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Plant immunity – cellular aspects of signaling

Immunity – the ability to withstand attack by pathogens – represents a basic ability of all organisms. During the last decade, striking analogies between the defence systems of mammalian cells and plant defence have emerged. Recognition of a pathogen triggers a complex cascade of events that on the one hand culminates in the synthesis of so-called pathogenesis-related proteins, on the other hand in an oxidative burst (K. Apel, H. Hirt, Annu. Rev. Plant Biol. 55: 373–399, 2004). The functional role of this oxidative burst seems to be complex – it is thought to serve as direct defence mechanism by oxidizing essential molecules of the intruder, in addition it will stimulate cross-linking of cell wall components, such that the invasion, for instance, of a fungal pathogen, is impaired. A third function of oxidative burst seems to be more indirect: it seems to be a signal that eventually will lead to a version of programmed cell death termed hypersensitive response (P. Wojtaszek, Biochem. J. 322: 681–692, 1997). The attacked cell will literally commit suicide, thus releasing the content of the vacuole and blocking the attack, such that the neighbouring cells will survive. Cellular aspects of this signaling function are investigated by K. Bóka et al. (pp. 89–97) and J. Chen et al. (pp. 13–21) in this issue.

How is the oxidative burst generated? The major source seems to be an NADPH-dependent oxidase located in the plasma membrane (H. Yoshioka, N. Numata, K. Nakajima, S. Katou, K. Kawakita, O. Rowland, J. D. Jones, N. Doke, Plant Cell 15: 706–718, 2003). Using a suspension culture of *Rubia tinctorum* as model system, Bóka et al. ventured to visualize the subcellular distribution of oxidative burst upon induction of plant defence by elicitors (fragments of fungal cell walls that participate in the recognition process) or the plant hormones jasmonic acid and salicylic acid (which are discussed as components of systemic resistance, i.e., a generalized immunity acquired by organs of a plant that were not in direct contact with the pathogen). By applying a set of histochemical and fluorescent assays, they not only are able to detect several peaks of oxidative burst that differ in timing depending on the corresponding trigger but can actually observe electron-dense precipitates at and adjacent to the plasmalemma, supporting the plasma membrane-located NADPH-dependent oxidase as primary source of oxidative burst.

What triggers the activity of the NADPH-dependent oxidase? In mammalian systems, where oxidative burst is observed in immunoresponses as well, it is under control of lipid second messengers (D. S. Regier, D. G. Greene, S. Sergeant, A. J. Jesaitis, L. C. McPhail, J. Biol. Chem. 275: 28406–28412, 2000). Chen et al. therefore investigate the role of the phospholipase C/diacylglycerol kinase pathway in the context of oxidative burst. They analyze cell death triggered by oxidative burst in rice suspension cultures. As a trigger, they use a chemical inducer of plant defence, benzothiadiazole (BTH), and in parallel a "real" pathogen, *Xanthomonas oryza* pv. *oryza*, the causal agent of rice leaf blight disease. Treatment with BTH triggered oxidative burst followed by hypersensitive cell death a few hours later. The same was observed when BTH was combined with the pathogen, but not when the pathogen attacked in the absence of BTH. Thus, BTH was clearly acting as trigger of defence responses in this system. Using scavengers and antioxidants of active oxygen species, Chen et al. were able to inhibit both oxidative burst and hypersensitive cell death, suggesting that active oxygen species are necessary to trigger plant defence. In the next step they manipulated the phospholipase C/diacylglycerol signaling pathway by overexpression of a diacylglycerol kinase and a specific phospholipase C from rice and were able to activate a defence-activated transcription factor that served as reporter. Similarily, treatment with phosphatidic acid, a product of this pathway, was able to activate the reporter as well as oxidative burst and hypersensitive cell death. Conversely, inhibition of the phospholipase C by neomycin had the opposite effect. Thus, oxidative burst mediates signaling through the phospholipase C/diacylglycerol pathway on activation of defence genes and hypersensitive cell death, which uncovers a further parallel to immune responses in mammalian cells (D. S. Regier et al., in the place cited).

The two publications confirm and extend the existence of a complex signaling network that is located in the plasma membrane and eventually decides whether a given cell will be able to withstand efficiently the attack of the pathogen.

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