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RESEARCH INTEREST

• Virology:

• Visualization by electron microscope

• Proteins detection using analytical and/or immuno-detection techniques

• Nucleic acids detection and identification by hybridization, PCR...etc.
Aly MOUSSA has obtained his BVSc from Cairo University, Egypt; Dr. Vet. Med. From Justus Liebig University, Germany and PhD from Claude Bernard University, France. He worked 4 years at IFFA-Mérieux Laboratory; Lyon- France, for 20 years was the chief of virology service at the French Bovine Pathology laboratory. Then for 8 years he was concerned at the national agency for sanitary security of aliments with research on the pathogenic prion proteins. He has published many papers in the fields of Virology and Transmissible Spongiform Encephalopathies. By the end of 2005 he is retired. During activity he was member of the biotechnology group at the Office International des Epizooties, member of the CEE group on Infectious Bovine Rhinotrachitis and he was a founding member of the European veterinary virology society.
EDWARD JENNER

- Vaccinations
- Cowpox
  - cross protection against small pox
    - Variola virus
      - Major
        - Blisters
        - Blindness
        - Death
      - Minor
    - Poxviridae
    - dsDNA
VIRUSES

Summary figure: Schematic representation of the evolution of viruses and evolutionary forces acting on them

- Define
- Classification
  - Group
    - NA
  - Family
    - -viridae
  - Genus
    - -virus
  - Species
    - Name
HOST RANGE: ANIMALS
## DNA ANIMAL VIRUS EXAMPLES

### TABLE 41-1 Chemical and Morphologic Properties of Animal Virus Families Relevant to Human Disease

<table>
<thead>
<tr>
<th>Family</th>
<th>Viral Genome: Type, Configuration and Number of Bases per strand (x 10^n)</th>
<th>Shape</th>
<th>Diameter (nm)</th>
<th>Enveloped</th>
<th>Capsid Symmetry</th>
<th>Number of Capsomers</th>
<th>Site of Capsid Assembly</th>
<th>Enzymes, e.g. Transcriptase present in Virion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Circoviridae</td>
<td>ssDNA, circular; 0.6-1.2</td>
<td>s</td>
<td>17-22</td>
<td>0</td>
<td>Icosahedral</td>
<td>32?</td>
<td>Nucleus</td>
<td>None</td>
</tr>
<tr>
<td>Paroviridae</td>
<td>ssDNA, linear; sense or antisense; 4-6</td>
<td>s</td>
<td>18-26</td>
<td>0</td>
<td>Icosahedral</td>
<td>32</td>
<td>Nucleus</td>
<td>None</td>
</tr>
<tr>
<td>Papoviridae</td>
<td>dsDNA, circular; 5.1 / 7.9</td>
<td>s</td>
<td>45 / 55</td>
<td>0</td>
<td>Icosahedral</td>
<td>72</td>
<td>Nucleus</td>
<td>None</td>
</tr>
<tr>
<td>Adenoviridae</td>
<td>dsDNA, linear; 35-40</td>
<td>s</td>
<td>75-80</td>
<td>0</td>
<td>Icosahedral</td>
<td>252</td>
<td>Nucleus</td>
<td>None</td>
</tr>
<tr>
<td>Herpesviridae</td>
<td>dsDNA, linear; 124-235</td>
<td>s</td>
<td>120-200</td>
<td>+</td>
<td>Icosahedral</td>
<td>162</td>
<td>Nucleus</td>
<td>Thymidine kinase DNA-dependent DNA polymerase</td>
</tr>
<tr>
<td>Iridoviridae</td>
<td>dsDNA, linear; 170-200</td>
<td>s</td>
<td>125-300</td>
<td>+</td>
<td>Icosahedral</td>
<td>ca. 1,500</td>
<td>Cytoplasm</td>
<td>DNA-dependent RNA polymerase Protein kinase</td>
</tr>
<tr>
<td>Poxviridae</td>
<td>dsDNA, linear, covalently closed; 130-370</td>
<td>x</td>
<td>240x300</td>
<td>+</td>
<td>Complex</td>
<td>-</td>
<td>Cytoplasm</td>
<td>DNA-dependent DNA polymerase</td>
</tr>
<tr>
<td>Hepadnaviridae</td>
<td>dsDNA, circular, 1 ss-region; 3.0-3.3/2.0</td>
<td>s</td>
<td>40-48</td>
<td>+</td>
<td>Icosahedral</td>
<td>180</td>
<td>Nucleus</td>
<td>DNA-dependent DNA polymerase</td>
</tr>
</tbody>
</table>
VIRAL CULTURE

- Tissue Culture
  - Chick Embryos
  - Animal Cells/Tissue

- Assays
  - Hemagglutination
  - Plaque
VIRAL CAPSID

**Function**
- Protect NA
- Aids in transfer to host

**Structure**
- Protein coat
- Capsomere arrange
  - Helical
  - Polyhedral
  - Complex
CAPSOMERES ARE CAPSID SUBUNITS

Polyhedral

Helical

Binal

(a) Mastadenovirus

(b) Herpesvirus
NAKED VS. ENVELOPED VIRUSES
**VIRAL ENV**

- **Presence**
  - Enveloped
  - Naked (non-enveloped)

- **Location**
  - Surrounds capsid

- **Source**
  - Host plasma membrane
  - Nuclear membrane
  - Endoplasmic reticulum

- **Components**
  - Phospholipid
  - Proteins
  - Glycoprotein spikes (+/-)

- **Examples**
  - Influenza
  - Rabies
  - Herpes
  - HIV
ENVELOPE GLYCOPROTEIN SPIKES

Diagram showing the structure of a virus with labels for Hemagglutinin, RNA, Envelope, Neuraminidase, gp120, Docking Glycoprotein, gp41, Transmembrane Glycoprotein, Lipid Membrane, Capsid, Matrix, Reverse Transcriptase.


VIRAL NA

- DNA OR RNA
- Shape
  - Circular
  - Linear
- Number
  - One
  - Or more
- Strands
  - ss
  - ds
  - + or - if RNA
VIRAL CLASSIFICATION

- dsDNA
  - Pox
  - Herpes
  - Papilloma
- ssDNA
  - Parvo
- DsRNA
  - Reovirus
  - Rotavirus
- ssRNA
  - Polio
  - Rhino
  - Corona
  - Measles, mumps
  - Rabies
  - Influenza
  - Parainfluenza
  - Retroviruses

<table>
<thead>
<tr>
<th>Characteristics/Dimensions</th>
<th>Virological family</th>
<th>Genera</th>
<th>Clinical or Special Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single-stranded DNA (ssDNA)</td>
<td>Paroviridae</td>
<td>Human parovirus B19</td>
<td>机化 chorionplacental villus (4% of term placentas) causes a transient form of anemia in newborns. Refer to Chapter 21.</td>
</tr>
<tr>
<td>Double-stranded DNA (dsDNA)</td>
<td>Adenoviridae</td>
<td>Adenovirus A</td>
<td>Mediated transmission in the respiratory tract by aspiration or ingestion. Refer to Chapter 21.</td>
</tr>
<tr>
<td>- 40-57 nm</td>
<td>Papovaviridae</td>
<td>Papillomavirus</td>
<td>HPV16 (causes cervical cancer)</td>
</tr>
<tr>
<td>- 200-520 nm</td>
<td>Picornaviridae</td>
<td>Picornavirus</td>
<td>Coxsackievirus A16 causes myocarditis.</td>
</tr>
<tr>
<td>- 53-40 nm</td>
<td>Caliciviridae</td>
<td>Calicivirus</td>
<td>Norovirus</td>
</tr>
<tr>
<td>- 55-50 nm</td>
<td>Flaviviridae</td>
<td>Flavivirus</td>
<td>Dengue virus</td>
</tr>
<tr>
<td>- 80-140 nm</td>
<td>Coronaviridae</td>
<td>Coronavirus</td>
<td>SARS virus</td>
</tr>
<tr>
<td>- 120-200 nm</td>
<td>Paramyxoviridae</td>
<td>Paramyxovirus</td>
<td>Respiratory syncytial virus (RSV)</td>
</tr>
<tr>
<td>- 60-80 nm</td>
<td>Retroviridae</td>
<td>Retrovirus</td>
<td>Human immunodeficiency virus (HIV-1) and -2 causes AIDS</td>
</tr>
</tbody>
</table>

Table 17.3: Families of Viruses That Affect Humans
# Viral Replication Differences

<table>
<thead>
<tr>
<th>Stage</th>
<th>Bacteriophages</th>
<th>Animal Viruses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Attachment</td>
<td>Tail fibers attach to cell wall proteins</td>
<td>Attachment sites are plasma membrane proteins and glycoproteins</td>
</tr>
<tr>
<td>Entry</td>
<td>Viral DNA injected into host cell</td>
<td>Capsid enters by endocytosis or fusion</td>
</tr>
<tr>
<td>Uncoating</td>
<td>Not required</td>
<td>Enzymatic removal of capsid proteins</td>
</tr>
<tr>
<td>Biosynthesis</td>
<td>In cytoplasm</td>
<td>In nucleus (DNA viruses) or cytoplasm (RNA viruses)</td>
</tr>
<tr>
<td>Chronic infection</td>
<td>Lysogeny</td>
<td>Latency; slow viral infections; cancer</td>
</tr>
<tr>
<td>Release</td>
<td>Host cell lysed</td>
<td>Enveloped viruses bud out; nonenveloped viruses rupture plasma membrane</td>
</tr>
</tbody>
</table>

*Table 13.3: Bacteriophage and Viral Multiplication Compared*
VIRAL INFECTIONS
REPLICATION OF ANIMAL VIRUSES

- Attach
- Entry
  - Direct Penetration
  - Membrane fusion
  - Endocytosis
- Uncoating
- Synthesis
- Assembly
- Release
VIRAL ATTACHMENT

Figure 2. Viral Attachment
Direct PENETRATION

herpesviruses, paramyxoviruses, HIV
ENDOCYTOSIS VS. MEMBRANE FUSION

(a) Entry of togavirus

(b) Entry of herpesvirus
RELEASE OF GENOME (UNCOATING)

Adenovirus uncoating

- Attachment
- Mature Virion
- Pentons
- DNA released
- Core enters nucleus
- Hexons
- Endosome formation
- pH drop due to H+ pump
- Fusion peptide to PM
- Conformational change
- Release of NA

Influenza Virus
NA SYNTHESIS

- dsDNA: usual replication (for most)
- ssDNA
  - complementary strand
  - Normal replication
- dsRNA
  - + strand = mRNA
  - Template and copy
- +ssRNA
  - + strand = mRNA
  - Complimentary strand for template
- -ssRNA
  - Viral enzymes make + strand
  - Template for mRNA and -ss
- Retroviruses
  - +ssRNA (mRNA to make DNA)
  - Reverse transcriptase
  - DNA is template for new +ssRNA
<table>
<thead>
<tr>
<th>Viral Nucleic Acid</th>
<th>Virus Family</th>
<th>Special Features of Biosynthesis</th>
</tr>
</thead>
<tbody>
<tr>
<td>DNA, single-stranded</td>
<td>Parvoviridae</td>
<td>Cellular enzyme transcribes viral DNA in nucleus</td>
</tr>
<tr>
<td>DNA, double-stranded</td>
<td>Herpesviridae</td>
<td>Cellular enzyme transcribes viral DNA in nucleus</td>
</tr>
<tr>
<td></td>
<td>Papovaviridae</td>
<td>Viral enzyme transcribes viral DNA in virion, in cytoplasm</td>
</tr>
<tr>
<td></td>
<td>Poxviridae</td>
<td></td>
</tr>
<tr>
<td>DNA, reverse transcriptase</td>
<td>Hepadnaviridae</td>
<td>Cellular enzyme transcribes viral DNA in nucleus; reverse transcriptase copies mRNA to make viral DNA</td>
</tr>
<tr>
<td>RNA, + strand</td>
<td>Picornaviridae</td>
<td>Viral RNA functions as a template for synthesis of RNA polymerase which copies − strand RNA to make mRNA in cytoplasm</td>
</tr>
<tr>
<td></td>
<td>Togaviridae</td>
<td></td>
</tr>
<tr>
<td>RNA, − strand</td>
<td>Rhabdoviridae</td>
<td>Viral enzyme copies viral RNA to make mRNA in cytoplasm</td>
</tr>
<tr>
<td>RNA, double-stranded</td>
<td>Reoviridae</td>
<td>Viral enzyme copies − strand RNA to make mRNA in cytoplasm</td>
</tr>
<tr>
<td>RNA, reverse transcriptase</td>
<td>Retroviridae</td>
<td>Viral enzyme copies viral RNA to make DNA in cytoplasm; DNA moves to nucleus</td>
</tr>
</tbody>
</table>
DNA VIRUS BIOSYNTHESIS

1. Virion attaches to host cell.
2. Virion enters cell and its DNA is uncoated.
3. A portion of viral DNA is transcribed, producing mRNA that encodes “early” viral proteins.
4. Viral DNA is replicated and some viral proteins are made.
5. Late translation; capsid proteins are synthesized.
6. Virions mature.
7. Virions are released.

Papovavirus

Host cell

Nucleus
Cytoplasm
RETROVIRUSES

1. Retrovirus enters by fusion between attachment spikes and the host cell receptors.

2. Uncapping releases the two viral RNA genomes and the viral enzymes reverse transcriptase, integrase, and protease.

3. Viral proteins are processed by viral protease; some of the viral proteins are moved to the host plasma membrane.

4. Transcription of the provirus may also occur, producing RNA for new retrovirus genomes and RNA that encodes the retrovirus capsid, enzymes, and envelope proteins.

5. The new viral DNA is transported into the host cell’s nucleus, where it is integrated into a host cell chromosome as a provirus by viral integrase. The provirus may be replicated when the host cell replicates.

6. Reverse transcriptase copies viral RNA to produce double-stranded DNA.

7. Mature retrovirus leaves host cell, acquiring an envelope and attachment spikes as it buds out.
VIRAL ASSEMBLY

- DNA
  - Nucleus
  - Moves to cytoplasm
- RNA
  - Cytoplasm
USE OF ER AND GOLGI

Herpes
VIRAL RELEASE

- Types
  - Budding
    - Acquire membranes
    - Envelope
  - Exocytosis
  - Lysis
- Latency
BUDDING

Figure 9. Viral Assembly / Budding
VIRAL EXOCYTOSIS
<table>
<thead>
<tr>
<th>Virus (Genus)</th>
<th>Cytopathic Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poliovirus (Enterovirus)</td>
<td>Cytocidal (cell death)</td>
</tr>
<tr>
<td>Papovavirus (family Papovaviridae)</td>
<td>Acidophilic inclusion bodies in nucleus</td>
</tr>
<tr>
<td>Adenovirus (Mastadenovirus)</td>
<td>Basophilic inclusion bodies in nucleus</td>
</tr>
<tr>
<td>Rhabdovirus (family Rhabdoviridae)</td>
<td>Acidophilic inclusion bodies in cytoplasm</td>
</tr>
<tr>
<td>Cytomegalovirus</td>
<td>Acidophilic inclusion bodies in nucleus and cytoplasm</td>
</tr>
<tr>
<td>Measles virus (Morbillivirus)</td>
<td>Cell fusion</td>
</tr>
<tr>
<td>Polyomavirus</td>
<td>Transformation</td>
</tr>
<tr>
<td>HIV (Lentivirus)</td>
<td>Destruction of T cells</td>
</tr>
</tbody>
</table>
ACUTE VS. LATENT

Diagram showing the timeline of acute, latent, and persistent infections.
Viral Infections

Acute infections
1. Cytolytic infection
   a. 
   b. 
   c. 

Persistent Infections
2. Chronic, e.g. steady state infection
3. Latent infection
4. Integrated infection

- Virus
- Viral genome
- Integrated virus genome

Reactivation
VIRUSES AND CANCER

- Definitions
  - Oncogenes
  - Activation
    - Mutation
    - Transduction
- Tumor
  - Types
    - Benign
    - Malignant
  - Characteristics
- Examples
  - DNA
    - Adenovirus
    - Herpes
    - Poxviruses
    - Papoviruses
    - Hepadnaviruses
  - RNA
    - Retroviruses
      - HIV
      - HTLV

Nature Reviews | Cancer
IMMUNE RESPONSE

Adaptive Immunity
ANTI-VIRAL DRUGS

- **Attachment antagonists**
  - Block attachment molecule
    - Arildone
- **Inhibit Uncoating**
  - Neutralize acid environment
    - Amantadine
    - Rimantadine
- **Inhibit DNA/RNA synthesis**
  - Activation by phosphorylation of drug by viral kinases
    - Acyclovir
    - Gancyclovir
PRION PROTEINS
TRIDIMENSIONAL STRUCTURE OF THE PRPC RICH IN ALPHA-HELICES (LEFT) AND THE PRPSC RICH IN BETA-SHEETS PR

PrPc
Sensitive to Proteinase K

PrPsc
proteinase k resistant

Detergant Insoluble

Insoluble
THE PRION IS AN AMYLOID PROTEIN WHICH INDUCES ALONE DISEASES;

THE TRANSMISSIBLE SPONGIFORM ENCEPHALITIS (TSE) ARE SUB-ACUTE, FATAL INFECTIONS AND CHARACTERIZED BY THE PRESENCE OF VACUOLES IN NEURONS

Exp:
Scrapie in sheep & goats, BSE in cattle, chronic wasting disease in deer and Creutzfeld-Jakob Disease in humans (CJD).
THE PRION PROTEIN (PRP):- PRPC & PRPSC

• The cellular Prion protein PrPc is coded by the prnp Gene situated on the chromosome 20 in humans, 13 in bovine and 2 in mice.
• This gene was found in all vertebrates and invertebrates and is expressed mainly in the CNS and the reticular-endothelial system.
• The gene product (PrPc) is transported outside the cell and anchored on the cell membrane and is associated with signal transduction.
• The pathogenic prion protein PrPsc is produced after conformational transformation of the PrPc induced either by gene mutation or after infection with a PrPsc.
PRION PRODUCTION

1. PrP\textsuperscript{c} produced by cells is secreted to the cell surface.
2. PrP\textsuperscript{Sc} may be acquired or produced by an altered PrP\textsuperscript{c} gene.
3. PrP\textsuperscript{Sc} reacts with PrP\textsuperscript{c} on the cell surface.
4. PrP\textsuperscript{Sc} converts the PrP\textsuperscript{c} to PrP\textsuperscript{Sc}.
5. The new PrP\textsuperscript{Sc} converts more PrP\textsuperscript{c}.
6. The new PrP\textsuperscript{Sc} is taken in by endocytosis.
7. PrP\textsuperscript{Sc} accumulates in endosomes.
8. PrP\textsuperscript{Sc} continues to accumulate as the endosome contents are transferred to lysosomes. The result is cell death.
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