Details

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PLANT SCIENCES

Ubiquitination of OsCSN5 by OsPUB45 activates immunity by modulating the OsCUL3a-OsNPR1 module

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The COP9 signalosome (CSN) is a highly conserved protein complex in eukaryotes, with CSN5 serving as its critical catalytic subunit. However, the role of CSN5 in plant immunity is largely unexplored. Here, we found that suppression of *OsCSN5* in rice enhances resistance against the fungal pathogen *Magnaporthe oryzae* and the bacterial pathogen *Xanthomonas oryzae* pv. *oryzae* (*Xoo*) without affecting growth. OsCSN5 is ubiquitinated and degraded by the E3 ligase OsPUB45. Overexpression of *OsPUB45* increased resistance against *M. oryzae* and *Xoo*, while dysfunction of *OsPUB45* decreased resistance. In addition, OsCSN5 stabilized OsCUL3a to promote the degradation of a positive regulator OsNPR1. Overexpression of *OsPUB45* compromised accumulation of OsCUL3a, leading to stabilization of OsNPR1, whereas mutations in *OsPUB45* destabilized OsNPR1. These findings suggest that OsCSN5 stabilizes OsCUL3a to facilitate the degradation of OsNPR1, preventing its constitutive activation without infection. Conversely, OsPUB45 promotes the degradation of OsCSN5, contributing to immunity activation upon pathogen infection.

INTRODUCTION

Pathogenic organisms can cause severe damages to crop plants, posing a threat to global food security. Plants have developed complex immune systems to protect against these pathogens (*1*, *2*). However, activating defense mechanisms in the absence of pathogens can be costly and harmful to plant growth and overall fitness (*3*). For example, removing the *Mildew Resistance Locus O* (*MLO*) gene in barley, *Arabidopsis* (*Arabidopsis thaliana*), and wheat (*Triticum aestivum*) can provide broad-spectrum resistance to powdery mildew, but it can also lead to unintended consequences such as premature aging (*4*). Similarly, knocking out *SPOTTED LEAF 11* (*SPL11*) and *ENHANCED BLIGHT AND BLAST RESISTANCE 1* (*EBR1*) in rice can enhance resistance to *Magnaporthe oryzae*, but it can also cause notable cell death (*5*, *6*), making it challenging to use these defense genes in practical agricultural settings. Recent studies have shown that some gene knockouts can provide disease resistance without adversely affecting plant growth (*7*). For instance, knocking-out *PUCCINIA STRIIFORMIS-INDUCED PROTEIN KINASE 1* (*TaPsIPK1*) in wheat, *BROAD-SPECTRUM RESISTANCE*

The COP9 signalosome (CSN) is a highly conserved protein complex found in higher eukaryotes, consisting of eight subunits known as CSN1 to CSN8 (*12*). Among these subunits, CSN5 plays a crucial role in removing "Related to Ubiquitin" (RUB) modification from the cullin subunit in Cullin (CUL)-RING ubiquitin ligase (CRL) complexes (*13*). In mammals, CSN5 positively regulates the Cul3/Keap1-mediated degradation of the nuclear factor E2-related factor 2 to control innate immune responses in macrophages (*14*). In *Arabidopsis*, mutations in either *CSN5A* or *CSN5B* lead to the inactivation of CSN and a loss of deRUBylation by CUL1 and CUL4 (*15*). *Arabidopsis* CSN5A interacts with NB-LRR proteins, RLKs, and 29 distinct effectors from *Hyaloperonospora arabidopsidis* (*Hpa*) and *Pseudomonas syringae* (*Psy*). Dysfunction of *CSN5A* enhances resistance to *Hpa* and *Psy* (*16*), indicating a critical role of CSN5 proteins in immunity. Furthermore, silencing or mutation of *TaCSN5* enhances wheat resistance against *Puccinia triticina* and multiple *Puccinia striiformis* f. sp. *tritici* isolates (*17*, *18*). Transient silencing of *VvCSN5* in grapevine (*Vitis vinifera*) boosts resistance to powdery mildew (*19*). These studies have shown that plant CSN5 proteins