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Receptor-like kinases BAK1 and SOBIR1 are required for necrotizing activity of Sclerotinia sclerotiorum necrosis-inducing effectors

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Sclerotinia sclerotiorum is the causative agent of stem rot in canola/oilseed rape, which is the most serious disease afflicting this crop in many regions of the world. The most distinct symptom of the disease are the necrotic lesions that eventually penetrate into the pith and result in collapse of the stem. Tissue necrosis may be caused by necrosis-inducing proteins, such as the necrosis and ethylene-inducing proteins (Nep1 and Nep2) that have been previously characterized in S. sclerotiorum and several other fungal and oomycete plant pathogens. To catalogue the wider suite of necrosis-inducing proteins and/or protein effectors, an informatics exercise was conducted to identify genes encoding small, secreted, cysteine-rich proteins. These were tested for their ability to induce necrosis in Nicotiana benthamiana via Agrobacterium-mediated infiltration. Six novel necrosis-inducing proteins were discovered, of which all but one required a signal peptide and secretion to the periplasmic place for activity. Localization studies using fusion to the green fluorescent protein and co-localization with organelle markers indicated that most of the necrosis-inducing proteins localized to the endomembrane system; however, endoplasmic reticulum stress and induction of the unfolded protein response were not involved in the necrosis phenotype. Interestingly, virus-induced gene silencing experiments revealed that all of the obligately-secreted necrosis-inducing proteins were dependent on the presence of the plant receptor-like kinases BAK1 and SOBIR1 for activity. This suggests that S. sclerotiorum necrosis-inducing proteins very likely interact with an extra-cellular receptor(s) to initiate the sequence of biochemical events required for necrosis and opens up the possibility that Brassica napus lines lacking these receptors might be more resistant to this pathogen.