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Commentary

Regulation of Plant Hypersensitivity Response

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DESCRIPTION

Hypersensitive response is a mechanism used by plants to prevent the spread of infection by microbial pathogens. Hypersensitivity is characterized by the rapid death of cells in the local region surrounding an infection and it serves to restrict the growth and spread of pathogens to other parts of the plant. It is a natural defense for plants in response to a variety of pathogens like viruses, bacteria, fungi and characterized by a programmed cell death (PCD) accompanied by an accumulation of toxic compounds within the dead cell. Hypersensitive reaction (HR) is taken into consideration as a biochemical rather than a structural protection mechanism however may be visible with the naked eye or with a microscope. There are varieties of hypersensitive responses such as structural and induced programmed cell death. Programmed cell death is seen in both structural as well as in induced hypersensitive response. Programmed cell death is extreme resistance shown by the plants in which it kills its cells (suicidal death), upon a perception of the pathogen to deprive it of nutritional supply and stops its growth. Apoptosis and autophagy are physiological techniques and kinds of biochemical programmed cell death.

Induced hypersensitivity reactions occur when plants recognize specific signalling molecules called elicitors produced by pathogens. Recognition of the elicitors by the host plant activates an army of biochemical reactions. These responses include oxidative bursts of reactive oxygen species (ROS), changes in the plant cell wall including cell wall immunity and damage-related molecular patterns, induction of phytoalexins. All of these are some of the front lines of plant defense that act on the recognition of conserved molecules characteristic of many microorganisms. These are called triggers and are known as microbial or pathogen-associated molecular patterns (MAMP or PAMP). The second line of defense of plants is the recognition of effectors by the plant disease resistance gene product known as the R gene, resulting in effect-induced immunity. This is supported by the gene-to-gene hypothesis. Non-pathogenic genes encode proteins that are specifically recognized by the genotype of the host plant, including the appropriate resistance genes. Hypersensitivity reactions are mechanisms used by plants to prevent the spread of infection by microbial pathogens. Hypersensitivity reactions are characterized by rapid death of cells in the local area around the infection and help limit the growth and spread of the pathogen to other parts of the plant. It resembles the animal's innate immune system and usually precedes a slower systemic (whole plant) response, ultimately leading to systemic acquired resistance. HR is observed in the majority of plant species and is induced by various plant pathogens such as oomycetes, viruses, fungi and even insects.

Hypersensitivity is widely recognized as an effective defense strategy against bio nutrient phytopathogens that require living tissue to obtain nutrients. For necrotic trophic pathogens, Hypersensitivity may also be beneficial to the pathogen, as it requires dead plant cells to obtain nutrients. The situation is complicated by pathogens such as Phytophthorainfestans, which act as bio nutrients in the early stages of infection but later switch to a necrotic nutritional lifestyle. It has been suggested that HR in this case may be beneficial in the early stages of infection but not in the later stages. The first idea of how the hypersensitivity reaction occurs came from Harold Henry Flor's Geneforgene model. He hypothesized that for every resistance gene (R) encoded by the plant, there is a corresponding non-pathogenic gene encoded by the microorganism. If both the Avr and R genes are present in the plant-pathogen interaction, the plant is resistant to the pathogen. Genes involved in plant-pathogen interactions tend to evolve very rapidly. The resistance mediated by the R gene is because they induce HR, which causes apoptosis. Most plant R genes encode NOD-like receptor (NLR) proteins. The NLR protein domain architecture consists of the NBARC domain, which is a nucleotide-binding domain involved in the conformational changes associated with NLR protein activation. In the inactive form, the NBARC domain binds to Adenosine Diphosphate (ADP). When the pathogen is recognized, ADP is exchanged for adenosine triphosphate (ATP), which causes a conformational change in the NLR protein, causing HR. At the N-terminus, there is either the Toll Interleukin Receptor (TIR) or the coiled coil motif. Both the TIR domain and the CC domain are involved in the cause of cell death.

The plant hypersensitive response (HR) is a rapid localized cell death that occurs at the point of pathogen penetration and is associated with disease resistance. While others had noted similar phenomena previously it is believed that E.C. Stakman (Stakman, 1915) first to use the term 'hypersensitive' mentioning the extreme resistance of certain grass hosts to Puccinia graminis, author said, "in such cases, the host plant is hypersensitive to fungi.

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