II. Viruses
   A. Viruses are obligate parasites that are not cells or organisms.
      1. The viral genome can be either DNA or RNA.
      2. Viruses, plasmids, and transposons may be related in that all are mobile genetic elements.
         a. Many viruses may be derived from "escaped gene sets."
         b. Large DNA viruses may be derived from former eukaryotic endosymbionts that took up a parasitic lifestyle.

   Picture Slide #1: Fig. 26.1 Smallpox virus
   Picture Slide #2: Fig. 26.3a Relative sizes of viruses, bacteria and a small eukaryotic cell
   Picture Slide #3: Fig. 26.3b Different shapes of viruses
   Picture Slide #4: Fig. 26.c Presence/absence of a phospholipid envelope important
   Sidetrack: Evolution
   Picture Slide: Farside

   B. Virus Replication
      1. Many viruses supply their own replication enzymes.
         a. DNA viruses code for DNA polymerase; RNA viruses code for RNA replicase.
         b. Retroviruses code for reverse transcriptase, which copies their RNA genome into double-stranded DNA.
      2. All viruses require host-cell ribosomes, ATP, and nucleotides to carry out replication.
      3. The viral replication cycle can be either lytic or lysogenic. (See Fig. 26.4)
         a. In the lytic cycle, the genome is copied and the genes are transcribed and translated to produce viral proteins.
            (1) New virus particles assemble in the cytoplasm.
            (2) Virus particles exit the cell, often by lysing (= rupturing) it.
         b. In the lysogenic cycle, the viral genome is incorporated into the host genome.
            (1) Each time the host chromosomes are copied and distributed to new cells, the viral genome is also copied and distributed to new cells.
            (2) The viral genome may excise itself from the host chromosome later and revert to lytic growth.

   C. The Human Immunodeficiency Virus (HIV)
      1. HIV is an emerging lentivirus that apparently arose from simian (= monkey) viruses in Africa.
         Picture Slide #5: Fig. 26a The Human Immunodeficiency Virus (HIV).
         Picture Slide #6: Fig 26b. Cutaway view emphasizing surface glycoproteins not easily seen in Fig 26.a
      2. HIV parasitizes human T-cells, rendering the immune system ineffective in clearing pathogens from the body
         Picture Slides #7 & 8 Role of T_{Helper}-cells (= T_{H}-cells)
         Picture Slide #9: Fig 26.6 T-cell Counts Decline over the Course of an HIV Infection
         Picture Slide #10: Fig 26.7 Geographic Distribution of HIV Infections

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3. HIV Infection, Replication, and Production of New Virus
   a. HIV enters helper T cells by binding to the membrane protein CD4 AND to a co-receptor protein (either CXCR4 or CCR5).
   Picture Slide #11 Fig 26.9a Molecules Involved in HIV’s entry into T-cells
   Picture Slide #12: Lock and Key Hypothesis: HIV can only enter cells after binding to CCR5 AND CD4 proteins
   b. Individuals that are resistant to HIV infection often have a defect in their co-receptor proteins.
   Picture Slides #13: Lock and Key Hypothesis: People with cells that do not express CD4 proteins do not develop AIDS.
   Quotation Slide HIV Resistance and Human History *Sci Amer.* Sept 1997
   Quotation Slide HIV Resistance and Human History *Sci Amer.* Sept 1997
   c. HIV reverse transcriptase copies HIV RNA into double-stranded cDNA.
   d. The cDNA is inserted into the host chromosome by the HIV integrase enzyme.
   e. HIV genes on the cDNA are transcribed and translated into proteins.
      (1). Proteins destined for the viral envelope are glycosylated in the Golgi apparatus and sent to the host-cell plasma membrane.
      (2). Proteins destined for the capsid are translated into long polyproteins in the cytoplasm, which are cleaved by HIV protease.
   Picture Slides #15 & 16 Figs 26.10a & 26.10b: Production of Envelope Proteins
   f. New HIV viruses leave the host cell by budding.
   Picture Slides 17 & 18: Figs 26.12a, left & right Budding of enveloped virus.
   Picture Slides 19 & 20: Figs 26.13a, left & right Bursting of non-enveloped virus

D. AIDS Drug Treatments and the Prospects for Vaccines
   1. Reverse transcriptase inhibitors are chain-terminating nucleotides that are incorporated into the cDNA by reverse transcriptase, but they block further cDNA synthesis.
   2. Protease inhibitors are synthetic molecules, developed through rational drug design, that block the activity of the HIV protease.
   3. Combination therapy involves treating the patient with multiple types of reverse transcriptase inhibitors and protease inhibitors.
   4. Patients rapidly develop resistance due to the high mutation rate of HIV.
   5. Attempts at producing vaccines against the HIV envelope proteins have not been successful, because few HIV particles contain identical envelope proteins.