Viruses: Pathogens of Plants

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- Overview of the process by which plant viruses cause disease
- Types of plant-virus interactions
- Physiological and cytological effects on host plants
- Positive effects due to virus infection

How do plant viruses initiate an infection?

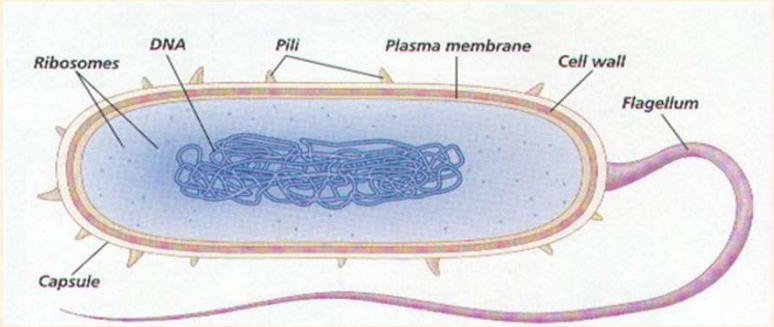
Plant viruses lack an active mechanism for cell entry.

No host receptors are known for plant viruses.

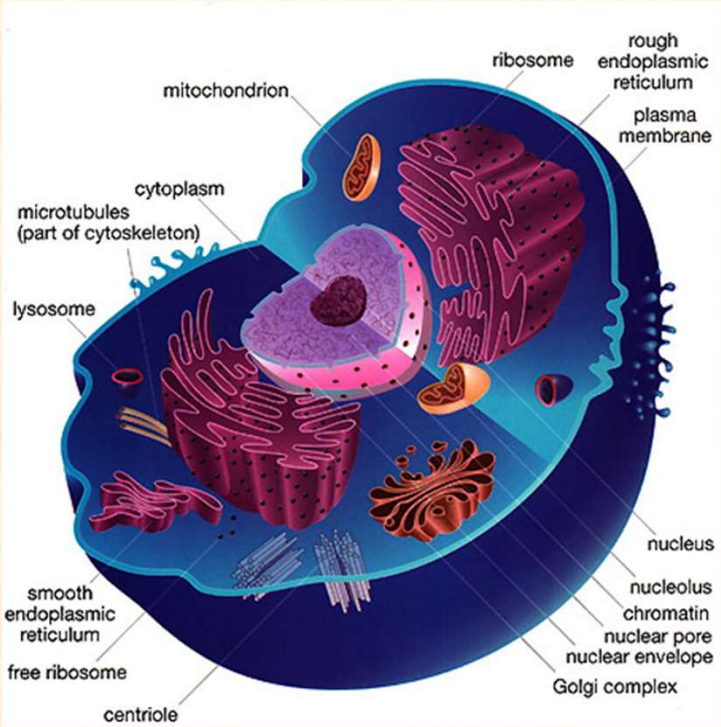
A plant virus must enter via a wound in the plant cell wall and must be at the site during injury OR acquired from a parent plant through fertilization or propagation of infected gametes.

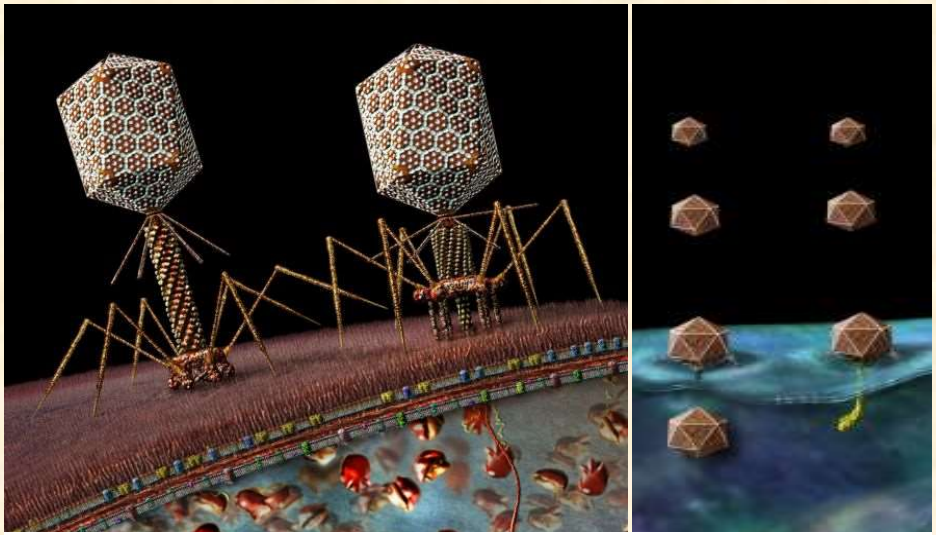
T4 phage injecting its genome into host cell

Prokaryotic cell



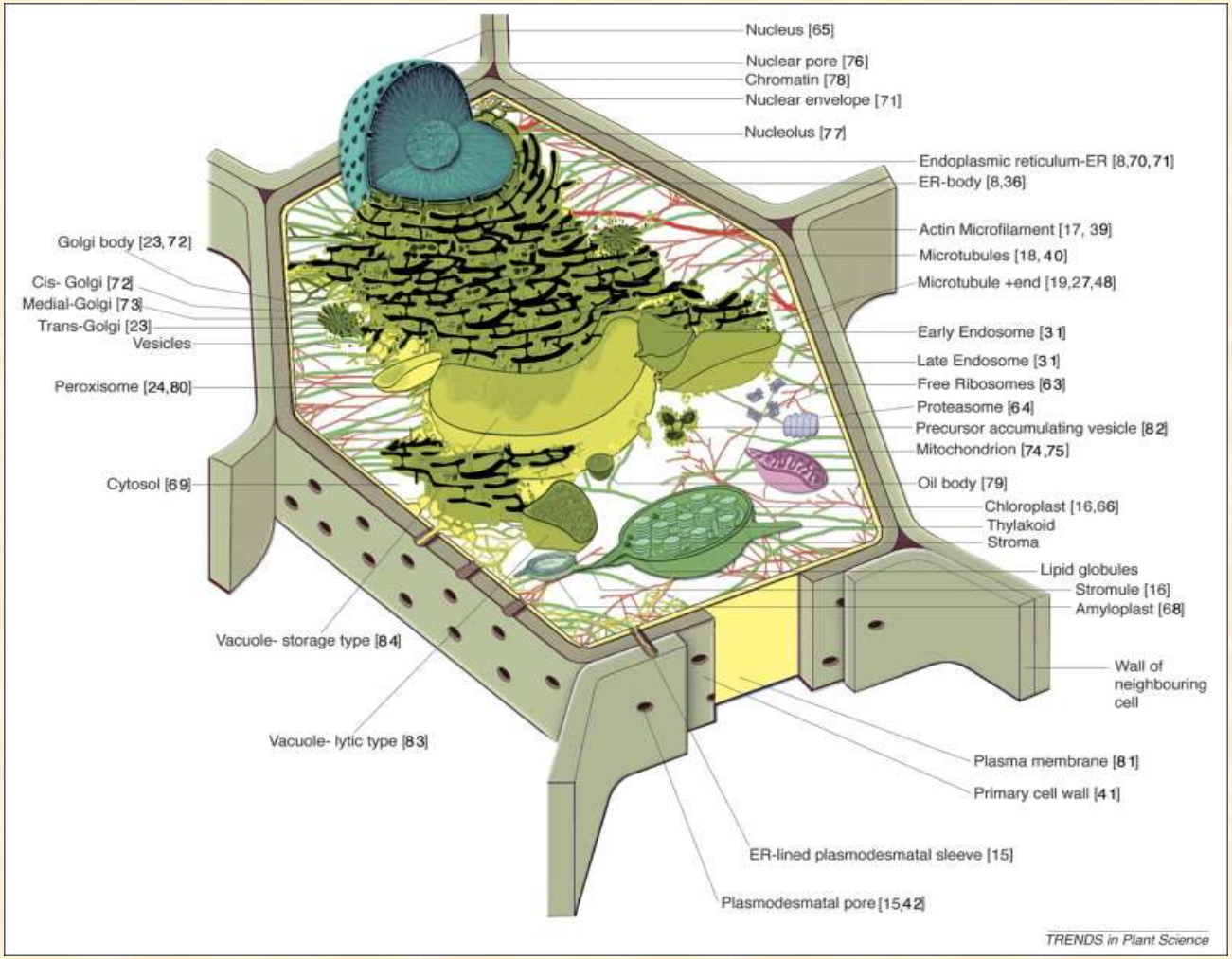
Eukaryotic cell



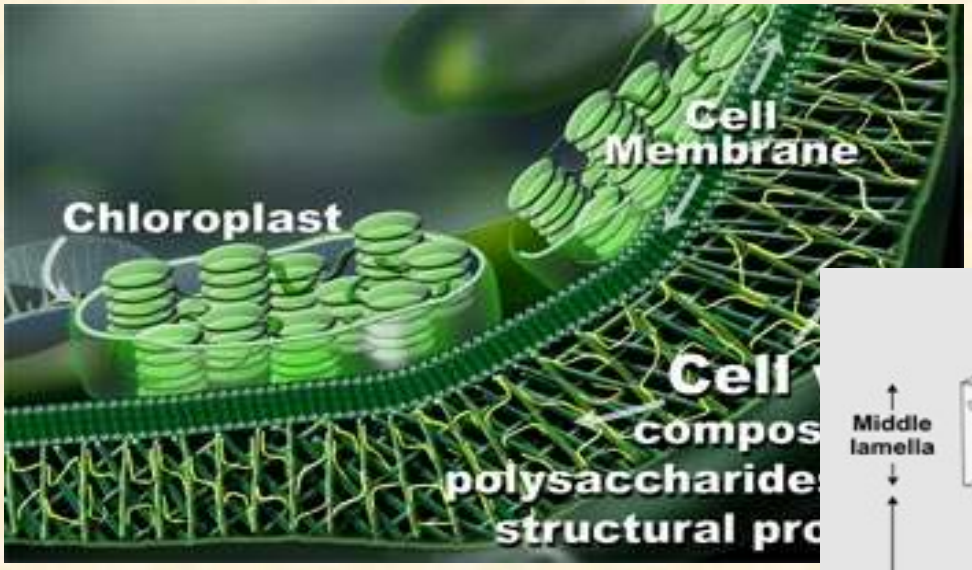


Examples of active means of infection (requires host receptors)

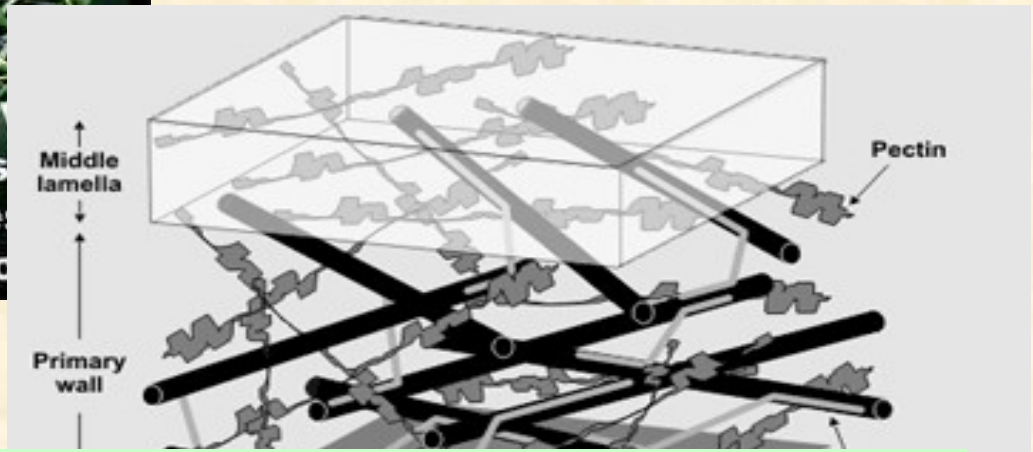
Eukaryotic Cell - Plant



Plant cell walls are thick compared with the sizes of the viruses infecting them (>10 µm versus <1 µm),



Plus, outer surfaces of plants have layers of waxes and pectin



No virus has evolved a mechanism to directly penetrate the plant cell wall

What types of interactions can occur once a virus is introduced into a plant?

1. There is no or very limited viral replication, no cell-to-cell movement - Immune, Immunity.

2. Virus replicates and moves through out the plant without obvious symptoms – Latent, Tolerance.

3. Virus replicates and causes symptoms in new leaves but with time and further plant growth symptoms disappear and virus cannot be detected – Recovery.

4. Virus replicates and plant responds with a hypersensitive response, may or may not move systemically (programmed cell death) - Local Lesions, Systemic Necrosis.

5. Virus replicates and moves through out the plant with obvious symptoms – "Susceptible".

Which of these possible responses do you think is the most common?

1. There is no or very limited viral replication, no cell-to-cell movement - Immune, Immunity.

2. Virus replicates and moves through out the plant without obvious symptoms – latent (tolerance)

3. Virus replicates and causes symptoms in new leaves but with time and further plant growth symptoms disappear and virus cannot be detected - recovery

4. Virus replicates and plant responds with a hypersensitive response, may or may not move systemically (programmed cell death) (local lesions, systemic necrosis)

5. Virus replicates and moves through out the plant with obvious symptoms

1. There is no or very limited viral replication, no cell-to-cell movement - Immune, Immunity.

2. Virus replicates and moves throughout the plant without obvious symptoms – Latent, Tolerance.

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Latent infections or Tolerance

- In some cases the virus/host combination does not result in obvious symptoms


TYLCV in tomato


TYLCV in tobacco

Latent infections or Tolerance

- In wild plants, virus infection often does not correlate with the presence of symptoms.

- Many of them have a persistent lifestyle and do not encode any proteins to aid in their dissemination.

- It is thought that these viruses may have been associated with their wild hosts for a long period on the viral evolutionary timescale.

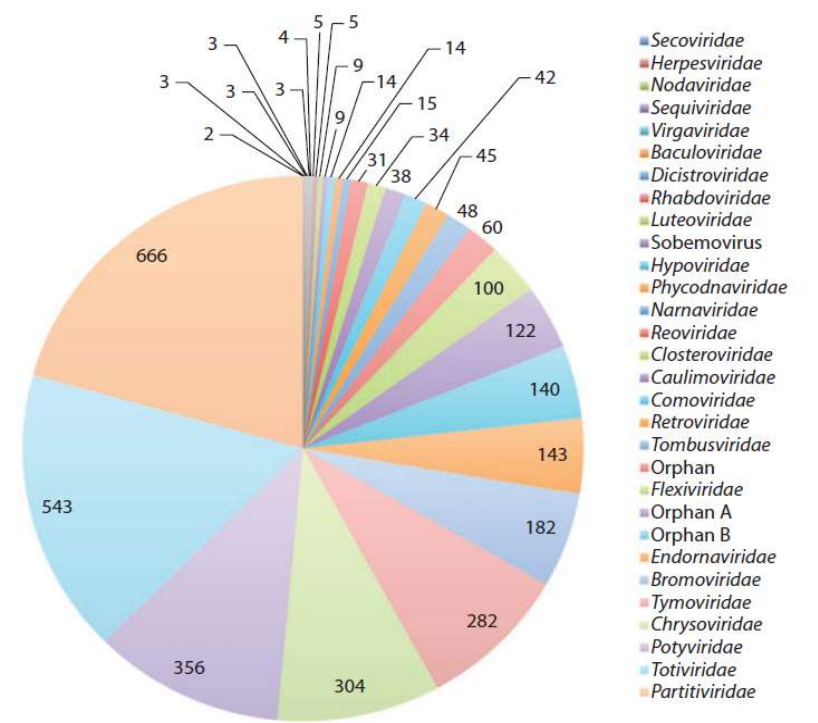

Figure 3

Distribution of plant virus families in wild plants. Numbers indicate the number of individual plants with viruses in the indicated families. Data are pooled from two study sites and are preliminary.

Roosinck 2012 Ann. Rev Plant Genetics 46:357

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Plant Recovery:

- Plant shows symptoms in leaves after infection but the symptoms become milder with each new leaf and within a few weeks there are no symptoms and no detectable virus in the new leaves. (first studied in early 1960's)

- The explanation for this was a mystery for many years

- Now known to be due to active plant responses to infection by viruses: post-transcriptional gene silencing (PTGS) which are activated upon infection and then are able to shut down virus replication over time.

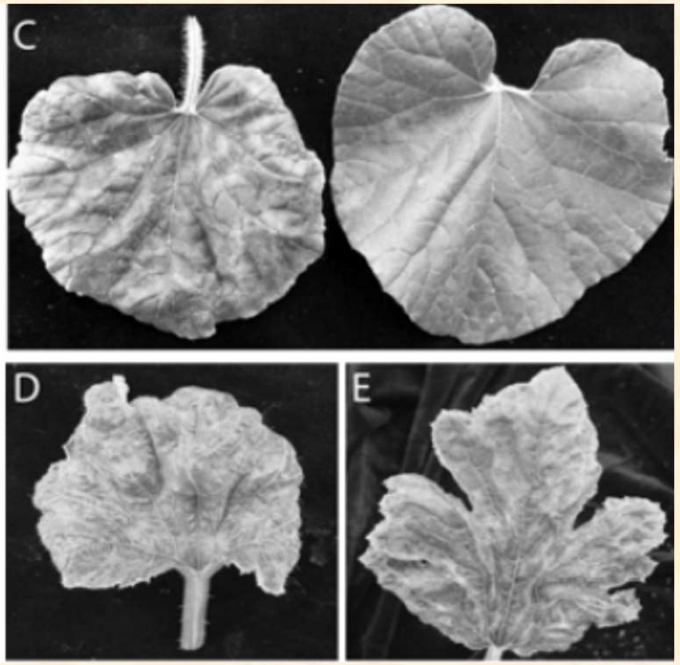
Plant Recovery:

- **Common in plants genetically engineered for resistance to virus using viral sequences**
- **Also can occur in without genetic intervention**

Example:

- **Cantaloupe and cucurbit leaf crumple virus**

Cantaloupe (recovery)



Zucchini (no recovery)

C: Cantaloupe leaves showing typical symptoms of CuLCrV infection at 10 dpi (non-recovered, left) and recovery from CuLCrV infection at 20 dpi (recovered, right);
D and E: Zucchini leaves showing typical symptoms at 10 and 20 dpi. [from Hagan et al 2008 Phytopathology 98:1029-1037].

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Virus replicates in the first infected cells, and the host produces a hypersensitive response (resulting in clusters of dead cells = local lesion).

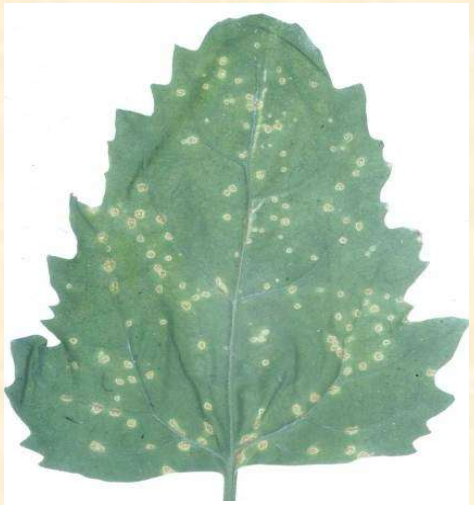
In some plant/virus combinations, this is the end of any symptom production, virus spread, and virus reproduction.

Local Lesions:

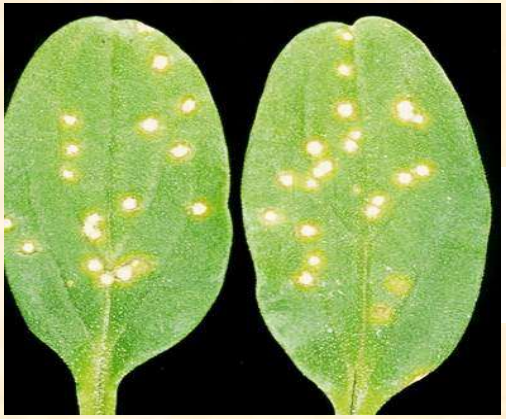
- **Occur on the inoculated leaf within a few days of inoculation**
- **Don't appear in all plant/virus/host combinations**
- **Some viruses never produce local lesions in any host**
- **Viruses can produce local lesions in some hosts but not in others**
- **Lesions vary in size, color, time to appearance**
- **Local lesions can be the only symptom or can be followed by systemic infection with or without systemic symptoms. (local lesions ≠ lack of systemic spread**

Local lesions vary in size, shape and color

Chenopodium quinoa inoculated with *Carnation mottle virus*



C. quinoa inoculated with Parsnip yellow fleck virus



Cucumis sativus inoculated with *Clover yellow vein virus*

Nicotiana tabacum 'Xanthi' inoculated with *Cherry leaf roll virus*

Phaseolus vulgaris 'Pinto 111' inoculated with *Bean pod mottle virus*

Necrotic local lesions extending along the veins. Caused by a strain of *Bean common mosaic virus* in primary leaves of *Phaseolus vulgaris* 'Monroe', 13 days after mechanical inoculation.

Uses of Local Lesions:

1. Number of lesions can be an indictor of the number of virus particles in the inoculum (similar to a plaque assay)

2. Biological indicator for identification of a virus

3. Rapid indicator of infectivity

4. Used to select and create host resistance in cultivated crops

Systemic necrosis: necrosis is not limited to the inoculated leaf.
Plant responds with a local necrosis, but virus continues to move and infected cells respond with programmed cell death and die, resulting in extensive necrosis.

Tobacco infected with *Tomato spotted wilt virus (Tospovirus)*

Tobacco mild green mosaic virus (*Tobamovirus*) in tropical soda apple

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"Susceptible":

- **Virus enters a plant cell,**
- **Replicates in the cell,**
- **Moves to neighboring cells,**
- **Moves into the phloem, then to sinks (root and shoot apices), and then spreads to neighboring cells**
- **Virus replication in apical cells may cause developmental abnormalities**
- **Cellular abnormalities give rise to foliar symptoms**

- Symptoms develop in leaves days/ weeks after infection

- Symptoms develop in leaves distant from site of inoculation

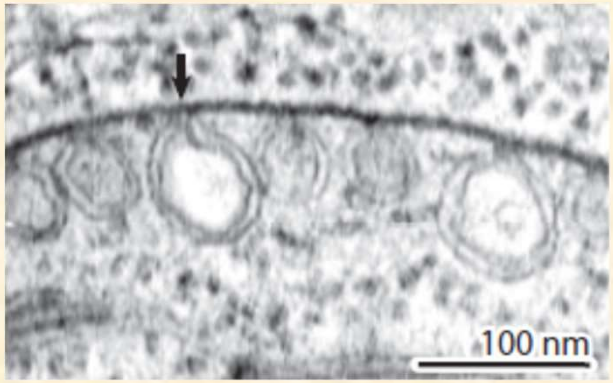
Systemic Infection with Symptoms:
Effects of Viruses on Plants

Effects of Viruses on Host Cell Structure

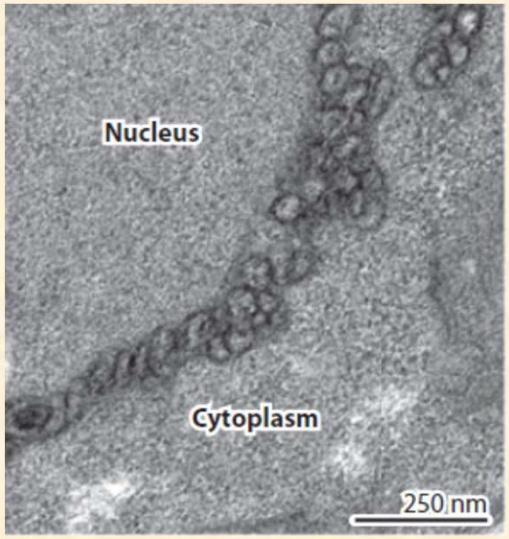
When viruses replicate they make many dramatic changes to the host cells.

Some viral proteins modify the cell to create virus “factories” also known as viroplasms. These are the locations where the progeny viruses are created.

Often these changes occur in/on host membranes resulting in vesicles and other alterations to membranes.

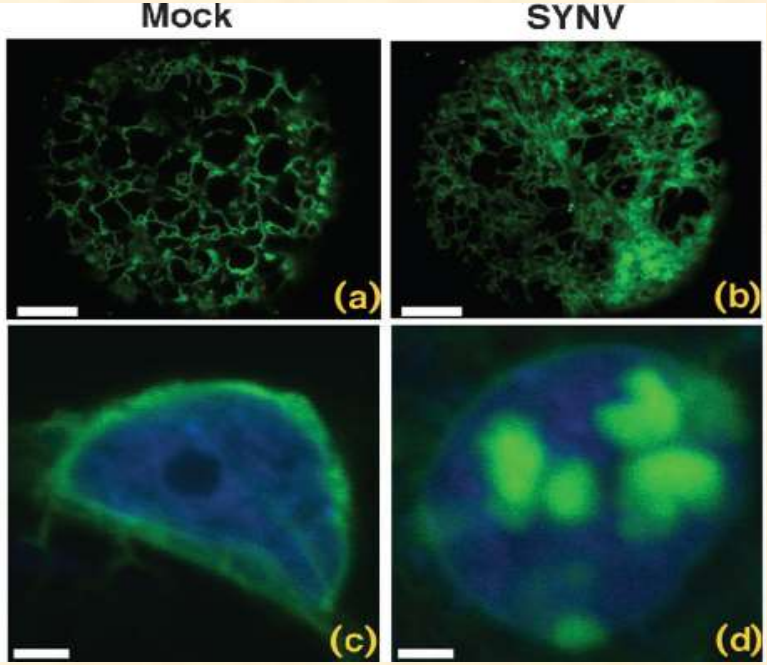


Vesicles associate with different membranes



Effects on the Cell and Nucleus:

Whole cell (Protoplast)



Green – Endoplasmic reticulum (ER) tubules

Nucleus

Green – nuclear membranes

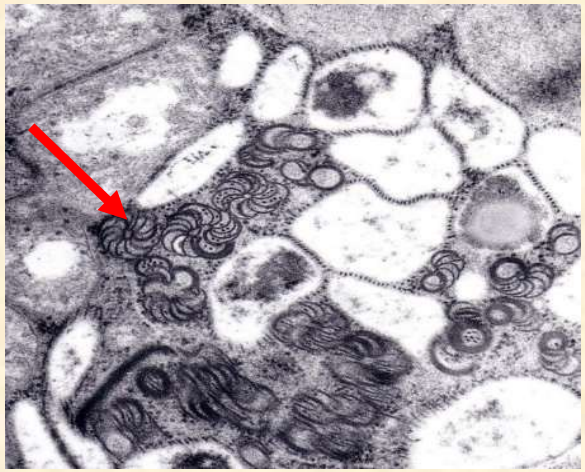
- Changes to a cell and a nucleus inducted by *Sowthistle yellow vein virus* (*Rhabdoviridae, Nucleorhabdovirus*)
- Distribution of ER tubules is altered in the protoplasts.
- Distribution of nuclear membranes is altered, instead of enclosing the nucleus it is clumped.

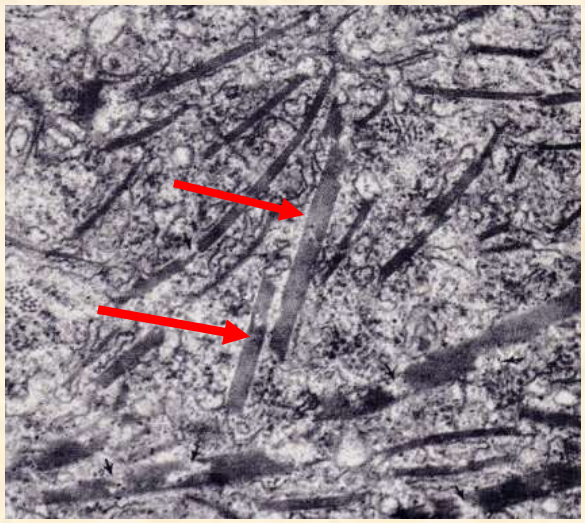
From: Goodin et al J. Gen.Virol. (2007) 88:1810–1820

Structures appear in cells:
Some can be seen with staining using a light microscope and with better resolution using an electron microscope.

These altered structures are known as "Inclusions" or "Inclusion bodies"

These can be found in the nucleus or cytoplasm depending upon where the virus replicates, they vary in shape and size as well as location.



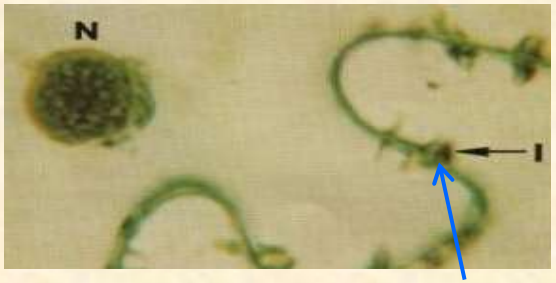


The location and type of inclusion varies with the virus (not so much with the host).

For a time these differences were exploited and used for diagnostic purposes.

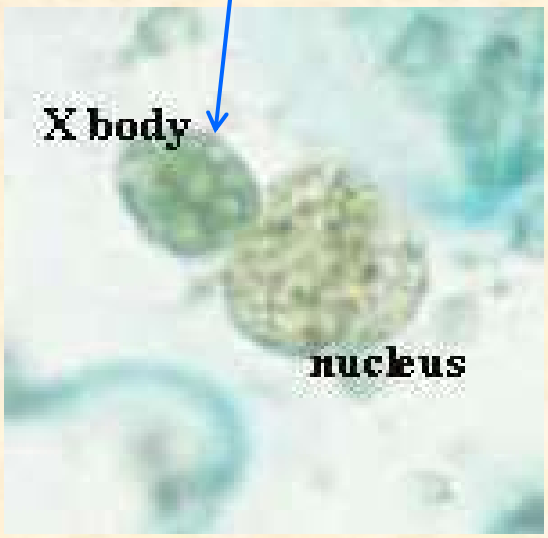
Although they are only rarely used today, they are still useful.

Light micrographs



Inclusion

Inclusion



Viral Inclusion Bodies Composed of:

- Virus particles
- Plus/or other proteins of the viral genome
- Plus/or modified host cell constituents

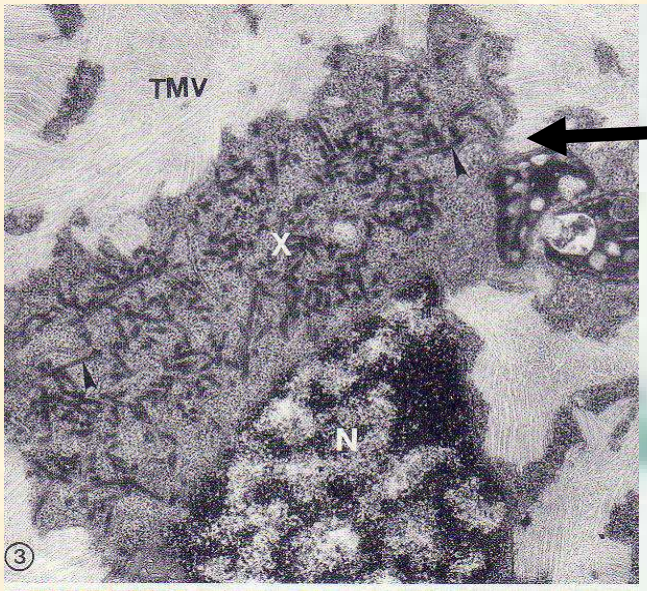
What are Inclusion Bodies?

- Sites of viral replication and/or assembly ("virus factories")
- Sites of accumulation of virus particles
- Sites of accumulation of excess viral protein(s)

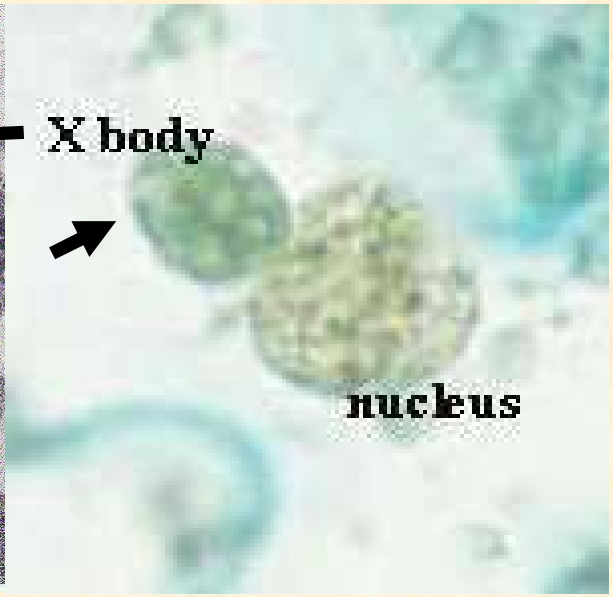
The function of the inclusion body varies among virus families and sometimes even genera in the same family

Viral Inclusions: Comparison of visualization in EM and light microscope

Electron micrograph



Light micrograph

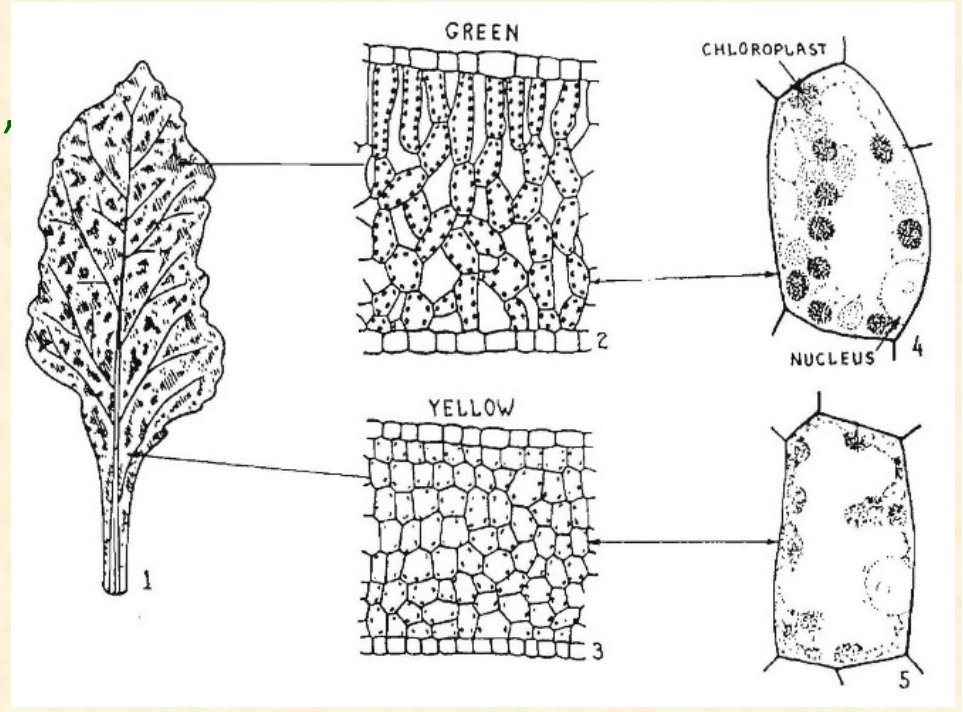


X body: Inclusion of *Tobacco mosaic virus* (TMV)

Effects of viruses on plant physiology:

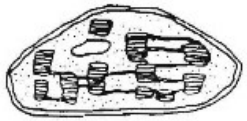
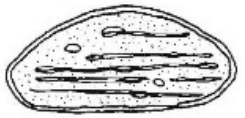
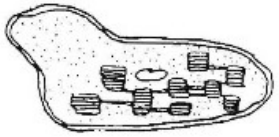
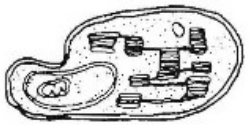
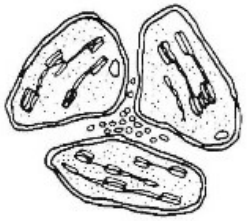
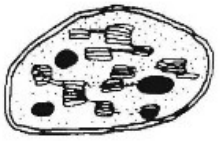
Changes at the cellular level result in changes to the plant's physiology

- Decrease in the rate of **photosynthesis** due to reduced amounts of chlorophyll, chloroplast ribosomes, and ribulose bisphosphate carboxylase



Effect of mosaic disease upon the sugar beet leaf. (1) Mosaic pattern on leaf. Green areas are shaded, yellow areas are left blank. (2) Mesophyll from a green area. It shows a loose arrangement of cells and numerous chloroplasts. (3) Mesophyll from a yellow area. It shows compact arrangement of cells like a young leaf. This underdevelopment is one of the expressions of hyperplasia. The chloroplasts are few. The deficiency in chloroplasts makes the tissue appear yellow. (4) Cell from green mesophyll with numerous chloroplasts. (5) Cell from yellow mesophyll. The chloroplasts have become partly or completely disorganized. (From Esau, K., *Am. J. Bot.*, 43, 739, 1956. With permission.)

Effects on chloroplasts in different virus/hosts:

Disease	Description	Diagrammatic Illustration
A chloroplast from a noninfected plant	The size, shape and distribution of lamellae are normal.	
Barley Stripe mosaic virus in young barley plants	Chloroplasts lack a developed grana system. They primarily have intergrana lamellae.	
Arabis mosaic virus in *Arabis sp.* Southern bean mosaic virus in bean	Chloroplasts hump out at one or more places. There is an increase in stroma and no lamellae in the hump.	
Some strains of tobacco mosaic virus in tobacco	An extrusion of the chloroplast which invaginates some of the ground cytoplasm of the cell. Mitochondria may even be found in the enclosure.	
Turnip yellows mosaic virus in Chinese cabbage	Clumping of chloroplasts with vesicles in the center of the array. Chloroplasts have a limited system of lamellae.	
Tobacco mosaic virus in the chlorotic halo area of local lesion — Datura	Large osmiophilic globules accumulate in the chloroplast and the lamellae are distrupted.	

Effects of viruses on plant physiology:

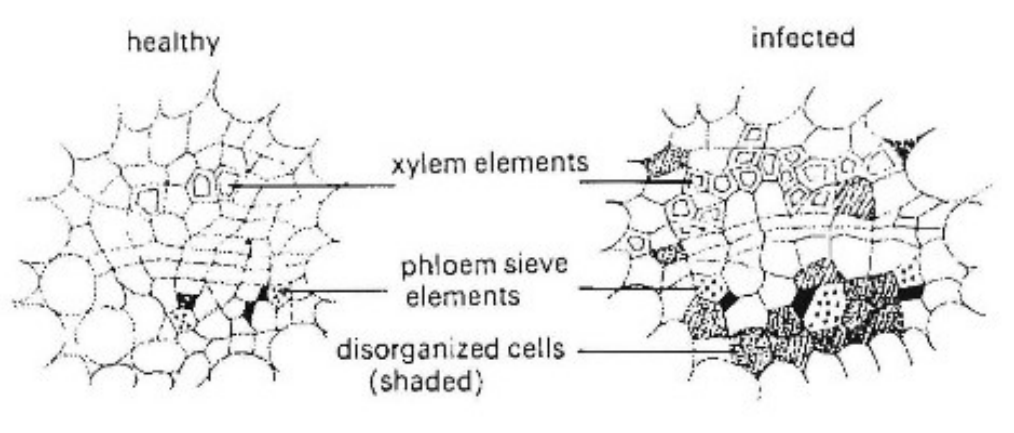
Changes at the cellular level result in changes to the plant's physiology

- Increase in rates of **respiration**
- Increase in **activity of some enzymes**, such as polyphenoloxidase (PPO)
    - PPO plays a role in biotic and abiotic stress responses
    - There is a close relationship between PPO gene expression and starch biosynthesis

Effects of viruses on plant physiology:

Changes at the cellular level result in changes to the plant's physiology

- Altered levels or ratios of various **plant hormones**
  (Symptoms such as epinasty and leaf abscission, may be due to increased levels of ethylene production)



Transverse sections of tobacco leaves showing xylem proliferation and cell enlargement (hypertrophy) resulting from virus infection. (From Stevens, W.A., *Virology of Flowering Plants*, Blackie & Sons, Glasgow, 1983, 28. With permission of Kluwer Academic Publishers.)

Hypertrophy
(cell enlargement and proliferation)
Results in galls

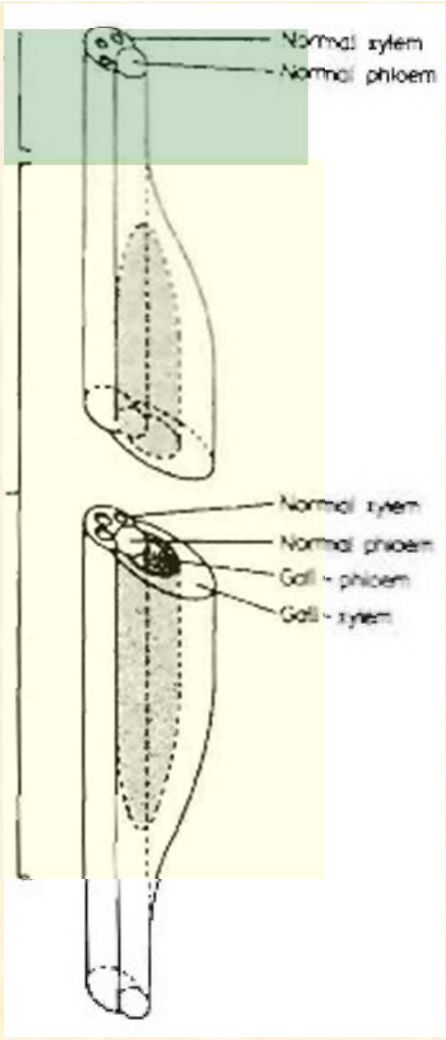


FIGURE 259
Vascular anatomy of a galled vascular bundle in a sugarcane leaf infected with Fiji disease virus. Only the distribution of xylem and phloem tissues is shown and a section of the bundle through the galled area has been removed to expose the tissues in transverse section. The galls appear to result from virus-induced cell proliferation. The proliferating cells develop into abnormal phloem (gall-phloem) and xylem (gall-xylem). Virus particles and viroplasms are confined to these tissues. (From Hatta, T., and Francki, R.I.B., *Physiol. Plant Pathol.*, 9, 321, 1976. With permission.)

Vascular galls impair movement of nutrients and water which can result in chlorosis, necrosis, reduced growth, reduced yields, plant death

Effects of viruses on plant physiology (how plant viruses reduce yields):

- **Diversion of host metabolic materials**
- Reduction in the availability of **fixed carbon**, which directly reduces photosynthesis, reduces the translocation of fixed carbon in the phloem.
- Impair the **movement of photo-assimilates** from the leaves and increase **starch accumulation, can change amounts and types of sugars**

How do viruses cause these changes??

Some of these effects are due to Post-transcriptional Gene Silencing (PTGS) –

- An ancient cellular process that plays an important role in management of cellular processes (elimination of mRNA)
- Plant viruses interact/interfere with the plant cell's PTGS processes
- So some genes that should be turned off (by normal PTGS) are not or are not turned off at the right time, this results in many of the typical virus symptoms

Do all virus infections result in a negative outcome?

Human:

Flower breaking caused by a number of different viruses

Foliar mosaics
Abutilon mosaic virus in *Abutilon spp.*

Cold tolerance induced in beets by
Cucumber mosaic virus

Human Con't:

♦ *Prune dwarf virus* causes a disease of cherry in which cherries are borne at the ends of the branches. This exposure results in larger and better colored fruit.

♦ *Citrus exocortis viroid* and *Citrus dwarfing viroid* (aka *Citrus viroid* III) have been used to dwarf citrus, thereby allowing a predictable degree of tree size control and the use of higher planting densities

Plant:

♦ Viruses can make the plant more resistant to other pathogens, insects, drought and increase tolerance to salt.

♦ Viruses can improve the ability of wild plants to compete with other species and survive.

It depends upon your perspective

Insect:

the plant journal

SEB Society for Experimental Biology

The Plant Journal (2014) doi: 10.1111/tpj.12417

The Nla-Pro protein of *Turnip mosaic virus* improves growth and reproduction of the aphid vector, *Myzus persicae* (green peach aphid)

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