P53 Dual Cf-2-mediated disease resistance in tomato requires a common virulence target of a fungus and a nematode

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Plants lack the seemingly unlimited receptor diversity of a somatic adaptive immune system as found in vertebrates and rely on only a relatively small set of innate immune receptors to resist a myriad of pathogens. Here, we show that disease-resistant tomato plants use an efficient mechanism to leverage the limited nonself recognition capacity of their innate immune system. We found that the extracellular plant immune receptor protein Cf-2 of the red currant tomato (*Solanum pimpinellifolium*) has acquired dual resistance specificity by sensing perturbations in a common virulence target of two independently evolved effectors of a fungus and a nematode. The Cf-2 protein, originally identified as a monospecific immune receptor for the leaf mold fungus *Cladosporium fulvum*, also mediates disease resistance to the root parasitic nematode *Globodera rostochiensis* pathotype Ro1-Mierenbos. The Cf-2–mediated dual resistance is triggered by effector-induced perturbations of the apoplastic Rcr3pim protein of *S. pimpinellifolium*. Binding of the venom allergen-like effector protein Gr-VAP1 of *G.rostochiensis* to Rcr3pim perturbs the active site of this papain-like cysteine protease. In the absence of the Cf-2 receptor, Rcr3pim increases the susceptibility of tomato plants to *G. rostochiensis*, thus showing its role as a virulence target of these nematodes. Furthermore, both nematode infection and transient expression of *Gr-VAP1* in tomato plants harboring *Cf-2* and *Rcr3pim* trigger a defense-related programmed cell death in plant cells. Our data demonstrate that monitoring host proteins targeted by multiple pathogens broadens the spectrum of disease resistances mediated by single plant immune receptors.