A SNARE-protein has opposing functions in penetration resistance and defence signalling pathways

Penetration resistance is often the first line of defence against fungal pathogens. Subsequently induced defences are mediated by the programmed cell death (PCD) reaction pathway and the salicylic acid (SA), jasmonic acid (JA) and ethylene (ET) signalling pathways. We previously demonstrated that full penetration resistance in Arabidopsis against the non-host barley powdery mildew fungus (Blumeria graminis f.sp. hordei) requires the syntaxin SYP121 (PEN1). Here we report that SYP121, together with SYP122, functions as a negative regulator of subsequently induced defence pathways. The SA level in the syntaxin double mutant syp121-1 syp122-1 is dramatically elevated, resulting in necrosis and dwarfism. This phenotype is partially rescued by introducing the SA-signalling mutations eds1-2, eds5-3, sid2-1 and npr1-1 as well as the NahG transgene. These partially rescued triple mutants have an unknown defence to Pseudomonas syringae pv. tomato, and have increased HR-like responses to non-host and host powdery mildew fungi. The HR-like responses cause efficient resistance to the latter. These defence pathways are SA-independent. Furthermore, the JA/ET signalling marker, PDF1.2, is highly upregulated in the triple mutants. Thus SYP121 and SYP122 are negative regulators of PCD, SA, JA and ET pathways through a molecular function distinct from that of SYP121 in penetration resistance. Our data suggest that individual cells preferentially express either penetration resistance or the subsequently induced defences.

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