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MECHANISM OF HOST PATHOGEN INTERACTION

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ABSTRACT
Unlike animals, plants defend invading pathogen by equipping a variety of resistance mechanisms. The programmed cell death is the one which restrict the pathogen to spread further. Plant also develops induced resistance mechanism such as hypersensitive and systemic acquired resistance upon pathogen infection. All these mechanisms are active according to the one gene one enzyme theory. Pathogenesis-related (PR) gene expresses themselves by synthesizing pathogenic related protein which intern activate the various kinds of resistance mechanism to ward off the pathogens.

Introduction

Plants are equipped with variety of defense system to effectively overcome the damage caused by pathogens. Plant developed induced resistance (RS) mechanism upon response to pathogen attack. There are two types of resistance mechanisms such as hypersensitive responses (HS) and systemic acquired resistance (SA) so far recognized in the basic defense response pathway as the mechanism of host pathogen interaction.

Hypersensitive response and defense mechanism

The hypersensitive response is a type of programmed cell death (PCD or apoptosis). It is characterized by that kill the cells in the areas of infection. It causes localized cell and tissue death at the site of infection. As a result, the pathogen remains confined to necrotic lesions near the site of infection. Cell death caused by pathogen infection is frequently associated with plant resistance. There appear to be two types of plant cell death associated with pathogen infection: a rapid, hypersensitive cell death localized at the site of infection during an incompatible a resistant plant and an avirulent pathogen, and a slow, "normosensitive" plant cell death that spreads beyond the site of infection

during some compatible interactions involving a susceptible plant and a virulent, necrogenic pathogen. A ring of cells surrounding necrotic lesions become fully refractory to subsequent infection, known as localized acquired resistance. The earliest reactions of plant cells include changes in plasma membrane permeability leading to calcium and proton influx, and potassium and chloride efflux, which subsequently induce extracellular production of reactive oxygen intermediates such as superoxide, hydrogen peroxide, and hydroxyl free radicals (McDowell and Dangl, 2000). The localized production of reactive oxygen intermediates and nitric oxide act to induce hypersensitive responses and expression of disease defense genes (**Fig. 1.**).

Types of induced resistance

Induced resistance are of three types viz. Local acquired resistance (LAR), Systemic acquired resistance (SAR) and Induced systemic resistance (ISR).

Systemic acquired resistance (SAR)

SAR refers to a distinct signal transduction pathway that plays an important role in the ability of plants to defend themselves against pathogens. After formation of necrotic lesion, either as a part of the hypersensitive response or as a symptom of disease, the SAR pathway is activated. SAR is more durable and relies on the plants endogenous defense, classically it is effective against biotrophic pathogens. Major changes in gene expression and broad-range resistance, leads to pathogenesis-related (PR) gene expression (Rajou *et al.*, 2006).

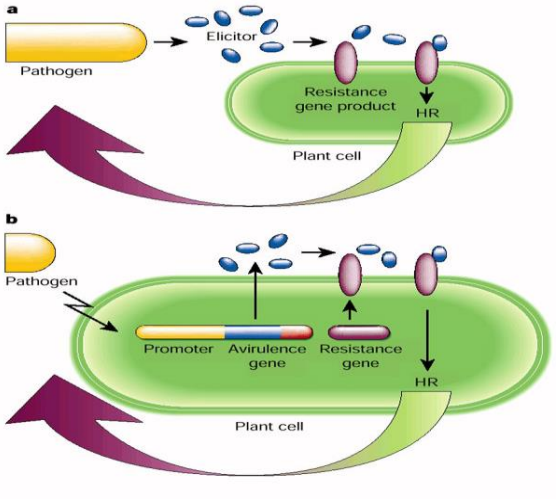

Fig. 1. Hypersensitivity response

a. The hypersensitive response (HR) is triggered by the highly specific recognition of a pathogen-derived elicitor by a plant resistance gene product. The powerful and concerted defense that constitutes the hypersensitive response stops the pathogen.

b. The components involved in the basic switch of the hypersensitive response can be used to create a more nonspecific defense system. A plant-derived pathogen-inducible promoter drives expression of a pathogen elicitor gene. The elicitor formed will trigger the hypersensitive response if the plants also contain the resistance gene.

History

SAR was first recognized in 1933 by Chester. He noted that infection of plants with necrotizing pathogens often results in enhanced resistance to subsequent infectious fungal, bacterial and viral pathogens. Ross in 1962 coined the term systemic acquired resistance. He showed viruses trigger SAR in tobacco. Kammen in 1970 proved that 4 new proteins made in TMV infected Tobacco. White in 1979 showed that aspirin induces resistance to TMV in tobacco. SAR is effective against some but not all pathogens e.g., Tobacco: *Phytophthora parasitica, Cercospora nicotianae, Peronospora tabacina, Pseudomonas syringae* pv. *tabaci, Erwinia carotovora,* tobacco mosaic virus, tobacco necrosis virus but not effective against: *Botrytis cinerea or Alternaria alternate.* Arabidopsis: *Phytophthora parasitica, Pseudomonas syringa*e pv. tomato DC3000, turnip crinkle virus.

Key events in understanding regulation of SAR

SAR associated with coordinated induction of a set of SAR genes encoding proteins known as Pathogenesis-related (PR) proteins. Acetyl salicylic acid application sufficient to induce PR gene expression and enhanced resistance to TMV. Salicylic acid application on tobacco leaves mimics pathogen induced expression of PR genes and pathogen resistance in treated tissues.

Criteria of chemicals to act for SAR

(i) The compound or its significant metabolites should not exhibit direct antimicrobial activity. (ii) It should induce resistance against the same spectrum of pathogens as in biologically activated SAR. (iii) It should induce the expression of the same marker genes as evident in pathogen-activated SAR.

Conclusion

Understanding the basic mechanisms involved in plant pathogen interaction could be necessary to find out the resistance gene and their introgression into the cultivars to effectively manage the pathogen induced stress.

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