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Hypersensitivity- A Combat Mechanism of Stress Tolerance

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Plants have specialized immune response system which has capacity to recognize and destroys infectious agents. There are numerous sophisticated and innate immune system that enable plant to restrain pathogen attack. Hypersensitivity is regarded as hallmark of pathogen resistance response in plants. The article helps to accentuate the immune response mechanisms in plant to cope-up stress condition.

Introduction

Under stress conditions, plants have special defense mechanism known as hypersensitivity. This response is accompanied with toxic compounds accumulation inside a dead cell and observed with naked eye. This type of effectively suicide as a part of plant's defense mechanism against pathogen attack called as Programmed cell death (PCD). The resistance mechanism solely dependent on incompatible interactions between plant and pathogens. There is complex signaling cascade in the resistant plant cells, which are coming from a virulent pathogen and result in cell death at the infection site. With this, the pathogen which penetrates already, would die within cells and infection not increase to other surrounding cells. Basically, it deprives the pathogen from nutrients in early steps of infection and they start starving, before they cause any damage. Hypersensitive mechanisms act as promising target in enhancing the overall pathogen resistance of crop plants and classified into two main categories termed asstructural hypersensitive and induced hypersensitive response.

Structural hypersensitive response

Hypersensitive is a biochemical reaction, but considered as structural response carried out via programmed cell death. PCD is a great resistance mechanism shown by plants in which it kills its own cells during initial stages of pathogen attack, so that it isolate pathogen from nutrients and its growth stops (Sarkar et al. 2015). It plays key role in defense and developmental mechanism, act as central component during innate immune response that mediated through intracellular program (Fig 1). There are two types of biochemical programmed cell death. i.e. Apoptosis and Autophagy.

- ***Apoptosis*:** Cell division and cell differentiation in a programmed manner leads to development of any multicellular organism. In this, intracellular organelles undergo fragmentation and whole cell collapse produce belbs, whereas membranous vesicles come out from cell surface. Outside, they are recognized by macrophages and they engulf it (Anonymous 2018).
- ***Autophagy*:** It is type of prosurvival mechanism that constrict the programmed cell death mechanism which is during plant innate immunity (Sarkar et al 2015). In this phenomenon, cytoplasmic components are engulfed by autophagosomes and transferred into vacuoles for degradation. Along with this, autophagy also regulates HR- PCD (Hypersensitive response-programmed cell death) by degrading the proteins produced during oxidative stress.

Induced Hypersensitive Response

This initial recognition event among host plant and pathogens, which result in activation of defense response mechanism. Hypersensitive response regulates various direct and indirect

interactions among avirulence gene products and resistance gene products. The specificity between specific host and pathogen is due to R genes (resistance), which recognizes, trigger into action by elicitor molecules produced by pathogens.

Processes other than Programmed cell death

- ***Necrosis*:** It is unprogrammed cell death, where upon injuries, cellextrudes various injurious components that cause damage to other surrounding cellsconsidered to be mass killing.
- ***Paraptosis*:** In this, cell swells and large vacuoles develop inside it, which causes cell death.
- ***Autoschizis*:** It is a novel type of cell death, where cell develop crates via which cell organelles move infected cells and destroyed by proteases. In healthy cellsremain unaffected during this process.
- ***Oncosis*:** In this, cells expand by absorbing lot of water in an uncontrolled manner. With this, cells intakes excess calcium into cells that leads to protein denaturation and ultimately leads to cell death.

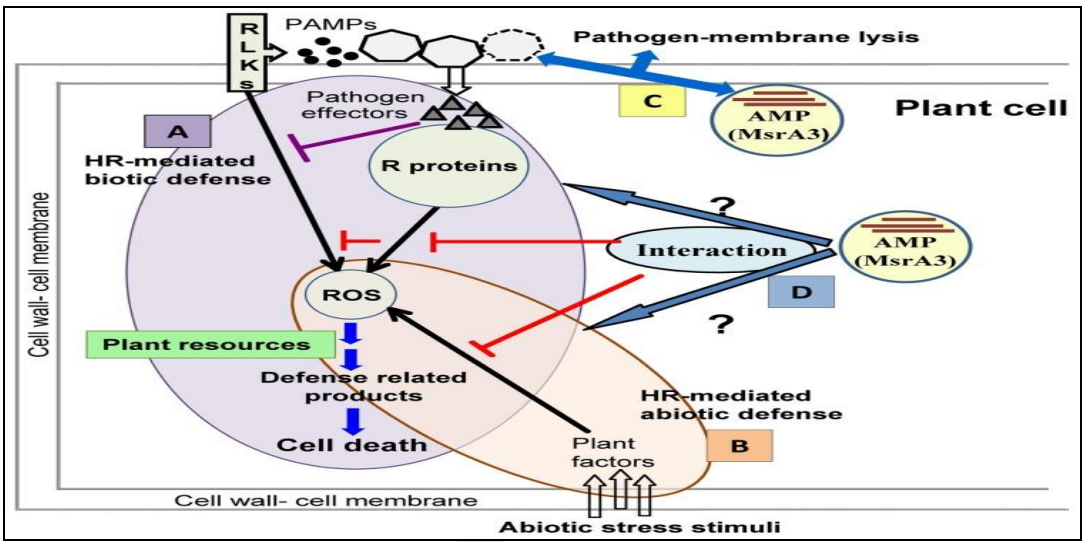

Illustration of pathways and processes during hypersensitive response (HR) against stress condition in pants. (Goyal *et al.*, 2013).

First line of defense

This first step in plant's immune response is pathogen perception through pathogen-associated molecular patterns (PAMPs) or pattern-recognition receptors (PRRs). PAMPs are derived from non-pathogens, basically called as microbe-associated molecular patterns or MAMPs. Host plant can also detect damage-associated molecular patterns (DAMPs) that are produced during invading pathogens and which are released by plants during pathogen attack.

- ***MAMPs*:** Microbe- associated molecular patterns (MAMPs) are highly conserved structures that are needed by microbes for survival. This is main reason that it conserved among pathogens, non-pathogenetic as well as saprophytic microorganisms. MAMPs are recognized by pattern recognition receptors that are present on plant's cell surface. This first phase of defense system is known as MAMP-triggered immunity (MTI).Second line of plant defensive response is recognition by R genes that is known as effector triggered immunity. Mostly, R proteins are intracellular receptor proteins of NR-LRR type (nucleotide-binding-leucine-rich-repeat).

- ***DAMPs*:** In this type of defense system initiated by microbial elicitors as well as plant derived molecules. This type of sensing is mediated by DMAPs (damage-associated molecular patterns) also named as microbe-induced molecular patterns.
- ***Oxidative Burst*:** MAMP-induced defense response also leads to production of ROS (reactive oxygen species). Oxidative burst result in rapid production of reactive oxygen species like hydrogen peroxide and super oxide radicals. ROS also strength the plant cell wall, for restricting pathogens. Both ROS and NO interact to produce reactive nitrogen species like ONOO-, NO_2, N_2O_3, and other NOX species. HR is initiated by balanced formation of NO and ROS, along NO interaction with hydrogen peroxide.
- ***Phytoalexins Induction*:** These are antioxidative substances produced within plant which accumulate very fastly at the pathogen infected areas. Basically, these are produced by uninfected cells. The major roles of phytoalexins are cell wall puncturing, delay in maturation, disrupt metabolism as well as reproduction of pathogens. Formation of phytoalexins only take place when plant comes in contact with pathogen and infection initiates.

Second line of defense

In this, R genes recognize the effectors via plant resistance gene products (Fig 1) (Goyal et al 2013). Second line of plant defense system is through plant resistance genes that leads in effector-triggered immunity. This concept was well supported by Gene-for-Gene Hypothesis given by Flor in 1942. According to this, each gene that is controlling resistance, there is corresponding gene controlling pathogenicity in pathogen. Whenever, host gene and pathogen gene matches for all loci, then host is susceptible to that, otherwise it shows resistant reaction.

Conclusion

Under stress conditions, such hypersensitivity or effectively suicidal system create so many questions on plant adaption behavior and their stress management strategies. The specificity between specific host and pathogen solely depends upon their genetic traits. The mystery of plant defense systems was dissected in recent years with advancement of noble molecular techniques but still pace on for enthusiasm.

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