Article

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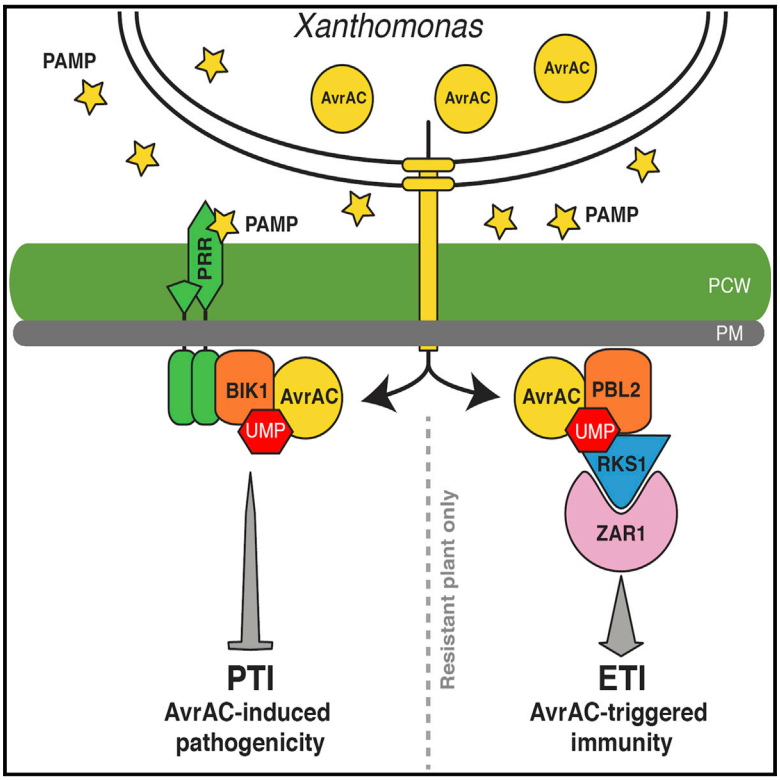
The Decoy Substrate of a Pathogen Effector and a Pseudokinase Specify Pathogen-Induced Modified-Self Recognition and Immunity in Plants

Graphical Abstract



Highlights

- BIK1 paralog PBL2 is uridylylated by *X. campestris* effector AvrAC/XopAC
- PBL2 uridylylation is not required for AvrAC-mediated virulence but triggers immunity
- AvrAC recognition requires the NLR ZAR1 and pseudokinase RKS1 that are in a complex
- The uridylylated PBL2 is specifically recruited to RKS1 to trigger immunity

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In Brief

A plant pathogen effector, AvrAC, uridylylates a host kinase to promote virulence. Wang et al. find that plants have evolved a decoy substrate which upon uridylylation by AvrAC is recognized by a pseudokinase-immune receptor complex to trigger immunity. Thus, decoy substrates and pseudokinases specify and expand immune capacity in plants.

Wang et al., 2015, Cell Host & Microbe *18*, 285–295
September 9, 2015
http://dx.doi.org/10.1016/j.chom.2015.08.004

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The Decoy Substrate of a Pathogen Effector and a Pseudokinase Specify Pathogen-Induced Modified-Self Recognition and Immunity in Plants

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SUMMARY

In plants, host response to pathogenic microbes is driven both by microbial perception and detection of modified-self. The *Xanthomonas campestris* effector protein AvrAC/XopAC uridylylates the *Arabidopsis* BIK1 kinase to dampen basal resistance and thereby promotes bacterial virulence. Here we show that PBL2, a paralog of BIK1, is similarly uridylylated by AvrAC. However, in contrast to BIK1, PBL2 uridylylation is specifically required for host recognition of AvrAC to trigger immunity, but not AvrAC virulence. PBL2 thus acts as a decoy and enables AvrAC detection. AvrAC recognition also requires the RKS1 pseudokinase of the ZRK family and the NOD-like receptor ZAR1, which is known to recognize the *Pseudomonas syringae* effector HopZ1a. ZAR1 forms a stable complex with RKS1, which specifically recruits PBL2 when the latter is uridylylated by AvrAC, triggering ZAR1-mediated immunity. The results illustrate how decoy substrates and pseudokinases can specify and expand the capacity of the plant immune system.

INTRODUCTION

Plants rely on cell-surface-localized pattern recognition receptors (PRRs) and intracellular NOD-like receptors (NLRs) for pathogen detection (Dodds and Rathjen, 2010; Maekawa et al., 2011; Monaghan and Zipfel, 2012). The former are comprised of receptor kinases (RKs) and receptor-like proteins that are functionally analogous to Toll-like receptors in animals and directly perceive molecular patterns derived from invading microbes or released from the plant upon infection and set off pattern-triggered immunity (PTI). Pathogenic microbes often deliver into the host cell effector proteins for virulence (Feng and Zhou, 2012). As a result of host-pathogen coevolution, the effectors are monitored by plants in a highly specific manner and confer effector-triggered immunity (ETI) when cognate NLRs are present (Cui et al., 2015; Jones and Dangl, 2006). Recent reports show that bacterial pathogen effector proteins are also detected by animal intracellular immune receptors including NLRs and pyrin (Keestra et al., 2013; Müller et al., 2010; Yarbrough et al., 2009), highlighting similarities between plant and animal innate immunity.

Plant NLRs recognize pathogen effectors either directly or indirectly (Cui et al., 2015; Jones and Dangl, 2006). Pathogen effectors often possess enzymatic activities that modify host proteins to their benefit. Several plant NLRs indirectly recognize pathogen effectors by interacting with other host proteins when the latter are modified by effectors (Axtell and Staskawicz, 2003; Chung et al., 2011; Kim et al., 2005; Liu et al., 2011; Shao et al., 2003). These modified proteins can either be virulence targets of the effectors or presumed decoys that mimic virulence targets (van der Hoorn and Kamoun, 2008; Zhou and Chai, 2008). Animal intracellular immune receptors may also detect pathogen effectors by indirectly interacting with a modified host protein, although this has not been convincingly shown (Vanaja et al., 2015). Thus the nature of host protein modification by pathogen effectors and mechanisms by which NLRs recognize pathogen effectors hold a key to our understanding of host-pathogen coevolution.

One class of such effector proteins contains the FIC (filamentation induced by cAMP) domain that is shared by several bacterial pathogens infecting plants and animals. The animal bacterial pathogen FIC domain effectors IbpA, VopS, and AnkX adenylylate or phosphocholinate human Rho GTPase for virulence (Mukherjee et al., 2011; Worby et al., 2009; Yarbrough et al., 2009). Interestingly, the modification of a subfamily of Rho by VopS and several other bacterial effectors indirectly triggers