

VIROLOGIA 2006/2007

APRESENTAÇÃO 6 **(Vírus de RNA+ com invólucro)**

Maria Filomena Caeiro

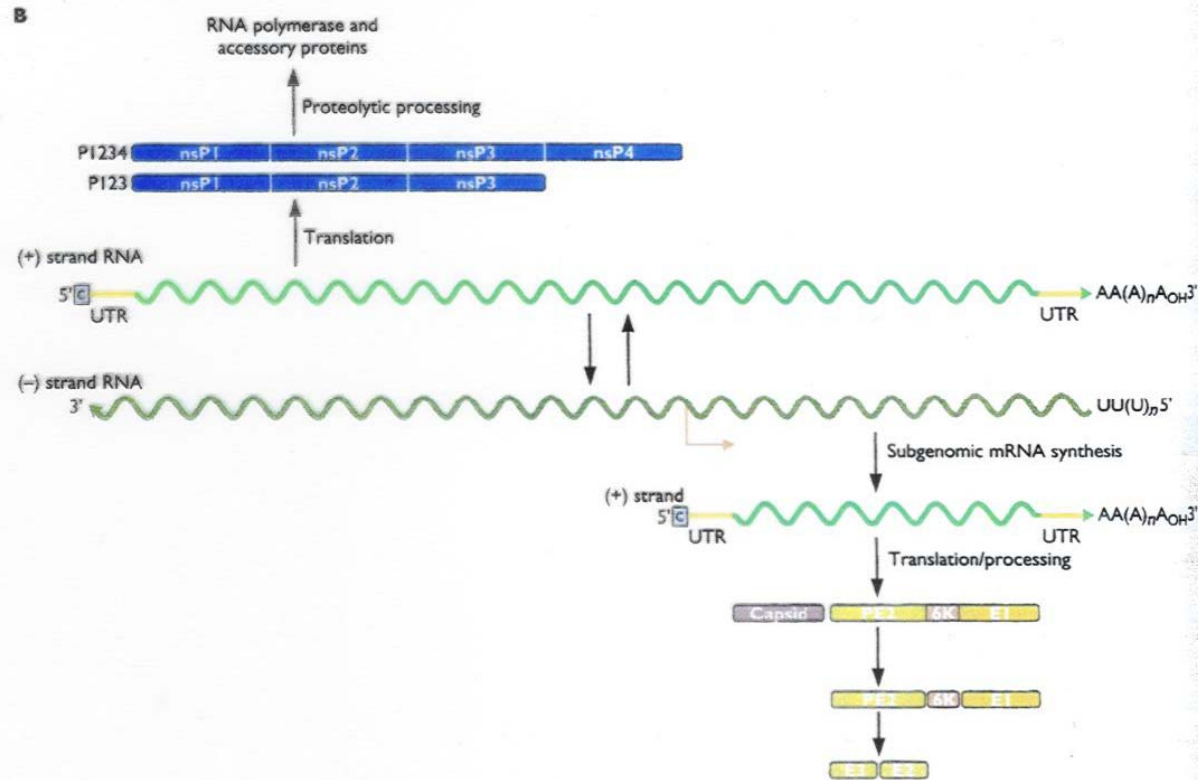
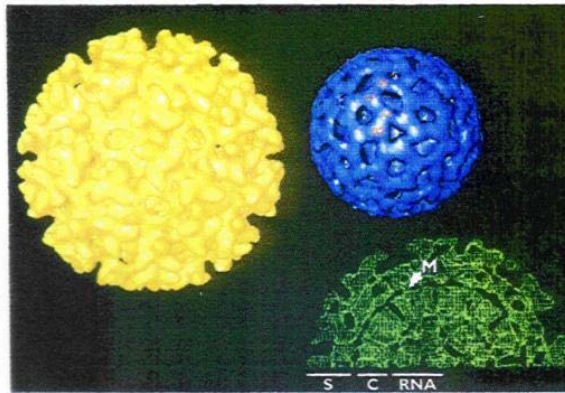
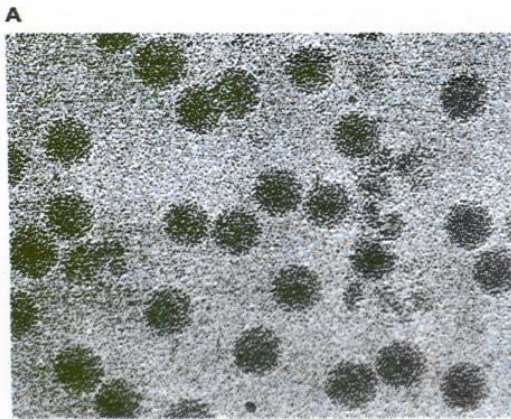


Figure 25 Structure and genomic organization

Flint, S. J., Enquist, L. W., Krug, R. M., Racaniello, V. R. and Skalka, A. M. (2004). "Principles of Virology. Molecular Biology, Pathogenesis, and Control". 2nd edition. ASM Press.

Togaviruses

Virus	Vector	Disease
Alphaviruses		
• Sindbis virus	Aedes mosquitoes	Subclinical
• Semliki Forest virus	Aedes mosquitoes	Subclinical
• Venezuelan equine encephalitis virus	Aedes, Culex mosquitoes	Mild systemic; severe encephalitis
• Eastern equine encephalitis virus	Aedes, Culiseta mosquitoes	Mild systemic; encephalitis
• Western equine encephalitis virus	Culex, Culiseta mosquitoes	Mild systemic; encephalitis
• Chikungunya virus	Aedes mosquitoes	Fever, arthralgia, arthritis
Rubella virus	None	Rubella

Epidemiology

Transmission

- Mosquito vectors
- Rubella virus: respiratory route

At risk

- Arthropod-borne viruses
- People in niche of vector
- Rubella virus
- Neonates <20 weeks old (congenital defects)
- Children (mild rash)
- Adults (more severe disease, arthritis, arthralgia)

Distribution of virus

- Arthropod-borne viruses: determined by habitat of vector
- Aedes mosquito: urban areas
- Culex mosquito: forest, urban areas
- More common in summer
- Rubella virus: ubiquitous

Vaccines or antiviral drugs

- Live, attenuated vaccine for rubella virus
- No antiviral drugs

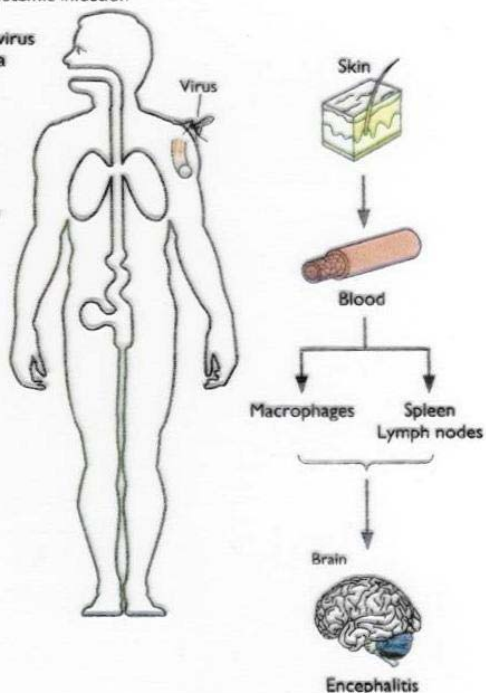
Disease mechanisms

Viruses are cytolytic (except rubella virus)

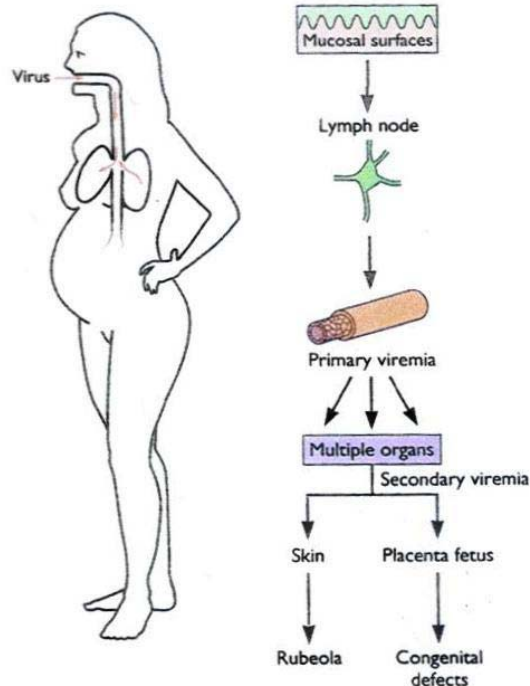
Cause viremia, systemic infection

Antibodies limit virus spread by viremia (e.g. to fetus in pregnant host)

Cell-mediated immunity important to resolve infection



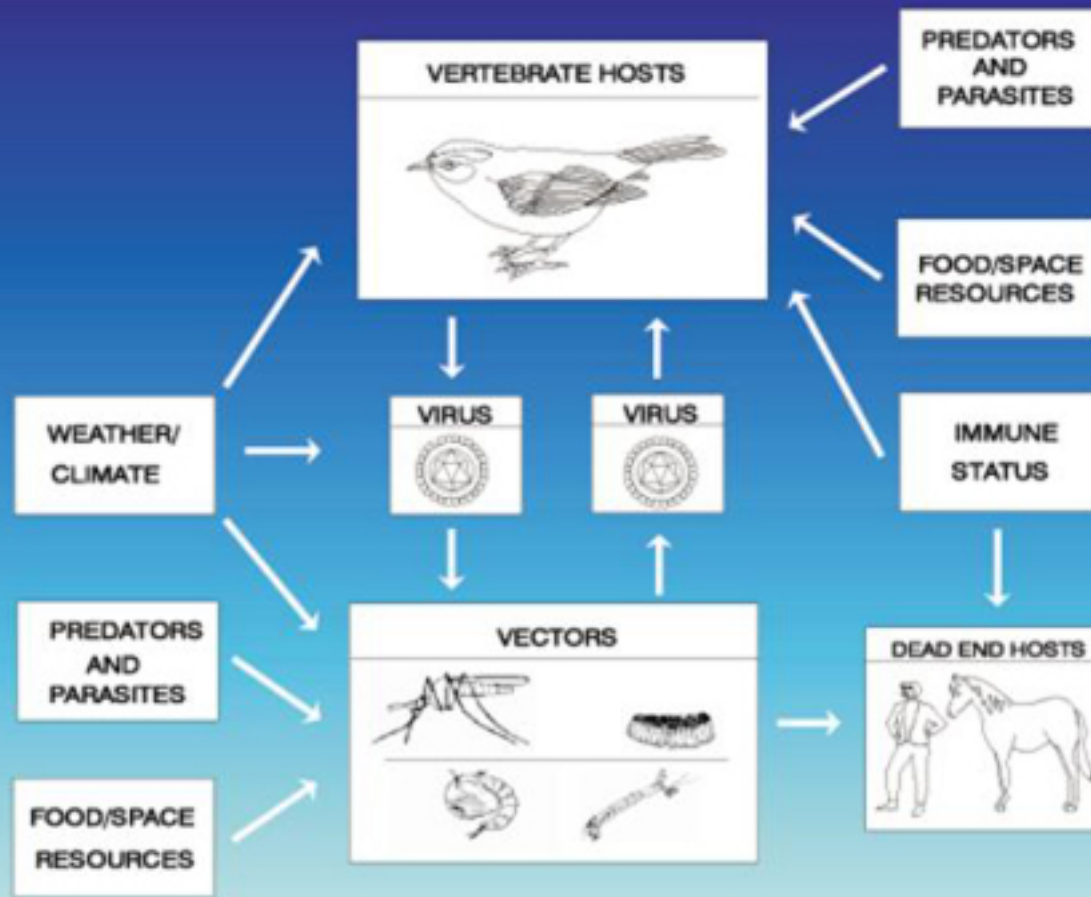
Mosquito borne



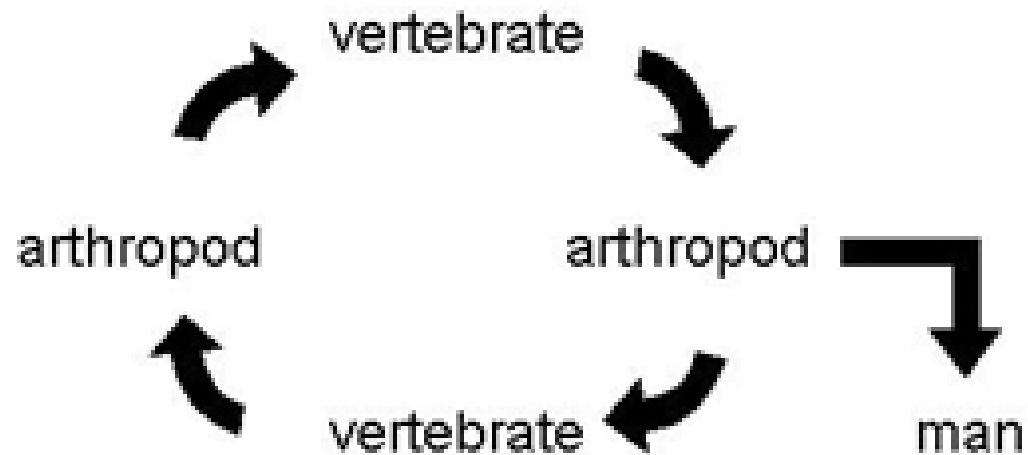
Rubella

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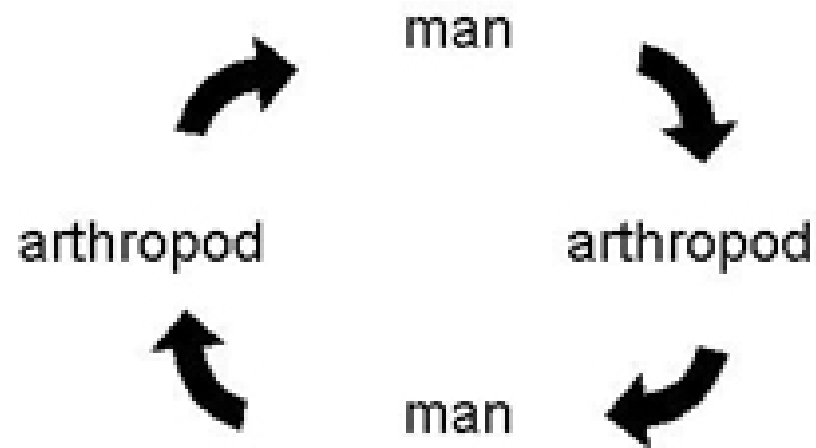
Components in the Transmission and Maintenance of Arboviral Encephalitis

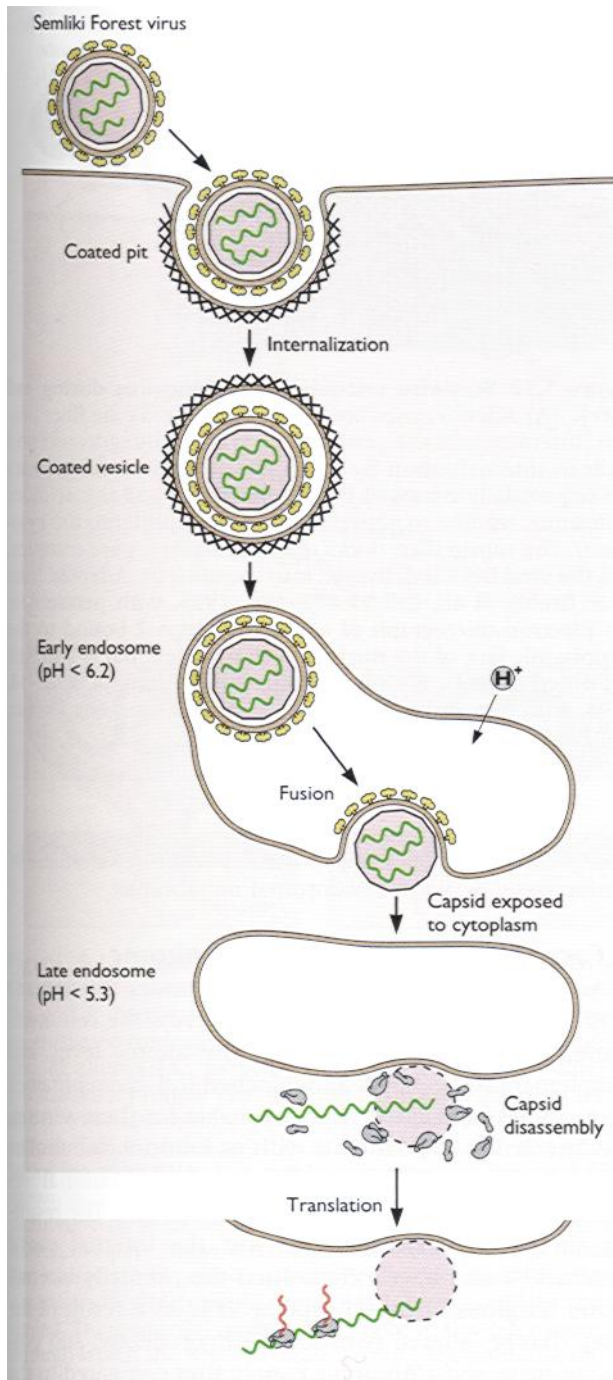


SYLVATIC (JUNGLE) CYCLE



URBAN CYCLE





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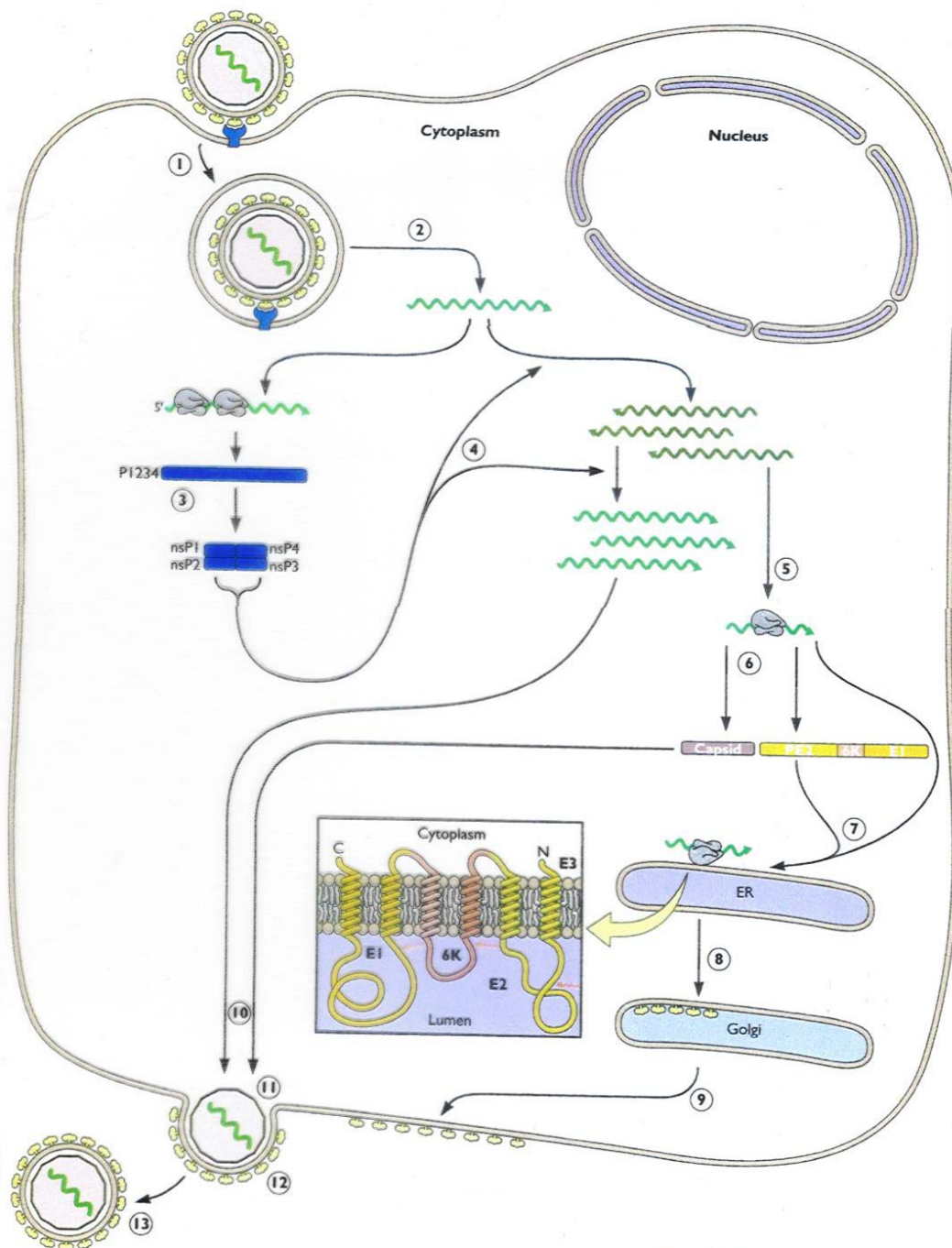


Figure 26 Single-cell reproductive cycle

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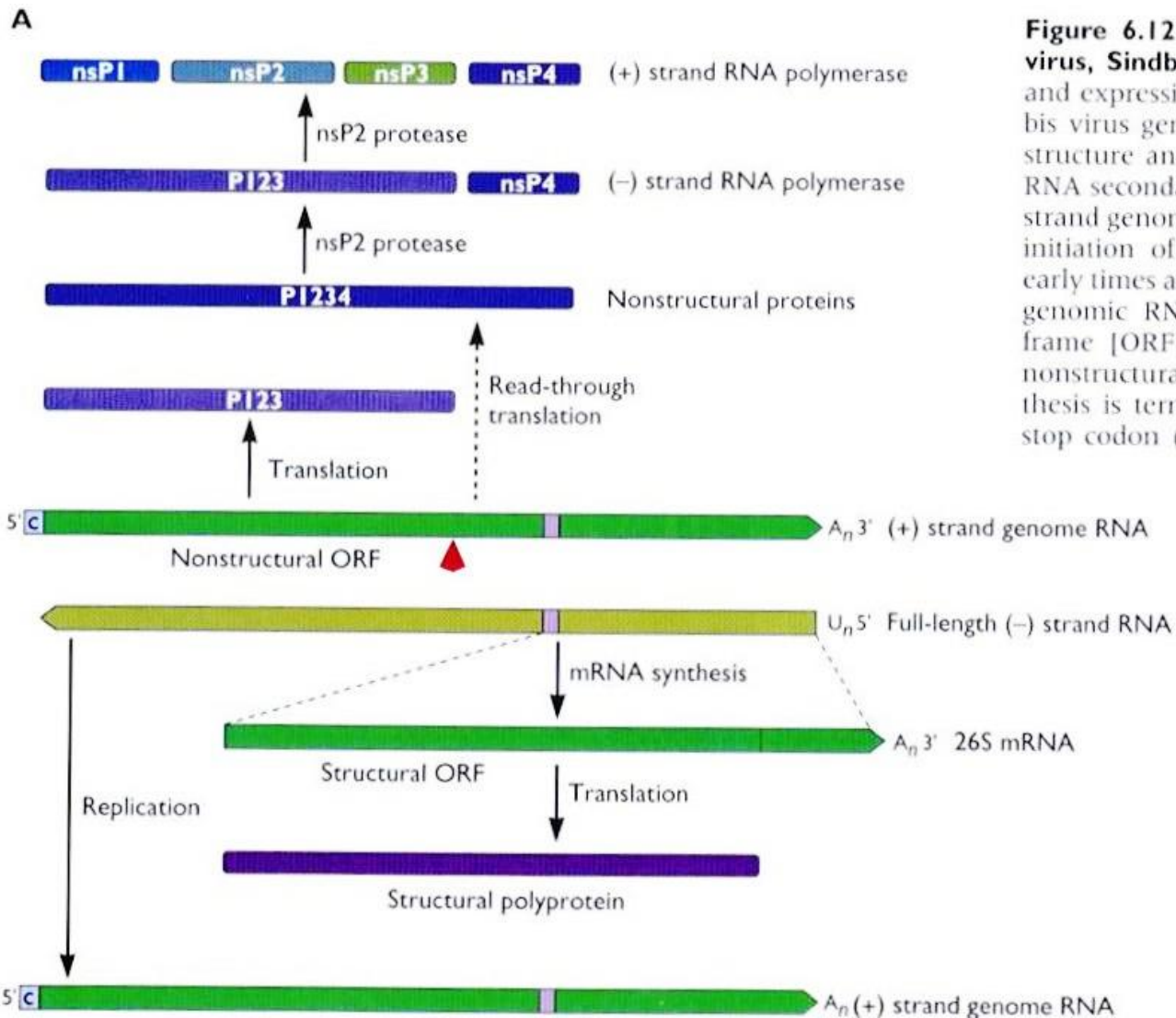


Figure 6.12 RNA replication of an alpha-virus, Sindbis virus. (A) Genome structure and expression. The 11,703-nucleotide Sindbis virus genome contains a 5'-terminal cap structure and a 3' poly(A) tail. A conserved RNA secondary structure at the 3' end of (+) strand genomic RNA is believed to control the initiation of (-) strand RNA synthesis. At early times after infection, the 5' region of the genomic RNA (nonstructural open reading frame [ORF]) is translated to produce two nonstructural polyproteins: P123, whose synthesis is terminated at the first translational stop codon (indicated by the red diamond),

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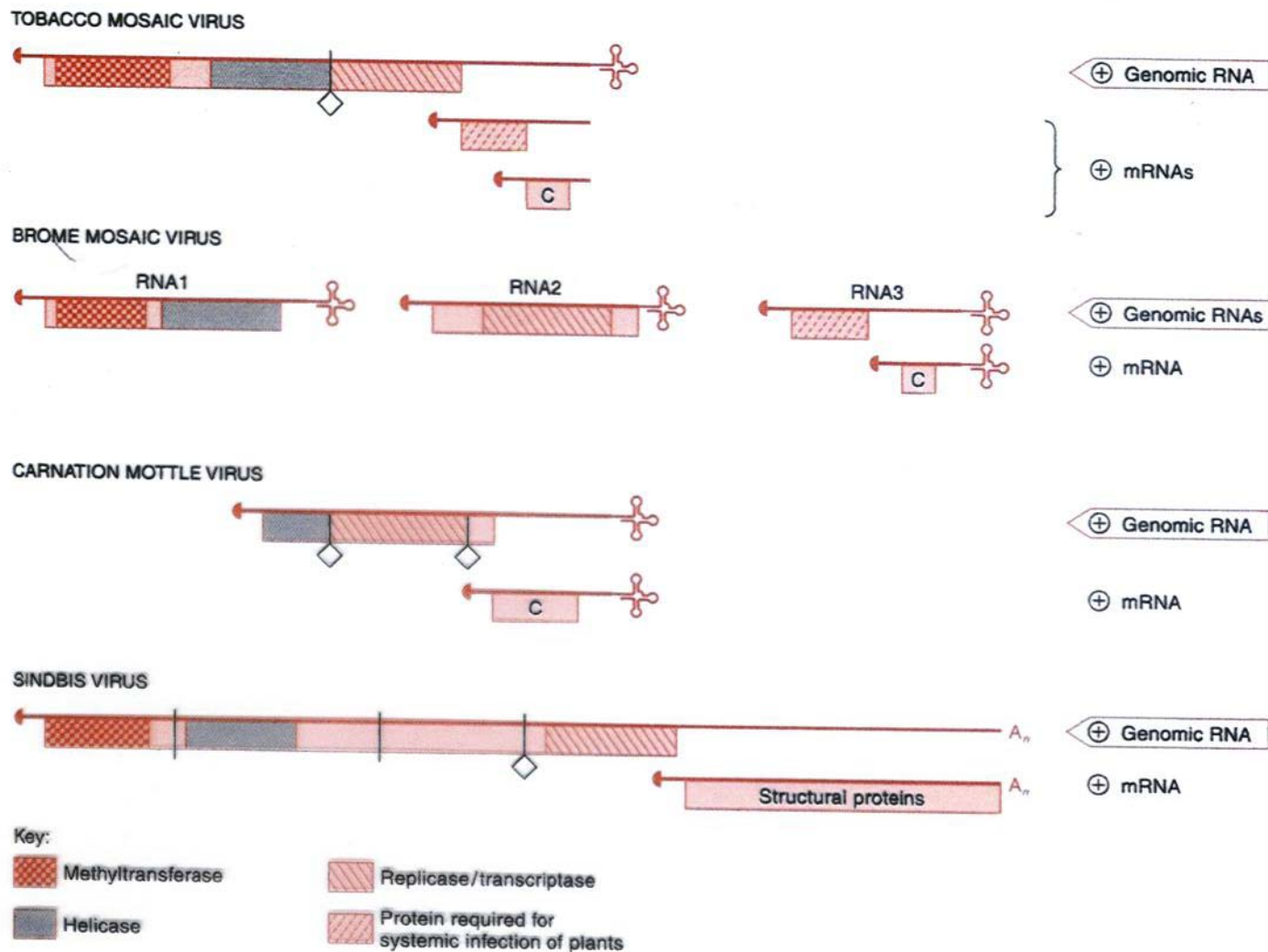
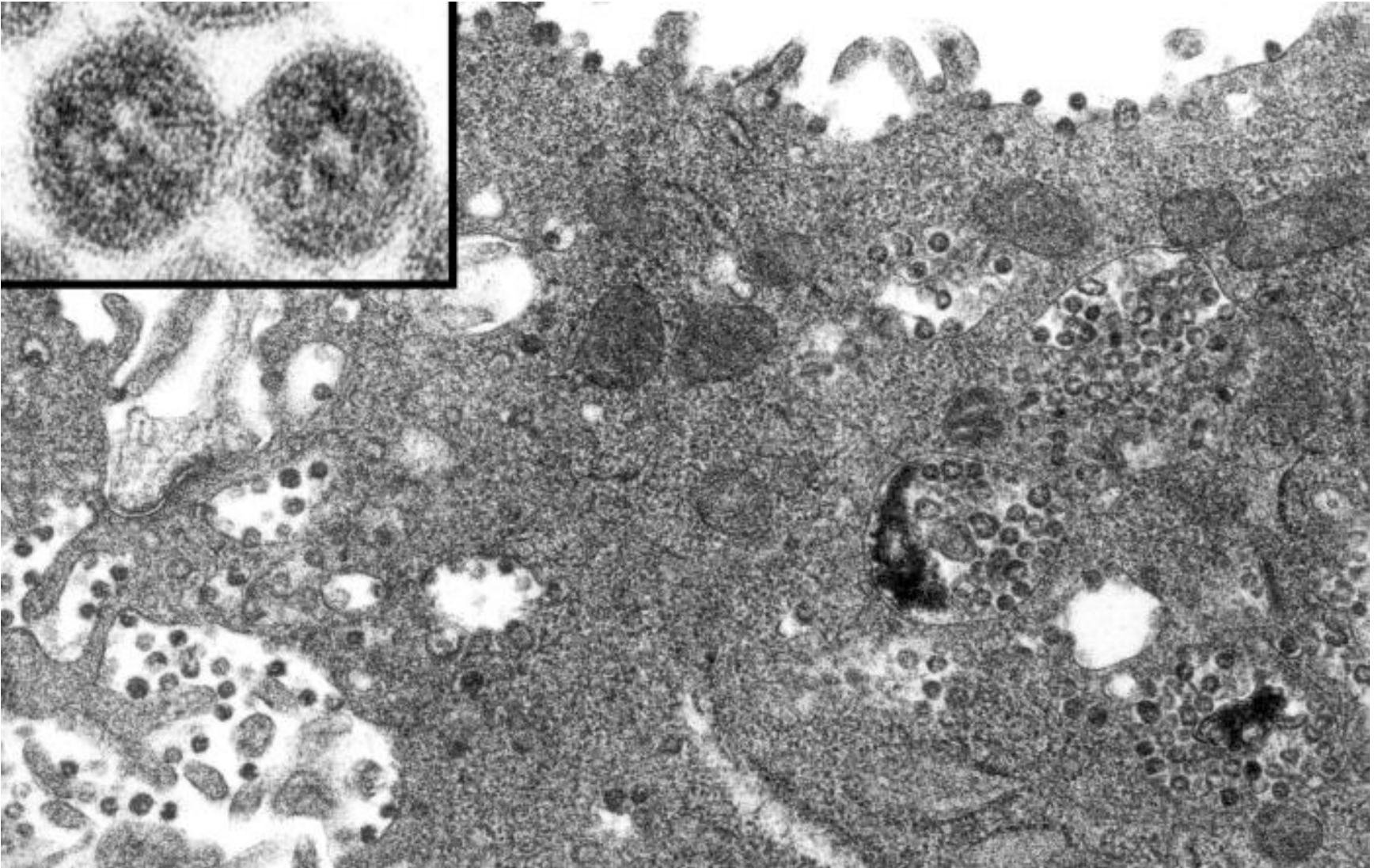


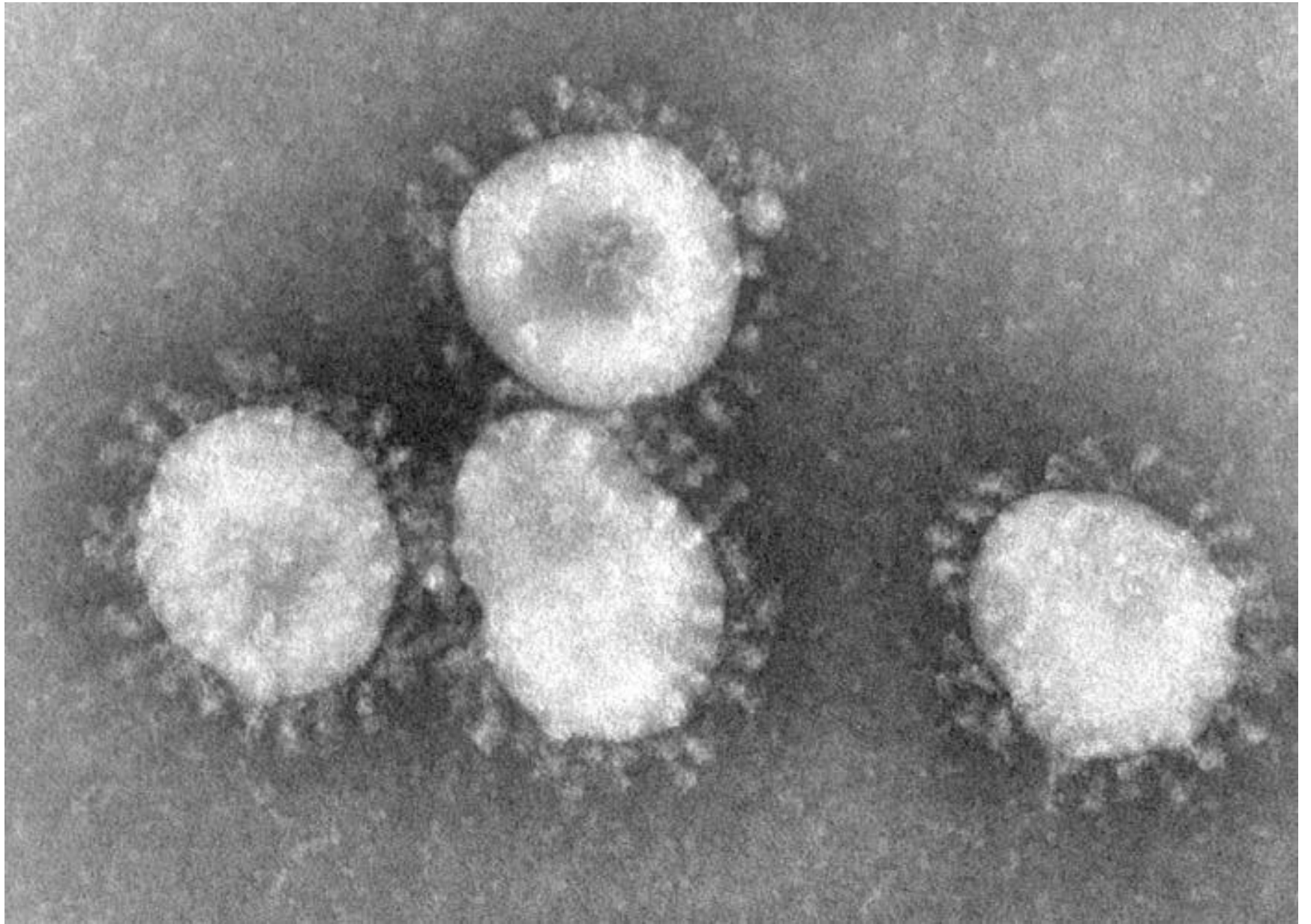
Figure 2.9

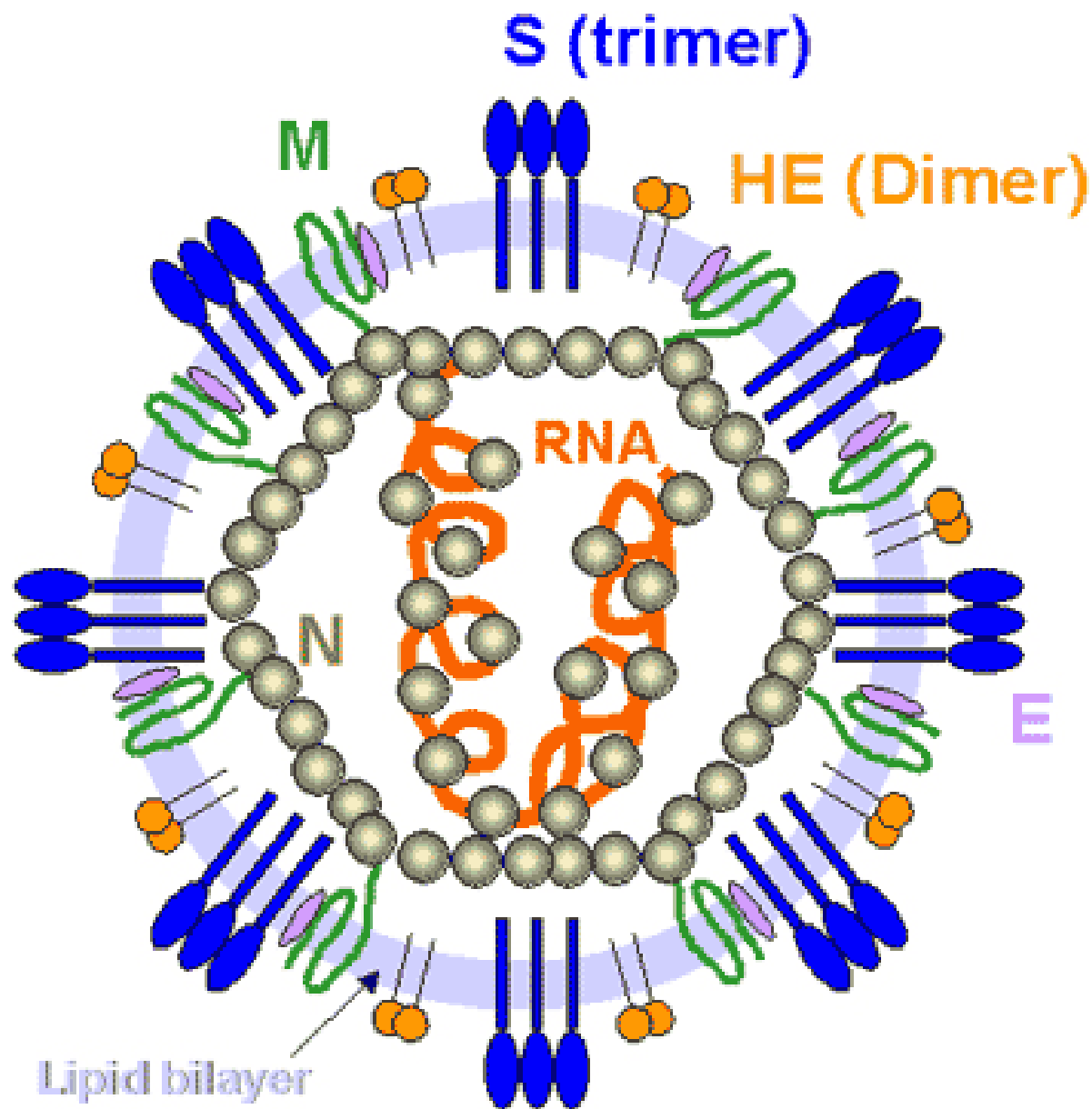
Homologies between the nonstructural proteins encoded by three plant viruses and Sindbis (SIN) virus. The translated regions of the genomes of tobacco mosaic virus (TMV), brome mosaic virus, and carnation mottle virus are shown diagrammatically and compared with that of Sindbis virus. (More detailed translation strategies for SIN and TMV are shown in Figures 2.5 and 2.7.) Three domains of significant sequence similarity in the nonstructural proteins within the helicase, methyl transferase, and replicase genes, respectively, are shaded differentially (see key).

Singer, M. and Berg, P. (1997).
"Exploring Genetic Mechanisms".
University Science Books. Sausalito,
California.

Coronaviridae







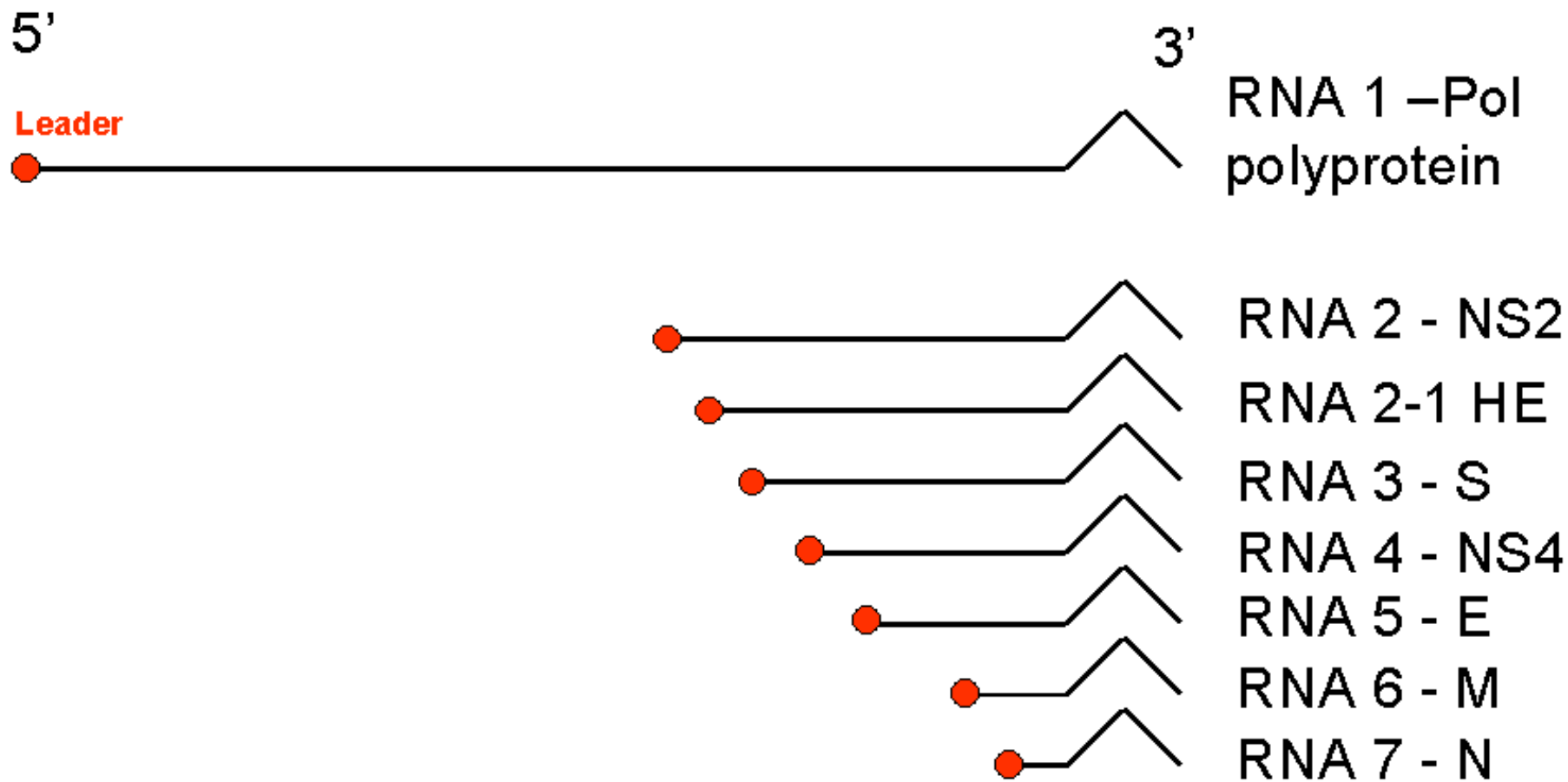
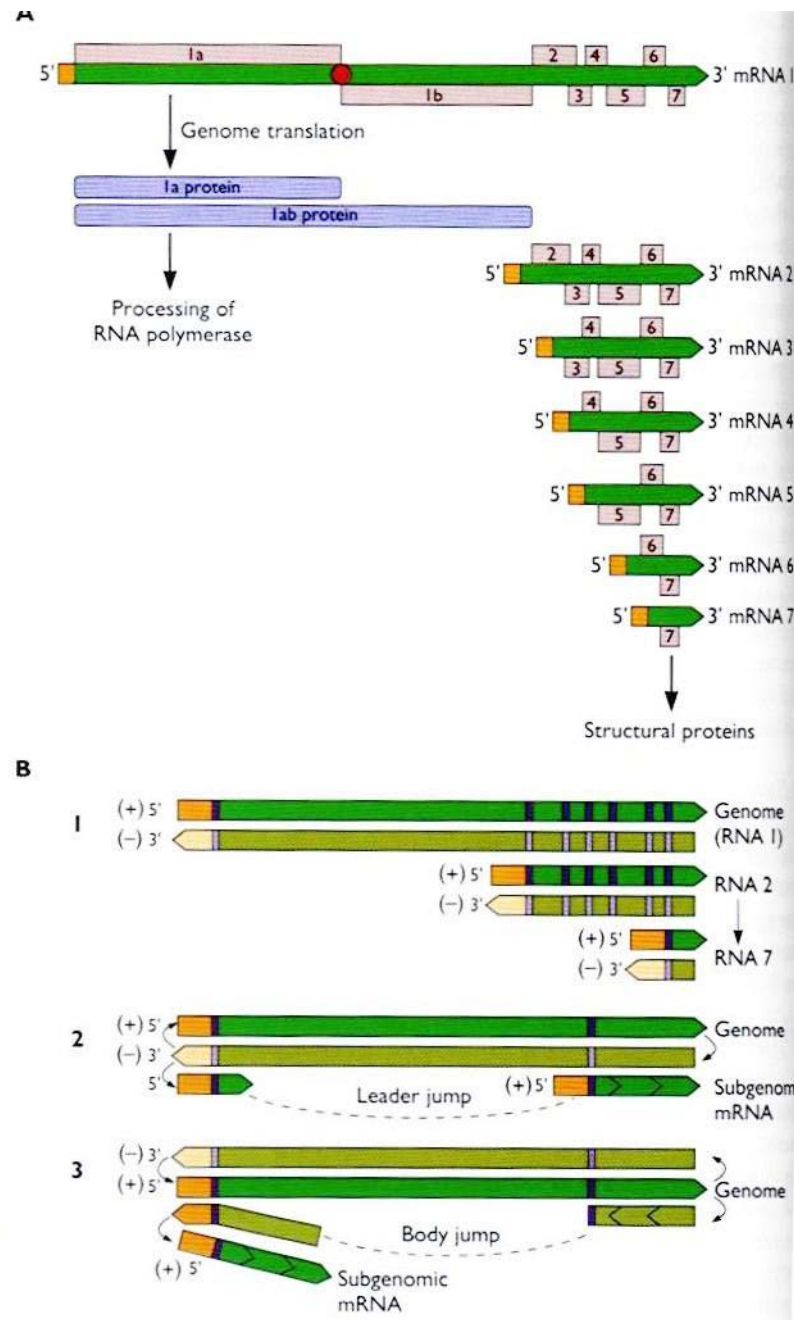


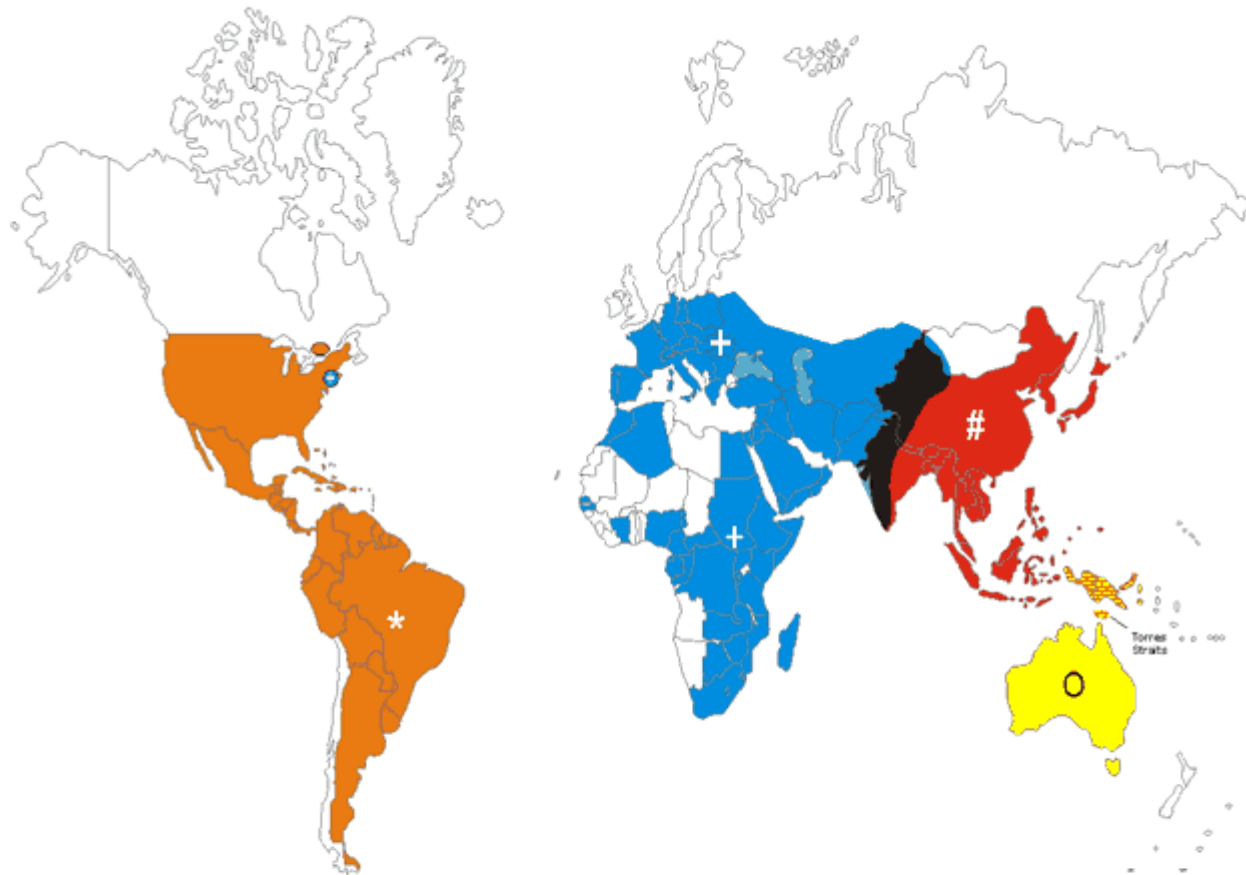
Figure 6.13 Nidoviral genome organization and expression.

(A) Organization of open reading frames. The (+) strand viral RNA is shown at the top, with open reading frames as boxes. The genomic RNA is translated to form proteins 1a and 1ab, which are processed to form the RNA polymerase. Structural proteins are encoded by six nested mRNAs that share a common 5' leader sequence (orange box). (B) Models of the synthesis of nested mRNAs. (1) Overview of (+) and (-) strand RNAs. (2) Discontinuous transcription was proposed to occur during (+) strand RNA synthesis by leader priming on a full-length (-) strand RNA template. Leader RNA (orange box) is joined to the mRNAs by a junction sequence also found adjacent to the leader RNA at the 5' end of the viral RNA. The (+) strand junction sequence at the 3' end of the leader can base-pair with the (-) strand junction sequence of the region that encodes each mRNA. (3) After a nested set of subgenomic replicative intermediates were discovered in infected cells [i.e., containing both (+) and (-) strand RNAs], a new model was proposed in which a (-) strand is synthesized on the (+) strand genomic RNA and then translocated to the 5' end of the genomic RNA to complete synthesis of the leader RNA. The resulting (-) strand RNAs, with leader sequences at the 3' ends, are then copied to form mRNAs. The nested set of mRNAs would be produced by translocation signaled by the individual junction sequences between mRNA coding regions. Adapted from E. J. Snijder et al., *J. Gen. Virol.* 79:961-979, 1998, with permission.



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The Geographic Distribution of the Japanese Encephalitis Serocomplex of the Family Flaviridae, 2000.



- St. Louis encephalitis
- * Rocio and St. Louis (Brazil)
- + West Nile virus
- # Japanese encephalitis
- West Nile and Japanese encephalitis
- Japanese and Murray Valley encephalitis
- O Murray Valley and Kunjin

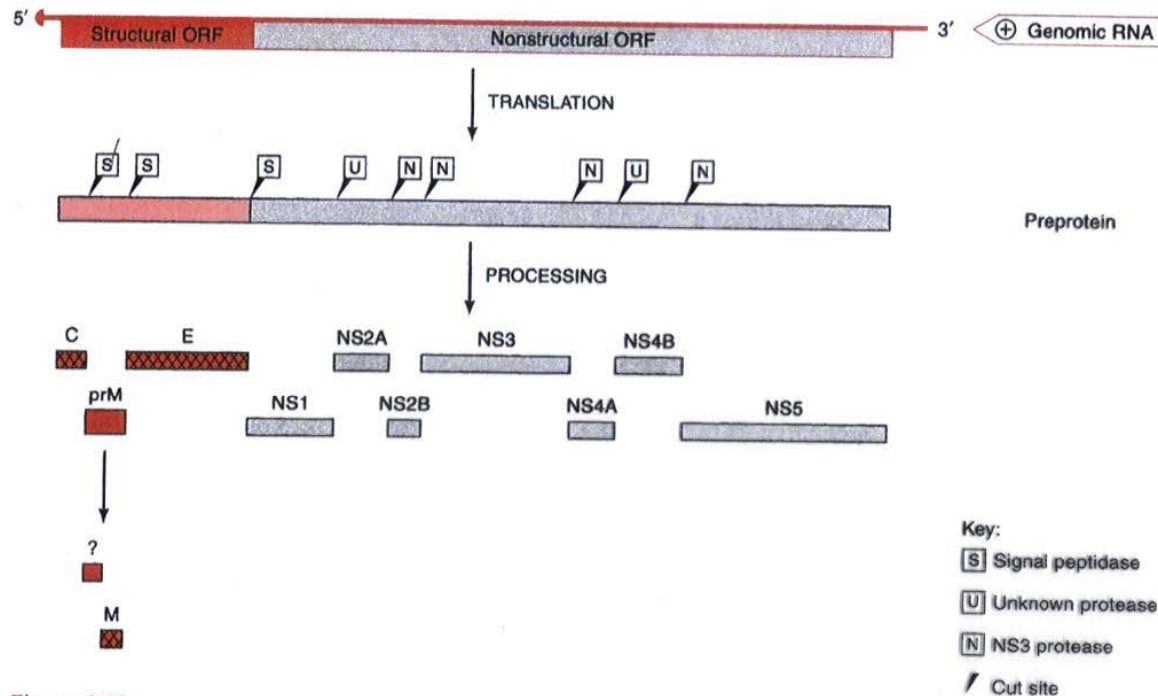


Figure 2.13

Map of the yellow fever virus genome. Conventions for translated regions are the same as those in Figure 2.10. The three crosshatched boxes represent polypeptides found in mature virions. The proteinases responsible for each cleavage are indicated with symbols above the site (see key).

Singer, M. and Berg, P. (1997). "Exploring Genetic Mechanisms". University Science Books. Sausalito, California.

Form 1 $\Delta G = -40$ kcal
Form 2 $\Delta G = -46$ kcal

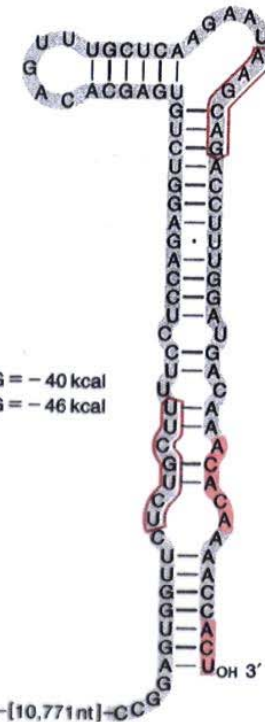


Figure 2.14

Structure at the 3' end of yellow fever virus RNA. A proposed secondary structure for the 3' terminal 87 nucleotides of yellow fever virus (strain 17D) is shown. Nucleotides shaded in color are shared with the 3' terminus of yellow fever vRNA. Form 1 is shown. Form 2 can be formed if the bases outlined in color are base paired to one another. (From C. M. Rice, et al. 1985. Nucleotide sequence of yellow fever virus: Implications for flavivirus gene expression and evolution. *Science* 229 726-733.)

Levy, J. A., Fraenkel-Conrat, H. and Owens, R. A. (1994). "Virology". 3rd edition. Prentice-Hall, Inc.

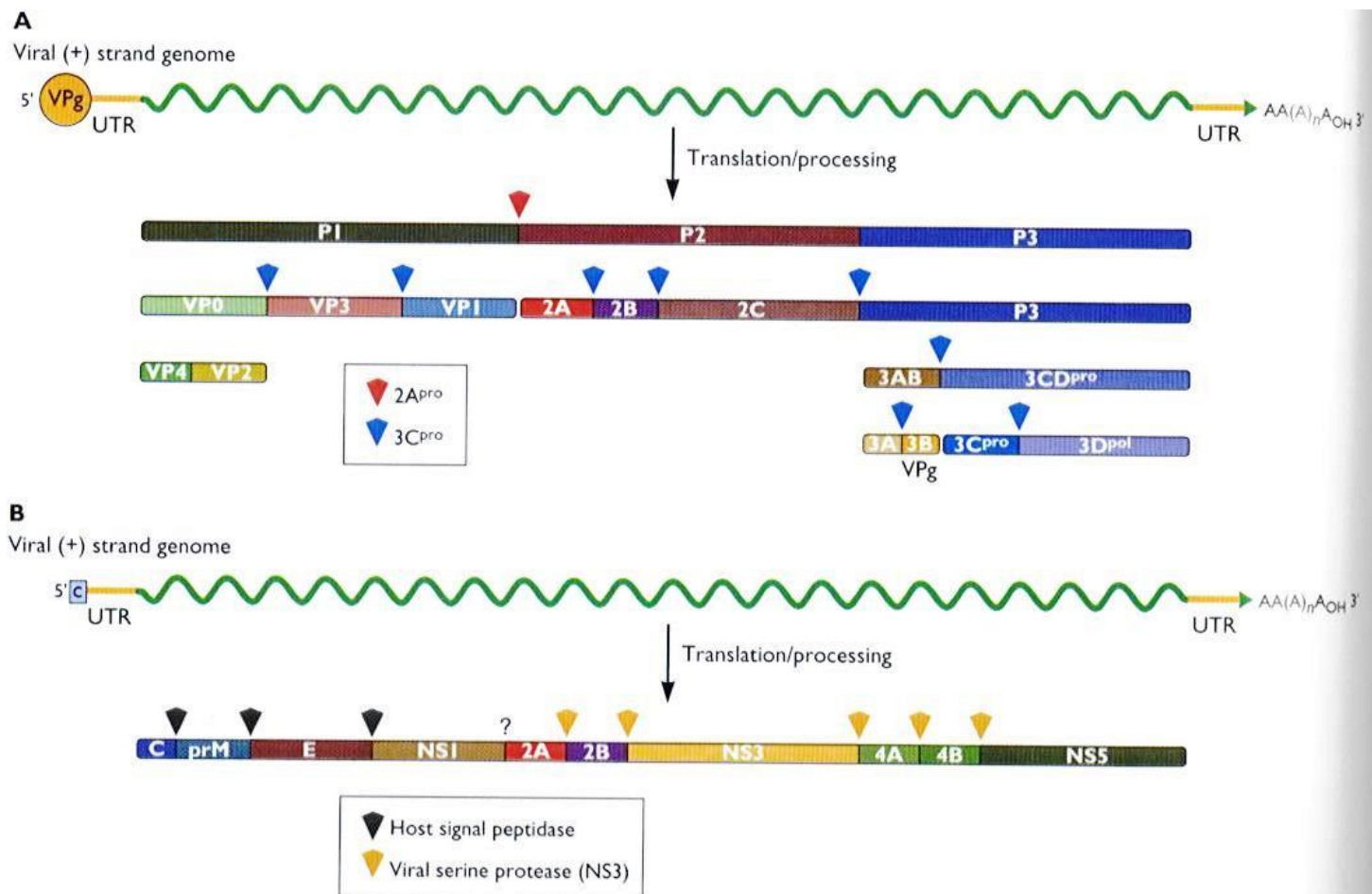


Figure 11.9 Polypeptide processing in enteroviruses and flaviviruses. (A) Protein processing map of poliovirus. The viral RNA is translated into a long precursor polypeptide that is processed by two viral proteases, 2A^{pro} and 3C^{pro}, to form viral proteins. Cleavage sites for each protease are shown. (B) Cleavage map of a flavivirus. Processing of the flavivirus precursor polypeptide is carried out either by the host signal peptidase, which occurs in the lumen of the endoplasmic reticulum, or by the viral protease NS3.

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Picornaviruses:



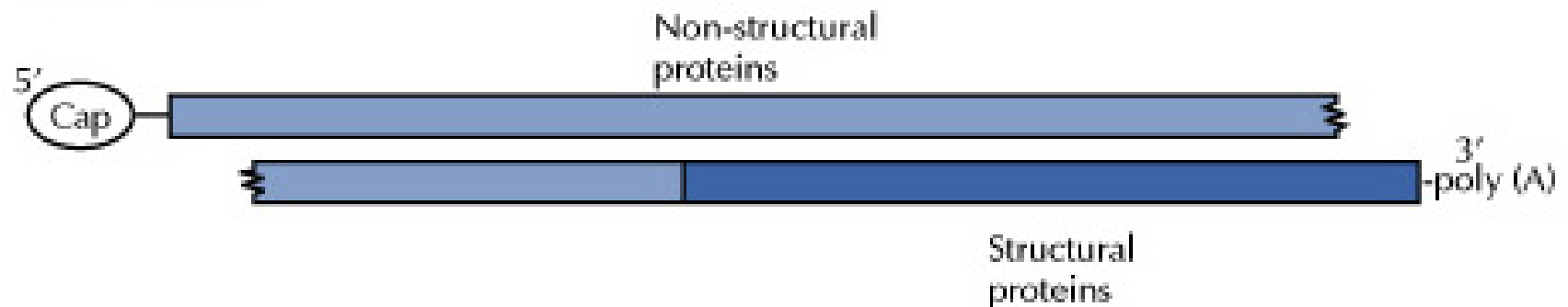
Togaviruses:



Flaviviruses:



Coronaviruses:



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