HIV / AIDS

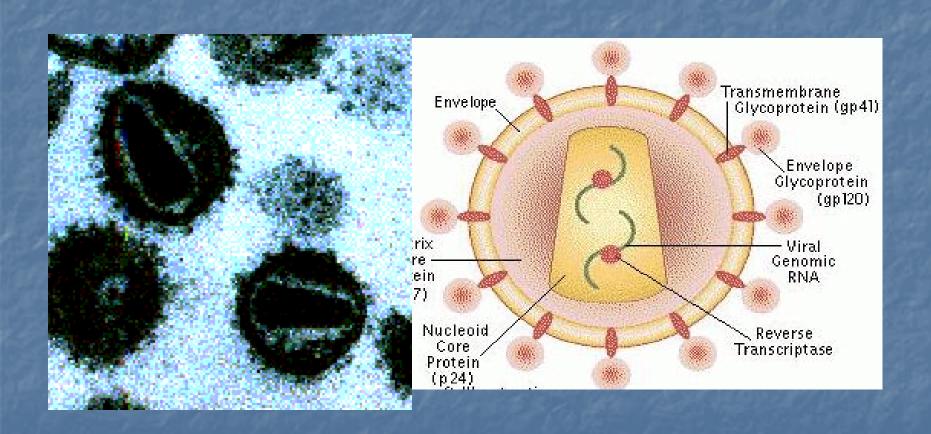
HIV = HUMAN
IMMUNODEFICIENCY VIRUS

AIDS = ACQUIRED IMMUNE DEFICIENCY SYNDROME

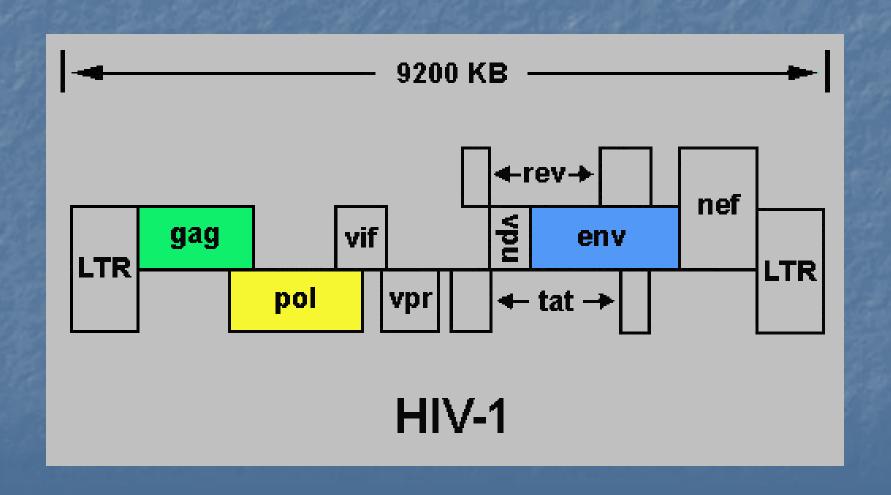
Human Immunodeficiency Virus

- Acquired Immunodeficiency syndrome first described in 1981
- HIV-1 isolated in 1984, and HIV-2 in 1986
- Belong to the lentivirus subfamily of the retroviridae
- Enveloped RNA virus, 120nm in diameter
- HIV-2 shares 40% nucleotide homology with HIV-1
- Genome consists of 9200 nucleotides (HIV-1):
- gag core proteins p15, p17 and p24
- pol p16 (protease), p31 (integrase/endonuclease)
- env gp160 (gp120:outer membrane part, gp41: transmembrane part)
- Other regulatory genes ie. tat, rev, vif, nef, vpr and vpu

HIV particles



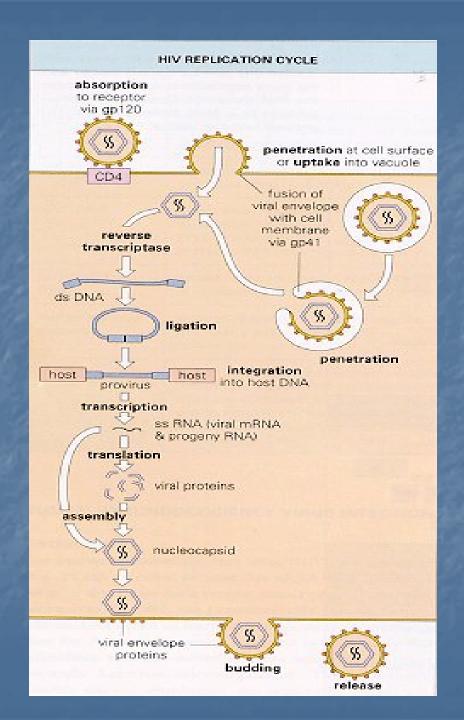
HIV Genome



Replication

- The first step of infection is the binding of gp120 to the CD4 receptor of the cell, which is followed by penetration and uncoating.
- The RNA genome is then reverse transcribed into a DNA provirus which is integrated into the cell genome.
- This is followed by the synthesis and maturation of virus progeny.

Schematic of HIV Replication



WORST CASE SCENARIO

- HIV CAUSES A SLOW, PROGRESSIVE AND PERMANENT DISEASE WITH NO RECOVERY, NO LOSS OF INFECTIVITY, NO DEVELOPMENT OF EITHER INDIVIDUAL OR GROUP IMMUNITY.
- THERE IS AT PRESENT, NO KNOWN BIOLOGICAL MECHANISM WHICH CAN STOP THE CONTINUING EXPANSION OF

- THE DISEASE UNLESS AN EFFECTIVE VACCINE WERE TO BECOME AVAILABLE, AND AT PRESENT THERE IS NO FEASIBLE DESIGN FOR SUCH ANEFFECTIVE VACCINE.
- THE PROGRESSIVE INCREASE IN THE POOL OF HIV CAN, IN THEORY, ONLY LEAD TO AN EXPONENTIAL INCREASE IN THE NUMBER OF INDIVIDUALS WHO WILL BECOME INFECTED UNTIL EVENTUALLY THE MAJORITY OF THE SEXUALLY ACTIVE POPULATION WILL BE INFECTED.

AIDS IS NOT A DISEASE BUT A SYNDROME AND IS NOT TRANSMITTED. PEOPLE DO NOT DIE OF AIDS. THEY DIE OF OPPORTUNISTIC INFECTIONS, CANCERS, AND ORGAN FAILURES BROUGHT ON BY A FAILED IMMUNE SYSTEM.

BECAUSE PERSONS CAN EXPRESS AIDS FOR REASONS OTHER THAN BECOMING HIV INFECTED, WE ARE ONLY GOING TO DISCUSS THAT FORM OF AIDS BROUGHT ON BY HIV AND REFERRED TO AS HIV/AIDS. IT COULD ALSO BE REFERRED TO AS HIV DISEASE OR HIV T4 HELPER CELL DISEASE, BECAUSE IT IS THE T4 HELPER CELLS THAT ARE EVENTUALLY DESTROYED.

IT IS IMPORTANT TO DISTINGUISH BETWEEN HIV INFECTION, HIV DISEASE AND AIDS. HIV INFECTION IS THE INITIAL STAGE WHEN HIV TAKES UP RESIDENCE IN THE BODY. THE CONTINUED PRESENCE OF HIV MAY EVENTUALLY CRIPPLE THE BODY'S ABILITY TO FIGHT OFF DISEASES AND IS REFERRED TO AS HIV DISEASE. AIDS IS THE END RESULT OF HIV DISEASE.

Clinical Features

- 1. Seroconversion illness seen in 10% of individuals a few weeks after exposure and coincides with seroconversion. Presents with an infectious mononucleosis like illness.
- 2. Incubation period this is the period when the patient is completely asymptomatic and may vary from a few months to a more than 10 years. The median incubation period is 8-10 years.
- 3. AIDS-related complex or persistent generalized lymphadenopathy.
- 4. Full-blown AIDS.

PRIMARY INFECTION (CATEGORY A) ACUTE CLINICAL ILLNESS RESEMBLING INFLUENZA OR MONONUCLEOSIS WITH AN ERYTHEMATOUS RASH THAT OCCURS IN ABOUT 50% OF CASES 1 – 4 WEEKS AFTER INFECTION. ASYMPTOMATIC HIV INFECTION NEVER CLEARS. THERE MAY BE SOME LYMPHADENOPATHY.

AFTER PRIMARY INFECTION THERE IS A PERIOD RANGING FROM A FEW MONTHS TO MORE THAN 10 YEARS WITH NO OR MILD SYMPTOMS BEFORE THE APPEARANCE OF SEVERE IMMUNODEFICIENCY.

HIV-RELATED CLINICAL SYMPTOMS (CATEGORY B) THE FOLLOWING SYMPTOMS OF DEFECTS IN CELL MEDIATED IMMUNITY OCCUR: ORAL CANDIDIASIS, HAIRY LEUKOPLAKIA, AND SHINGLES. NIGHT SWEATS, FATIGUE AND MALAISE.

AIDS (CATEGORY C) THE AVERAGE TIME TO AIDS IS 7-11 YEARS. AIDS IS ONE LIFE-THREATENING OPPORTUNISTIC INFECTION OR KAPOSI'S SYMPTOMS INCLUDE FEVER, DIARRHEA, SARCOMA PLUS BEING HIV POSITIVE. THE AVERAGE SURVIVAL TIME AFTER THE DIAGNOSIS OF AIDS HAS BEEN MADE IS ABOUT 1 YEAR IF NO ANTIVIRAL THERAPY IS GIVEN.

HIV Pathogenesis

- The profound immunosuppression seen in AIDS is due to the depletion of T4 helper lymphocytes.
- In the immediate period following exposure, HIV is present at a high level in the blood (as detected by HIV Antigen and HIV-RNA assays).
- It then settles down to a certain low level (set-point) during the incubation period. During the incubation period, there is a massive turnover of CD4 cells, whereby CD4 cells killed by HIV are replaced efficiently.
- Eventually, the immune system succumbs and AIDS develop when killed CD4 cells can no longer be replaced (witnessed by high HIV-RNA, HIV-antigen, and low CD4 counts).

- IN ORDER TO BE CONSIDERED POSITIVE FOR HIV/AIDS A PERSON MUST
- 1. TEST POSITIVE FOR HIV AND
- 2. HAVE ONE OR MORE OF THE FOLLOWING 26 CONDITIONS OR DISEASES:

- 1. CANDIDIASIS OF BRONCHI, TRACHEA, OR LUNGS
- 2. CANDIDIASIS, ESOPHAGEAL
- 3. CERVICAL CANCER, INVASIVE
- 4. COCCIDIOIDOMYCOSIS, DISSEMINATED
 OR EXTRAPULMONARY
- 5. CRYPTOCOCCOSIS, EXTRAPULMONARY

- 6. CRYPTOSPORIDIOSIS, CHRONIC INTESTINAL (>1 MONTH DURATION)
- 7. CYTOMEGALOVIRUS DISEASE (OTHER THAN LIVER, SPLEEN, OR NODES)
- 8. CYTOMEGALOVIRUS RETINITIS (WITH LOSS OF VISION)
- 9. HIV ENCEPHALOPATHY
- 10. HERPES SIMPLEX CHRONIC ULCERS (>1 MONTH DURATION) OR BRONCHITIS, PNEUMONITIS, OR ESOPHAGITIS

- 11. HISTOPLASMOSIS, DISSEMINATED OR EXTRAPULMONARY
- 12. ISOSPORIASIS, CHRONIC INTESTINAL (>1 MONTH DURATION)
- 13. KAPOSI'S SARCOMA
- 14. LYMPHOMA, BURKITT'S
- 15. LYMPHOMA, IMMUNOBLASTIC
- 16. LYMPHOMA, PRIMARY IN BRAIN
- 17. MYCOBACTERIUM AVIUM COMPLEX OR M. KANSASII DISSIMINATED OR EXTRAPULMONARY

- 18. MYCOBACTERIUM TUBERCULOSIS, DISSEMINATEDOR EXTRAPULMONARY
- 19. MYCOBACTERIUM TUBERCULOSIS ANY SITE
- 20. MYCOBACTERIUM ANY OTHER SPECIES DISSEMINATED OR EXTRAPULMONARY
- **21.** *PNEUMOCYSTIS JIROVECI* PNEUMONIA
- 22. PNEUMONIA, RECURRENT

- 23. PROGRESSIVE MULTIFOCAL LEUKOENCEPHALOPATHY
- 24. SALMONELLA SEPTICEMIA, RECURRENT
- **25.** TOXOPLASMOSIS OF THE BRAIN
- 26. WASTING SYNDROME DUE TO HIV

OR

HAVE A T4 LYMPHOCYTE COUNT OF LESS THAN 200 PER MICROLITER OF BLOOD (<200/uL) OR A T4 LYMPHOCYTE PERCENT LESS THAN 14% OF TOTAL LYMPHOCYTES.

- MANY HIV INFECTED PEOPLE CAN LIVE 10 YEARS OR LONGER WITHOUT EXPERIENCING ILLNESS.
 AND USING THE NEW ANTI-HIV DRUGS, PEOPLE MAY LIVE 20 OR MORE YEARS AFTER INFECTION
- IT IS BELIEVED THAT EVENTUALLY ANYONE WHO IS CORRECTLY DIAGNOSED WITH HIV/AIDS WILL DIE OF IT. BUT ALL WHO BECOME HIV-INFECTED MAY NOT PROGRESS TO AIDS. ESTIMATES ARE THAT SOME 5% OF THE HIV-INFECTED POPULATION WILL NOT PROGRESS TO AIDS.

- FACTS ABOUT AIDS
- 1. AIDS IS A PANDEMIC.
- 2. DURING THE 22 YEARS SINCE HIV/AIDS WAS DEFINED AS A NEW DISEASE, MORE MANPOWER AND MONEY HAS BEEN POURED INTO HIV/AIDS RESEARCH THAN INTO ANY OTHER DISEASE IN HISTORY.
- 3. INFECTION BY HIV CAN BE PREVENTED.

- 4. WORLDWIDE, THE VIRUS IS PRIMARILY SPREAD THROUGH HETEROSEXUAL CONTACT.
- 5. ASYMPTOMATIC INFECTED PERSONS CAN INFECT OTHERS.
- 6. HIGH HIV INFECTION RISK IS ASSOCIATED WITH HAVING MANY SEXUAL PARTNERS.
- 7. HIV IS FRAGILE AND EASILY DESTROYED BY ENVIRONMENTAL AGENTS WHEN OUTSIDE THE HUMAN BODY.

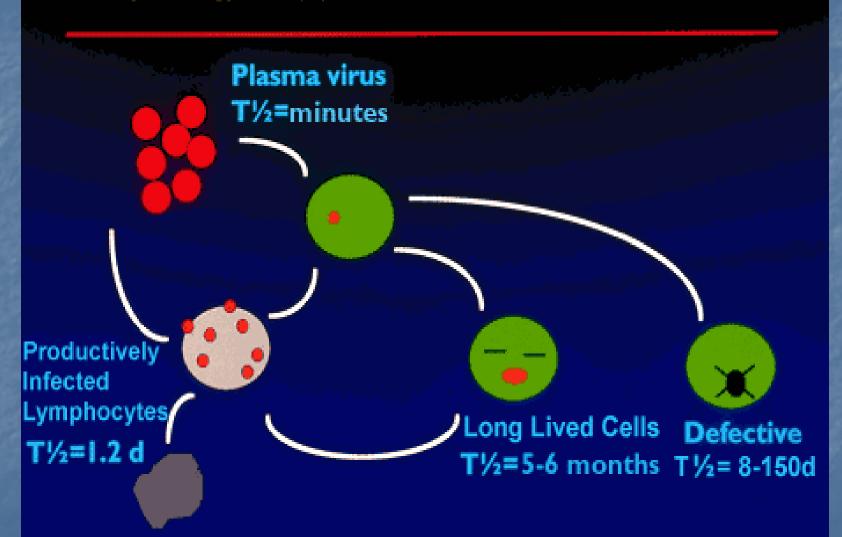
- 8. CASUAL CONTACT DOES NOT SPREAD HIV.
- 9. HIV IS NOT TRANSMITTED TO HUMANS VIA INSECTS.
- 10. THE CURRENT RISK OF ACQUIRING HIV VIA BLOOD TRANSFUSIONS IN THE U.S. IS LOW.
- 11. THERE IS NO CURE FOR HIV DISEASE OR AIDS.
- 12. IN THE U.S. ABOUT 57% OF ALL PERSONS EXPRESSING AIDS HAVE DIED; WORLDWIDE IT IS ABOUT 60%.

- 13. AIDS MAY BE CLOSE TO 100% LETHAL SOONER OR LATER A PERSON WHO EXPRESSES AIDS DIES PREMATURELY.
- 14. IT IS BELIEVED THAT THE MAJORITY OF THOSE INFECTED WITH HIV WILL EVENTUALLY PROGRESS TO AIDS.
- 15. THERE ARE PEOPLE WHO ARE GENETICALLY RESISTANT TO HIV INFECTION.

HIV half-lives

- Activated cells that become infected with HIV produce virus immediately and die within one to two days.
- Production of virus by short-lived, activated cells accounts for the vast majority of virus present in the plasma.
- The time required to complete a single HIV life-cycle is approximately 1.5 days.
- Resting cells that become infected produce virus only after immune stimulation; these cells have a half-life of at least 5-6 months.
- Some cells are infected with defective virus that cannot complete the virus life-cycle. Such cells are very long lived, and have an estimated half-life of approximately three to six months.
- Such long-lived cell populations present a major challenge for antiretroviral therapy.

HIV-I half-lives



HISTORY OF AIDS IN THE UNITED STATES

THE FIRST RECORDED AIDS CASE IN AMERICA WAS THAT OF A 15-YEAR-OLD MALE PROSTITUTE WHO DEMONSTRATED KAPOSI'S SARCOMA AND DIED IN 1969. FROZEN TISSUE SAMPLES CONTAINED HIV ANTIBODIES.

In the second of the second of

1981 NEW YORK CITY, NEW YORK 26 GAY YOUNG MEN DEVELOPED KAPOSI'S SARCOMA (KS)

HISTORY OF AIDS IN AFRICA

THE EARLIEST KNOWN CASE OF HIV/AIDS OCCURRED IN AN AFRICAN MALE IN 1959. HE WAS A BANTU LIVING IN LEOPOLDVILLE, BELGIAN CONGO -WHAT IS NOW KINSHASA, REPUBLIC OF CONGO. IT WAS IDENTIFIED IN BLOOD SAMPLES THAT HAD BEEN SAVED.

- IN AFRICA TO BE THE RESULT OF RECENT IMPORTATION FROM OUTSIDE; THE AFRICAN EPIDEMIC CLEARLY PRECEDES THE WESTERN ONE.
- IT PROBABLY GOT INTO PEOPLE IN THE 1940'S OR EARLY 50S.

- HIV-1 IS FOUND IN EAST AND CENTRAL AFRICA, EUROPE, NORTH AMERICA, SOUTH AMERICA AND ASIA WAS FIRST ISOLATED IN 1983.
- RECENT EVIDENCE CLEARLY INDICATES THAT HIV-1 EVOLVED IN A SUBSPECIES OF CHIMPANZEE AND WAS PRESENT IN THAT SUBSPECIES FOR CENTURIES. IT "JUMPED" SPECIES TO HUMANS - PROBABLY BY BLOOD CONTAMINATION OF AN OPEN WOUND DURING BUTCHERING OF THE ANIMALS FOR FOOD.

EVOLUTION OF HIV

- DATA SUGGEST THAT HIV BEGAN AS ONE INTRODUCTION OF HIV INTO PEOPLE RATHER THAN MANY CROSSOVERS FROM ANIMAL TO HUMANS.
- HIV HAS MUTATED OVER THE YEARS TO FORM 11 DISTINCT SUBTYPES, LETTERED A THROUGH K.

SUBTYPE B IS THE DOMINANT STRAIN IN THE UNITED STATES AND EUROPE, WHILE SUBTYPE D IS THE MOST COMMON IN AFRICA. HIV-2 IS FOUND IN WEST AFRICA AND WAS FIRST ISOLATED IN 1985. IT IS FOUND MOSTLY IN PROSTITUTES AND RESEMBLES A CLOSELY RELATED MONKEY VIRUS (ISOLATED FROM A SPECIES OF WEST AFRICAN MONKEY, THE SOOTY MANGABEY), SIMIAN IMMUNODEFICIENCY VIRUS (SIVsm), AND PROBABLY AROSE FROM IT. IT MAY HAVE INFECTED HUMANS 20 TO 30 YEARS AGO. THERE HAVE BEEN MANY REPORTED CASES OF TYPE-2 IN THE UNITED STATES AND EUROPE.

Estimated number of HIV infected people alive: to end 1998 by region (percentage of total of 33.4m)



CDSC Source: UNAIDS

HIV INFECTIONS 2003 WORLDWIDE (WHO)

- 1. AT THE END OF 2003, IT IS ESTIMATED THAT THERE WILL HAVE BEEN AT LEAST 70 MILLION PEOPLE INFECTED WORLDWIDE WITH HIV. 44 MILLION PEOPLE WILL STILL BE ALIVE - 26 MILLION WILL HAVE ALREADY DIED. 50% OF THOSE INFECTED WILL BE WOMEN.
- 2. AFRICA:
- NORTH AFRICA & MIDDLE EAST
- 519,200
- SUB-SAHARAN AFRICA
- 31 MILLION +

- 3. LATIN AMERICA1, 892,000
 - 4. THE CARIBBEAN 484,000
- 5. ASIA:
- EAST ASIA & PACIFIC 1.1 MILLION
- SOUTH AND SOUTH-EAST ASIA
 7.3 MILLION
- AUSTRALIA AND NEW ZEALAND14,000

- 6. NORTH AMERICA1,074,000
- 7. WESTERN EUROPE616,000
- 8. EASTERN EUROPE AND CENTRAL ASIA1.2 MILLION

SUB-SAHARAN AFRICA

- 1 IN 11 ADULTS INFECTED WITH HIV58% ARE WOMEN
- IN BOTSWANS, LESOTHO, NAMBIA, ZAMBIA, ZIMBABWE, AND SWAZILAND 1 IN 5 ADULTS AGED 15 TO 49 HAVE HIV.
- SOUTH AFRICA BIGGEST PROBLEM -1,600 NEW INFECTIONS / DAY – IN 5 YEARS, 15% OF POPULATION WILL BE INFECTED.

■ INCIDENCE OF AIDS IN THE UNITED STATES - 2002

•	GROUP	PERCENTAGE
_	1. MEN WHO HAVE SEX WITH MEN	46
•	2. INJECTION DRUG USERS (IDU)	25
	3. HOMOSEXUAL/IDU	5
	4. HEMOPHILIAC	0.2
-	5. HETEROSEXUAL CONTACT	11
_	6. TRANSFUSION RELATED	0.4
	7. NONE OF THE ABOVE	11

- THERE HAVE NEVER BEEN ANY DOCUMENTED CASES OF HIV/AIDS BY:
- 1. TEARS
- 2. SWEAT
- 3. COUGHING AND SNEEZING
- 4. CLOTHING
- 5. TELEPHONES

- 6. TOILET SEATS
- 7. EATING UTENSILS
- 8. DRINKING GLASSES
- 9. DAILY CONTACT WITH INFECTED WORKER
- 10. DAILY CONTACT WITH INFECTED STUDENT
- 11. INSECTS
- 12. CPR

NUMBER OF HIV VIRUSES NEEDED TO ESTABLISH AN INFECTION

HUMAN TISSUE CULTURE CELLS = APPROXIMATELY 1,000

PERSON = APPROXIMATELY 10,000 to 15,000

EXAMPLE

- HEPATITIS B VIRUS
- ONE MILLILITER INTO A SWIMMING POOL (APPROXIMATELY 24,000 GALLONS OF WATER), INJECT ONE MILLILITER INTO PERSON THERE IS A VERY GOOD CHANCE THAT DISEASE WILL DEVELOP
- HIV
- ONE MILLILITER INTO A QUART OF WATER, INJECT ONE MILLILITER INTO PERSON THERE IS A 10% CHANCE THAT DISEASE WILL DEVELOP

MYTHS ABOUT THE ORIGIN OF THE AIDS VIRUS

■ 1. UFO'S

2. BIOLOGICAL WARFARE RESEARCH

3. DOMESTIC CAT

4. THE CIA

- HIV IS PRESENT IN:
- *1. BLOOD

*2. SEMEN

*3. VAGINAL SECRETIONS

*4. CEREBROSPINAL FLUID

*5. BREAST MILK

6. URINE

7. TEARS

8. SALIVA

9. LUNG FLUIDS

* HIGH NUMBERS

TRANSMISSION

- **1. BLOOD**
- A. BLOOD TRANSFUSION
- B. NEEDLE SHARING (IV DRUG USE)
- C. ACCIDENTAL NEEDLE STICKS (HEALTH CARE WORKERS)
- D. MUCOUS MEMBRANE EXPOSURE (HEALTH CARE WORKERS)
- E. TATTOOS AND PIERCING (EARS, NOSE, TONGUE, NAVEL, ETC.)
- F. INJECTIONS WITH UNSTERILIZED NEEDLES
- G. ACUPUNCTURE NEEDLES

- 2. SEXUAL CONTACT
- A. HOMOSEXUAL
- B. BISEXUAL
- C. HETEROSEXUAL
- **3. PERINATAL**
- **A. WITHIN THE UTERUS**
- B. DURING BIRTH
- C. SHORTLY AFTER BIRTH (BREAST FEEDING)
- **4. ORGAN TRANSPLANTS**

AIDS AND HEALTH CARE WORKERS

- HIV/AIDS
- WAS TRANSMITTED BY ONE HEALTH CARE PROVIDER
- DENTIST DAVID J. ACER Stuart, Florida Died September 1990
- PATIENT KIMBERLY BERGALIS Died December 1991. At least 4 other patients also developed HIV/AIDS.

Laboratory Diagnosis

- Serology is the usual method for diagnosing HIV infection. Serological tests can be divided into screening and confirmatory assays. Screening assays should be as sensitive whereas confirmatory assays should be as specific as possible.
- Screening assays EIAs are the most frequently used screening assays. The sensitivity and specificity of the presently available commercial systems now approaches 100% but false positive and negative reactions occur. Some assays have problems in detecting HIV-1 subtype O.
- Confirmatory assays Western blot is regarded as the gold standard for serological diagnosis. However, its sensitivity is lower than screening EIAs. Line immunoassays incorporate various HIV antigens on nitrocellulose strips. The interpretation of results is similar to Western blot it is more sensitive and specific.

ELISA for HIV antibody



Microplate ELISA for HIV antibody: coloured wells indicate reactivity

Western blot for HIV antibody



Figure:

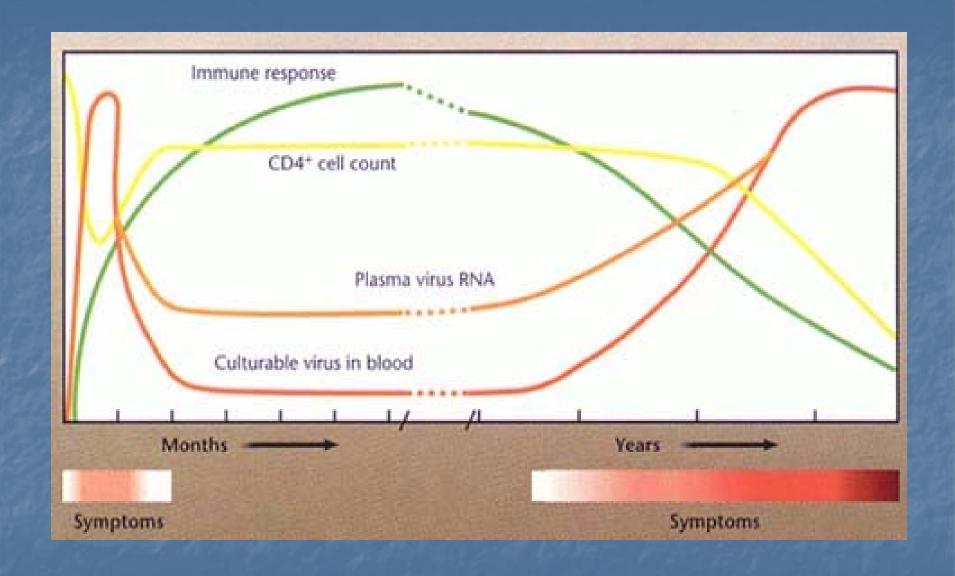
Examples of reactions by an HIV-1 Western blot:

- Positive control (strong)
- Positive control (weak)
- 3. Negative control
- 4. Indeterminate profile
- Indeterminate profile (highly suggestive)

- There are different criteria for the interpretation of HIV Western blot results e.g. CDC, WHO, American Red Cross.
- The most important antibodies are those against the envelope glycoproteins gp120, gp160, and gp41
- p24 antibody is usually present but may be absent in the later stages of HIV infection

Other diagnostic assays

- It normally takes 4-6 weeks before HIV-antibody appears following exposure.
- A diagnosis of HIV infection made be made earlier by the detection of HIV antigen, pro-DNA, and RNA.
- However, there are very few circumstances when this is justified e.g. diagnosis of HIV infection in babies born to HIV-infected mothers.



NUCLEOSIDE REVERSE TRANSCRIPTASE INHIBITORS

- a.* zidovudine (ZDV) (RETROVIR) (1987) formerly azidothymidine (AZT)
- b.* didanosine (VIDEX) (1991)
 formerly dideoxyinosine (ddl)
- c.* zalcitabine (HIVID) (1994)
 formerly dideoxycytidine (ddC)
 - d.* stavudine (ZERIT) formerly d4T (1994)

e.* lamivudine (3TC; EPIVIR) (1995)

f.* abacavir (ZIAGEN) (1998)

g. tenofovir (VIREAD) (2001)

- h.* lamivudine/zidovudine (COMBIVIR)
- i. zidovudine/limivudine/abacavir (TRIZIVIR)

NON-NUCLEOSIDE REVERSE TRANSCRIPTASE INHIBITORS

a.* nevirapine (VIRAMUNE) (1996)

b.* delavirdine mesylate (RESCRIPTOR) (1997)

c.* efavirenz (SUSTIVA) (1998)

PROTEASE INHIBITOR DRUGS

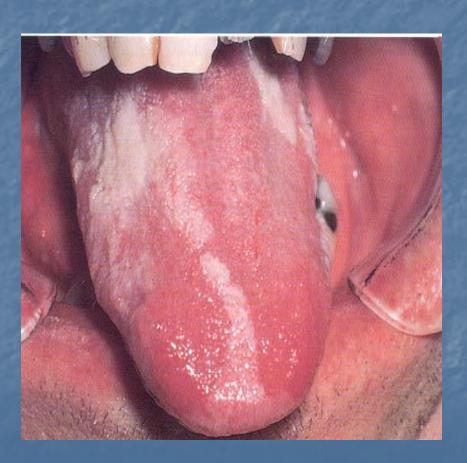
- a.* saquinavir mesylate (INVIRASE) (1995)
- b.* ritonavir (NORVIR) (1996)
- c.* indinavir (CRIXIVAN) (1996)
- d.* nelfinavir (VIRACEPT) (1997)

e*. sequinavir (FORTOVASE) (1997)

f.* amprenavir (AGENERASE) (1999)

g.* lopinavir (KALETRA) (2000)

ORAL MANIFESTATIONS OF HIV INFECTION.



 Oral hairy leukoplaxia due to Epstein-Barr virus infection on the dorsal surface of the tongue is shown.

ORAL MANIFESTATIONS OF HIV INFECTION



The pseudomembranous form of oral candidiasis also known as thrush is shown



Multiple elongated and irregularly shaped violet-to-brown plaque lesions of Kaposi's sarcoma on the upper extremities are shown.



 A 60-year-old homosexual Hispanic man presented with patch and plaque lesions on his face, some of which were developing into nodules.

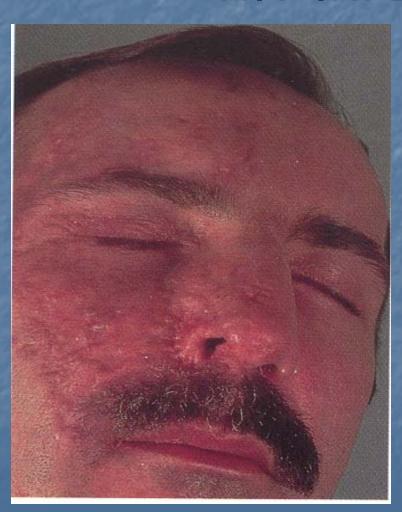


A deep brown nodule Kaposi's sarcoma on the lateral shaft of the penis is shown.



A long- standing case of Kaposi's sarcoma affecting the lower extremitiy with ulceration of the tumor on the arch of the foot. There are rounded nodules extending over the ankle.

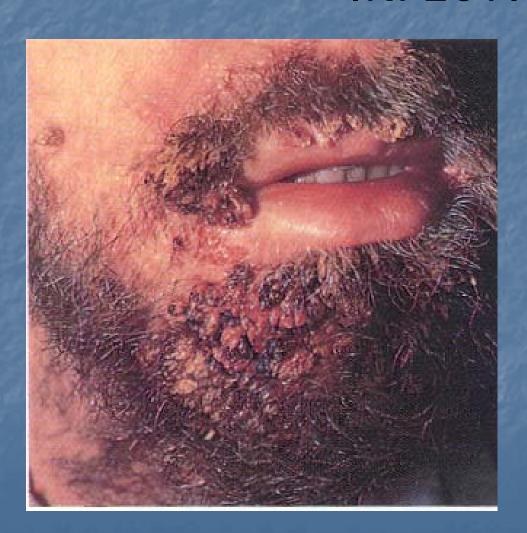
CUTANEOUS MANIFESTATIONS OF HIV INFECTION



Herpes zoster. Severe residual scarring of the face occurring as a sequela to herpes zoster. This complication is more frequent in patients with AIDS.



Necrotizing nonhealing perianal ulcerations in this case were due to herpes simplex virus infection and infection with cytomegalovirus in a patient with AIDS.



Extensive human papillomavirus infection of the bearded area of the face in a patient with AIDS.



Condylomata
 acuminata. Multiple
 vegetating
 coalescing
 condylomata in the
 perianal area of a
 patient with AIDS.

EPIDERMODYSPLASIA VERRUCIFORMIS IN AIDS

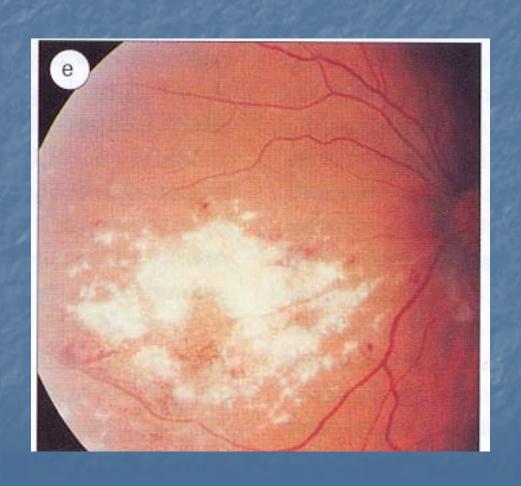


This is a manifestatiom of a widespread human papillomavirus infection that is associated with the development of squamous cell carcinoma.



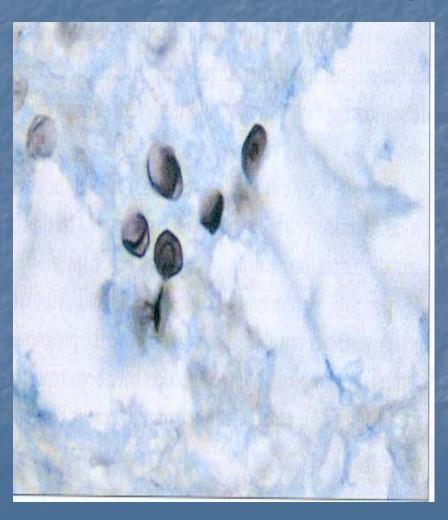
Numerous large umbilicated waxy papules of molluscum contagiosum are seen on the face of a patient with AIDS. They have coalesced to form crusted plaques.

OCULAR COMPLICATIONS OF HIV INFECTION



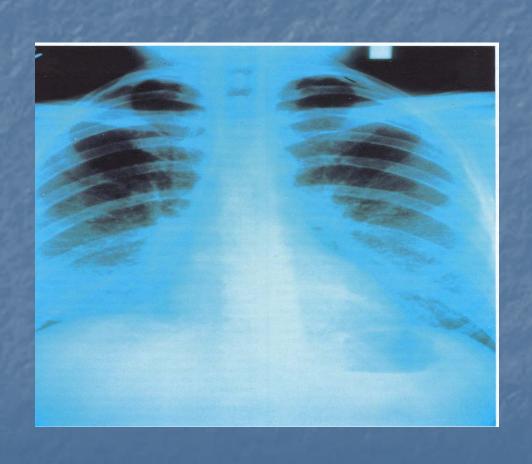
 Cytomegalovirud retinitis showing scattered exudates and hemorrhages, with sheathing of vessels.

CLINICAL MANIFESTAIONS OF HIV INFECTION



Darkly staining cysts of Pnemocysyis carinii in an open lung biopsy from an AIDS patient with pneumonia. silver stain.

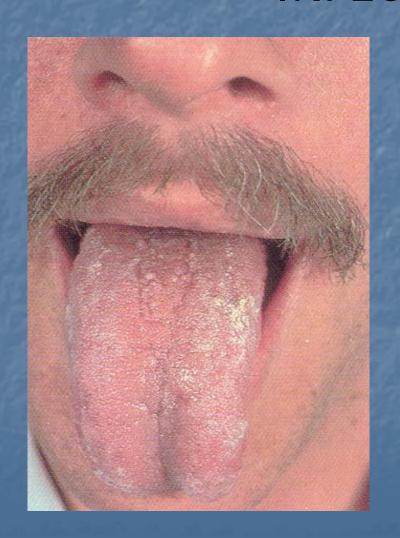
CLINICAL MANIFESTATION OF HIV INFECTION



Chest
 radiograph
 showing
 Pneumocystis
 pneumonia
 with extensive
 infiltrates in
 both lungs.



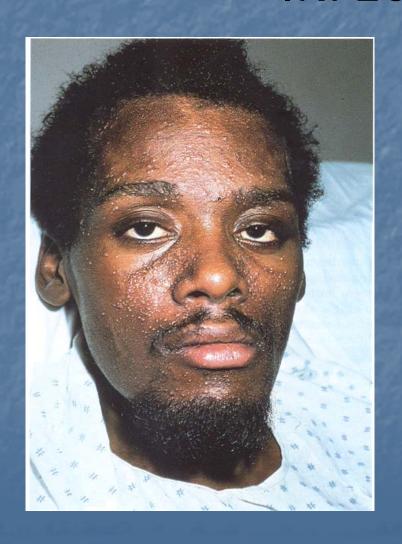
Cutaneous **Pneumocystis** carinii infection. There are scattered translucent papules present on the trunk and extremities.



Candidiasis. A whitish, curd-like exudate present on the tongue may be an indication that the patient will develop AIDS.



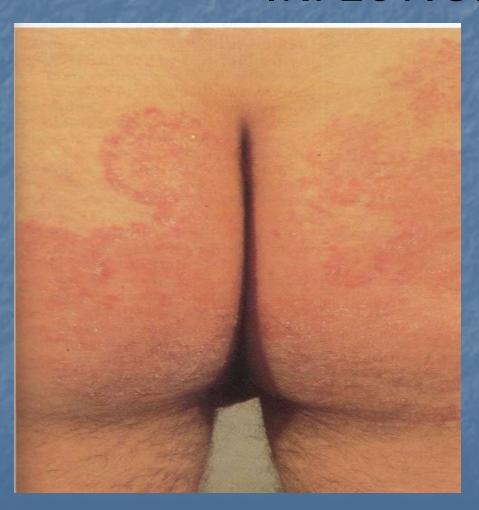
Chronic
 Mucocutaneous
 candidiasis seen in
 patient with HIV
 infection. Note the
 extensive crusting
 and scaling.



Cryptococcosis. Note crusted umbilicated follicular and perifollicular papules.
 They are often multiple and may tend to confluence.

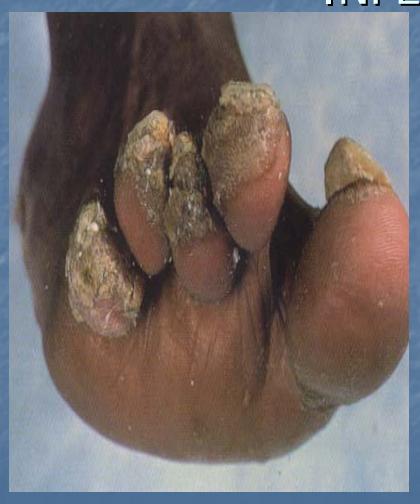


 Histoplasma capsulatum infection. Note the widespread papules in this AIDS patient.



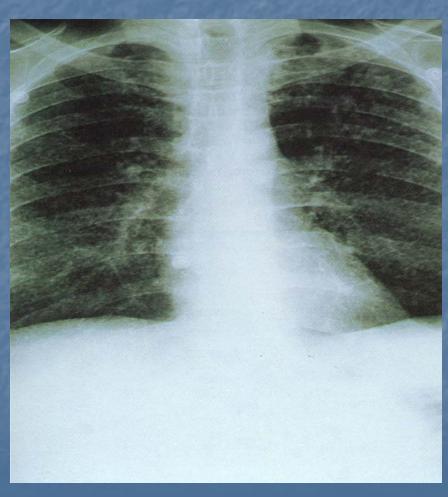
 Common dermatophyte infection in patient with HIV infection or AIDS.

CLINICAL MANIFESTATIONS OF HIV INFECTION



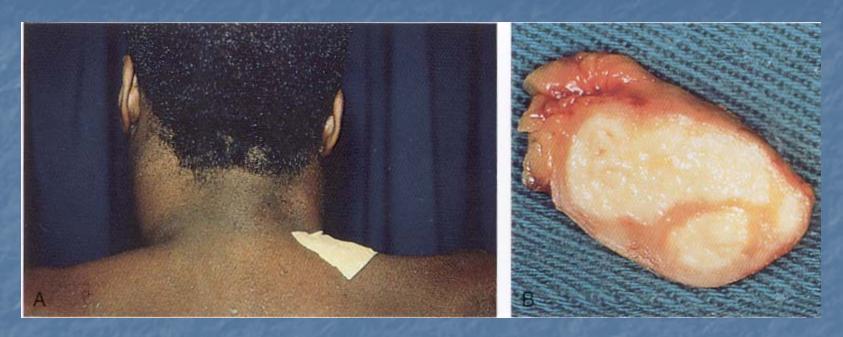
Onychomycosis.
 Severe
 onchomycosis with
 marked subungual
 hyperkeratosis and
 nail dystrophy may
 be seen in patients
 with AIDS.

OPPORTUNISTIC INFECTION IN PATIENT WITH HIV INFECTION.



 Tuberculosis may manifest itself as miliary tuberculosis

TUBERCULOSIS ADENITIS

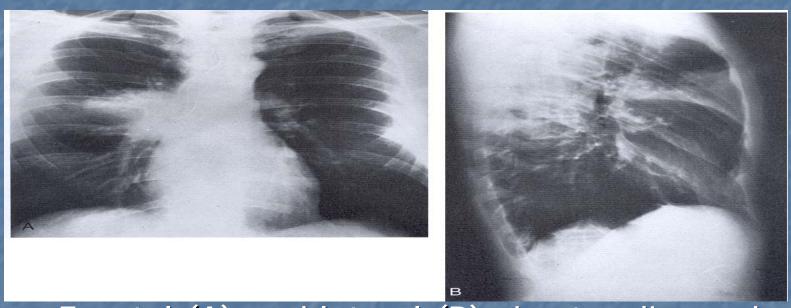


A 26-year-old HIV-infected Zairian man with a large cervical polyadenopathy. A lymph node biopsy from the patient is also shown. The lymph node is full, of caseum because of M. tuberculosis infection.



Bacillary angiomatosis. This rare and unusual vascular proliferation is sometimes seen in patients with AIDS or HIV infection.

OPPORTUNISTIC INFECTION OF PATIENT WITH HIV INFECTION



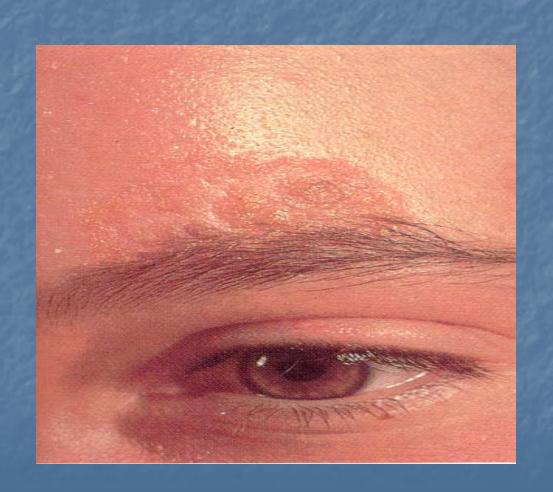
Frontal (A) and lateral (B) chest radiographs show an infiltration in the anterior segment of the right upper lobe caused by M. avium complex.

IMPETIGO



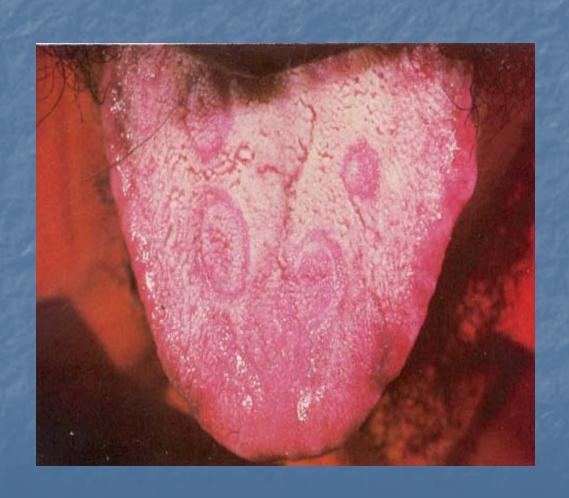
Impetigo may be widespread and fulminant in patients with HIV infection or AIDS. Note: impetigo is caused by staphylococci, streptococci or a combination of both.

IMPETIGO



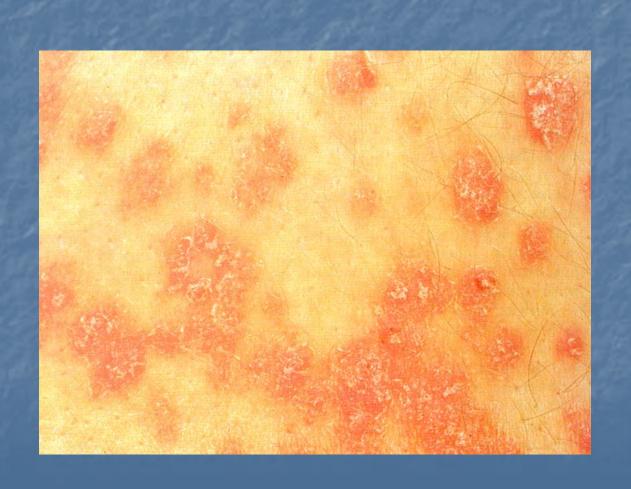
 The characteristic erosion covered by a honeycolored crust is pictured here.

SECONDARY SYPHILIS



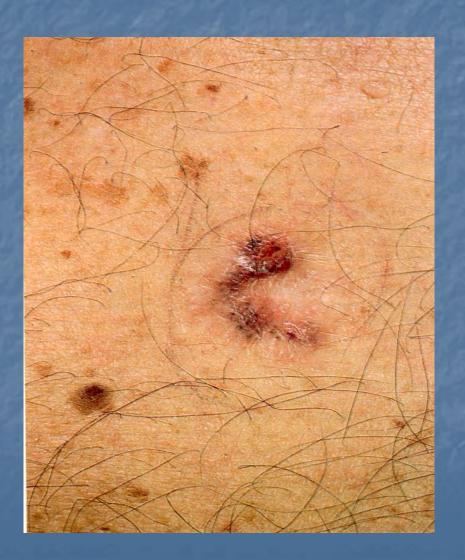
Multiple mucous patches as well as "coated" tongue in an HIV-infected person with secondary syphilis.

ERUPTIVE PSORIASIS

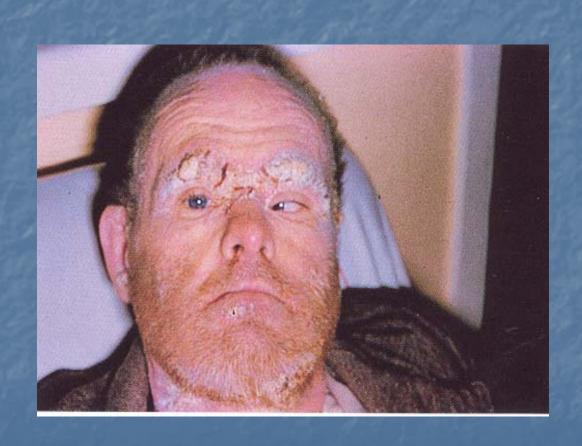


 Close up view of eruptive psoriasis in a patient with AIDS.

BASAL CELL CARCINOMA



This multifocal pigmented basal cell carcinoma is another example of the type of lesions that may be seen in patients with HIV infection.



Norwegian scables. These crusted plaques were found to have innumerable mites of Sarcoptes scabei.