LEARNING OBJECTIVES:

1. List the viruses contributing to human cancers and describe their common features.
2. Describe how HPV is “typed” and indicate the key features distinguishing high cancer-risk HPV types.
3. Describe how HPVs are transmitted.
4. Note the structure of HPVs and their infection cycles in skin epithelia.
5. Discuss how HPV dysregulates host cell cycle and contributes to dysplastic epithelia.
6. Name the HPV oncoproteins and describe how they operate to reduce activities of host cell cycle checkpoint proteins.
7. Contrast HPV oncoproteins from the more directly-acting oncogene products of transforming retroviruses.
8. Describe HPV diagnosis, therapy and vaccines.

KEY CONCEPTS:

1. HPVs are DNA tumor viruses.
2. HPVs are nonenveloped viruses, entering through skin abrasions, replicating in basal epithelia, requiring epithelial cell differentiation to produce progeny viruses.
3. Viral DNA replication requires modulation of host cell cycle.
4. Permanent modulation of host cell cycle can arise upon integration of viral DNA into host genome (a rare event).
5. Modulation of host cell cycle is by virus-encoded proteins (E6 and E7) that target cell cycle checkpoint proteins (p53 and Rb).
6. HPV is diagnosed by identifying hyperplastic or dysplastic tissue (koilocytes) and by PCR or DNA probe-based identification of HPV DNA.
7. HPV therapies use surgery to remove warts or topical antiviral creams.
8. Best current control of HPV cancers is through vaccination with recombinant HPV capsids.