Agri Articles
(e-Magazine for Agricultural Articles)
Volume: 02, Issue: 06 (NOV-DEC, 2022)
Available online at http://www.agriarticles.com
©Agri Articles, ISSN: 2582-9882

Strategies Adopted by Plants to Resist Pests and Diseases for Surviving in a Hostile World

(*Jagdish Yadav, Ashok Kumar Mahawer and Prashantha S.T.)
ICAR-Indian Agricultural Research Institute, New Delhi
*Corresponding Author's email: yadavjagdish335@gmail.com

Abstract

As primary producers, plants are under constant pressure to defend themselves against potentially deadly pathogens and herbivores. To resist pests and diseases, plants employ internal and external strategies that are based on plant immunological pathways and can involve interactions with beneficial organisms. These strategies occur over varying timescales ranging from innate immune responses within seconds to epigenetic and symbiotic adaptations that occur over multiple plant generations. The two pillars of the plant innate immune system, PTI and ETI, provide instant protection against the majority of potential pests and pathogens. Attack by pests and/or pathogens induces the release of plant metabolites that recruit and support beneficial organisms, which can help plants and their progeny resist pests and diseases.

Introduction

Our planet is inhabited by a wide range of different plant species, from short-lived desert angiosperms to long-lived coniferous species that dominate boreal forests. Although variable in phenotype, generation time, and geographical range, all plants are primary producers. Consequently, they face constant pressure from opportunistic attackers, such as viruses, bacteria, fungi, nematodes, arthropods, and large herbivores. Despite this pressure, land plants have continued to thrive for ages, which would not have been possible without sophisticated defense strategies. All plants have an innate immune system, which provides instant protection against most attackers. Plants can also acquire resistance after the perception of specific environmental stimuli. This acquired resistance (AR) is typically long-lasting and can

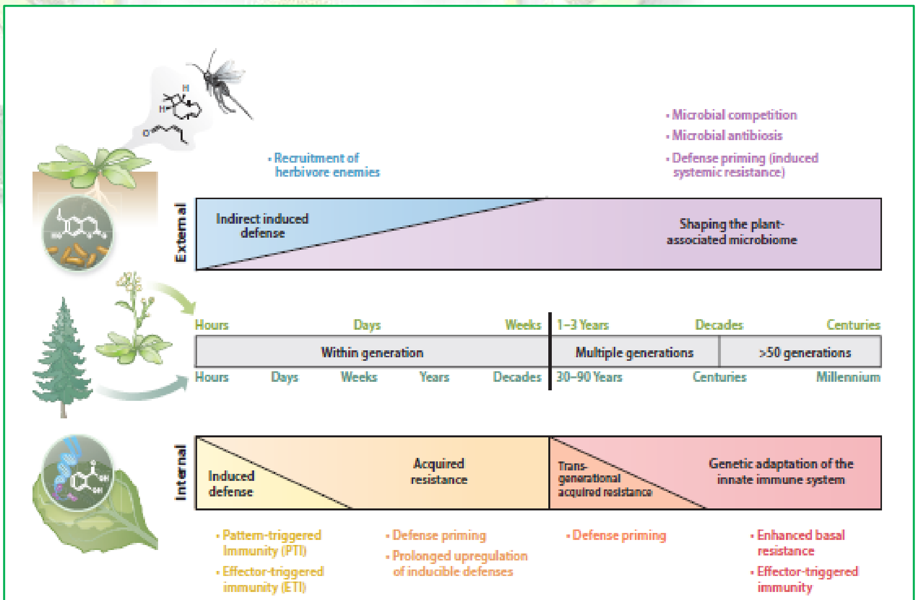

Fig 1. Short- and long-term strategies by which plants adapt to stress from pests and diseases.

even be transmitted to following generations through changes in DNA methylation and associated chromatin density. There is increasing evidence that these epigenetic processes can influence genetic mutations and the rate at which new defense genes evolve. In addition to these internal strategies, plants are capable of orchestrating multitrophic ecological interactions for their protection. These external strategies involve above- and belowground recruitment of beneficial insects and microbes. Recruitment of beneficial microbes can lead to the formation of disease-suppressive microbiomes that offer long-term protection to individual plants and their progeny.

Internal Strategies

The Innate Immune System: Pre-existing constitutive defense structures, such as secondary cell walls, trichomes, and thorns, provide the first layer of protection against attackers. Although these structures are efficient against non-adapted opportunistic aggressors, they can also be costly and are typically ineffective against more specialized attackers. Consequently, plants have evolved a regulatory system for more efficient exploitation of defense resources: the plant innate immune system. This genetically controlled system regulates the perception of attack and subsequent activation of inducible defenses and is subject to an evolutionary arms race with virulence strategies of pathogens and herbivores. There are the two major pillars of the plant innate immune system: Pattern-triggered immunity (PTI) and effector-triggered immunity (ETI), both of which provide instant protection over relatively short timespans ranging from hours to days. PTI protects plants against most potential attackers and involves multiple defense layers that are induced after recognition of specific molecular patterns. This recognition is mediated by pattern recognition receptors (PRRs), which are receptor kinases and receptor-like proteins that are receptor-like proteins that are localized to the surface of plant cells. PRRs detect attackers via molecular patterns. Some PRRs detect pathogen-/microbe-/herbivore-associated molecular patterns (PAMPs, MAMPs, and HAMPs), which indicate the presence of chemical signatures that are not from the host plant itself. Regulation of PTI is controlled by a complex signalling web that varies between plant species and the molecular patterns perceived. Perception of PTI-eliciting molecular patterns induces fluxes of defense hormones, which regulate defenses that are effective against different groups of attackers Jasmonic acid (JA)-dependent defenses are generally more effective against necrotrophic pathogens and herbivores, whereas salicylic acid (SA)-dependent defenses are mostly effective against biotrophic pathogens.

Effector-triggered immunity: To counter immune-suppressing effectors from hemi-biotrophic pathogens, plants have evolved resistance genes (*R* genes). Most *R* genes encode nucleotide binding site–leucine-rich repeat (NLR) receptor proteins, which directly or indirectly detect pathogen effector activity. Activation of NLR receptors elicits ETI, which often leads to a form of programmed cell death at locally infected tissues, the hypersensitive response (HR). The HR is very effective against biotrophic pathogens that rely on living plant cells for their growth. The limitation of ETI is its narrow range of effectiveness. Each R protein recognizes a limited number of effectors, thereby providing protection against one or a small number of pathogen isolates. This reliance on single *R* genes allows pathogens to rapidly overcome ETI, as a single mutation can give rise to a virulent pathotype.

Acquired Resistance: Enhanced resistance to pests and/or pathogens following exposure to specific stimuli is known as induced or acquired resistance (AR). AR is an example of phenotypic plasticity because it allows plants with the same genotype to have different resistance phenotypes. The classic example is systemic AR (SAR). SAR is a long-lasting resistance response, which can still be detected at 42 days after induction. Colonization of roots by beneficial rhizobacteria or mycorrhizal fungi can result in induced systemic resistance (ISR) and mycorrhiza-induced resistance (MIR), respectively. AR is generally based on two nonexclusive mechanisms: prolonged upregulation of inducible defenses and defense priming. Following exposure to a resistance-inducing stimulus, inducible defenses can remain upregulated, providing AR against subsequent attack.

Epigenetic mechanisms of defense priming: Genomic DNA in the nucleus is tightly wrapped around histone protein octamers called nucleosomes, which form the basic unit of eukaryotic chromatin. The density of chromatin regulates to what extent the DNA is available for the transcriptional machinery. Heterochromatin is often associated with silenced genetic areas, whereas lightly packed chromatin (euchromatin) is more associated with transcriptionally active areas. Chromatin density is controlled by methylation and acetylation of histone tail residues and the presence of specific histone variants inside nucleosomes. Histone modifications and related changes in chromatin density *cis*-regulate priming of defense genes. In addition to histone modifications, there is increasing evidence that DNA methylation regulates priming. Unmethylated DNA is often associated with euchromatin, whereas methylated DNA is more likely to be associated with heterochromatin

External Strategies

Plants constantly interact with beneficial organisms, such as insect pollinators, animal seed dispersers, nitrogen-fixing bacteria, and nutrient-providing fungi. These organisms can assist plants in their battle against pests and pathogens. In this section, we explore how plants enhance their survival by enlisting beneficial organisms for their defense. This includes both short-term strategies, such as recruitment of natural enemies of attacking herbivores, and long-term strategies, such as shaping rhizosphere and soil microbiomes.

Indirect Induced Defense: In response to feeding or egg deposition by herbivores, plants often emit herbivore-induced plant volatiles (HIPVs). HIPVs can mediate tritrophic interactions by attracting predators and/or parasitoids of the attacking herbivore. The recruitment of natural enemies of plant attackers by HIPVs is commonly referred to as indirect induced defense because plants are not directly antagonizing their attackers. Indirect induced defense is controlled by the plant innate immune system, as HIPVs are induced by defense-eliciting molecular patterns (DAMPs and HAMPs). Accordingly, this plant defense strategy operates over relatively short timescales. Many early reports about indirect induced defense involved maize. In response to feeding by caterpillars, maize releases a rich blend of volatiles that can recruit parasitoid wasps.

Shaping the Plant-Associated Microbiome: The rhizosphere, phyllosphere, and spermosphere of plants are heavily colonized by commensal and mutualistic microbes. These plant-associated microbiomes can help suppress biotic stress via different mechanisms, including antibiosis, nutrient competition, and induction of AR. Consequently, changes in the plant-associated microbiome can improve the long-term survival of plants and even their progeny.

Rhizosphere: Plants control their rhizosphere microbiome through root exudates, which change the chemical composition of the rhizosphere to attract and support beneficial microbes, such as plant growth–promoting rhizobacteria (PGPR) and mycorrhizal fungi. Although the exact mechanisms driving these stress-induced changes remain unclear, key defense signaling chemicals, including SA, JA, and benzoxaxinoids, have been shown to influence microbial communities in the Rhizosphere. Microbes recruited to roots of pathogen- or herbivore-infested plants can contribute to defense via both direct mechanisms, such as biocidal activity against the attackers and indirect mechanisms, such as competition for nutrients or induction of AR. Stress-induced changes in root exudation can also benefit the progeny of the attacked plant. This transgenerational effect acts through soil conditioning, during which plant-beneficial microbiota are vertically transmitted from parent to offspring via the soil.

Conclusions

With ever-increasing global interconnectedness and our rapidly changing climate, the distribution of pests and pathogens is expanding. Reliance on a limited number of crop and tree species for food and wood products makes our production systems vulnerable to these shifts in pest and pathogen ranges. Understanding the strategies by which different plants adapt to biotic stress will aid with the selection of new crop and tree varieties. Apart from the burden of low genetic diversity, agriculture and forestry remain heavily reliant on pesticides. Because of stringent regulations on pesticide usage, the evolution of pesticide resistance, and public concerns about environmental sustainability and chemical residues, this reliance will need to change. It is widely believed that an integrated approach to plant protection can improve the environmental sustainability of the global bioeconomy. The plant defense strategies outlined here seem promising for exploitation in such an integrated approach. However, more research is needed to understand the underpinning mechanisms, ecoevolutionary drivers, and complementarity of these strategies.

References

1. Ahmad S, Van Hulten M, Martin J, Pieterse CMJ, Van Wees SCM, Ton J. 2011. Genetic dissection of basal defence responsiveness in accessions of Arabidopsis thaliana. Plant Cell Environ. 34(7):1191–206.
2. Berendsen RL, Vismans G, Yu K, Song Y, De Jonge R, et al. 2018. Disease-induced assemblage of a plant-beneficial bacterial consortium. ISME J. 12(6):1496–507.
3. Wilkinson, S. W., Magerøy, M. H., Lopez Sanchez, A., Smith, L. M., Furci, L., Cotton, T. A., & Ton, J. (2019). Surviving in a hostile world: plant strategies to resist pests and diseases. Annual review of phytopathology, 57, 505-529.