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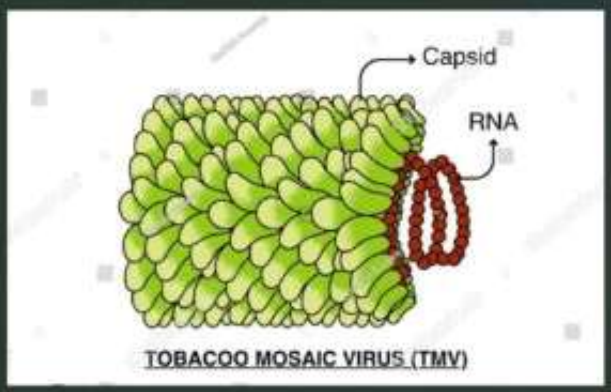
TOBACCO MOSAIC VIRUS (TMV)

BOTANY (MAJOR)
SEMESTER -1
UNIT -1 TOPIC



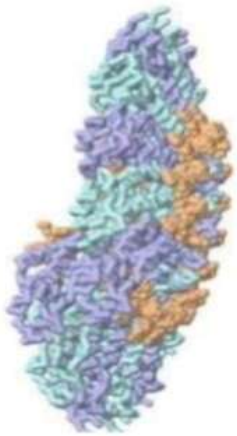
TOBACOO MOSAIC VIRUS (TMV)

Tobacco Mosaic Virus

What is TMV?

TMV is a virus that infects plants. In fact, it was the first virus to be discovered (by Martinus Beijerinck in 1898) and has since been used in many studies to work out how viruses replicate and move between cells.

- TMV (Tobacco mosaic virus) is the most serious pathogen causing mosaic on tobacco leaves . It is transmitted by artificial inoculation but not by insect vectors.
- TMV is the most resistant virus known so far of which the thermal death point is 90°C for 10 minutes.
- This is the first virus that was crystallized in 1935 by *W.M. Stanley* in the U.S.A.



Symptoms

This virus is able to infect over 199 plant species (not just tobacco), these include: tomato, pepper, cucumber and several ornamental flowers.

- The virus causes a mosaic pattern of brown spots on the surface of leaves. The virus doesn't usually cause the plant to die, but can seriously stunt its growth.
- Lower leaves can suffer from 'mosaic burn' in hot and dry weather, where large areas of the leaf die.

Symptoms :

- First appear on new leaves
- Mottling
- Greening & yellowing of leaves
- Green parts - normal growth
- Yellow parts – growth stop
- Result – green parts form pits
- If infection in early stage - leaf twist & malformed
- Old leaves – yellow parts – brown
- Partial sterility in pollens

How does it infect?

- This virus cannot get into plants on its own. Plants are usually infected via plant wounds after human handling or via contaminated equipment.
- The virus can also contaminate seeds, which then grow into infected plants.
- People smoking tobacco can even carry the virus to new plants on their hands.

- Once inside the plant, the virus releases its genetic code (RNA). The plant gets confused by this code, mistaking it for its own, and starts to produce virus proteins.

How does it spread?

One of the viral proteins is a special movement protein, which widens the channels between the plant cells (plasmodesmata). This allows the virus to spread to neighboring plant cells. Eventually the virus can enter the plant's main water and sugar tubes (xylem and phloem) and spread to the whole plant.

What are the effects on the plant?

Viral replication disrupts the plant, distracting it from its own processes, such as photosynthesis and results in the yellowing of leaves. The virus is able to replicate quickly and can account for 10 – 40% of all protein in infected leaves.

What does it look like?

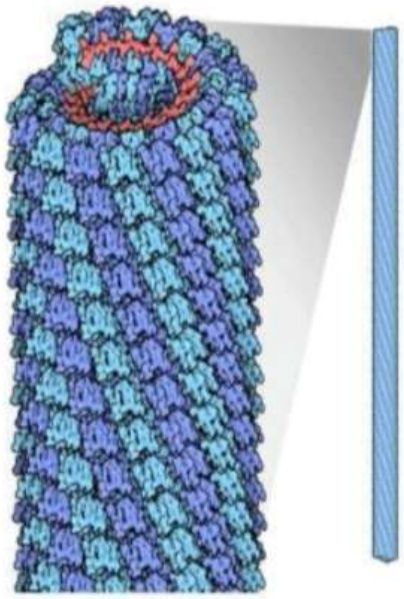
It is rod-shaped, and composed of two main parts:

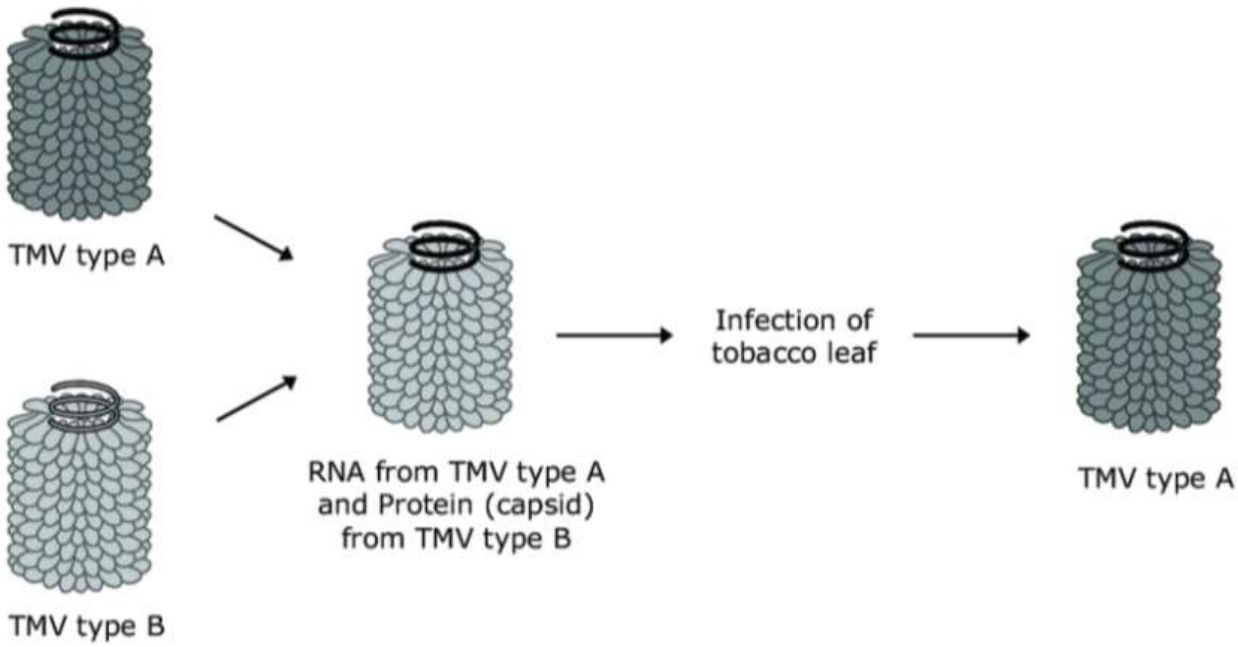
1. Coat proteins 2139 identical proteins coat and protect the RNA code. These proteins make up 95% of viral weight.

2. RNA code A single strand of RNA code forms a spiral structure inside the protein coat. The RNA hijacks plant cells, by forcing them to produce viral proteins. Once the coat protein and RNA come into contact, they quickly assemble themselves into new viruses. These can then spread to other parts of the plant or to new plants.

Description of Molecule

- TMV has one strand of RNA, shown in red.
- RNA is wrapped with stacks of protein, shown in blue.
- Protein Coat: 2,130 copies of a small protein stack on top of each other, spiraling downward.
- RNA strand is encoded in four proteins, transporting the RNA from cell to cell and allowing the virus to spread.

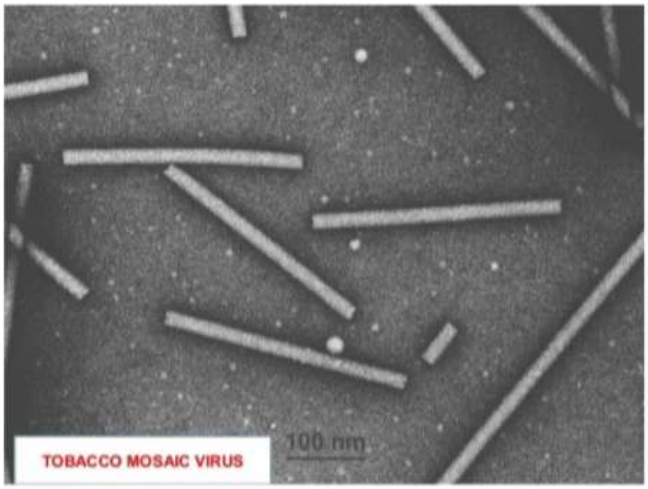




How small is it?

Viruses are very small!

The TMV virus is 300 nanometers long and 18 nanometers wide. This means that 500,000 TMV viruses could fit into the length (15cm) of 1 unsharpened pencil! Why is it a problem? The virus is very stable; it can withstand temperatures of 4°C up to 50°C. It is also able to survive outside the plant for up to 50 years. It is particularly good at spreading between plants grown close together in greenhouses. Finally TMV's wide host range means in can spread between fields of different crops.



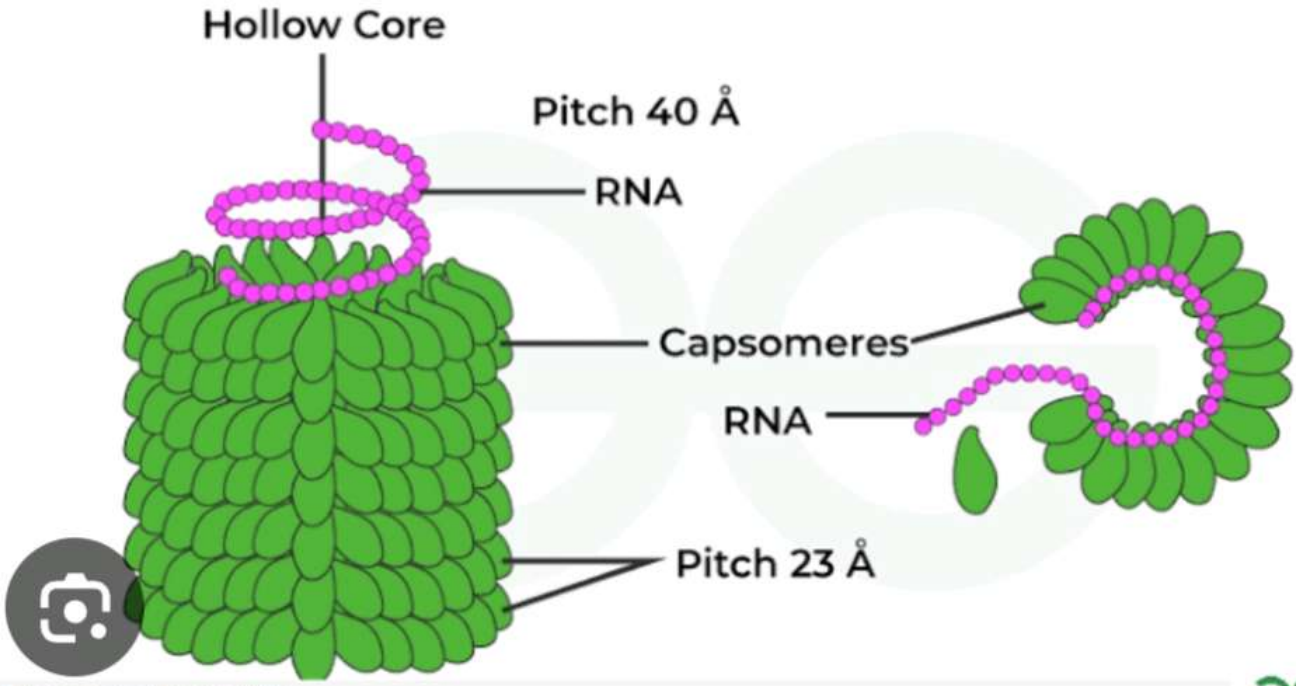
How can it be controlled?

Removing infected plants, washing hands in between planting, and rotating crops can all help to control the spread of TMV.

Farmers can also choose to grow TMV resistant plants.

Disease Management

- Removal of crop resides & infected plants
- Removal of solanaceous weeds
- Crop rotation of 2 years
- Wash hand with Trisodium phospate
- Wash agril. equipments in Trisodium phospate
- No smoking
- Disease resistant varieties- Ambalema



Tobacco mosaic virus(TMV)

Tobacco Mosaic Virus (TMV): Structure and Replication

Structure of Tobacco Mosaic Virus (TMV):

TMV is a simple rod-shaped helical virus (Fig. 13.20) consisting of centrally located single-stranded RNA (5.6%) enveloped by a protein coat (94.4%). The rod is considered to be 3,000 Å in length and about 180 Å in diameter.

The protein coat is technically called 'capsid'. R. Franklin estimated 2,130 sub-units, namely, capsomeres in a complete helical rod and 49 capsomeres on every three turns of the helix; thus there would be about 130 turns per rod of TMV.

The diameter of RNA helix is about 80 Å and the RNA molecule lies about 50 Å inward from the outer-most surface of the rod. The central core of the rod is about 40 Å in diameter. Each capsomere is a grape like structure containing about 158 amino acids and having a molecular weight of 17,000 dalton as determined by Knight.

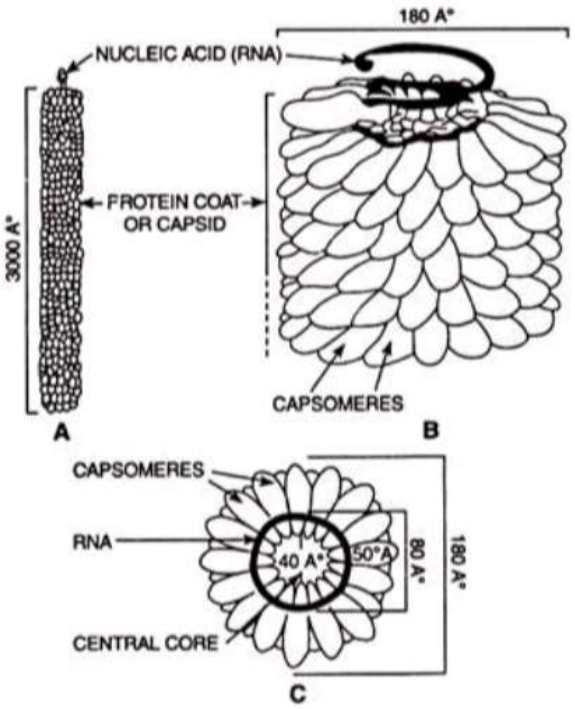


FIG. 13.20. Tobacco mosaic virus (TMV). **A.** surface view; **B.** an enlarged portion showing RNA-capsomere arrangement; **C.** view in section.

The ssRNA is little more in length (about 3300 Å) slightly protruding from one end of the rod. The RNA molecule consists of about 7300 nucleotides; the molecular weight of the RNA molecule being about 25,000 dalton.

Life-Cycle (Replication) of Tobacco Mosaic Virus (TMV):

Plant viruses like TMV penetrate and enter the host cells in toto and their replication completes within such infected host cells (Fig. 13.21). Inside the host cell, the protein coat dissociates and viral nucleic acid becomes free in the cell cytoplasm.

Although the sites for different steps of the viral multiplication and formation of new viruses have not yet been determined with absolute certainty, the studies suggest ha alter becoming free in the cell cytoplasm the viral-RNA moves into the nucleus (possibly into the nucleolus).

The viral-RNA first induces the formation of specific enzymes called 'RNA polymerases' the single-stranded viral-RNA synthesizes an additional RNA strand called replicative RNA.

This RNA strand is complementary to the viral genome and serves as 'template' for producing new RNA single strands which is the copies of the parental viral-RNA. The new viral-RNAs are released from the nucleus into die cytoplasm and serve as messenger-RNAs (mRNAs). Each mRNA, in cooperation with ribosomes and t-RNA of the host cell directs the synthesis of protein subunits.

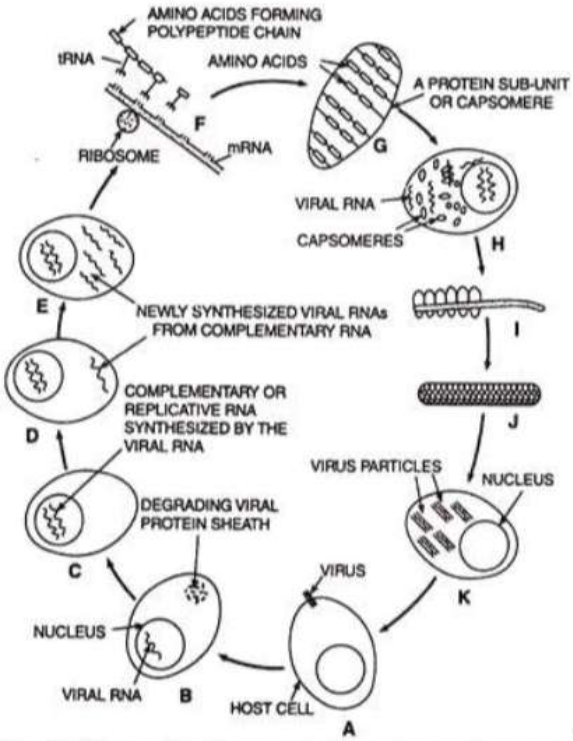


FIG. 13.21. Replication of TMV (diagrammatic). **A.** Virus particle entering inside the cell of the host plant; **B. & C.** Viral RNA enters inside the nucleus and synthesizes its complementary copy; **D. & E.** Complementary RNA synthesizes new viral RNA that comes in the cytoplasm; **F.** Polypeptide chain synthesis; **G., H. & I.** Arrangement of capsomeres around viral-RNA; **J.** Complete virus particle; **K.** Host cell containing many virus particles.

After the desired protein sub-units (capsomeres) have been produced, the new viral nucleic acid is considered to organize the protein subunit around it resulting in the formation of complete virus particle, the virion.

No 'lysis' of the host cell, as seen in case of virulent bacteriophages, takes place. The host ells remain alive and viruses move from one cell to the other causing systemic infection. When transmitted by some means the viruses infect other healthy plants.