative. Blood tests for HSV-2 are not recommended in the genital area as a general STD screen in low risk populations.

TREATMENT

There is no permanent cure for the herpes simplex virus; once infected, patients will remain a carrier of the virus for the rest of their lives. Some remedies, however, can reduce the duration of the epidemic. Timely treatment with drugs like oral Aciclovir (Zovirax®), Famciclovir (Famvir®) and Falacyclovir (Valtrex®) will often abort the outbreak of blisters.

The best way to prevent recurrence is to avoid direct contact with an open lesion. People with herpes simplex virus should avoid sexual contact when active lesions are present. Using condoms and not sharing ones personal articles (like towels, soaps, kerchiefs, etc) can be helpful in reducing the chance of infection.

CHLAMYDIA

INTRODUCTION

Lymphogranuloma venereun (LGV) or Chlamydia is a sexually transmitted infection caused by a certain immunotypes of the bacterium *Chlamydia trachomatis*. The bacterium is gram negative and like virus it grows only inside the host body. The word ***chlamys*** hails from a Greek word which means "cloak draped around the shoulder." This describes how the intracytoplasmic inclusions caused by the bacterium are "draped" around the infected cell's nucleus. Because the symptoms of the disease resemble that of other disease, Chlamydia was not recognized as a sexually transmitted disease until recently. Isolation of the bacterium from embryonated eggs in 1957 and from cell culture in 1963 confirmed its existence as a bacterium. Owing to the fact that *Chlamydia trachomatis* is an obligate intercellular parasite that only infects humans it was also thought to be a virus. The organism is extremely sensitive to temperature and is refrigerated at 4°C as soon as a sample is obtained for laboratory purpose.

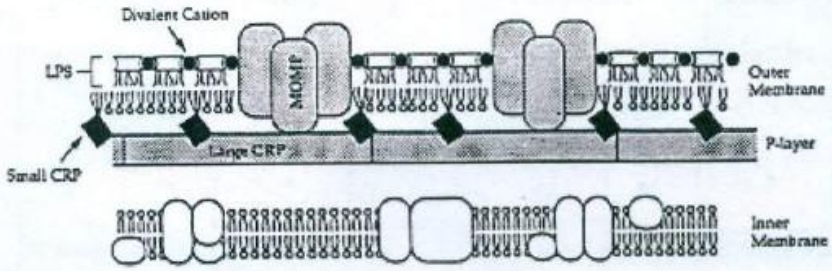
THE BIOLOGY OF *Chlamydia trachomatis*

(A)STRUCTURE

Chlamydia has a genome size of approximately 500-1000 kilobases and contains both types of nucleic acids (DNA & RNA). The bacteria invade and replicate in the columnar epithelium cells of humans. It persists at body sites that are inaccessible to phagocytes, T-cells, and B-cells. There

are 15 different serotypes. These serotypes cause four major diseases in humans namely endemic trachoma (caused by serotypes A and C), sexually transmitted disease and inclusion conjunctivitis (caused by serotypes D and K), and lymphogranuloma venereum (caused by serotypes L1, L2, and L3). Endemic trachoma leads to blindness, whereas inclusion conjunctivitis is associated with the sexually transmitted form and does not lead to blindness.

The unique cell wall structure contributes majorly to the virulence of the bacterium. It is now known that Chlamydia, because of its cell wall, is able to inhibit phagolysosome fusion in phagocytes. The Chlamydial cell wall is gram-negative in that it contains an outer lipopolysaccharide membrane, but it lacks peptidoglycan in its cell wall. This lack of peptidoglycan is shown by the inability to detect muramic acid (which is an essential component of the peptidoglycan layer of the bacterial cell wall) and antibodies directed against it. It may, however, contain a carboxylated sugar other than muramic acid. The cell wall structure consists of a major outer membrane protein cross-linked with disulfide bonds. It also contains cysteine-rich proteins (CRP) that may be the functional equivalent to peptidoglycan. This unique structure allows for intracellular division and extracellular survival.



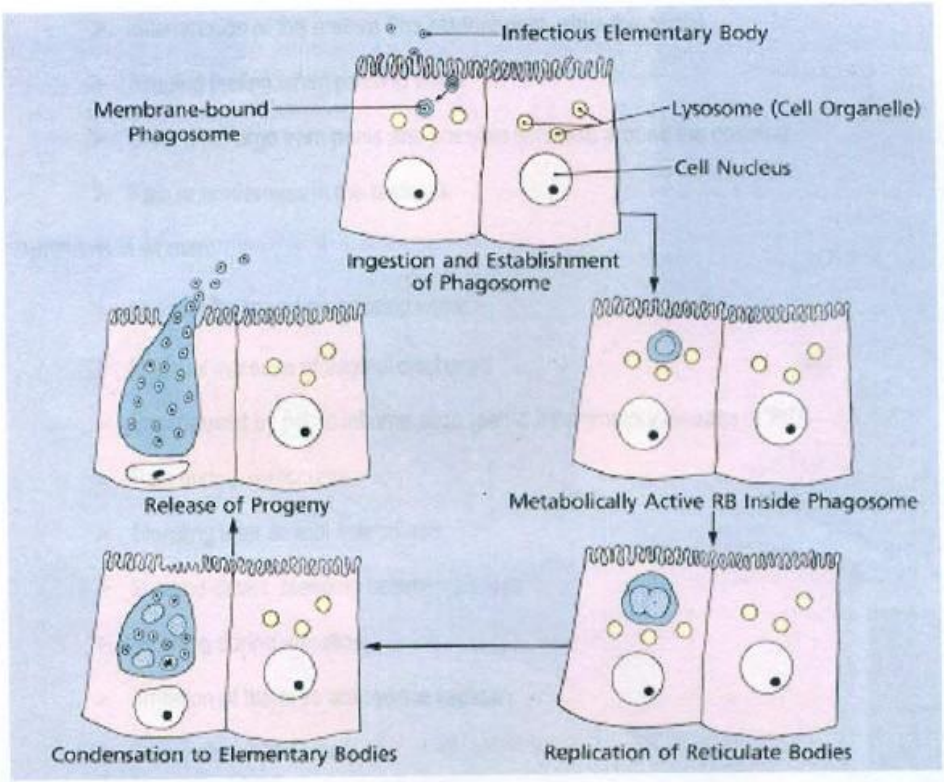
Structure of the Cell Wall (From Hatch 1996)

(B) LIFECYCLE OF *C. trachomatis*

Chlamydia trachomatis is taxonomically classified in a separate order because of its unique life cycle. The life cycle of *C. trachomatis* basically consists of two stages: elementary body and reticulate body.

The elementary body is the dispersal form and is equivalent to a spore of a spore forming bacteria. It is approximately 0.3 um in diameter and induces its own endocytosis upon exposure to target cells. It is this form that prevents phagolysosomal fusion and hence allows for intracellular survival.

Once the elementary body enters the endosome, the glycogen produced causes it to "germinate" into the vegetative form, which is called as the **reticulate body**. The reticulate body divides by binary fission at approximately 2-3 hours per generation. It has an incubation period of 7-21 days in the host. It contains no cell wall and is detected as a cell inclusion when stained with iodine. After division, the reticulate body transforms back to the elementary form and is released by the cell by exocytosis. One phagolysosome usually produces 100-1000 elementary bodies.



(Source: **Infections Caused by *Chlamydia trachomatis* Chapter 6 in Morse, et al, *Sexually Transmitted Diseases*. R C BARNES)**

Chlamydia is transmitted through infected body secretions only. It infects mainly mucosalmembranes, such as the cervix, rectum, urethra, throat, and conjunctiva. It is primarily spread via sexual contact and manifests as the sexually transmitted disease. The bacterium is not easily spread among women, so the disease is mainly transmitted by heterosexual or male homosexual contact. However, infected secretions from the genitals to the hands and eventually to the eyes can cause trachoma.

SYMPTOMS

Symptoms for Chlamydia usually appear approximately 7 to 21 days after infection and differ for men, women and children. Women infected with Chlamydia can also infect their newborn infant during delivery.

Symptoms in men:

- Inflammation of the urethra (the bladder duct within the penis)
- Stinging feeling when passing water
- Clear discharge from penis and possible itchiness around the opening
- Pain or tenderness in the testicles.

Symptoms in women:

- Stinging feeling when passing water
- Unusual increase of vaginal discharge
- Pain caused by pelvic inflammation (pelvic inflammatory disease or PID)
- Pain during intercourse
- Bleeding after sexual intercourse,
- In some cases, bleeding between periods.
- Burning during urination,
- Irritation of the area around the vagina,
- Lower abdominal pain

Symptoms in infants:

- Inflammation of the eye (conjunctivitis) at birth
- Problems breathing
- Premature birth
- In rare cases, pneumonia.

DIAGNOSIS

Detection of the bacterium in body secretions can be done by using both non-culture and culture tests.

Non-culture tests include the following:

1. **Fluorescent Monoclonal Antibody Test (FMAT)**: Detects either the major outer membrane protein characteristic of every bacterial cell wall or the lipopolysaccharide (LSP).
2. **Enzyme immunoassay**: Detects a colour product converted by an enzyme linked to an antibody.
3. **DNA probes**: Uses DNA complementary to specific ribosomal RNA sequences. Nucleic acid amplification using polymerase chain reaction and ligase chain reaction are also under experimentation
4. **Rapid Chlamydia tests**: Uses antibodies against the lipopolysaccharides (LPS).
5. **Leukocyte esterase tests**: Detects enzymes produced by leukocytes containing the bacteria in urine.

Unfortunately, certain non-culture tests are not specific and hence leads to a wrong conclusion similarly, antibodies can cross-react with non-chlamydial species to give a positive result for Chlamydia.

Culture tests identify intracytoplasmic inclusions in cells stained with monoclonal fluorescent antibodies. The cells are subsequently amplified on cyclohexamide-treated McCoy cells (a mouse cell line easily infected with the bacterium). Culture tests are 100% specific and thus the disease can be efficiently diagnosed. The sample is stored a -70C if the collection is delayed by more than 48 hours. As Chlamydia is normally found in association with the normal flora, samples must be treated with gentamycin to kill other microorganisms. Dead microorganisms or effect of the gentamycin on Chlamydia may bias results.

TREATMENT

Various tropical or oral doses of antibiotics like Tetracycline, Chloramphenicol, Rifampicin, Doxycycline and Fluroquinones can be administered to treat Chlamydia. Usually Doxycycline is used because it is used for extended treatment, can be taken with food, and is inexpensive. Pregnant women are advised to take Erythromycin as a part of the treatment procedure. Recently, Azithromycin has been proven as an effective single-dose therapy. Though this improves patient compliance it is more expensive than the other antibiotics. The treatment is provided to the infected person as well as his/hers sexual partner.

More stress should be laid on prevention rather than cure of the disease. Habits like using condoms during sex, delaying the age of first intercourse, monogamy must be practiced. Sexual history should be discussed with partners and educating oneself about sexually transmitted diseases is a must.

GENITAL WARTS (Condylomata Acuminataare)

INTRODUCTION

A DNA containing virus called the Human Papillomavirus (HPV) causes Genital Warts or Condylomata Acuminataare. Recent studies and developments in sciences have shown that certain types of HPV can cause cervical cancer. However this cancer is preventable through regular screening of blood for the virus and proper treatment of abnormally changed cells. There are over 100 serotypes of HPV and the genomes of almost 70 have been sequenced completely. Only certain types of HPV (usually serotypes 6, 11, 30, 42, 43, 44, 45, 51, 52 and 54) cause genital warts. About 30 of these types are sexually transmitted and cause genital warts. Other types are not related to genital warts and can cause abnormal cell changes on the genital skin, usually on a female's cervix. Genital warts are spread through skin-to-skin contacts and not through body fluids. Unlike other sexually transmitted diseases, genital warts cannot be completely prevented by the use of condoms. The HPV is usually asymptomatic and the infections often go unnoticed. Only about after nine months from the time of infection, the actual development of warts can be seen. Genital warts, like normal warts on other parts of the body, are dry and painless, firm and rough in texture, and usually greyish or skin colour. Warts can be small and difficult to detect. Small or flat warts on a man's penis or a woman's cervix are particularly likely to go unnoticed. They may itch slightly or give an irritated feeling. When warts are present, the virus is considered to be in active phase. The symptoms may vanish after sometimes and is often mistaken to be cured but usually in this phase the virus is latent (sleeping) in the skin cells. The virus is not contagious at this phase. Genital warts may or may not return after the first episode. Warts may appear within several weeks after intercourse with the infected person, or it may also take several months or years to appear. It is also possible that warts may never appear. This makes it hard to know exactly when or from whom someone got the virus.

BIOLOGY OF HUMAN PAPILLOMAVIRUS (HPV)

(A)STRUCTURE

Papillomaviruses are highly species specific and infect only human cells. Papillomavirus is approximately 55nm in diameter. These viruses has a caspid of icosahedral symmetry contain 72 capsomeres clustered in groups of five (pentamers) and six (hexamers) surrounding a genome containing a double-stranded circular DNA with approximately 8000 base pairs. The capsomers located at each of the 12 vertices, are pentamers (i.e. each is surrounded by five adjacent capsomers), and the other 60 capsomers are hexamers (i.e. each adjacent to six capsomers). The HPV does not have any envelop surrounding its inner components like the one present in HIV. The papillomavirus can survive extreme conditions (e.g. very low temperature) and for a long time as a virion i.e. in the absence of a host cell.

The strains of HPV, which cause genital warts, are considered to be low risk HPV and are generally transmitted through sexual contacts. The HPV genome exists as a circular episomal DNA separate from the host cell nucleus in low-risk or non-malignant HPV lesions, such as those

typically associated with HPV types 6 and 11. The genomes of high-risk HPV types 16 and 18 are typically integrated into the host cell DNA in malignant lesions. Integration of the viral genome into the host cell genome is considered a hallmark of malignant transformation. HPV proteins E6 and E7 of high-risk serotypes have been shown to inactivate the host's tumour suppressor proteins p53 and Rb, thereby resulting in unregulated host cell proliferation and malignant transformation.

(B)MODE OF REPLICATION

Condylomata Acuminataare infections typically occur when basal cells are exposed to infectious virus through a disturbed epithelial layer as would occur during a sexual intercourse or after minor skin abrasions. The incubation period of the virus may range from 2-3 months or more.

Generally HPV has 2 modes of replication:

1. Stable replication of the episomal genome in basal cells and
2. Vegetative, replication in more differentiated cells to generate progeny virus.

HPV lesions arise from the proliferation of infected basal keratinocytes. All cells of a lesion contain the viral genome but the expression of viral genes depends on the state of cellular differentiation. Most viral genes are activated only when the infected keratinocyte leaves the basal layer. Production of daughter virions can occur only in highly differentiated keratinocytes. Therefore viral replication occurs only at the epithelial layer where the cells are ultimately sloughed into the environment.

Virus multiplication takes place only in the host cell nucleus. Consequently, infected cells exhibit a high degree of nuclear atypia. Perinuclear clearing (halo) with a pyknotic or shrunken nucleus appears which is a characteristic feature of productive papillomavirus infection. This process is called Koilocytosis, which hails from a Greek word *koilos* meaning empty.

HPV infections have not been shown to be cytolytic, rather viral particles are released as a result of degeneration of desquamating cells.

SYMPTOMS

The symptoms are raised, rough, wart-like growths that may occur singly or in clusters. In men, they are usually found around the head of the penis and tend to be drier. In women, they appear most often around the vaginal opening and may spread to the rectal area. It is also possible for the virus to appear on or near the cervix as whitish, flat-like lesions, usually only detectable through close visual examination of the cervix (colposcopy). In both men and women, lesions may also be present in the mouth and throat. In general, symptoms can intensify if the immune system is weakened, or during pregnancy or if the person has diabetes.

DIAGNOSIS

The diagnosis of the disease can involve any one of the following procedures:

(A) PHYSICAL EXAMINATION: A diagnosis is made when a characteristic lesion is visible. But the fairly common small or flat warts can pass unnoticed. By swabbing the skin with 5 per cent acetic acid (VINEGAR), 'invisible' warts will emerge as white-coloured patches especially if viewed through a magnifying lens, such as a colposcope.

(B) HPV DNA TYPING: HPV DNA typing using polymerase chain reaction (PCR) methods may be used to help clarify the diagnosis if doubt exists about the cause of a lesion. This modality may also be used to help guide management by distinguishing between high- and low-risk types of HPV. However, little data exist to support the routine use of this modality.

(C) TISSUE BIOPSY: Tissue biopsy can be used to confirm HPV infection if the diagnosis is uncertain, particularly if warts are abnormally pigmented, ulcerated, indurate or possess any unusual character in general. A biopsy is usually taken of a warty lesion if the patient is immuno compromised.

TREATMENT

Although there is no permanent cure for HPV, there are several treatment methods for genital warts. When choosing what treatment to use, the health care provider will consider the size, location and number of warts, changes in the warts, patient preference, and cost of treatment, convenience, adverse effects, and their own experience with the treatments.

Several treatments, some of which are provider-administered or self-administered, are available. Genital warts can be chemically burned or frozen with liquid nitrogen (cryotherapy). Although these techniques for freezing the warts are effective, they can cause a temporary irritation of tissue. Applying caustic chemicals to the infected area treats some cases of genital wart infection. More than one application is usually needed. Most of these treatments must be repeated several times, sometimes over several months. All methods of treatment have high failure rates. Therefore, re-examinations sometimes are recommended, even after the warts seem to go away. Genital warts cannot be effectively treated with any over-the-counter preparations. The applications of certain chemicals like Podophyllin and TCA (trichloracetic acid) on the warts can be effective in treating the disease and the latter being preferred by doctors now. Podofilox cream or gel (Condylox®) is a self-applied treatment for external genital warts. Besides being cost effective this treatment is easy to use and is safe, but it must be used for about 4 weeks. Another self applied treatment involves the use of Imiquimod cream (Aldara®).This cream is different than other commonly-used treatments, which work by destroying the wart tissue. Aldara actually boosts the immune system to fight HPV.

Also a Laser therapy (using an intense light to destroy warts) may be administered to treat the disease. This is used for larger or extensive warts, especially those that have not responded well to other treatments. Laser can also cost a lot of money. The unavailability of proper machineries and training to the doctors is hampering the use of this technique.

Section B: Database on STD's in Sikkim.

RESEARCH DESIGN

(a) THE PROBLEM

A view into the history of mankind reveals that the problem of sexually transmitted diseases, also sometime referred to as venereal diseases, aggravated with the increase in the number of many brief sexual relationships in ones life. The advent of prostitution further elaborated the sexual life of an individual and centres of prostitution thus emerged as storehouse of the disease.

The advent of 90's also marked the emergence of these diseases as a matter of major health concern in the state of Sikkim. The growing population of people infected with HIV and other common STD's like syphilis and gonorrhoea in the state pose a serious threat for the people of the state. Though the reports of the Health Department, Government of Sikkim, Sikkim State AIDS Control Society and various other organizations show a gradual increase in the number of STD cases each year, however with the progress of this research it was found that the total number of STD cases could probably be beyond count in the state.

(b) AIMS AND OBJECTIVES OF THE STUDY

The overall aim of the investigation was to study the various aspects of sexually transmitted diseases in details and to study its rate of growth in Sikkim. The study also involves the detailed study of factors responsible for the diseases, their diagnostic features and their latest treatment procedure (section A of the project).

The objective will be to delineate the role of professional social worker for helping the STD patient, to study the present awareness level amongst the people of Sikkim and to suggest measures for facilitating the effective management, prevention and control of the disease. Specifically the objectives can be stated as below:

- To introduce the disease and their causation factors in details,
- To analyze the STD knowledge and sexual habits of the sample population,
- To find out the role of education and modes of media like television and radio in the awareness and control of the diseases,
- To find the best possible way to reach the masses to create awareness,
- To locate the major risk groups of the disease.

(c) METHODOLOGY

(i) LOCALE OF STUDY

The present has been carried out in the state of Sikkim. Sikkim is the 22nd state of the Indian union. The state with hills criss-crossed by green valleys, towering peaks, rippling rivers and exotic species of flora and fauna covering over 7096sq km with a population of 540493 people (2001 census) boasts of huge natural collection of life forms.

The prostitution level in the state is very low though not negligible. No "RED LIGHT STREETS" in the state were found and despite all this the constantly growing rate of STD cases highlights the seriousness of the problem.

FACTS ABOUT SIKKIM USEFUL FOR THE PRESENT STUDY

TOTAL POPULATION (2001 CENSUS)	540493 MALE: 288217, FEMALE: 252276
LITERACY RATE	69.68% MALES:76.73%, FEMALES:61.46%
SEX RATIO (FEMALES PER 1000 MALES)	875 (2001)
PERCENT USING ANY MODERN METHOD OF CONTRACEPTIVES	41.4%
PERCENT USING CONDOMS	1.5%
PERCENT OF UNPROTECTED SEXUAL PRACTICE	12.2%

(ii) SOURCES OF DATA

The data to be collected was both of primary and secondary types. The primary data is basically collected from people at random. The data with the help of questionnaires has been collected from various parts of the state to enable the sample to represent the whole population. The areas visited include Jorethang, Manpari Busty and Namchi from the south, Mangan from the north and Pakyong, Ranipool, Singtam, Rangpo and Gangtok from the east district. A visit was also made to the NHPC workstation at Balwatar for data collection. Care was taken to interact with people from west in places like the Taxi stand near the State assembly from where taxis ply to various places to the west and the bus stops. Also people were extensively interviewed at places like the Secretariats, taxi stands, hospitals, and courts of law to ensure that the data is uniformly collected from all parts of the state and the respondents include all section of the society and from all occupational levels. Also all possible secondary data were collected from various governmental organizations like the STNM hospital, Gangtok and Sikkim State AIDS control society, Kazi road for reference. The data collection procedure began on the 15th of May 2005 and was brought to an end on the 29th of May 2005. The total numbers of respondents were 550.

(iii) TOOLS AND TECHNIQUES

The interview schedule has been the tool in the investigation for collecting pertinent information. The questionnaire had been finalized after series of revisions and corrections striking out inappropriate questions and making way for new additions. A copy of the questionnaire is attached below:

QUESTIONNAIRE

1. Gender
(a) Male
(b) Female

2. Age group
(a) 15-25
(b) 26-35
(c) 36-45
(d) 46 -55
(e) 56-65

3. Occupation
(a) Government Employee
(b) Entrepreneur
(c) Farmer
(d) Small self owned business
(e) Daily wage labour
(f) Drivers
(g) Students
(h) Business
(i) Others

4. Educational qualification
(a) None
(b) Primary
(c) Middle
(d) High
(e) Intermediate
(f) Graduate
(g) Post Graduate
(h) Post Graduate +

5. Which district are you from?
(a) North
(b) South
(c) East
(d) West
(e) Migratory

6. Are you aware of sexually transmitted diseases? Yes/no

7. Are you aware of any of the following diseases?
(a) Chlamydia
(b) Gonorrhoea

(c) Genital Herpes
(d) HIV&AIDS
(e) Genital warts
(f) Syphilis (feranga *rog*)
(g) Any other sexually transmitted diseases
(h) ALL
(i) NONE

8. **Are you aware of various programmes being held by the govt/state AIDS control society to create a mass awareness regarding sexually transmitted diseases and AIDS?** – Yes/no

9. **Have you/do you attend any such awareness programmes?** - Yes/no

10. **Which of the following appeals the most to you?**
    (a) Television
    (b) Radio
    (c) Newspapers
    (d) Local group discussion/ seminars
    (e) None

11. **If you come across a HIV+/STD patient would you accept that person into the society?**

12. **Have you been involved in extra marital sexual affair? – yes/ no/ DNS**
    - Protected/unprotected
    - Number of exposures
    - Disease treatment? Yes/no
    - If yes when and how long?
    - Present status

13. **What do you know about STD/HIV/AIDS?**

14. **Have you seen anyone with STD/HIV/AIDS?**

15. **Your opinion.**

16. **Remarks.**

(iv) TABULATION OF DATA

As the information collected from the respondents was basically is descriptive form, it was tabulated and had to undergo several steps of statistical calculations so that the raw data would be available in an organized form to ensure proper analyzing and interpretation of the data. Due to a large sample size of 550 respondent's automated software (SPSS v7.5.1 for windows) was used to organize the data and was later manually tabulated.

(d)LIMITATIONS OF THE STUDY

Since the sexual habits of the respondents were also to seek for there is a likelihood that some respondents may not have submitted to certain questions correctly, even though a maximum effort was put in by the investigator to collect the most accurate data and personally interviewed all the respondents. Some of the information sought is based on the individual's capacity to recall which has its own limitations. Some people even hesitated to be a part of the investigation. People hesitant to answer certain questions were flexibly dealt with and if they refused/hesitated to answer certain portions of the questionnaire, they were not forced to do so.

ANALYSIS OF THE DATA

(1) GENDER: Table I shows the frequency of the total male and female respondents and their respective percentages.

TABLE I

SRL. NO.	GENDER	FREQUENCY	PERCENTAGE
1.	MALE	269	48.9%
2.	FEMALE	281	51.1%
TOTAL		550	100%

OBSERVATION: As evident from the table there were total of 550 respondents out of which 48.1% are male and the 51.1% female.

(2) AGE GROUP: Sexually transmitted disease is one such disease that is prevalent in all age groups. The questionnaire divides the respondents into six different age groups as shown in Table II which shows the frequency of male and female respondents from different age group.

TABLE II

SRL.NO.	AGE GROUP	MALE	FEMALE	TOTAL	%
a.	15-25	99	154	253	46.0%
b.	26-35	69	49	118	21.5%
c.	36-45	44	30	74	13.5%
d.	46-55	37	35	72	13.1%
e.	56-65	20	13	33	6.0%
TOTAL		269	281	550	100%

OBSERVATION: The table reveals that the maximum numbers of respondents (46.0%) were aged 15-25. the youngest respondent in the study universe was 15 years old and the oldest respondent happened to be 62 years old. The mean (X) age of the study universe was found to be 31.4.

(3) OCCUPATION: The type of occupation of the independent respondents under the study has been analysed. There are ten occupational sub-groups namely (a) Government Employee, (b) Entrepreneur, (c) Farmer, (d) Small self owned business, (e) Daily wage labour, (f) Drivers, (g) Students, (h) Business, (i) Teachers and (j) others. The last sub group includes all minor categories like housewives, mechanics, carpenters, unemployed, etc. Table III shows the frequency of all the respondents from the various sub groups.

TABLE III

SRL.NO	OCCUPATION	MALE	FEMALE	TOTAL	%
a.	Government Employee	29	24	53	9.6%
b.	Entrepreneur	2	1	3	0.5%
c.	Farmer	20	11	31	5.6%
d.	Small self owned business	23	29	52	9.5%
e.	Daily wage labour	24	11	35	6.4%
f.	Drivers	59	0	59	10.7%
g.	Students	39	138	177	32.2%
h.	Business	19	19	38	6.9%
i	Teachers	7	7	14	16.0%
j.	Others	47	41	88	2.5%
TOTAL		269	281	550	100%

OBSERVATION: The table reveals that the students have the maximum frequency (32.2%) in the sample population of 550 respondents. Occupation is believed to have an impact on the infection of STDs. For example, during the process of data collection many respondents submitted that they had met STD patients who were generally drivers, daily wage labours and youths.

(4) EDUCATIONAL QUALIFICATION: According to some researchers in this field, education has a direct impact on sexually transmitted diseases. Some say that the uneducated ones are more afflicted to various STDs and the others have a different view. Thus, to study the role of education on the sexual habits of the respondents and also on the amount of awareness the respondents have regarding STDs; this parameter is being studied. Table IV shows all the data pertaining to the educational qualification of the respondents.

Table IV

SRL.NO	EDUCATIONAL QUALIFICATION	MALE	FEMALE	TOTAL	%
a.	NONE	22	25	47	8.5%
b.	PRIMARY	62	16	78	14.2%
c.	MIDDLE	46	24	70	12.7%
d.	HIGH	38	86	124	22.5%
e.	INTERMEDIATE	32	67	99	18.0%
f.	GRADUATE	62	51	113	20.5%
g.	POST GRADUATE	5	10	15	2.7%
h.	POST GRADUATE +	2	2	4	.7%
TOTAL		269	281	550	100.0%

OBSERVATION: As evident from the table the study population is more or less evenly poised amongst various educational levels with respondents with high school education having maximum frequency (22.5%).

(5) DISTRICT TO WHICH THE RESPONDENTS BELONG: In Sikkim there are only four districts (North, South, East and West) .In additions there are migratory people who have come in for jobs

or business and reside in Sikkim. The present investigation was carried out at the district level and the data is as shown in table V.

TABLE V

SRL.NO.	DISTRICT	MALE	FEMALE	TOTAL	%
a.	NORTH	35	41	76	13.8%
b.	SOUTH	51	44	95	17.3%
c.	EAST	94	149	243	44.2%
d.	WEST	52	34	86	15.6%
e.	MIGRATORY	37	13	50	9.1%
TOTAL		269	281	550	100%

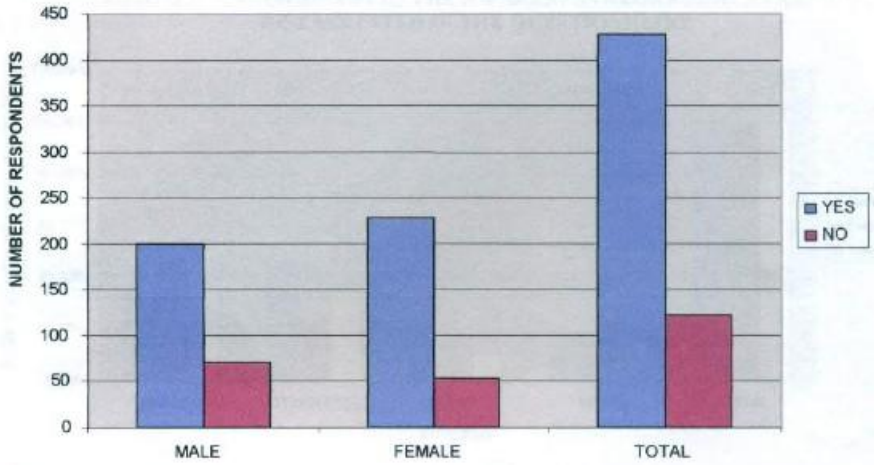
OBSERVATION: From the table we find that the maximum numbers of the respondents (44.2%) come from the east district. This is possibly because the capital is the centre of all activity in the state with government secretariats, major hospitals, etc situated in the city. Also the health department reports the maximum HIV+ cases and STD cases from places like Rangpo, Singtam and Pakyong, which are all, located in the east district.

(6) AWARENESS PERTAINING TO STDs: The questionnaire contains a question (nos.6) "Are you aware of sexually transmitted diseases"; the answer to which is optional (yes/no). All the respondents were personally interviewed and were intensely cross questioned, especially for this question and "yes" was marked only when the investigator satisfactorily felt that the respondent were actually aware of the diseases. Table VI shows the awareness level among both the gender. This section will later be broadly studied under various aspects like the awareness amongst various age group, educational qualification, etc.

TABLE VI

SRL.NO.	AWARENESS	MALE		FEMALE		TOTAL	OVERALL
		f	%	*f*	%		%
a.	YES	199	74.0%	228	81.1%	427	77.6%
b.	NO	70	26.0%	53	18.9%	123	22.4%
TOTAL		269	100%	281	100%	550	100%

GRAPH 1: AWARENESS OF STDs



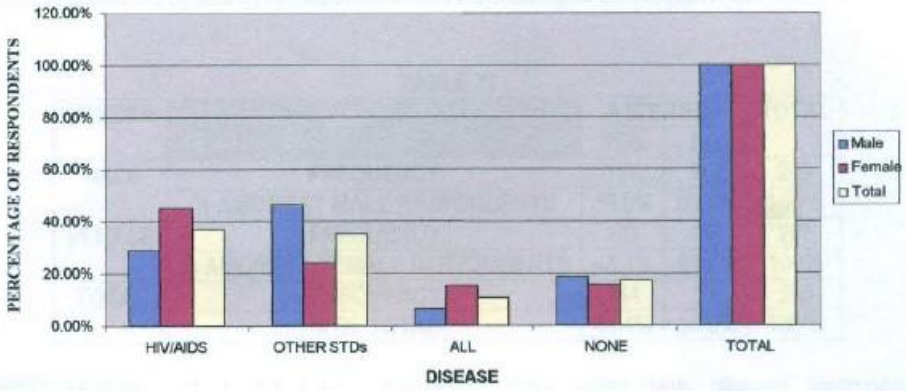
OBSERVATION: From the total sample size 77.63% of the respondents were aware of sexually transmitted diseases. Amongst the male population 74% are aware of sexually transmitted diseases and unexpectedly 81.1% of the female population are aware of the same. Graph 1 makes the picture of awareness levels clearer.

The awareness relating to six common sexually transmitted diseases, namely Chlamydia, Gonorrhoea, Genital Herpes, HIV/AIDS, Genital Warts and Syphilis was also sought for to see if the respondents knew any disease other than AIDS as it the most popular STD after syphilis and gonorrhoea. As many respondents were not familiar with the clinical names of the diseases, they were explained to the respondent by their usual symptoms and their popular local names. Table VII shows the frequency of the respondents aware of only AIDS and frequency of respondents who have knowledge of other sexually transmitted diseases along with or without AIDS.

Table VII

GENDER		HIV/AIDS	OTHER STDs	ALL	NONE	TOTAL
MALE	FREQUENCY	78	124	17	50	269
	% AMONGST MALE RESPONDENTS	29.0%	46.1%	6.3%	18.6%	100%
FEMALE	FREQUENCY	126	68	43	44	281
	% AMONGST FEMALE RESPONDENTS	44.9%	24.1%	15.3%	15.7%	100%
TOTAL	FREQUENCY	204	192	60	94	550
	% OF TOTAL	36.95%	35.1%	10.8%	17.15%	100%

GRAPH 2: GRAPH SHOWING THE AWARENESS REGARDING A PARTICULAR DISEASE CITED IN THE QUESTIONNAIRE



OBSERVATION: From the table it is apparent that 35.1% of the total sample size is aware of other sexually transmitted diseases apart from AIDS. In addition 10.8% of the respondents are aware of all the diseases cited in the questionnaire thus raising the total percentage to 45.9%. Also 17.15% of the respondents are completely unaware of the diseases. We can also see from the table that females have better numbers then males which imply that females are more well-informed about the diseases then males.

From the above graph we can also make out that the male respondents are better informed of STDs other than HIV/AIDS (mostly gonorrhoea) the reverse holding true for the females.

(7) AWARENESS OF VARIOUS PROGRAMMES BEING HELD BY THE GOVERNMENT/STATE AIDS CONTROL SOCIETY TO CREATE MASS AWARENESS CONCERNING STDs AND AIDS:

To check the performance of the present machinery to create a mass awareness for STDs and AIDS, the respondent's awareness of various programmes of the same was sought for. Table VIII shows the frequency of the respondents knowing about awareness programmes and table XI shows the frequency of respondents who have attended such programmes.

TABLE VIII

GENDER		AWARE	UNAWARE	TOTAL
MALE	FREQUENCY	184	85	269
	% AMONGST MALE RESPONDENTS	68.4%	31.6%	100%
FEMALE	FREQUENCY	221	60	281
	% AMONGST FEMALE RESPONDENTS	78.6%	21.4%	100%
TOTAL	FREQUENCY	405	145	550
	% OF TOTAL	73.6%	26.4%	100%

OBSERVATION: Among the 269 male respondents 68.4% males are aware of various awareness programmes and out of 281 females 78.6 % are aware of the same. As a whole 73.6% of the respondents are aware of the programmes.

Now let us see the frequency of respondents who have attended awareness programmes/camps. Table XI shows the frequency of respondents who have attended the programmes.

TABLE XI

GENDER		ATTENDED		TOTAL
		YES	NO	
MALE	FREQUENCY	107	162	269
	% AMONGST MALE RESPONDENTS	39.8%	60.2%	100%
FEMALE	FREQUENCY	137	144	281
	% AMONGST FEMALE RESPONDENTS	48.8%	51.2%	100%
TOTAL	FREQUENCY	244	306	550
	% OF TOTAL	44.4%	55.6%	100%

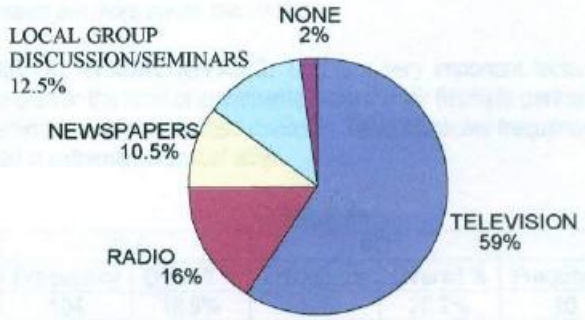
OBSERVATION: Out of 269 male respondents 39.8% males have attended awareness programmes and out of 281 females 48.8% have attended the same. As a whole 44.4% of the respondents have attended awareness programmes/camps.

(8) BEST MODE OF MASS COMMUNICATION: To find the best mode of communication to reach the masses a question was set (Q. no. 10) with five options namely television, radio, newspapers and local group discussion/seminars. The results are as shown in table X.

TABLE X

SRL.NO	MODE OF COMMINUCATION	MALE	FEMALE	TOTAL	PERCENTAGE
a.	TELEVISION	137	188	325	59.1%
b.	RADIO	52	34	86	15.6%
c.	NEWSPAPERS	39	19	58	10.5%
d.	LOCAL GROUP DISCUSSION/SEMINARS	34	35	69	12.5%
e.	NONE	7	5	12	2.2%
TOTAL		269	281	550	100%

PIE CHART 1: CHART SHOWING THE BEST MODE OF COMMUNICATION



OBSERVATION: As seen in the table and the pie chart an overwhelming 59.1% of respondents find television more appealing and can also be the most effective tool in reaching the masses for awareness.

(9) RESPONDENTS ACCEPTANCE OF HIV+/STD PATIENTS INTO THE SOCIETY: There is always a social stigma associated with STDs and unaware people are by and large reluctant to take patients into the society. The belief that even touching can spread AIDS/STDs or staying or sharing toilets with the diseased person may be the reason for the bad social treatment usually melted out on the patients. So it is clear that there exists a definite relationship between the awareness level of the people and the treatment melted out on the patients. More the awareness betters the behaviour towards the patients.
All the data related to this parameter of the investigation are as shown in table XI.

TABLE XI

GENDER		PATIENTS ACCEPTANCE		TOTAL
		YES	NO	
MALE	FREQUENCY	182	87	269
	% AMONGST MALE RESPONDENTS	67.7%	32.3%	100%
FEMALE	FREQUENCY	221	60	281
	% AMONGST FEMALE RESPONDENTS	78.6%	21.4%	100%
TOTAL	FREQUENCY	403	137	550
	% OF TOTAL	73.3%	26.7%	100%

OBSERVATION: Table XI reveals that 73.3% of the entire sample size would accept a HIV+/STD patient. The female have better percentage of patient's acceptance than males thus hinting towards that females are more aware than males.

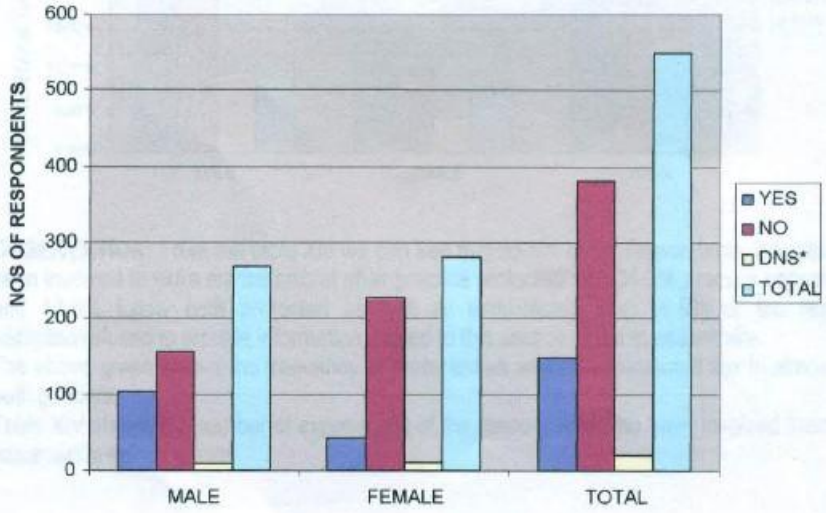
(10) EXTRAMARITAL SEXUAL AFFAIRS: This is a very important factor to find out the risk groups because greater the level of extramarital sexual affair (multiple partners) greater will be the chance of contacting sexually transmitted diseases. Table XII shows frequency of respondents who are/were involved in extramarital sexual affair.

TABLE XII

GENDER	YES		NO		DNS*	
	Frequency	Overall %	Frequency	Overall %	Frequency	Overall %
MALE	104	18.9%	155	28.2%	10	1.8%
FEMALE	44	8.0%	226	41.1%	11	2.0%
TOTAL	148	26.9%	381	69.3%	21	3.8%

***DID NOT SUBMIT.**

GRAPH 3: EXTRAMARITAL SEXUAL AFFAIR

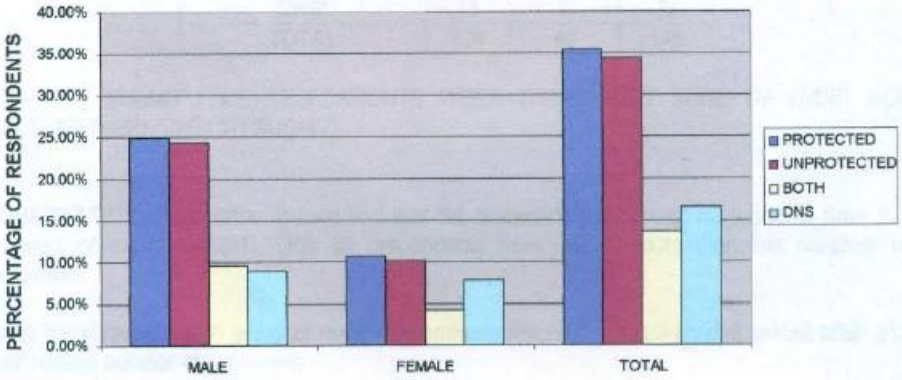


OBSERVATION: As seen in table XII, 26.9% of the respondents have involved themselves in extramarital sexual affairs; the majority of the percentage going to the male population. 3.8% of respondent's hesitated/refused to provide information related to this section of the questionnaire. Graph 3 makes the representation simpler to have a bird's eye view on the data.
Table XIII shows the type of sexual practice (protected/unprotected) followed by the respondents.

Table XIII

GENDER	PROTECTED		UNPROTECTED		BOTH		DNS	
	f	%	*f*	%	*f*	%	*f*	%
MALE	42	24.9%	41	24.3%	16	9.5%	15	8.9%
FEMALE	18	10.7%	17	10.1%	7	4.1%	13	7.7%
TOTAL	60	35.5%	58	34.3%	23	13.6%	28	16.6%

GRAPH 4: GRAPH SHOWING THE PERCENTAGES OF PROTECTED/UNPROTECTED/BOTH TYPE OF SEXUAL PRACTICE



OBSERVATION: From the table XIII we can see that 35.5% of the respondents, admitting to have been involved in extra marital sexual affair practice protected sex. 34.3% practice unprotected sex and 13.6% follow both protected as well as unprotected sex. 16.6% of the respondents hesitated/refused to provide information related to this section of the questionnaire.

The above graph shows the frequency of protected as well as unprotected sex in almost equal in both genders.

Table XIV shows the number of exposure(s) of the respondents who have involved themselves in extramarital sexual affairs.

TABLE XIV

NOS. OF EXPOSURES	MALE	FEMALE	TOTAL
1	20	14	34
2	23	14	37
3	14	6	20
4	6	0	6
5	3	0	3
6	4	1	5
7	4	1	5
8	2	1	3
9	1	0	1
>9	11	1	12
DNS*	16	6	22
TOTAL	104	44	148

**DID NOT SUBMIT (THE RESPONDENTS WERE EITHER NOT SURE OF IT/DID NOT REMEMBER/REFUSED TO SUBMIT).*

OBSERVATION: From table XIV we find that the maximum numbers of respondents have 1-3 numbers of exposures (91). Only 35 respondents have four or more than four numbers of exposures.

Table XV shows the age group of **male respondents** involved in extra marital sexual affair and their relative number of exposures.

TABLE XV

AGE GROUP	FREQUENCY OF NOS.EXPOSURE (S)										TOTAL	%
	1	2	3	4	5	6	7	8	9	>9		
15-25	2	8	5	3	2	1	1	0	1	4	27	30.6%
26-35	9	7	5	0	1	1	1	2	0	4	30	34.1%
36-45	5	4	2	1	0	1	1	0	0	1	15	17.1%
46-55	4	2	2	2	0	1	1	0	0	1	13	14.8%
56-65	0	2	0	0	0	0	0	0	0	1	3	3.4%
TOTAL	20	23	14	6	3	4	4	2	1	11	88	100%

OBSERVATION: From the table XV we find that 34.1% of the total male who have admitted of being involved in extramarital sexual affair (once or more than once) belongs to the 26-35-age group and 30.6% belonging to the 15-25 age groups. The detail of number of exposure(s) is given in the table (XV).

Table XVI shows the age group of **female respondents** involved in extra marital sexual affair and their relative number of exposures.

TABLE XVI

AGE GROUP	FREQUENCY OF NOS.EXPOSURE (S)										TOTAL	%
	1	2	3	4	5	6	7	8	9	>9		
15-25	4	2	2	0	0	0	0	1	0	1	10	26.3%
26-35	5	7	1	0	0	0	0	0	0	0	13	34.2%
36-45	3	2	2	0	0	0	1	0	0	0	8	21.0%
46-55	2	3	1	0	0	1	0	0	0	0	7	18.5
56-65	0	0	0	0	0	0	0	0	0	0	0	0%
TOTAL	14	14	6	0	0	1	1	1	0	1	38	100%

OBSERVATION: From the table XVI we find that 34.2% of the total female who have admitted of being involved in extramarital sexual affair (once or more than once) belongs to the 26-35-age group and 26.3% belonging to the 15-25 age groups. The detail of number of exposure(s) is given in the table (XVI).

During the course of data collection few of the respondents were found to be suffering from some type of sexually transmitted diseases or were infected by these diseases in the past. Out of the total sample size of 550 respondents 7 male and 6 females admitted of contacting one of the sexually transmitted diseases. All the 7 males had been suffering from gonorrhoea and four of them had undertaken self-treatment either by traditional methods of simply by purchasing the medicines from the pharmacy for the same (the disease is locally called *bhirengey*). The other underwent treatment at government hospitals or the primary health centres (PHCs) in Mangan, Namchi and STNM hospital in Gangtok respectively. Out of the 6 females admitting to have been infected by one STD or the other four refused to speak of the disease although they did mention of being treated at private clinics in or outside Gangtok. One case of syphilis and one case of oral chancre was found, both being treated at government hospitals.

Interestingly, it was observed that all the 13 respondents who admitted of being infected to STDs had practised unprotected sex once or more than once in their life, thus hinting that condoms are actually effective against the diseases though the number of diseased respondents is very low to concretely derive a conclusion.

(11) DISTRICT WISE AWARENESS: Since the project involves district wise study of the diseases it is important to analyse the awareness levels at a district level. Table XVII shows the district wise report of the respondents who are aware of the diseases.

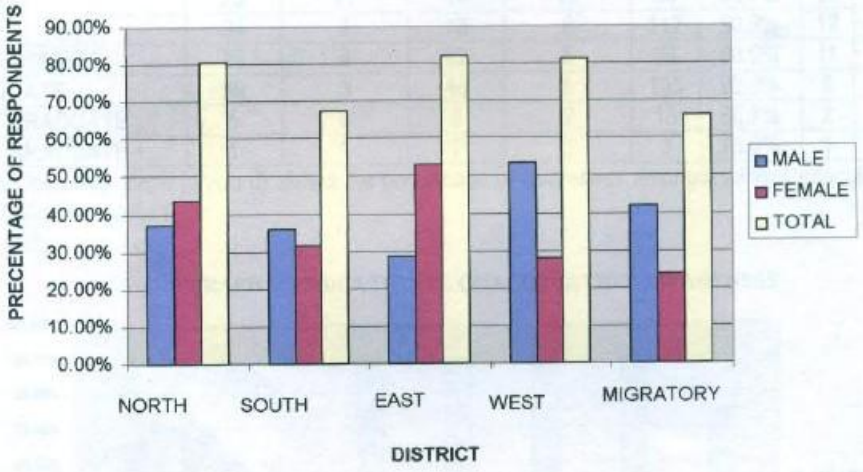
Table XVII

DISTRICT	MALE				FEMALE				TOTAL			
	Aware		Unaware		Aware		Unaware		Aware		Unaware	
	f	%	f	%	f	%	f	%	f	%	f	%
NORTH	28	36.9%	7	9.2%	33	43.4%	8	10.5%	61	80.3%	15	19.7%
SOUTH	34	35.8%	17	17.9%	30	31.5%	14	14.7%	64	67.3%	31	32.6%
EAST	70	28.8%	24	9.8%	129	53.1%	20	8.3%	199	81.9%	44	18.1%
WEST	46	53.5%	6	7%	24	27.9%	10	11.6	70	81.4%	16	18.6%
MIGRATORY	21	42.0%	16	32.0%	12	24.0%	1	2.0%	33	66.0%	17	34.0%

Note: All percentage calculations are done taking in consideration the total number of respondents of particular district and not the entire sample size. Here, f =frequency.

OBSERVATION: It is clear from the table that the general awareness is very good in the state. The South district has 67% awareness and the rest shows around 80% awareness. The male population of the east district and the female population of the west show quite low awareness with 28.8 % and 27.9% awareness respectively.
The following graph would provide a clearer representation of the district wise percentage aware population.

GRAPH 5: DISTRICT WISE PERCENTAGE OF AWARE RESPONDENTS



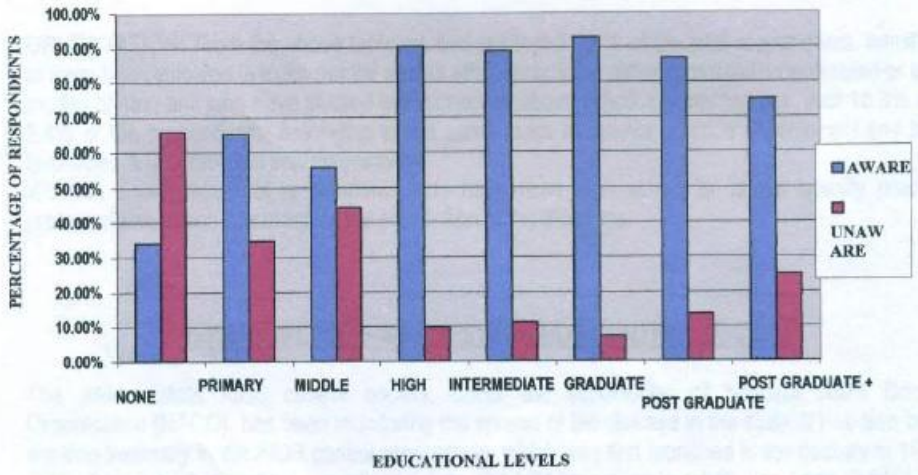
(12) ROLE OF EDUCATION IN CONTROLING THE DISEASE: Education is believed to have a major impact on the mindset of man. The present system of academic education involves the study of sexually transmitted diseases especially AIDS at some level. It is thus important to analyse the role of education for creating an awareness and thus in the control of these diseases.
Table XVIII shows the awareness levels of the respondents belonging to various educational qualification classes.

Table XVIII

EDUCATIONAL QUALIFICATION	MALE		FEMALE		TOTAL			
	Aware	Unaware	Aware	Unaware	Aware		Unaware	
					f	%	*f*	%
NONE	8	14	8	17	16	34.0%	31	66.0%
PRIMARY	41	21	10	6	51	65.4%	27	34.6%
MIDDLE	25	21	14	10	39	55.7%	31	44.3%
HIGH	34	4	78	8	112	90.3%	12	9.7%
INTERMEDIATE	26	6	62	5	88	88.9%	11	11.1%
GRADUATE	59	3	46	5	105	92.9%	8	7.1%
POST GRADUATE	5	0	8	2	13	86.7%	2	13.3%
POST GRADUATE +	1	1	2	0	3	75.0%	1	25.0%

The following graph (graph 6) shows the percentage of awareness amongst various educational qualification classes.

GRAPH 6: EDUCATIONAL QUALIFICATION-AWARENESS



OBSERVATION: The awareness percentages down the table are very good. Amongst all the respondents who have read high school and above (N= 355), 90.4% are aware of sexually transmitted diseases and amongst those who have read middle school and below (N= 195), only 54.4% are aware of the same. Education, thus, is a major tool in fighting the diseases.

With the increase in the literacy rate of the state, 100% awareness looks very attainable.
Now let us now also analyse the frequency of protected sex amongst the respondents belonging to various educational qualification groups. Table XIX shows the frequency of the respondents belonging to various educational qualification groups, who practice protected, unprotected or both types sex.

Table XIX

EDUCATIONAL QUALIFICATION	PROTECTED		UNPROTECTED		BOTH	
	f	%	*f*	%	*f*	%
NONE	3	2.1%	9	6.4%	3	2.1%
PRIMARY	3	2.1%	19	13.5%	5	3.5%
MIDDLE	9	6.4%	7	5.0%	3	2.1%
HIGH	11	7.8%	11	7.8%	4	2.8%
INTERMEDIATE	11	7.8%	7	5.0%	5	3.5%
GRADUATE	19	13.5%	5	3.5%	3	2.1%
POST GRADUATE	4	2.8%	0	0.0%	0	0.0%
POST GRADUATE +	0	0.0%	0	0.0%	0	0.0%
TOTAL	60	42.6%	58	41.2%	23	16.2%

OBSERVATION: From the above table we find out that 31.9 % of the total respondents, admitting to have been involved in extra marital sexual affair (practicing either protected, unprotected or both modes of sex) and who have studied high school or above practice protected sex. Just 16.3% and 8.4% of the respondents, belonging to the same class as above practice unprotected and both (protected & unprotected) sex respectively.
We can clearly see that respondents who have read high school or above usually practice protected sex, which is a must for the prevention of the diseases.

HIV/AIDS REPORT- SIKKIM STATE AIDS CONTROL SOCIETY

The Sikkim state AIDS control society, under the supervision of National AIDS Control Organisation (NACO), has been monitoring the spread of the disease in the state. It has also been working intensely in the AIDS control programme, which was first launched in the country in 1991-1992. Based on the sentinel surveillance data, the HIV frequency in adults has been divided into three groups as follows:

- GROUP 1: Includes states where the HIV infection has crossed 1% or more in antennal women. Maharastra, Tamil Nadu, Andhra Pradesh, Manipur, etc fall in this group.
- GROUP 2: Includes states like Goa, Pondicherry and Gujrat where the HIV infection has crossed 5% among the high-risk group but below 1% in antenatal women.
- GROUP 3: Includes rest of the states where the HIV infection has not crossed 5% among the high-risk group and is still below 1% in antenatal women. Sikkim falls in this group.

HIV SENARIO IN THE COUNTRY:

ESTIMATED NUMBER OF HIV+ CASES IN THE COUNTRY	5.1 MILLION
TOTAL NUMBER OF REPORTED AIDS CASES –TILL MARCH, 2005	7,2943
TOTAL NUMBER OF REPORTED AIDS CASES-AS ON 31ST MARCH, 1993	310

Thus we can see how the disease scenario has worsened in the country with just 310 cases of full blown AIDS in 1993 to 7, 2943 cases in 2005.

HIV SENARIO IN THE STATE:

There are a total of 36 HIV+ cases in Sikkim with 26 males and 10 females. Nine cases of full-blown AIDS have been reported so far. The present data has been collected from the Sikkim state AIDS control society, Kazi road, Gangtok. All the data is as on April 2005.

The total number of samples screened in the state for HIV is 7689. Out of 36 cases of HIV+ 24 are known to be sexually transmitted and the rest are through other routes of transmission like infected needles or syringe or through infected blood transfusion.

Table 1 shows the number of HIV+ cases reported annually.

Table 1:

SL.NO	YEAR	MALE	FEMALE	TOTAL	CUMULATIVE FREQUENCY	GROWTH %
1	1995	2	0	2	2	-
2	1996	0	0	0	2	0.0%
3	1997	1	0	1	3	33.3%
4	1998	3	3	6	9	66.7%
5	1999	5	1	6	15	40.0%
6	2000	1	0	1	16	6.3%
7	2001	3	0	3	19	15.8%
8	2002	3	2	5	24	20.8%
9	2003	2	3	5	29	17.2%
10	2004	5	0	5	34	14.7%
11	2005	1	1	2	36	5.6%
TOTAL		**26**	**10**	**36**		

OBSERVATION: From the table we can find the average annual rate of growth, which is 22.0%.

Now let us see the trend of HIV+ cases in various age groups. Table 2 shows the number of HIV+ cases reported in various age groups.

Table 2

SL.NO	AGE GROUP	MALE	FEMALE	TOTAL
1	0-14	1(10months)	0	1
2	15-29	12	5	17
3	30-44	11	5	16
4	45+	2	0	2
TOTAL		26	10	36

OBSERVATION: The table reveals that majority of reported cases are from the younger age group (15-29).

Table 3 shows the route of transmission of HIV among the 36 HIV+ cases.

Table 3:

SL.NO	ROUTE OF TRANSMISSION	TOTAL NO. OF POSITIVE CASES	%
1	SEXUAL	24	66.7%
2	INFECTED NEEDLE & SYRINGE	4	11.1%
3	BLOOD &BLOOD PRODUCTS	1	2.8%
4	PARENTAL	1	2.8%
5	OTHERS	6	16.7%
TOTAL		36	100%

OBSERVATION: The table reveals that 67.7% of the HIV+ cases in Sikkim have acquired the disease through sexual contacts.

CONCLUSIONS

1. 77.63% of the total sample sizes are aware of sexually transmitted diseases.
2. The females are more aware than the males. 81.1% of the female population are aware of the diseases while the male percentage is 74%.
3. 45.9% of the total sample size is aware of other sexually transmitted diseases apart from AIDS. 10.8% of the respondents are aware of all the diseases cited in the questionnaire. The male respondents are better informed of STDs other than HIV/AIDS (mostly gonorrhoea).
4. 39.8% males have attended awareness programmes and 48.8% of the females have attended the same. As a whole 44.4% of the respondents have attended awareness programmes/camps. Thus here we can easily conclude that the females are better aware than males though the figures are not satisfactory.
5. The best way to communicate with the masses is through the medium television. 59.1% of the respondents find television most appealing.
6. In the present investigation we find that 77.6% of the respondents say that are aware of sexually transmitted diseases and 73.3% of the respondents say that they would accept a HIV+/STD patient into the society. This shows that validity of the present investigation and we can safely conclude that more that 70% of the people in the state are aware of the diseases.
7. The age group of 15-35 has been identified as the major risk group in the present investigation. This group has higher chances of contacting the diseases. This conclusion has been drawn taking into consideration the sexual characteristics of the people.
8. It is also observed that all the 13 respondents who admitted of being infected by any STDs had practised unprotected sex once or more than once in their life, thus hinting that condoms are actually effective against the diseases.
9. The overall awareness is very good in the state with all the districts except the south district showing approximately 80% awareness.
10. Education definitely has a positive impact on the awareness and thus the control of the diseases. Higher the educational qualification better is the knowledge about sexually transmitted diseases and better is the sexual habits of the people.
11. Many people go for self-treatment or any traditional methods of treatment thus making the controlling and monitoring of the diseases difficult.
12. The percentage of protected sexual habits is unsatisfactory for both male and females. Thus the use of condoms/other protective measures is not being well practiced in the state probably due to reluctance to purchase them or the unavailability during the time of intercourse.
13. The secondary data collected from various sources pertaining to Sexually Transmitted Diseases cannot be considered authentic due to already mentioned reasons and various other reasons.

RECOMMENDATIONS

1. Hospitals to maintain proper reports on STDs. The data pertaining to STDs from the Sikkim State AIDS control society were found to be inadequate to define the present status of the diseases.
2. Private clinics should be asked to submit their monthly reports on the number of STD patients treated to the respective department to help better monitoring of the disease in the state. Also nearby private clinics outside the state should also be requested for database for such diseases to have proper track of disease, if norms provide..
3. A more intense campaign for the awareness of STDs other than AIDS to be launched in the state. It was notices that less importance was being given to other STDs like Gonorrhoea, Syphilis, herpes, etc.
4. The awareness programmes can be aired on local T.V channels to reach the masses.
5. Sex education a must at school level so that people don't hesitate to speak about these diseases.
6. The voluntary confidential counselling and testing centres (VCCTC) should be more localised as most of the youths admitted that they fear themselves to be infected by HIV or other STDs but are also scared of the social dilemma and are not aware of VCCTC. There are precisely just three voluntary confidential counselling and testing centres in the state.
7. A compulsory education module can be started at high school level about various diseases like tuberculosis and sexually transmitted diseases.
8. Awareness programmes should be held keeping in mind the various risk groups and their free time. For examples taxi drivers cannot attend the programmes during the day so a suitable time must be fixed for them to attend the programmes.
9. NGO's must be encouraged.
10. The major risk groups must be actively involved in the awareness campaigns so that they acquire a first hand knowledge about the STDs.
11. The male population of the east district and the female population of the west show quite low awareness with 28.8 % and 27.9% awareness respectively and need immediate attention.
12. An instant check must be put to the circulation of pornographic movies and literatures, which often becomes responsible for indiscriminate indulgence in sex and consequently STDs.
13. The rate of distribution of condoms must be immediately increased and should be done at random amongst the various risk groups.

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