

**Investigating LysM effector function and the biotrophic growth phase
of *Magnaporthe oryzae***

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Abstract

During intracellular biotrophic growth, the rice blast fungus *Magnaporthe oryzae* secretes a large battery of effector proteins, which are thought to suppress host cell defence responses. Although a number of these effector proteins have been identified, their precise biological functions and contribution towards plant infection remains unclear. In this thesis, I report that during biotrophic growth, the secretion of a LysM effector protein, Slp1, is required for rice blast disease. I show that Slp1 binds chitin and is able to suppress the chitin-induced oxidative burst and defence gene-expression in rice cells. Slp1 competes with the membrane-localised chitin receptor CEBiP in rice, and this competitive interaction results in a reduction in virulence associated with $\Delta slp1$ null mutants. Slp1 is secreted by intracellular hyphae specifically during biotrophic growth, and accumulates around hyphal tips at the plant-fungal interface. Using transgenic rice lines which express fluorescent marker proteins targeted to the plasma membrane and endoplasmic reticulum, I investigate the biotrophic growth phase of *M. oryzae*. I show that the rice host plasma membrane becomes tightly apposed to invasive biotrophic intracellular hyphae. I also show that the rice host plasma membrane and endoplasmic reticulum accumulate around the Biotrophic Interfacial Complex (BIC), a bulbous structure attached to the sub-apical region of intracellular fungal hyphae, which accumulates fluorescently-labelled avirulence effector proteins. Using a fungal plasma membrane marker, I show that the BIC resides outside the fungal plasma membrane and cell wall is made exclusively of plant cellular material.

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