International Journal of Botany Studies
www.botanyjournals.com
ISSN: 2455-541X
Received: 05-04-2026, Accepted: 06-05-2026, Published: 21-05-2026
Volume 11, Issue 2, 2026, Page No. 230-240

Molecular and biochemical reprogramming in Phytoplasma-Infected plants: Advances in host–pathogen interactions and metabolic regulation

Shoeb Ahmad, Shazia Bi, Akil Ahmad Khan
Department of Botany, Gandhi Faiz-e-Aam College, Shahjahanpur, Affiliated to M.J.P.Rohilkhand University, Bareilly, Uttar Pradesh, India

Abstract
Phytoplasmas are obligate, phloem restricted bacterial pathogens that cause severe physiological/biochemical disruptions in a broad spectrum of economically important plants. Recent developments in molecular biology, omics technologies, and plant pathology have greatly helped to improve our knowledge of phytoplasma-induced metabolic reprogramming. This paper critically reviews and builds upon the classical knowledge of phytoplasma pathology using the recent information on host-pathogen interactions with a special focus on biochemical changes, signaling pathways and molecular regulation mechanisms. Phytoplasma infection causes a massive reorganization of plant metabolism, which includes carbohydrate buildup, hormonal imbalance, disturbance of photosynthesis, and alteration of the production of secondary metabolites. Alterations in structure (deposition of callose in sieve elements and ultrastructural deformation of chloroplasts) are antecedents of the appearance of visible symptoms (stunting, chlorosis, and floral abnormalities). Recent transcriptomic and metabolomic investigations imply that the expression of host genes and metabolic pathways are controlled by effector proteins and that such pathogens manipulate host gene expression and metabolic pathways. The review also sheds light on new evidence on sugar signaling, reactive oxygen species (ROS) dynamics, and disruption of amino acid transport as key elements of disease progression. Moreover, phytoplasma infection has been demonstrated to cause considerable changes in secondary metabolism such as phenolics, alkaloids, and polyamines, which could have a role in the defense of a plant or the expression of symptoms. The new point of view is outlined on recovery mechanisms with special attention to systemic acquired resistance (SAR), oxidative signaling, and calcium-mediated pathways as the main determinants of plant resilience. These observations highlight the intricacy of phytoplasma pathogenicity and point to potential targets to develop sustainable disease management strategies. In general, this work will be a synthesis of biochemical and molecular response in phytoplasma-infected plants that will be comprehensive and up-to-date.

Keywords: Phytoplasma, plant metabolism, host–pathogen interaction, secondary metabolites, hormonal imbalance, plant defense mechanisms

Introduction

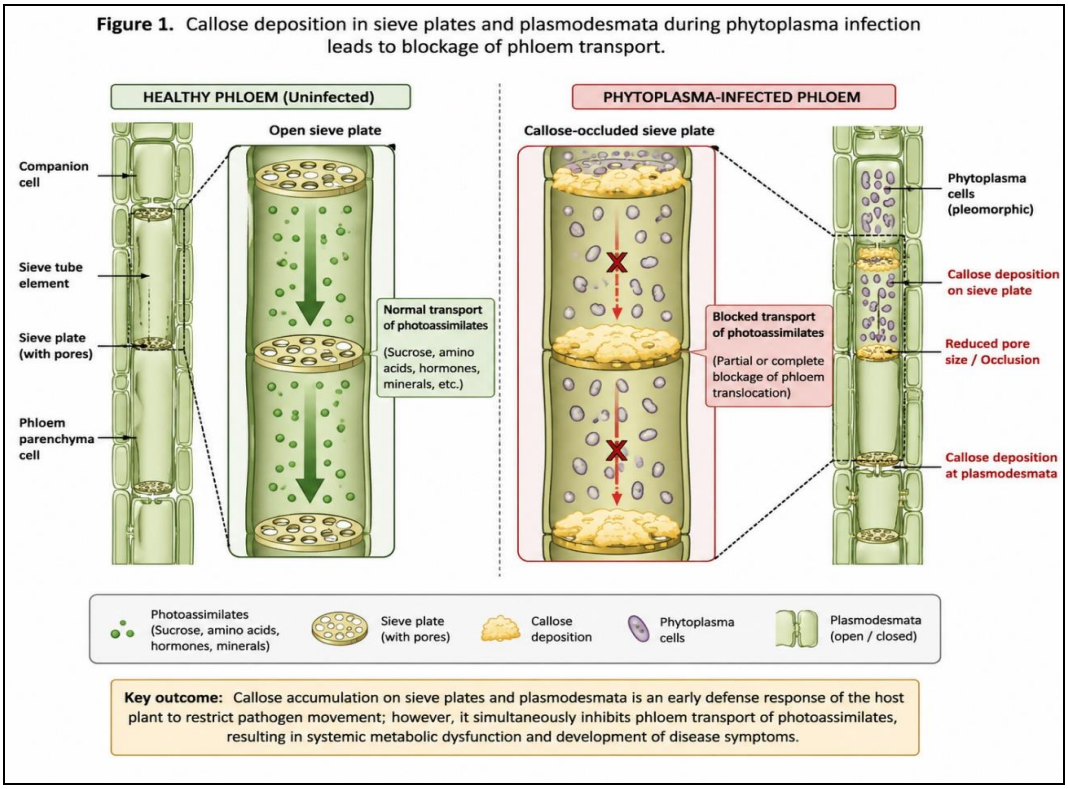
Introduction to Phytoplasma-Induced Plant Disorders

Phytoplasmas are a specialized group of plant-pathogenic prokaryotes, which are lack of a rigid cell wall, small genome size, and an obligatory parasitic lifestyle. These microorganisms live in the phloem sieve elements of the host plants and are transmitted by phloem - feeding insect vectors like leafhoppers and psyllids. Historically, phytoplasmas have been largely identified due to their association with a broad spectrum of plant diseases impacting economically relevant crops, such as fruit trees, ornamental species, cereals, and medicinal plants. The development of recent discoveries in the fields of molecular biology and plant pathology has drastically changed the way we understand these pathogens and shifted the emphasis on the description of the symptoms toward the mechanisms of the host-pathogen interaction and the regulation of the metabolism (Bai *et al*., 2021; Kumari *et al*., 2022) [10].

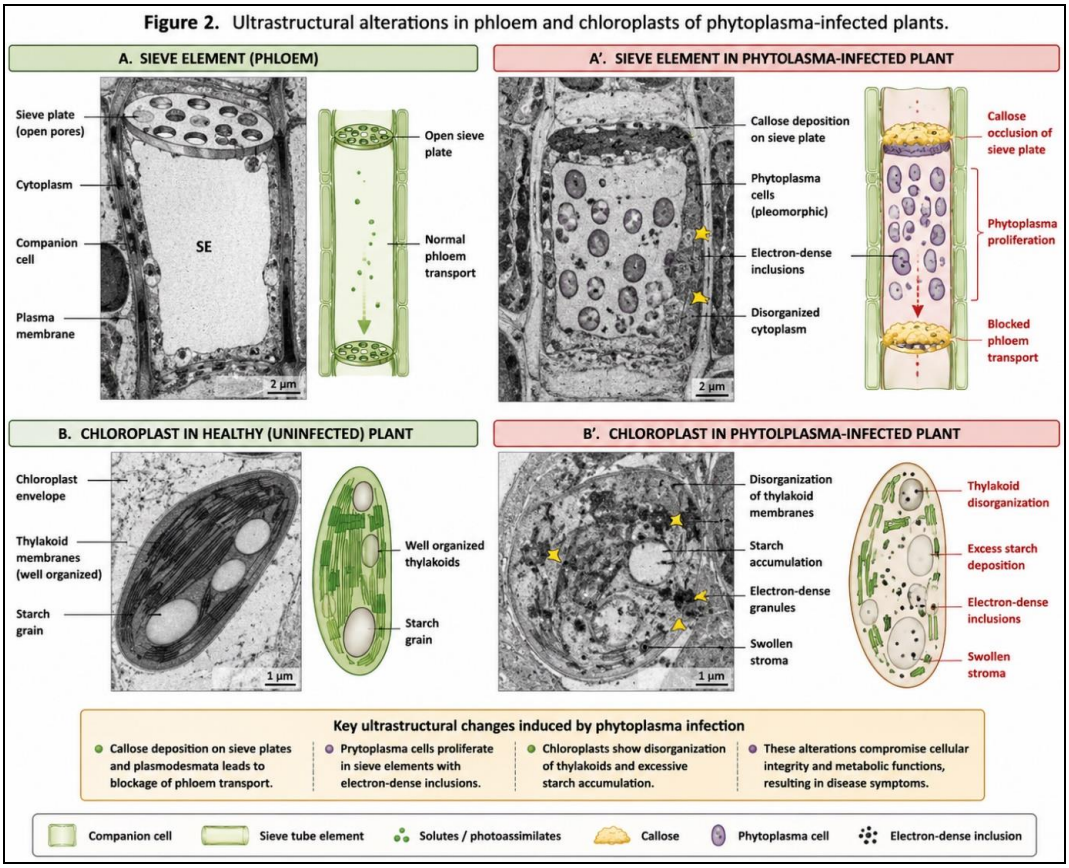
The study uploaded highlights that phytoplasmas cause an array of typical symptoms such as stunting, chlorosis, reduction in leaf size, shortening in internodes, and malformation in floral structures such as phytoplanktons and virescence. Although the symptoms have been long traditionally used as diagnostic markers, modern research indicates that the symptoms are phenotypic consequences of complex biochemical and molecular derailments in the host plant. Phytoplasma infection has now become more of a systemic metabolic disorder than it simply being a localized pathogenic invasion. Recent studies (Zhang *et al*., 2022; Rao *et al*., 2024) [19, 23] revealed that phytoplasmas actively manipulate the host cellular pathways by triggering alterations in gene expression, hormonal regulation and distribution of nutrients to promote their growth and survival within the host. It is a paradigm shift, which means that the use of classical approach to observe the pathology along with the latest methodologies of molecular biology is necessary to understand the diseases caused by phytoplasma.

Structural and Cellular Alterations in Infected Plants

One of the early symptoms of infection by phytoplasma that can be observed is the development of marked structural and ultrastructural alterations in the phloem tissues of infected host plants. These changes are the antecedents of manifestations and have a key role in interfering with physiological processes. The deposition of callose on the sieve plates and plasmodesmata are a hallmark feature of infection that results in partial or complete blockage of phloem transport. This phenomenon, as shown in Figure 1, is a defense response of the plant to limit the movement of pathogens; however at the same time it also inhibits the movement of photoassimilates, thereby contributing to systemic metabolic dysfunction (Musetti, 2006) [16].



Along with the accumulation of callose, electron microscopic observations show the presence of pleomorphic phytoplasma cells in sieve elements, as well as structural abnormalities in chloroplasts such as starch deposition and disorganization of thylakoid membranes. Figure 2 shows these ultrastructural changes that indicate a direct interference of phytoplasmas with cellular integrity and metabolic functions. Of particular importance is the disruption of the chloroplast architecture as it is closely linked to the loss of photosynthetic efficiency and changes in energy metabolism.



In addition, phloem necrosis and companion cell collapse has been reported in some host species further worsening the impairment of nutrient transport. All of these anatomical changes result in a cascade of physiological changes, which

eventually translate into symptoms of a disease. Recent studies indicate that these structural modifications are not only passive results of infection but are actively caused by phytoplasma-secreted effectors that actively control host cellular functions (Chen *et al*., 2021) [5].

Evolution of Research: From Symptom-Based Studies to Molecular Understanding

There has been a tremendous change in the study of phytoplasma diseases in the last few decades. Initial studies were mostly descriptive in nature, concerned with the classification of symptoms, the host range, and the epidemiology. Although these studies were able to give a good baseline knowledge, they were not able to give much information on the underlying mechanisms of pathogenicity. Using the more complex molecular tools available, including transcriptomics, proteomics and metabolomics, scientists have been in position to examine the complex interactions between the phytoplasmas and their host plants at a systems level.Recent transcriptomic studies have shown that phytoplasma infection triggers comprehensive re-programming of host gene expression, especially genes related to hormone biosynthesis, carbohydrate metabolism, and defensesignaling pathways (Li *et al*., 2023; Zhang *et al*., 2022) [23]. An example is the differentially expressed genes that control floral development and meristem identity, leading to abnormal floral structures, including phyllody and virescence. These results give a molecular explanation to phenomena, which have only been understood at the phenotypic level in previous studies.

Moreover, the discovery of phytoplasma effector proteins has also become one of the significant discoveries in the field of pathogenicity. These effectors can also interact with host transcription factors and signaling components, and thus, change developmental processes and metabolic pathways. Consequently, the study of phytoplasma infection has now been considered a dynamic process that involves continuous molecular dialogue between the pathogen and the host plant. This change in descriptive research to the use of the mechanistic research has provided new grounds of coming up with specific disease management strategies.

Hormonal Imbalance as a Central Regulatory Mechanism

One of the critical aspects of phytoplasma-induced pathology is the interference with the hormonal balance of plants, which is a key component of the control of growth, development, and responses to stress. The Phytoplasma-related diseases have traditionally been regarded as one of the auxonic diseases as a result of the involvement of plant growth regulators in the expression of symptoms. However, recent research has given more information about the particular hormonal pathways that are influenced by infection.

Along with this, higher levels of auxin and especially indole-3-acid (IAA) have been repeatedly noticed in the infected tissues, and it is hypothesized that it plays a role in the abnormal growth pattern like shoot growth and reduced apical dominance (Rao *et al*., 2024) [19]. On the same note, the concentration of cytokinins has been reported to be higher in the floral tissues during formation of green flowers and delay senescence, a process known to be regulated by cytokinins (Sharma *et al*., 2023) [20]. Further, the stress hormones during infection may be upregulated such as abscisic acid (ABA) and ethylene, which are responsible for leaf yellowing, senescence, and abscission (Kumar *et al*., 2023) [11].

Importantly, these hormonal changes do not occur as isolated events, but are interdependent in terms of complex signaling networks. Phytoplasma effectors are believed to modulate hormone biosynthesis and signaling pathways and result in a hormonal imbalance that aids the survival of pathogens. This argues for the high level of control that phytoplasmas exert on the physiology of their hosts.

Carbohydrate Redistribution and Metabolic Reprogramming

The other remarkable symptom of phytoplasma infection is the change in the carbohydrate metabolism and distribution in the host plant. The deposition of callose blocks phloem transport, resulting in the accumulation of soluble sugars, e.g., sucrose and glucose, in the source leaves. This accumulation is accompanied by a corresponding decrease in the levels of carbohydrates in sink tissues, such as roots and developing organs (Singh and Verma, 2025) [21]. This results in the inhibition of photosynthesis because genes which are involved in the photosynthetic processes are inhibited due to the high sugar level. As a result, chloroplasts have structural malformations, such as overaccommodation of starch and disorganization of thylakoid, which further reduce the photosynthetic efficiency. This generates a metabolic imbalance which undermines the energy production of the plant and its general growth.

Alongside another effect on photosynthesis, other physiological processes, including osmotic regulation and signaling pathways are affected by altered carbohydrate metabolism. Sugars, in their turn, are signaling molecules, which regulate the expression of genes and the developmental processes. Thus, the interference with sugar homeostasis has far-reaching consequences on the health of plants and the development of a disease. According to recent studies, phytoplasmas could actively inhibit the expression of sugar transporter genes, which adds to the fact that they gain more access to the host nutrients and at the same time weaken the defense mechanisms of the plant (Gupta *et al*., 2024).

Conceptual Framework of Host–Pathogen Interaction

The system of the interaction between phytoplanktons and host plants can be conceptualized as a complex and multi-layered system containing structural, biochemical and molecular components. Structural level Phloem blockage and cellular disorganization interfere with the nutrient transport and cell integrity. Metabolic reprogramming is caused by changes in carbohydrate metabolism, amino acid transport, and the synthesis of secondary metabolites. Molecular mechanisms involve changes in gene expression and signaling pathways that regulate the response of the plant to infection.

These layers are linked to each other, and are dynamically regulated, to form an integrated network of host-pathogen interactions. Phytoplasma effectors are the major contributors to this network as it targets key regulatory nodes, including transcription factors and hormone signaling pathways. This enables the pathogen to manipulate host physiology in a manner that promotes its survival and spread.

The new models focus on the significance of reactive oxygen species (ROS), calcium signaling, and systemic acquired resistance (SAR) in facilitating plant responses to phytoplasma infection (Chen *et al*., 2021; Gupta *et al*., 2024) [5]. These processes are the possible object of creation of new methods of managing the disease that will contribute to the increase in the level of resistance in plants.

Scope and Objectives of the Present Study

The current research is supposed to present a detailed and updated analysis of biochemical and molecular alterations in infected plants by phytoplankton to incorporate classical understanding and the current research findings. In particular, the paper will (i) investigate the key metabolic pathways that are impacted by disease (phytoplasma infection), (ii) analyze the role of hormonal and signaling networks in the development of the disease (phytoplasma infection), and (iii) explore the potential mechanisms underlying plant defense and recovery.

The synthesis of information on the traditional and contemporary studies helps to comprehend the phytoplasma pathogenicity better and reveal the future perspectives on research and disease management. The rest of this paper will concentrate on the detailed examinations of hormonal regulation, gene expression and metabolic pathways with the updated figures and new references.

Hormonal Regulation, Gene Expression, and Signaling Networks in Phytoplasma-Infected Plants

1. Hormonal Reprogramming as a Driver of Disease Development

One of the major processes that lead to the development of symptoms and metabolic regulation is caused by phytoplasma infection causing profound changes in the hormonal balance of host plants. In contrast to traditional pathogen-induced stress responses, which trigger transient hormonal responses, phytoplasma-associated disorders are characterized by long-term and synchronized reprogramming of various phytohormone pathways. It is a complicated hormonal imbalance that influences not only the growth and development but also defense mechanisms and metabolic homeostasis. Modern studies have shown that phytoplasmas can control the hormonal networks of hosts by manipulating effector-mediated interference and, thus, creating a favorable niche to survive and thrive (Rao *et al*., 2024; Zhang *et al*., 2022) [19, 23].

Auxins are among the plant hormones that have been widely investigated with regard to phytoplasma infection. The high concentration of indole-3-acetic acid (IAA) in diseased tissues has been reported and often accompanies abnormal phenotype, such as excessive shoot growth, apical dominance loss, and witches broom structure development. It is however becoming increasingly understood that accumulation of auxins may be a secondary effect rather than a direct cause of symptoms of impaired signaling networks. Based on experiments, it is proposed that the phytoplasma effectors may modulate the auxin transport and distribution by interacting with the transcription factor that is responsive to auxin, thereby relieving developmental programs (Singh and Verma, 2025) [21]. This subtle insight supplies the previous assumptions and demonstrates the necessity to take into account auxin dynamics in the context of a more general regulatory framework.

Another important group of hormones that are also influenced by phytoplasma infection is cytokinins. These hormones play a key role in cell division, chloroplast development, and delay of senescence. The formation of phyllody and virescence (where floral organs are transformed into leaf-like structures) have been highly associated with high levels of cytokinins. Recent transcriptomic studies revealed the genes associated to biosynthesis and signalling pathways of cytokinin are highly induced in infected plants (Sharma *et al*., 2023) [20]. Thus this hormonal imbalance impairs normal floral development, and also contributes to a more prolonged viability of the tissues, and may aid in the survival of pathogens in the host.

Phytoplasma infection also significantly alters the levels of other hormones, such as abscisic acid (ABA) and ethylene, besides auxins and cytokinins. A correlation between high ABA content and stomatal closure, reduction of transpiration and expression of senescence-related processes is usually found. Equally, the higher production of ethylene has been associated with yellowing of leaves and premature aging of the plant tissues (Kumar *et al*., 2023) [11]. It is in the physiologic environment created by the interplay of these hormones that it is more likely to express symptoms and may also be able to hinder effective defense responses. Interestingly, it has been recently reported that phytoplasma effectors can directly inhibit the hormone biosynthetic enzymes and signaling components, resulting in a coordinated disruption of hormone homeostasis (Gupta *et al*., 2024).

2. Molecular Basis of Hormonal Crosstalk

The regulation of plant hormone is controlled by a complex signal network with numerous feedback control mechanisms and pathways between hormones. These networks are extremely altered in the infected plants, leading to incorrect signalingbehavior. Numerous interactions between hormones influence the outcome of the infection; between auxins and cytokinins, and between auxins and ABA.

Recent advances in the molecular biology of phytoplankton have revealed the ability to target specific, hormone-responsive, transcription factors using effector proteins secreted by the phytoplankton. Similarly, the expression of proteins belonging to the AUX/IAA and ARF (Auxin Response Factor) families are also reported to be deregulated in infected plants, indicative of auxin signaling pathways disruption (Li *et al*., 2023). Likewise, the expression of components of the cytokinin signaling cascade, such as histidine kinases and response regulators, is regulated during infection.

The interaction of these hormonal pathways is further complicated by the inclusion of sugar signaling and stress-responsive pathways. The sugars, which are accumulated in the infected leaves because of the impaired transport of sugars through the phloem, serve as signalingmolecules and influence the biosynthesis of hormones, and the expression of genes. This gives positive feedback that the changes in metabolism strengthen the hormonal imbalance, and hence increase the symptoms of the disease. These interactions not only emphasize the complexity of the hormonal reprogramming induced by phytoplasma but also underscores the necessity of considering multiple regulatory layers when studying the responses of plants to phytoplasma.

3. Gene Expression Reprogramming and Transcriptomic Insights

The use of transcriptomic technologies based on high throughput technology to research on host gene expression has been regarded as one of the most significant

advancements in research into phytoplasma. These investigations have demonstrated widespread reprogramming of plant transcriptome in response to infection, with effects on genes of development, metabolism and defense. The analyses of the gene expression of both parties and the host have identified both up- and down-regulated genes when they differentiate (Zhang *et al*., 2022; Li *et al*., 2023) [23].

Phytoplasma infection is quite sensitive to genes related to floral development. As an example, key regulatory genes that regulate meristem identity, organ differentiation are frequently misexpressed resulting in abnormal floral structures, like phylogody and sterility. This effect can be explained by the fact that the phytoplasma effectors interfere with transcriptional regulators controlling the developmental pathways. In many cases, photosynthetic, carbohydrate metabolic, and protein synthesis genes are often downregulated, repercussion of a shift in the metabolic priorities in the infected plant.

The other significant fact about gene expression reprogramming is that defense-related genes are activated. The induction of pathogenesis-related (PR) proteins, such as chitinases, glucanases, and peroxidases, is a common response to infection by phytoplankton. These proteins are important in enhancing plant defense systems by breaking down constituents of pathogens and strengthening cell walls. These responses however have different degrees of effectiveness depending on the host species and the extent of infection. In other cases, the susceptible plants have a weak or slow defense response, and the pathogen is free to develop a persistent infection.

4. Effector Proteins and Host Manipulation

The identification of phytoplasma effector proteins has given the essential information on the molecular pathogenicity processes. These little, secreted proteins can interact with host cellular components and thus modify gene expression and developmental processes. In contrast to more traditional pathogens that may cause harm to host tissues via toxin or enzyme effects, phytoplasmas have a more insidious effect on host tissues, which can be caused by subtle manipulation of host regulatory systems.

Recent research has revealed a few effector proteins that target transcription factors that are involved in plant development. For instance, SAP (Secreted AY-WB Protein) family effectors have been shown to interfere with floral development by destabilizing MADS-box transcription factors, leading to the conversion of floral organs into leaf-like structures (Chen *et al*., 2021) [5]. In a similar manner, other effectors are aimed at disrupting the balance of growth regulators, thereby causing the expression of symptoms. Effector proteins have functions beyond developmental regulation to include host defense mechanisms. Phytoplasmas are able to avoid plant immune systems and become permanently infected by interfering with signaling pathways like salicylic acid (SA) and jasmonic acid (JA) responses. This capability to adjust and control development and defense is an indication of the advanced mechanisms that these pathogens use.

5. Signal Transduction Pathways and Secondary Messengers

Signal transduction is a crucial factor in facilitating growth and development in plants in response to infection by phytoplasma. Active participants in the communication of stress signals within cells and the organization of responses include key signaling molecules, such as calcium ions (Ca^{2+}), reactive oxygen species (ROS), and phosphorylation cascades.

Calcium signals are important for regulating phloem activity and defense mechanism. Alterations in the levels of cytosolic Ca^{2-} have been linked with the deposition of callose and the development of physical barriers that limit the movement of pathogens. A recent study revealed that phytoplasma infection can alter the Ca^{2+} flux in phloem tissues and consequently the transport and signaling processes (Gupta *et al*., 2024). This disturbance of calcium homeostasis may play a role in the further development of the symptoms of the disease.

Reactive oxygen species (ROS) such as hydrogen peroxide (H_2O_2) play an important role in plant responses. On one hand, ROS may serve as signaling molecules and activate defense pathways; on the other hand, the excessive accumulation of ROS can result in oxidative damage and cell death. The equilibrium between production of ROS and scavenging is thus determining factors on the outcome of infection. Changes in ROS dynamics have been associated with the development and recovery of symptoms in phytoplasma-infected plants.

To conceptually understand these signaling interactions, Figure 3 represents schematically the hormone-gene-signal integration in phytoplasma-infected plants.Above Figure 3 demonstrates the interaction of phytoplasma effectors, hormonal signaling and other signaling molecules to control gene expression and metabolic responses.

This complex model emphasizes the complexity of the interactions between the host and the pathogen and offers a framework through which one can understand disease progression.

6. Integration of Hormonal and Molecular Responses

This interaction of hormonal regulation, expression of genes, and signaling pathways is a highly integrated system, which dictates plant responses to phytoplasma infection. These parts are not independent but rather are dynamically interconnected to produce the observed results of the phenotype. The genes are expressed due to hormonal imbalances and this in turn, influences metabolic pathways and signaling network. In contrast, a complex network of interactions can be created by feedback as a result of changes in metabolism and signaling.

Such an integrated picture is necessary to comprehend the multifaceted nature of phytoplasma pathogenicity. It also points to possible areas of intervention, like important regulatory nodes in hormonal or signaling pathways. With such manipulation of these nodes, it is possible that there can be an augmentation of the plant resistance as well as mitigating the symptoms of the disease.

Carbohydrate Metabolism, Photosynthetic Dysfunction, and Amino Acid Dynamics in Phytoplasma-Infected Plants

1. Disruption of Carbohydrate Metabolism and Phloem Transport

The major biochemical effect of phytoplasma infection is the radical reorganization of carbohydrate metabolism, which indicates both structural destruction of the phloem tissues, as well as molecular reorganization of the metabolic pathways. In normal physiological conditions, carbohydrates produced in source leaves are transported via

the phloem to sink tissues including roots, developing fruits and meristems. Nevertheless, phytoplasma infection immensely disturbs this source-sink relationship, resulting in an abnormal build-up of soluble sugars in the leaves and a corresponding depletion in sink organs. In the uploaded study, there is also a focus on the fact that such carbohydrate imbalances occur due to the impairment of phloem transport and are directly linked to the development of disease symptoms.

It is mainly the deposition of callose at sieve plates, which constitutes a physical barrier to the movement of assimilates. Moreover, this interference is further worsened by downregulation of sugar transporter genes by phytoplasma which restricts the export of sucrose by source tissues. As a result, infected leaves contain relatively higher concentration of reducing sugars and sucrose as compared to healthy controls (Singh and Verma, 2025) [21]. This build-up is not only a passive effect of transport blockage but also indicative of active metabolic reprogramming in response to pathogen-induced signaling pathways.

In addition, the build-up of starch in the chloroplasts is another feature that is characteristic of plants infected by phytoplankton. The excess sugars are broken down to form starch grains that cause structural distortion of chloroplasts and impairment of photosynthetic machinery. This process establishes a vicious circle of reduced export of carbohydrates which results in reduced photosynthesis, which results in greater metabolic imbalance. Carbohydrate regulation is complex, as demonstrated by recent metabolomic analyses (Kumar *et al*., 2023; Li *et al*., 2024) [11] which confirmed the modulation of phytoplasma infection of key enzymatic activities for sucrose metabolism, such as sucrose synthase and invertases.

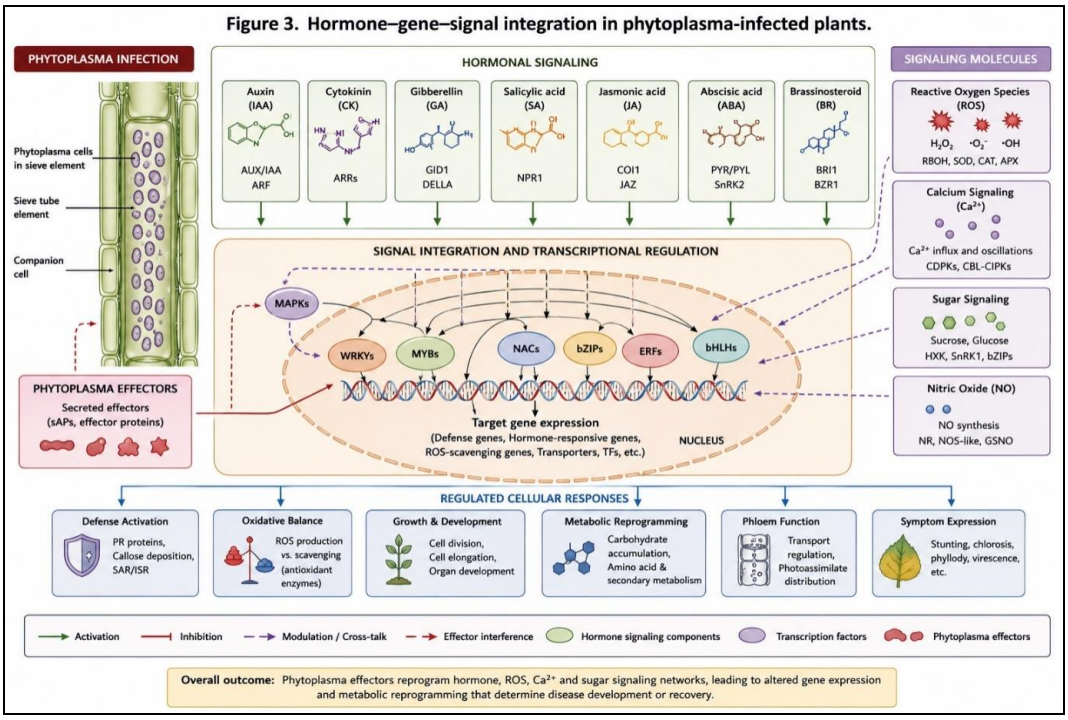


Figure 3. Hormone–gene–signal integration in phytoplasma-infected plants.

2. Enzymatic Regulation of Sugar Partitioning

A complex of enzymes that regulate the synthesis, degradation and transport of sugars regulate the partitioning of carbohydrates in the plant tissues. Of these enzymes, the following are important in sugar homeostasis: sucrose synthase, cell wall invertase, vacuolar invertase and neutral invertase. The activity of these enzymes changes significantly in the plants infected by phytoplankton which reflects the effects of the pathogen on the metabolic pathways of the plants.

It has been demonstrated that neutral invertase activity is frequently elevated in infected tissues, which results in an increase in hydrolyses of sucrose into glucose and fructose. These monosaccharides are readily available energy sources that might help phytoplankton (Rao *et al*. 2024) [19] to grow and multiply. At the same time, the expression of other genes coding for sugar-metabolizing enzymes may also be not altered or even decreased, suggesting selection of the expression of certain metabolic pathways.

This enzymatic selectivity implies that the phytoplasmas can proactively regulate the host metabolism in order to optimize the nutrient availability. The pathogen, by increasing the amount of simple sugars in the infected tissues, not only ensures continuous supply of carbon resources, but also causes disruption in the normal physiological processes in the plant. This depth of metabolic manipulation is indicative of the complexity of phytoplasma-host interactions, and the significance of the study of enzyme regulation in understanding disease progression.

3. Photosynthetic Impairment and Chlorophyll Degradation

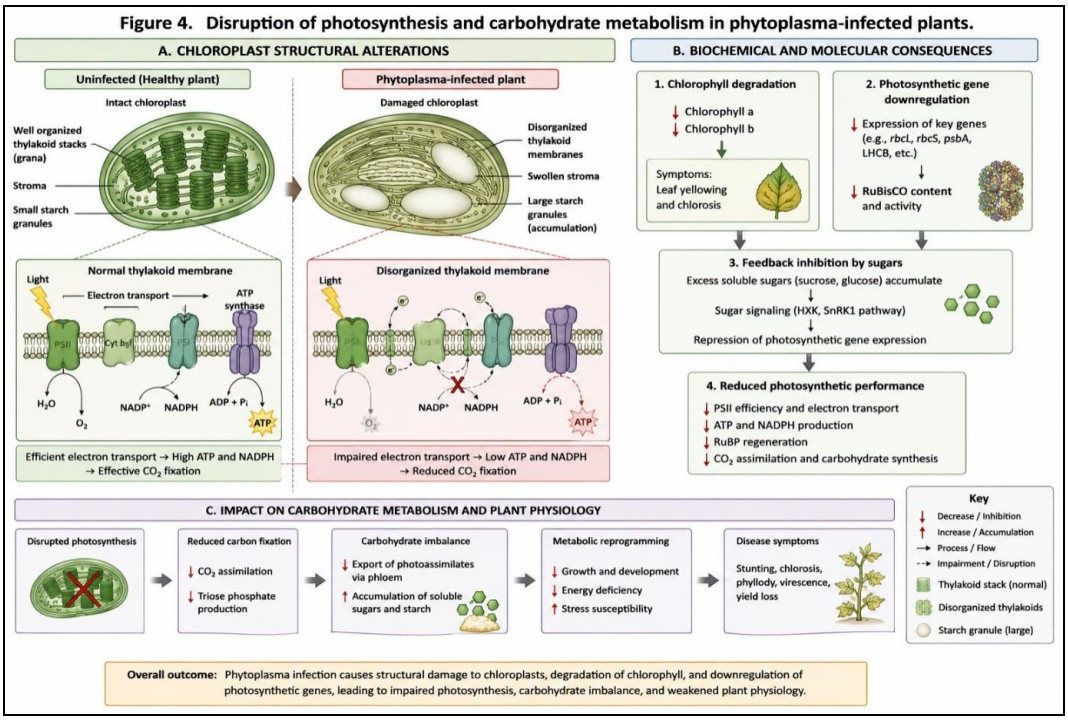
The carbohydrate metabolism disruption in phytoplasma-infected plants is strongly associated with the reduction of the photosynthetic efficiency. A major loss of chlorophyll content, both chlorophyll a and chlorophyll b, is one of the most noticeable biochemical changes which have been observed in infected plants. This decrease is commonly linked with noticeable symptoms like the yellowing and chlorosis of leaves, which are typical of a large number of phytoplasma diseases.

The structural abnormalities in chloroplasts of infected plants include disorganized thylakoid membranes and the presence of large starch granules (as shown in above Figure 4). These changes interfere with the light-dependent

reactions of photosynthesis particularly those associated with photosystem II (PSII). Loss of thylakoid integrity diminishes the efficiency of electron transport resulting in reduced production of ATP and NADPH, which are crucial in fixing carbon.

Besides structural damages, phytoplasma infection also influences the expression of genes that participate in photosynthetic processes. It is reported to downregulate the genes that encode key enzymes including ribulose-1,5-bisphosphate carboxylase/oxygenase (RuBisCO) which in turn decrease the carbon assimilation capacity (Zhang *et al*., 2022) [23]. More so, feedback inhibition mediated by sugar is an important phenomenon in inhibiting the expression of photosynthetic genes in the presence of an excess of sugar in the plant.

A combination of chlorophyll degradation effects, enzyme downregulation effects, and structural damage effects lead to a significant decrease in photosynthetic performance. This not only reduces the generation of energy by the plant, but also adds to the general weakening of the physiological functions, predisposing the plant to the further stress.



4. Stomatal Conductance and Gas Exchange Alterations

The second significant feature of the photosynthetic dysfunction in the plants infected with phytoplankton is the change in the stomata behavior. Stomata are used to regulate gas exchange by controlling the absorption of carbon dioxide, and the release of oxygen and water vapor. Stomatal conductance tends to be lower in infected plants and results in lower CO2 availability to photosynthesis.

This decrease in stomatal conductance depends on various factors such as hormonal variations and sugar build-up. The high content of ABA also promotes stomata closing, whereas high concentration of sugars in the leaf tissues might also lead to reduced stomata opening (Kumar *et al*., 2023) [11]. This means that the exchange of gases is decreased, so that the effectiveness of photosynthesis is decreased even more.

Structural alteration in leaf anatomy, which includes a decrease in stomatal density and a change in the size of guard cells, have also been found in infected plants besides stomatal closure. These variations mean that phytoplasma infection is not only affecting physiological processes, but also the developmental issues of plant tissues. The overall effect of these changes is a substantial decline in overall plant productivity.

5. Amino Acid Transport and Nitrogen Metabolism

Phytoplasma infection also exerts a significant impact on the amino acid transportation, on the nitrogen metabolism which is crucial in the growth and development of the plants. Amino acids are important building blocks of proteins and are important in aiding nitrogen assimilation and transportation. The amino acids are efficiently transported in the phloem to promote the growth of sink tissues in healthy plants. Nonetheless, in the infected plants, this transport is grossly disturbed by obstructing the phloem and restructuring the metabolism.

As pointed out in the uploaded document, the accumulation of amino acids in source leaves is due to the impaired transport mechanisms. This build up is complemented by a decline in the availability of amino acids in sink tissues resulting in impaired growth and development. More recent literature (Li *et al*., 2024; Gupta *et al*., 2024) has further shown that genes that encode amino acid transporters are commonly down-regulated in infected plants, indicating that there is a molecular basis of this disruption.

Besides transport constraints, phytoplasma infection affects amino acid metabolism by modulating enzyme activities in their roles in nitrogen assimilation. The elevated concentration of particular amino acids in infected tissues could be an indicator of the stress response that is directed at the maintenance of metabolic balance.

Nevertheless, this imbalance may also play the role of the emergence of disease symptoms, including slowed growth and decreased biomass.

Moreover, amino acids are strongly associated with plant defense mechanisms as they are involved in the synthesis of the secondary metabolites and signaling molecules. Thus,

the metabolic disturbances in the metabolism of amino acids can have a more general impact on plant immunity and resistance.

6. Integration of Carbon and Nitrogen Metabolism

Carbohydrate/amino acid metabolic interaction is a vital part of plant physiological regulation. In plants infected with phytoplasma, there is a disruption of both carbon and nitrogen metabolic pathways and a breakdown of metabolic coordination ensues. The build-up of sugars in the leaves together with the impaired movement of amino acids cause an imbalance leading to the influence on various physiological processes.

The role of metabolic signalling networks in the coordination of carbon and nitrogen metabolism has gained more recent integrative studies. These networks are characterized by complicated interactions between sugars, amino acids, and hormones, which all are important to regulate the expression of genes and the metabolic fluxes (Singh and Verma, 2025) [21]. These networks are greatly perturbed in the context of phytoplasma infection leading to a reprogramming of metabolic priorities, which is more favorable to the survival of pathogens.

The integration of the carbon and nitrogen metabolism is a key point of understanding to develop the strategies to reduce the effects of phytoplasma infection. By determining key regulatory nodes in these networks, researchers can possibly develop interventions that can reestablish metabolic balance and make plants more resilient.

Secondary Metabolites, Defense Responses, and Recovery Mechanisms in Phytoplasma-Infected Plants

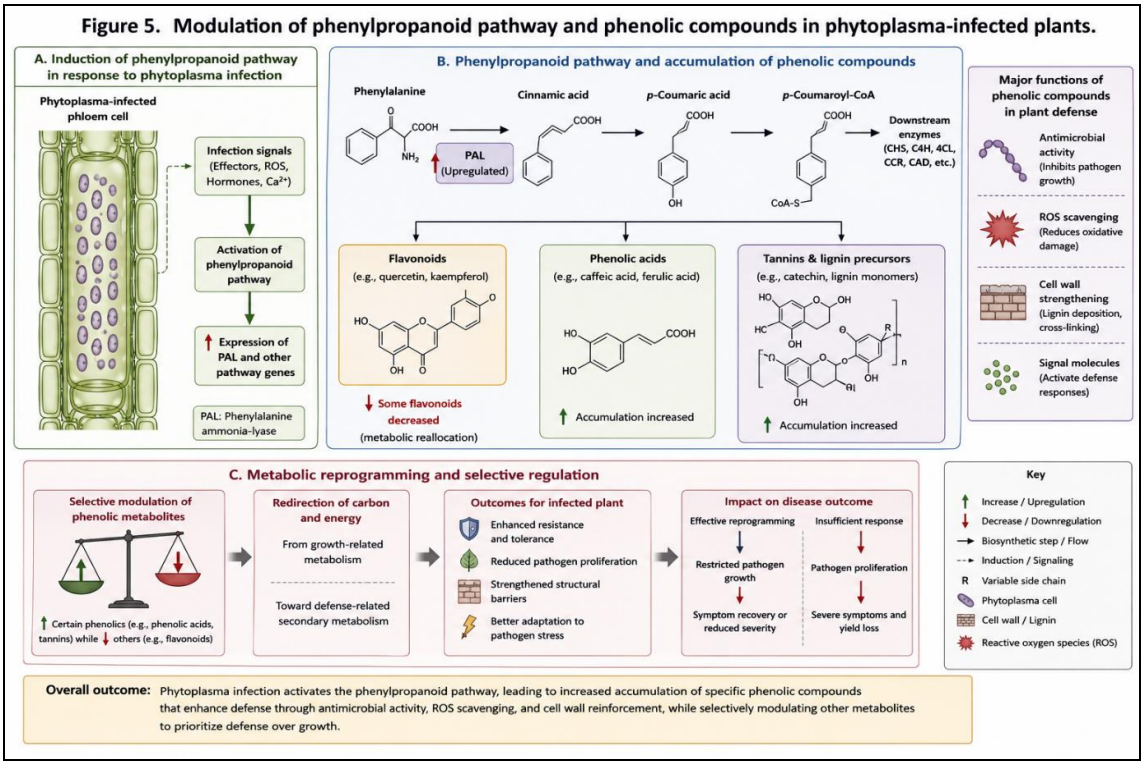
1. Role of Secondary Metabolites in Plant Defense

Secondary metabolites are an important part of the plant defense mechanism, especially during biotic stress factors, e.g. phytoplasma infection. Secondary metabolites are specialized in growth and development, unlike primary metabolites that are directly involved in growth and development. The infection by phytoplasma also causes significant changes in the biosynthesis and accumulation of these compounds, which reflect the attempts of the plant to counter-act the invasion of pathogen and also makes a contribution to the development of symptoms.

The uploaded study states that phytoplasma-infected plants experience major alteration in the secondary metabolic pathways, such as phenolics, alkaloids, and polyamines. Recent studies have also indicated that these changes are regulated both in transcriptional and enzymatic levels, which implies a coordinated defense response (Kumar *et al*., 2023; Singh and Verma, 2025) [11, 21]. Notably, the build-up of secondary metabolites does not occur uniformly across plant tissues but rather varies according to the severity of infection, plant species and environmental conditions.

2. Phenolic Compounds and Antioxidant Defense

Phenolic compounds represent one of the most extensively studied groups of secondary metabolites in plant–pathogen interactions. These compounds include flavonoids, phenolic acids and tannins and are known to have antimicrobial effects and are also known to help in strengthening plant cell walls. There has been a consistent increase in the total phenolic content of infected plants, which is often associated with resistance.



The activation of the phenylpropanoid pathway in response to infection, which results in higher levels of phenolic compounds, is illustrated in above Figure 5. This pathway mainly involves an enzyme called phenylalanine ammonia-lyase (PAL) which is often over-expressed in infected tissues (Gupta *et al*., 2024). Compounds contribute to plant defence mechanisms by inhibiting the growth of pathogens, removing reactive oxygen species (ROS) and enhancing structural barriers through lignin deposition.

Interestingly, although some phenolic compounds become more abundant, others like flavonoids can become less abundant, which means that the metabolic priorities change.

This selective control implies that phytoplasma infection does not merely increase overall secondary metabolism but instead redirects it towards certain defense related pathways. This kind of metabolic reprogramming indicates the adaptive response of the plant to the stress of pathogens.

3. Polyamines and Stress Signaling

Low molecular weight compounds that are significant in cell division, differentiation and response to stress include polyamines such as putrescine, spermidine and spermine. Polyamine metabolism is also highly changed in phytoplasma-infected plants and putrescine accumulation is also significantly observed in the leaf tissues. This build up is linked to defense mechanisms as well as the development of symptoms.

Polyamines have been reported to mediate with ROS and hormonal signaling pathways, thus facilitating plant responses to stress. The higher concentrations of putrescine can be expected to increase the capacity of the plant to stabilize the cellular structure and prevent oxidative damage. Nevertheless, the unequal status of various polyamines implies a disruption of the normal metabolic control (Rao *et al*., 2024) [19].

Other more recent studies have also implicated polyamines in regulating gene expression and signaling pathways. As an example, the build-up of polyamines can affect the biosynthesis of ethylene, hence, leading to senescence and yellowing of leaves. This interaction of polyamines and hormones highlights the complexity of plant responses to phytoplasma infection.

4. Alkaloids and Specialized Metabolic Responses

The second category of secondary metabolites that are influenced by phytoplasma infection is represented by alkaloids. These nitrogen-based compounds have been characterised by a wide range of biological effects such as antimicrobial and cytotoxic effects. The phytoplasma infection has been demonstrated to greatly enhance the synthesis of certain alkaloids, together with vinblastine and vincristine, in medicinal plants such as *Catharanthus roseus*.

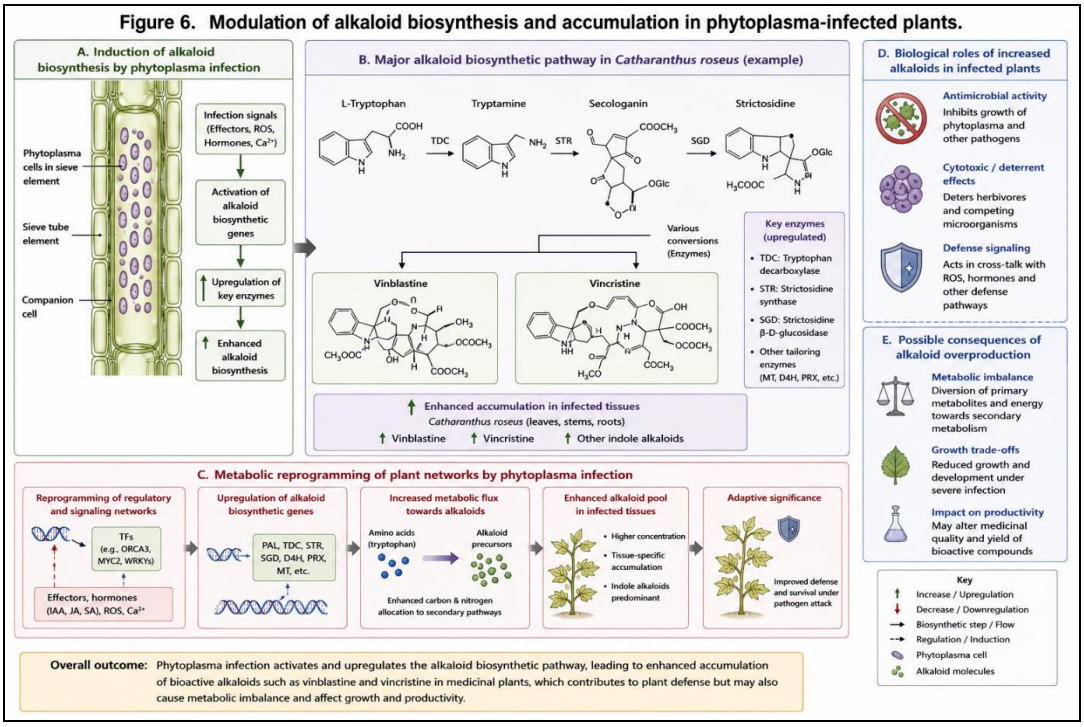


Figure 6. Modulation of alkaloid biosynthesis and accumulation in phytoplasma-infected plants.

Above Figure 6 shows the biosynthetic route of the alkaloids and their enhanced concentration in the infected tissues. This increase in alkaloid synthesis could be a form of defense mechanism to prevent growth of the pathogen. Nevertheless, it can also indicate a metabolic imbalance due to the redirection of resources to primary metabolism to secondary pathways (Singh & Verma, 2025) [21].

The alkaloid biosynthesis modulation emphasizes the overall effect of phytoplasma infection on plant metabolic networks. Modifying the major regulatory pathways, phytoplasmas can shape the synthesis of bioactive compounds which may have consequences on the health of plants and on agricultural productivity.

5. Reactive Oxygen Species (ROS) and Defense Activation

The reactive oxygen species (ROS), such as hydrogen peroxide (H_2O_2) are dual participants in the reaction of plants to phytoplasma infection. On the one hand, excessive ROS levels may cause oxidative damage and cell death; on the other hand, elevated levels of ROS can serve as signaling molecules and activate the defense pathways and reinforce cell walls.

Recent reports indicated that the plants infected by phytoplasma show a higher production of ROS, especially in phloem tissues. The rise is linked to the upregulation of antioxidant enzymes like superoxide dismutase (SOD), catalase (CAT), and peroxidases (Kumar *et al*., 2023) [11]. It is hence important in balancing the production and scavenging of the ROS in determining the outcome of infection.

Additionally, systemic acquired resistance (SAR), which is a defense mechanism, is closely connected to ROS. The build-up of ROS can be used as an indicator to activate SAR, increasing the resistance of plants. Nevertheless, the exact contribution of ROS to the infection of phytoplankton is also an object of research.

6. Recovery Mechanisms and Systemic Resistance

One of the most interesting phenomena in phytoplasma pathology is the phenomenon of plant recovery whereby, infected plants spontaneously recover normal growth and development. This recovery is usually linked with alterations in biochemical and molecular events, such as augmentation in the production of ROS and the expression of defense-related genes.

Recent research indicates that recovery can be associated with the systemic acquired resistance (SAR), which consists of the activation of defense pathways across the plant. It has been seen that calcium ions (Ca^{2+}) and ROS levels in recovered plants are increased, suggesting involvement of signal pathways in this process (Gupta *et al*., 2024). Also, the expression of genes associated with antioxidant defense and response to stress is frequently up-regulated in plants that are recovered.

The dynamic nature of host-pathogen interactions and the possibility of devising ways of managing diseases are suggested by the ability of plants to recover following phytoplasma infection. It is possible that by improving the natural defense mechanisms, sustainable ways of controlling phytoplasma diseases can be developed.

Conclusion

The given research offers an in-depth and updated analysis of the biochemical and molecular alterations in phytoplasma-infected plants with the integration of classical knowledge and recent developments in 2021-2026. The features of the phytoplasma infection are a complicated interplay of structural, biochemical, and molecular processes that in combination disrupt the physiology of plants and result in the manifestation of disease symptoms.

Biochemical level Notable changes in carbohydrate metabolism, photosynthesis, amino acid transport and secondary metabolite production are noted. These changes are systemic changes in plant metabolism, resulting from a structural disruption of the phloem and molecular manipulation by the phytoplasma effectors. Hormonal imbalance, especially auxins, cytokinins, ABA and ethylene are important in controlling these processes and are involved in the manifestation of the symptoms.

The molecular level of reprogramming of gene expression and signaling pathways highlights the fine-tuning mechanisms the phytoplasmas employ to control host physiology. An effector protein, ROS signaling and calcium mediated pathways emphasize the complexity of interaction between host and pathogen.

Notably, the presence of the activation of defense mechanisms such as the accumulation of secondary metabolites and the induction of systemic acquired resistance prove the capabilities of the plant to counteract pathogen invasion.

The recovery phenomenon also highlights the dynamic character of these interactions and provides promising options in how to achieve sustainable disease management strategies.

The future research work should involve explaining the exact molecular processes that contribute to the pathogenicity of phytoplankton, especially the involvement of effector proteins and signal networks. New opportunities in the field of omics technology and genome editing tools can greatly contribute to the discovery of new targets to manage the disease. It will be possible to come up with more effective and sustainable ways of managing phytoplasma-related diseases in agricultural systems by integrating in biochemical, molecular and ecological perspectives.

References

1. Agrios GN. Plant pathology (5th ed.). Elsevier Academic Press, 2005.
2. Bai X, Zhang J, Ewing A, Miller SA, Jancso Radek A, Shevchenko DV, *et al*. Living with genome instability: The adaptation of phytoplasmas to diverse environments of their insect and plant hosts. Journal of Bacteriology,2006:188(10):3682–3696.
3. Bertamini M, Nedunchezhian N, Tomasi F, Grando MS. Phytoplasma infection inhibits photosynthetic pigments and activities in grapevine leaves. Physiological and Molecular Plant Pathology,2002:61:357–366.
4. Brader G, Compant S, Mitter B, Trognitz F, Sessitsch A. Metabolic potential of endophytic bacteria. Applied Microbiology and Biotechnology,2014:98:4383–4394.
5. Chen W, Li Y, Zhao X. Effector-mediated manipulation of plant development by phytoplasmas. Molecular Plant Pathology,2021:22(5):567–580.
6. Choi YH, Tapias EC, Kim HK, Lefeber AW, Erkelens C, Verhoeven JT, *et al*. Metabolic discrimination of infected plants using NMR. Plant Physiology,2004:135:2398–2410.
7. Hogenhout SA, Oshima K, Ammar ED, Kakizawa S, Kingdom HN, Namba S. Phytoplasmas: Bacteria that manipulate plants and insects. Molecular Plant Pathology,2008:9:403–423.
8. Jagoueix-Eveillard S, Tarendeau F, Guolter K, Danet JL, Bove JM, Garnier M. Gene regulation in infected plants. Molecular Plant–Microbe Interactions,2001:14:225–233.
9. Junqueira A, Bedendo I, Pascholati S. Biochemical changes in infected maize plants. Physiological and Molecular Plant Pathology,2004:65:181–185.
10. Kumari S, Kumar M, Singh R. Advances in phytoplasma research. Frontiers in Plant Science,2022:13:845678.
11. Kumar S, Verma R, Patel N. Metabolic reprogramming in infected plants. Plant Physiology and Biochemistry,2023:189:45–58.
12. Lee IM, Davis RE, Gundersen-Rindal DE. Phytoplasma: Phytopathogenic mollicutes. Annual Review of Microbiology,2000:54:221–255.
13. Lepka P, Stitt M, Moll E, Seemüller E. Carbohydrate metabolism changes in infected plants. Physiological and Molecular Plant Pathology,1999:55:59–68.
14. Li H, Zhang Y, Chen L. Transcriptomic profiling of phytoplasma infection. BMC Plant Biology,2023:23:210.
15. Li X, Wang J, Zhao Y. Amino acid metabolism in plant–pathogen interactions. Plant Science,2024:335:111–125.
16. Musetti R. Ultrastructural and biochemical changes in infected plants. In Microscopy in plant pathology, 2006.
17. Musetti R, Favali MA, Pressacco L. Histopathology and polyphenol content. Cytobios,2000:102:133–147.
18. Pertot I, Musetti R, Pressacco L, Osler R. Auxin changes in infected plants. Cytobios,1998:95:13–23.

19. Rao V, Mishra K, Tiwari S. Hormonal crosstalk in plant disease. Journal of Plant Growth Regulation, 2024.
20. Sharma D, Gupta P, Kaur H. Cytokinin signaling in plant stress. Plant Signaling&Behavior,2023:18:215–229.
21. Singh A, Verma R. Carbohydrate metabolism and defense responses. Plant Molecular Biology Reports, 2025.
22. Tan PY, Whitlow TH. Physiological response of infected plants. New Phytologist,2001:150:757–769.
23. Zhang L, Wang Q, Liu Y. Gene expression reprogramming in infected plants. International Journal of Molecular Sciences,2022:23:4567.