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Dynamic cross-talk between host primary metabolism and viruses during infections in plants

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Upon infection plant viruses modulate cellular functions and resources to survive and reproduce. Plant cells in which the virus is replicating are transformed into strong metabolic sinks. This conversion gives rise to a massive reprogramming of plant primary metabolism. Such a metabolic shift involves perturbations in carbohydrates, amino acids and lipids that eventually lead to increase respiration rates, and/or decrease in photosynthetic activity. By doing so, plants provide metabolic acclimation against cellular stress and meet the increased demand for energy needed to sustain virus multiplication and defense responses against viruses. This review will highlight our current knowledge pertaining to the contribution of primary metabolism to the outcome of viral infections in plants.

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Introduction

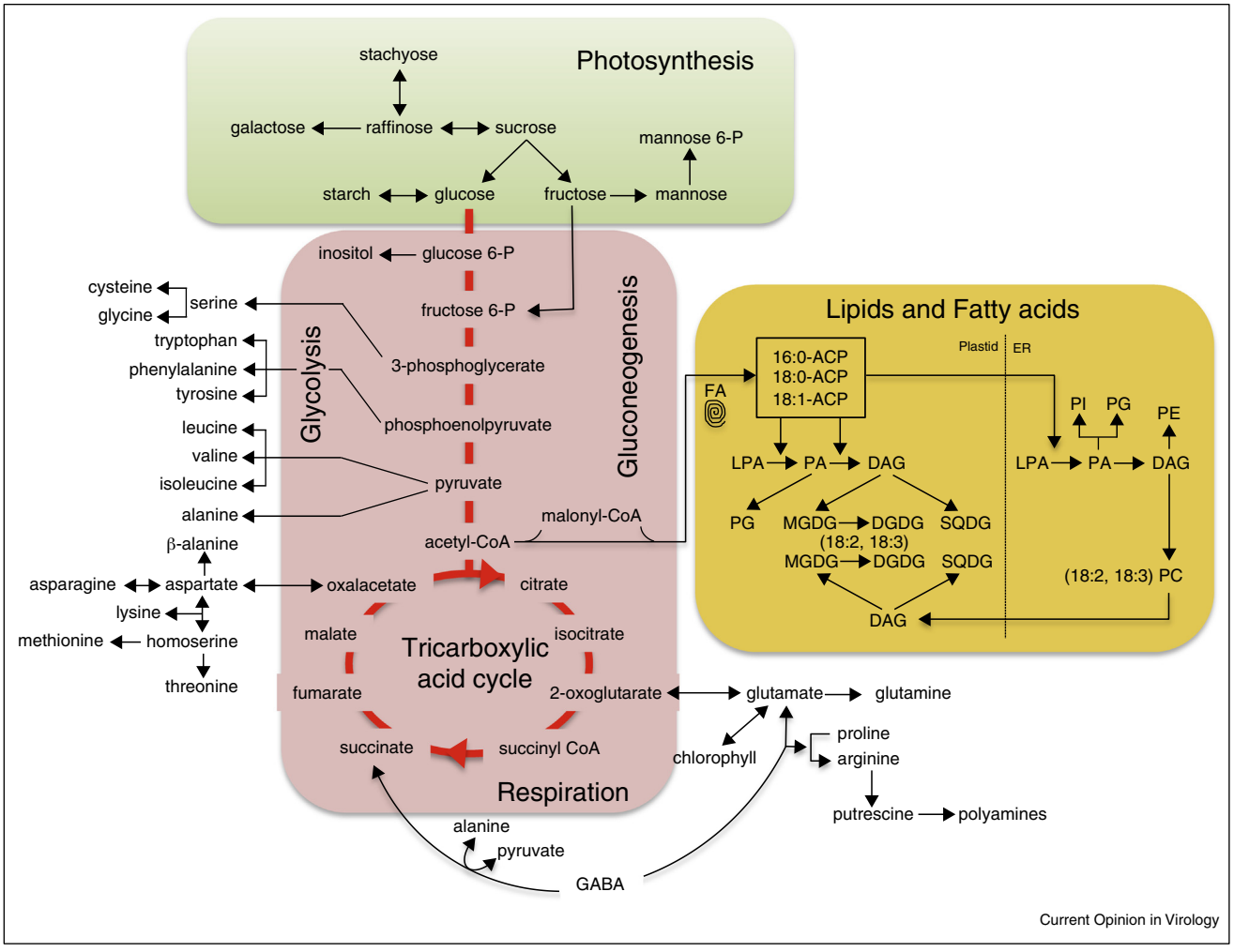
A viral infection is a highly dynamic process in which infected plant cells are transformed into major metabolic consumers (sinks) for products of photosynthesis. During the infection, nitrogen and carbon skeletons are required for the synthesis of new molecules and energy is necessary to fuel biosynthesis. The source-to-sink transition in the infected tissue elicits reallocation and increases demands for photosynthetic assimilates, increases respiration rates, and/or decreases in photosynthetic activity. Such a metabolic shift contributes to sustain viral proliferation but is also necessary to activate defense mechanisms. Due to the uneven nature of viral infections in plants and that different virus–host interactions have been studied, it is complicated to depict an unequivocal generalized picture of the metabolic responses triggered by plant viruses. In addition, many reactions in central metabolism are reversible and complex, hindering the perception of major changes in metabolite levels. Nevertheless, a considerable effort has been made to elucidate alterations in metabolite contents linked to viral infections in plants of which significant commonalties arise (Figure 1). In this review, the current knowledge on the modulation of plant primary metabolism during viral infections and its importance for plant compatibility and/or resistance are discussed.

Metabolomics strategies based on chromatography, mass spectrometry (MS) or nuclear magnetic resonance (NMR) spectroscopy in combination with multivariate data analysis have provided an excellent platform to understand the input of certain metabolites in the plant's response to viral infections [1–7]. Recent studies have gone steps further by using system biology approaches to study primary metabolism in plant–virus interactions. For instance, time-course transcriptomics and GC–MS-based metabolomics supported by functional reverse genetics were used to study the reciprocal influence of primary metabolism and *Tobacco rattle virus* (TRV) infection in *Arabidopsis thaliana* [8••]. GC–MS-based metabolomics and gene expression data identified altered and unique metabolic signatures characteristic of two tomato inbred lines that exhibited susceptibility or resistance to *Tomato yellow leaf curl virus* (TYLCV) [9•], or in response to mild and aggressive isolates of *Potato virus Y* (PVY) in potato leaves at different times of disease development [10•].

Plant virus, carbohydrate metabolism and photosynthesis

The carbohydrate status has profound implications in mobilization and synthesis of storage compounds, symptoms development and defense functions, and its alteration is diagnostic for perturbations in photosynthesis and respiration. The accumulation of sugars in the infected tissue causes an imbalance in the ratio of nitrogen and carbon, and the sensing of such changes results in a feedback transcriptional regulation of photosynthesis genes, and occasionally, photosynthetic repression [11–13,14•,15–17]. Even though repression of photosynthesis and induction of sink metabolism is a general response to viral infection, the effects on sugar levels varies considerably between different host–virus interactions. Changes in sugar levels involving the accumulation of enlarged starch grains in the chloroplast have been reported for some compatible interactions [18–21], whereas starch content decreases for some others [22•,23]. Interestingly, viral accumulation is unaffected in starch-depleted *Arabidopsis*

Figure 1



A simplified schematic representation of plant primary metabolism showing the major compounds that are altered in response to viral infections. Further details are given within the text and literature cited herein. ACP (acyl carrier protein), ER (endoplasmic reticulum), FA (fatty acids), LPA (lysophosphatidic acid), PA (phosphatidic acid). Glycolipids: DAG (diacylglycerol), DGDG (digalactosyl diacylglycerol), MGDG (monogalactosyl diacylglycerol), SQDG (sulfoquinovosyl diacylglycerol). Phospholipids: PC (phosphatidylcholine), PE (phosphatidylethanolamine), PI (phosphatidylinositol), PG (phosphatidylglycerol).

mutants suggesting that starch catabolism is not strictly required for virus multiplication in this species [8••,20]. Metabolism of sink tissues, where sugar is used, is mainly sustained by sucrose synthesized in source leaves and transported through the phloem into sink tissues. Sucrose and soluble sugars are abundant in different host species infected with *Tomato mosaic virus* (ToMV), *Cauliflower mosaic virus* (CaMV), and TRV [2,8••,24], whereas soluble sugar contents decrease upon infection with PVY, *Turnip yellow mosaic virus* (TYMV), *Jatropha mosaic virus* (JMV), *Ageratum enation virus* (AEV) or *Squash mosaic virus* (SqMV) [5,6,10•,25,26].

Changes in carbohydrates accumulation respond to different causes that include physical disturbance of the transport path (e.g. modification of plasmodesmata by viral movement proteins), inhibition of sugar transport proteins, induction of starch hydrolysis or cell wall invertases. *Cucumber mosaic virus* (CMV) increases sucrose concentration in the phloem sap of CMV-infected melon plants likely by altering sucrose localization [27]. Sucrose export routes can be severely affected by callose deposition at the cell-to-cell interfaces observed in multiple plant–virus interactions [28]. Infection of cotyledon of marrow plants by CMV causes a gradual increment of soluble sugars and a detriment of starch likely due to enhanced starch hydrolase activities [29,30]. The elevated hexose levels observed in tobacco plants infected with PVY^N are concomitant with increased invertase activity, which cleaves sucrose into glucose and fructose [31]. Interestingly, cell-wall invertase-overexpressing transgenic tobacco or *Arabidopsis* plants accumulate large