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Cell Death in Plant Immune Response to Necrotrophs

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Plant pathogens can be generally divided into two kinds, necrotrophs and biotrophs. Necrotrophs can kill the host cells and feed on the contents, while biotrophs complete their life cycle depending on the living host cells. Microbial necrotrophy is often associated with production of toxins, and necrotrophs are further divided into Host–specific necrotrophs (HSNs) and broad host–range necrotrophs (BHNs) according the toxins they secret. HSNs produce host–specific toxins (HSTs) that is essential for their pathogenicity and virulence and can be recognized by the immune system of theirs hosts. The fungal pathogen *Cochliobolus carbonum* is archetypical HSN, which produces HC–toxin and limitedly infects the susceptible genotypes, causing the Northern corn leaf spot. There are several broad host–range necrotrophs (BHNs) reported previously, such as the fungal pathogens *Sclerotinia sclerotiorum, Alternaria brassicicola, Botrytis cinerea*, and *Plectosphaerella cucumerina*, and the bacterial pathogen *Erwinia carotovora*. Plant responds to necrotrophs differentially according to the primary determinant of virulence [1,2]. The attempted infection of plant pathogens, both biotrophs and nerotrophs, can activate plant immune responses, which include complex histological, cellular, biochemical, and molecular events that the pathogen proliferation or disease spread is limited. At the early stages of necrotrophic infections, host cell death is associated with, as well as the production of various secondary metabolites, antimicrobial peptides, and hormones, such as salicylic acid (SA), jasmonate (JA), ethylene (ET), and abscisic acid (ABA). In addition, accumulation of reactive oxygen species (ROS), callose, and some other cell wall modifications are also involved. The kinetics of these responses and the relative abundance and timing may vary but are common responses to different infections [2].

Interactions between pathogens and their hosts are complicated and dynamic. The plant innate immune system is composed of pathogen–associated molecular pattern (PAMP)–triggered immunity (PTI) and effector–triggered immunity (ETI) pathways. Plants recognize pathogens through two major groups of receptors. Initially, plants sense pathogens via perception of their conserved PAMPs by pattern–recognition receptors (PRRs) located on the cell surface. This first level of recognition results in PAMP–triggered immunity (PTI), which is sufficient to ward off most pathogens. Different from PTI, diverse plant pathogens independently evolved mechanisms to secrete and release effector proteins into host cells evolutionarily. These effectors interact with cellular host targets and regulate PTI and/or host metabolism in a manner conducive to pathogen multiplication and dispersal. Nevertheless, these specific effectors can be recognized by a second set of polymorphic intracellular immune receptors in plants, which mostly belongs to the nucleotide–binding site–leucine–rich repeat (NB–LRR) protein family, analogous to animal innate immune NLR proteins [3]. NB–LRR proteins can be activated upon direct recognition of an effector or indirectly by the action of an effector protein on a specific host target. NB–LRR activation causes ETI, which is essentially a high–amplitude PTI response that results in robust disease–resistance responses that often include localized host cell death and systemic defense signaling [4]. In brief, the plant immune system can be explained using a famous "Zigzag" model. PTI is basically evolved to recognize general feature of plant pathogens, while it is subsequently suppressed by the pathogen acquired effector–triggered susceptibility (ETS), and then the plant resistance (R) proteins are evolved to recognize pathogen ETS, consequently the advanced ETI response initiate in plant [5]. Commonly ETI is regard as an amplified and accelerated PTI response, associated with programmed cell death, a response which is referred to as hypersensitive response (HR) [4]. HR in plant is a unique and specific type of cell death different from animal cell death. When HR cell death is induced by pathogens, the plant–specific cell morphology changes, including chromatin condensation, cytoplasmic shrinkage, mitochondrial swelling, in combined with some other characteristics, such as vacuolization and chloroplast disruption during the final stages [6]. However, the mechanism of cell death in plants stimulated by the effector recognition via NB–LRR receptors is not completely understood. It is supposed that signaling modules regulate NB–LRR proteins and integrate redox signals downstream of NADPH oxidase leading to SA accumulation, which plays an important role in defense responses. Consequently SA and ROS act synergistically to drive HR [6].

Cell death plays an essential role in innate immune responses in both plants and animals, and they both can respond to infection and pathogen recognition with inducing programmed cell death (PCD) [6]. Cell death is a common phenomenon in both resistant and susceptible responses of plant–pathogen interactions, although its temporal regulation may differ with respect to infection. In order to limit the pathogen growth, plants evolve HR–associated cell death to confine pathogens by abolishing nutrient supply. However cell death has distinctly different roles in plant responses to biotrophs and necrotrophs on account of their entirely different living styles. Necrotrophs secret phytotoxins and cell wall degrading enzymes that induce the host cell death, which results in the formation or expanding of necrotic lesions in the infected plant tissue, thereby some necrotrophs promote virulence by using the plant HR machinery as a strategy [2]. If plant HR is invalid in limiting pathogen growth, it may be adaptive for the production of long range signals, which is mediated by SA and ROS and induce the systemic acquired resistance that protects a plant from secondary infection [6].

What role dose ROS play in the cell death defense to necrotrophs in plants? ROS, as an important signaling molecule, can possess antimicrobial activity, induce resistance, or facilitate cell death. Plant–produced ROS are meaningful for resistance to biotrophs and

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hemibiotrophs by regulating the cell death; however it is suggested that ROS is a virulence factor for necrotrophs. It was reported that pathogenicities of the two necrotrophs, *S. sclerotiorum* and *B. cinerea*, were directly dependent on the level of superoxide (OH−) and hydrogen peroxide in cells. At early stages of infection, *S. sclerotiorum* suppresses the oxidative burst beneficial for plant hosts, through an oxalate–dependent mechanism. However, once infection is established, the pathogen induces ROS, resulting in cell death in host tissue, and directly benefits from it. Therefore ROS contributes in different ways depending on the kinetics and context; continuous production of ROS may lead to cell death and then promote susceptibility, nevertheless early activation may induce the disease–resistance. At early stages of infection, the role of ROS in resistance to biotrophs and necrotrophs may be similar and is seemingly based on activation of different immune responses [2,6].

In our recent study, near–isogenic *Brassica napus* lines, including a resistant and a susceptible line to *S. sclerotiorum*, were used in combination with the proteomic technique. A comparison of protein expression profiles in a susceptible line with those in a resistant line during the interaction of *B. napus* with *S. sclerotiorum* resulted in the identification of 20 important proteins related to disease resistance. These proteins were determined to be involved in various functions, including pathogen resistance, antioxidation, and transcription regulation. We also found that the activities of antioxidative enzymes, including ascorbate peroxidase (APX), peroxidase (POD), catalase (CAT), and superoxide dismutase (SOD), were higher, the content of ROS was lower, and DNA laddering was delayed in the resistant line. These findings imply that the ability to remove ROS in the resistant line was stronger than that in the susceptible one. It was therefore possible that the plants responded to pathogen infection by increasing the cellular levels of antioxidant enzymes. It seems that the antioxidative system in the resistant line was more effective than that in the susceptible one and that this helped resist the invasion of *S. sclerotiorum* and delay the onset of PCD in the resistant line. Our finding suggests that ROS was helpful for *S. sclerotiorum's* invading into *B. napus*, and the more efficient antioxidative system helped to slow the spread of the Sclerotinia disease in plants [7].

It is generally believed that HR–associated cell death enhances susceptibility of necrotrophs, but it is unclear whether this extends to all necrotrophs. Interestingly, HR is connected with resistance to the hemibiotrophs *Magnaporthe oryzae* and *Phytophthora infestans*, despite their necrotrophic nutrition at later stages of infection; this may indicate a more complicated association of cell death to susceptibility and resistance [2].

Are the phytochemicals involved in the cell death response to necrotrophs? Plant secondary metabolites are known to facilitate defense to a variety of plant pathogens, and some phytochemicals have multiple functions in ecological interactions [8]. It is reported that Glucosinolates (GS), a group of amino acid–derived secondary metabolites found throughout the Cruciferae family, are commonly synthesized and stored in healthy cells, may also be mobilized to pathogen challenge cells [9]. Upon tissue disruption, glucosinolates are converted to biologically active compounds by myrosinases (plant glycosyl hydrolases). Although best known as insect deterrents, glucosinolate breakdown products, such as desulfo–derivatives, have potent antimicrobial activity [10]. Camalexin, another kind of phytoalexin, is also essential for resistance to the two typical necrotrophs, *S. sclerotiorum* and *B. cinerea*, in *Arabidopsis*. Ren et al. reported that camalexin production is not a secondary effect of cell death, and cell death is independent of camalexin production because cell death was not inhibited in camalexin–deficient mutant [11]. It seems that the syntheses of these phytochemicals in plant cells have less relationship to cell death, while the transportations of them are more meaningful to plant defenses. However, in *Arabidopsis*, it is found interestingly that the genes associated with syntheses of GS and camalexin are induced when challenged with *S. sclerotiorum*, without responding to *B. cinerea*. It may be deduced that specific differences in gene expression between these closely related necrotrophs raise the possibility that specific pathogen elicitors trigger induction of those biosynthetic genes [8].

How are hormones involved in the plant defense to necrotrophs? Plant hormones are integral to plant immune responses. Mengiste reviewed that ET and JA can impact resistance to necrotrophs significantly, whereas SA primarily regulates resistance to biotrophic and hemibiotrophic pathogens. JA–mediated immunity to necrotrophic pathogens is associated with the regulation of protease inhibitor and secondary metabolite biosynthesis, including that of anthocyanin, an antimicrobial flavonoid. ABA modulates immune responses to necrotrophs through multifaceted mechanisms, such as modulation of defense gene expression, cuticle permeability, callose accumulation, and ROS production/scavenging. Gibberellin (GA) is commonly a negative regulator of defense against necrotrophs [2]. However how changes in hormonal levels during infection are translated into specific immune responses is still obscure.

The finding and understanding of plant R genes resistant to necrotrophs are eagerly needed. Plant resistance proteins and pathogen effectors are key genetic determinants of the HR. However with the exception of *Arabidopsis* resistance to *Leptosphaeria maculans* 3 (RLM3), a Toll/interleukin 1 receptor domain R–protein associated with wide immunity to several necrotrophs, no R–gene has been found responsible for resistance to necrotrophs [2]. Unfortunately, the primary genetic determinants of ETI and its relevant immune responses cannot resist to necrotrophs effectively. Actually responses to some HSNs are inversely corresponding to ETI, and a gene–for–gene relationship between HSTs and the host resistance protein leads to disease and is specific ETS [12]. Many necrotrophs secrete HSTs as virulence factors recognized by host defense proteins that induce the cell death in plant. At the genetic level, simply inherited resistance traits to HSNs are controlled by host genes to detoxify toxins, encode toxin–insensitive alleles to lose recognition specificity, or enhance cellular processes that are important for restricting toxin–induced cell death. Differently, resistance to BHNs is quantitative, requiring many genes for defense, and tolerance to general toxins may be more effective to quantitative resistance to BHNs [2]. However, the biochemical and molecular mechanisms regulating the highly specific (HSNs) or the broad (BHNs) resistance to necrotrophs remain unclear.

In the coming decades, efforts for integration of proteomics, transcriptomics, and metaboliomics data will help elucidate the mechanism of gene expressions and their regulation in plant–necrotroph interactions. While there are many challenges for those OMICS in this field, the chances are also close at hand for a superior understanding of plant defense to necrotrophs, the association to associated dysfunction and pathology in plants.

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