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The altered photosynthetic machinery during compatible virus infection

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As an organelle only found in plant cells and some protists, the chloroplast is not only the main metabolic energy originator, but also the abiotic/biotic stress sensor and defense signal generator. For a long time, chloroplasts have been recognized as a common target by many plant viruses. Viruses may directly modify chloroplast membranes to assemble their replication complex for viral genome replication. Viruses may downregulate chloroplast-related and photosynthesis-related genes via an as yet unknown mechanism to support their infection. Viruses may also interrupt functionality of the photosynthetic machinery through protein–protein interactions. This review briefly summarizes current knowledge about modifications of the photosynthetic machinery by plant viruses, highlights the important role of chloroplasts in the infection process and discusses chloroplast-associated pathogenesis.

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Introduction

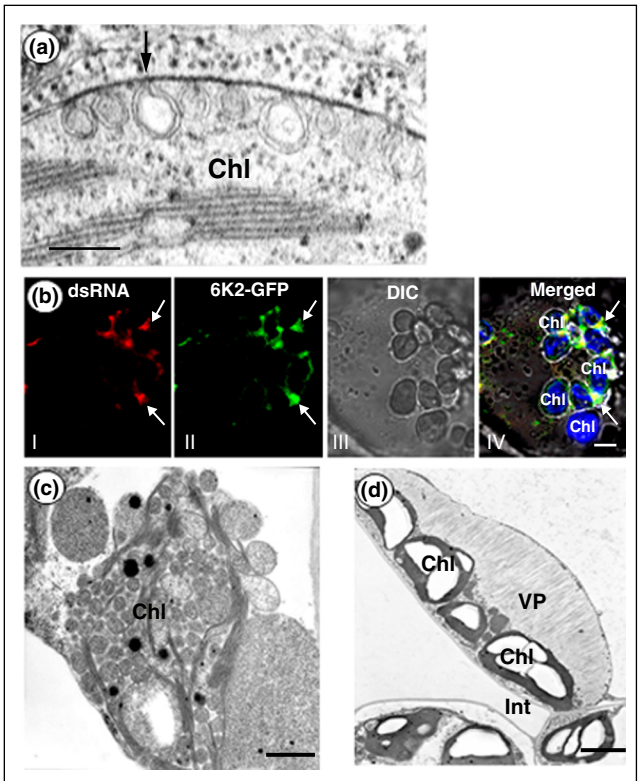
Viruses are obligatory-intracellular parasites that exclusively live in their host cells, and usurp cellular machineries and resources to reproduce their progeny therein [1–3,4••]. Viral infections induce transcriptomic, proteomic, metabolomic, and morphological changes. In infected plants, common physiological and biochemical alterations include a decrease in photosynthesis, an increase in respiration, the accumulation of nitrogen compounds, and expanded oxidase activities [5]. These modifications are accompanied by the appearance of a variety of disease symptoms, particularly associated with plant foliage. Accumulated evidence suggests that among cellular organelles, chloroplasts are a common target by plant viruses. As a metabolically versatile organelle unique to plant cells and some protists (such as algae), the chloroplast, responsible for photosynthesis, is the main energy sources [6]. Chloroplasts are also the sites where fatty acid metabolism, nitrate assimilation and amino-acid biosynthesis take place [7], extending their essential functionalities to defense response via biosynthesis of signaling molecules such as fatty acids and their derivatives, salicylic acid (SA), and jasmonic acid (JA) [8–13]. Therefore, it is not surprising that the interaction between the chloroplast and the invading virus plays a critical role in viral infection and pathogenesis. This short review intends to address alterations of the photosynthetic machinery by plant viruses. As virus–chloroplast interactions represent only one portion of the big picture about the plant–virus interactions, readers are referred to articles published in this themed issue for several other aspects of viral pathogenesis in plants.

Ultrastructural modifications of chloroplasts

Although viral symptoms may depend on host plant, environmental conditions such as temperature, and virus strains, viral infection generally induces mosaic, mottle or chlorosis, and necrosis symptoms. One of a few best cytologically studied viruses is *Turnip yellow mosaic virus* (TYMV), the type member of the genus *Tymovirus*. TYMV induces chlorotic local lesions and systemic yellow mosaic symptoms on cruciferous hosts such as Chinese cabbage. In infected cells, chloroplasts develop numerous 50–60-nm vesicles along their peripheries via invagination of both their outer membranes (Figure 1a), and then become swollen, rounded, vacuolized and clumped together in groups forming chloroplast aggregates [14•,15]. In contrast, in the healthy cells, the chloroplasts are oblong-shaped and evenly distributed. Similar to TYMV, *Barley strip mosaic virus* (BSMV), the type member of the *Hordeivirus* genus, also induce many chloroplast invaginations along the chloroplast periphery [16]. Hosts develop local lesions, and mosaic symptoms [17].

Infections by potyviruses such as *Potato virus Y* (PVY), *Turnip mosaic virus* (TuMV), *Tobacco etch virus* (TEV),

Figure 1



Modification of chloroplasts for viral replication. **(a)** Electron microscopy of an ultrathin section of part of a chloroplast from a TYMV-infected Chinese cabbage leaf cell. The arrow indicates a vesicle in which an open channel is apparently connecting the interior to the cytoplasm. Bar = 100 nm. **(b)** Confocal colocalization of virus replication vesicles with 6K2 bodies associated with aggregated chloroplasts in protoplasts isolated from *Nicotiana benthamiana* leaf tissues infected by a recombinant TuMV containing additional 6K2 tagged by GFP. dsRNA, signals derived from staining with monoclonal antibody J2; DIC, differential interference contrast; Chl, chlorophyll autofluorescence (in blue). Bar = 8 μm. **(c and d)** Electron microscopy of an ultrathin section of tobacco mesophyll cells infected by TRV. (C) Disorganized chloroplast (Chl) structure with numerous vesicles. Bar = 0.2 μm. **(d)** Large, organized TRV particle (VP) inclusion. Chloroplast (Chl) with disorganized lamella structure and increased number of starch grains. Int: intercellular space. Bar = 0.1 μm. Panel a is adapted with permission from Ref. [14•] © 2001 Academic Press; Panel b is adapted with permission from Ref. [22•] © 2010 American Society for Microbiology; Panels c and d are adapted with permission from Ref. [27•] © 2015 Elsevier.

Soybean mosaic virus (SMV), and *Plum pox virus* (PPV) usually induce mosaic, chlorosis and leaf distortion symptoms as well. In host cells, chloroplast ultrastructural changes include vesicular invagination, membrane-bound extrusion, abnormal distortion, dilated thylakoids, chloroplast-associated vesicles, and the viral particle-containing elongated tubular structures between the clustered chloroplasts [18–23]. Large chloroplast aggregates are also evident [18,22•] (Figure 1b).

Infection by the *flavum* strain of *Tobacco mosaic virus* (TMV), a member of tobamoviruses, induces the chlorotic symptom in the mature leaves too, where chloroplasts are swollen to nearly or fully globular, have distorted thylakoid membranes, and often lack the grana [24]. Similarly, abnormal thylakoid membranes and reduced granum stacks are also evident in the chlorotic leaf tissues of tobacco plants infected by *Cucumber mosaic virus* (CMV), a cucumovirus, and the degree of thylakoid membrane abnormality is correlated with chlorosis severity [25,26••]. Interestingly, *Tobacco rattle virus* (TRV), a member of the genus *Tobravirus*, is a widely used virus-induced gene silencing vector. TRV-infected tobacco and potato show systemic necrosis and leaflet deformation. In TRV-infected cells, the ultrastructure of the main cell organelles including the nucleus, endoplasmic reticulum (ER), mitochondrion and chloroplast all undergoes remarkable changes [27•]. Chloroplasts usually display strongly disturbed arrangement of thylakoids with highly accumulated starch grains. Taken together, these observations clearly establish a correlation between typical foliage symptoms and the malformations of the structure or function of chloroplast, or its major components thylakoid and grana.

Targeting of the viral protein/replication complex to chloroplasts

Viral replication is closely associated with virus-induced intracellular membranous structures [1,2,4,28]. Depending on the type of virus, the virus-induced membranes that house the viral replication complex (VRC) are derived from distinct organelles such as the ER, endosomes, mitochondria, peroxisomes, and chloroplasts. As mentioned above, TYMV induces the formation of many vesicular invaginations of the chloroplast outer membranes for viral genome replication (Figure 1a). TYMV encodes two replication proteins, 140 K and 66 K. The latter encompasses the RNA-dependent RNA polymerase (RdRp) domain, whereas the former contains domains indicative of methyltransferase, proteinase, and NTPase/helicase. During viral infection, the localization of the 66 K to chloroplastic membrane vesicles is dependent on the presence of the 140 K, which targets chloroplast and can induce chloroplast clumping alone [29]. In the case of BSMV, viral replication takes places in the vesicular invaginations of chloroplast too [16]. It is still unclear how the chloroplast peripheral vesicles are induced for TYMV and BSMV replication.

The ER was thought to be the site for potyviral replication. It was not established until a few years ago that potyviruses recruit both the ER and chloroplasts for their genome replication [22•]. Confocal and ultrastructural studies suggest that the potyviral 6K2 membrane protein alone can induce ER proliferation, vesicular structures, and chloroplast clumping [22•,30]. In potyvirus-infected cells, the full-length negative-strand viral RNA, viral