Fitness Costs and Trade-offs in Plant-Parasite Interactions

BSPP Presidential Meeting 2012
16th - 18th December
Norwich UK

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Stomatal lock-up contributes to the cost of resistance to foliar fungal pathogens

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Pathogen attack can reduce yield in plants with effective major gene resistance. In barley genotypes Pallas (susceptible) and P01 (with Mla1) attacked by Blumeria graminis f. sp. hordei (Bgh) stomatal behaviour was disrupted. In Bgh infected Pallas, stomata exhibited a “lazy” phenotype where only opening in the light was restricted but in P01, stomata were “locked” open in both dark and light and failed to respond to drought or the exogenous application of ABA (1). Pallas genotypes with Mla1, Mla3, or MIlA R genes conferred distinctive spatiotemporal patterns of HR following challenge with Bgh and which correlated between the speed of cell death and stomatal locking. Locking occurred at inoculation rates of \(~10\) conidia/mm\(^2\) and in response to the non-host pathogen B. graminis f. sp. avenae (Bga) (2). Examining rust pathogens, in barley genotypes exhibiting major gene resistance against brown rust (Puccinia hordei), stomata locked shut. This was also the case in a wheat cultivar expressing \(R\) gene \(Lr24\) following inoculation with Puccina triticina (3). NO plays a role in bacterial effects on stomata but analysis of transgenic lines of barley cultivar Golden Promise over-expressing a non-symbiotic form of haemoglobin (catalysing the oxidation of NO; \(NO + O_2 \rightarrow NO_3\)), suggested a negligible role for NO in Bga-induced lock-open. Metabolomic and photosynthetic analysis suggests that stomatal locking may be symptomatic of wider-ranging effects on primary metabolism which are likely to contribute to a cost of resistance.