

Genetic control of the resistance of *Erysiphe graminis* f.sp. *hordei* to five triazole fungicides

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The genetics of resistance to ergosterol demethylation inhibitor (DMI) fungicides of the triazole (conazole) group was examined in a cross between two isolates of the barley powdery mildew fungus, *Erysiphe graminis* (= *Blumeria graminis*) f.sp. *hordei*. One isolate, E1, was previously identified as being resistant to the triazole fungicide triadimenol, while the other, HL3/5, was sensitive. The 56 progeny tested were classified into two distinct groups, either being resistant to triadimenol, like E1, or sensitive, like HL3/5. The segregation ratio was not significantly different from 1:1, consistent with responses to triadimenol being controlled by a single gene. In further tests with cyproconazole, epoxiconazole, propiconazole and tebuconazole, all the progeny classified as resistant to triadimenol were also more resistant to each of these other triazole fungicides than were any of the triadimenol-sensitive progeny. This is consistent with the triadimenol resistance allele also conferring cross-resistance to the other triazoles. The ratio between the responses of the resistant and sensitive progeny (the resistance factor, RF) was greatest for triadimenol, followed by tebuconazole, propiconazole, epoxiconazole and cyproconazole, in that order. The RF for triadimenol was much greater when the fungicide was applied as a seed treatment than when it was sprayed. Five isolates, covering the five levels of responses to triadimenol identified previously in the UK population of *E. graminis* f.sp. *hordei*, were used as standards; a triadimenol-sensitive isolate and one with the lowest level of resistance were sensitive to all four of the other fungicides, while three isolates with higher levels of triadimenol-resistance were also more resistant to the other chemicals.

Introduction

Resistance to systemic fungicides has been recognized as a major challenge for crop protection since the early 1970s (Russell, 1995). Farmers may respond to the development of fungicide resistance (and hence of reduced control of the target disease) by switching to a different chemical treatment. However, the benefit of doing so may be limited by the existence of cross-resistance, when genes that cause resistance to one fungicide also confer resistance to other, usually related, compounds.

One of the most important classes of fungicide are C14 demethylation inhibitors (DMIs), which act on the ergosterol biosynthesis pathway. These include the triazole (conazole) fungicides, widely used to control

powdery mildew of cereals caused by *Erysiphe graminis* (= *Blumeria graminis*). The triazole group binds to the haem group of cytochrome P450, a cofactor required by the C14-demethylase (Gadher *et al.*, 1983), while the halogenated phenol group is thought to interact with the substrate binding site of the demethylase enzyme (Hollomon & Butters, 1991). Triadimenol, introduced commercially in 1977, has been the most widely used triazole, while propiconazole, tebuconazole, cyproconazole and epoxiconazole were introduced for cereal mildew control in the late 1980s and early 1990s. A sixth triazole used against cereal mildews, flutriafol, is not considered in this paper.

Resistance to triadimenol is present in populations of barley powdery mildew (*E. graminis* f.sp. *hordei*) throughout Europe (Limpert, 1987; Wolfe *et al.*, 1992), and is severe in that populations of the pathogen are nowadays much less sensitive to this fungicide than they were when it was first introduced. Four distinct levels of resistance to triadimenol have been detected in the UK population of *E. graminis* f.sp. *hordei*, in addition to sensitive isolates (Brown & Wolfe, 1991; Brown *et al.*, 1991). The highest three levels of resistance are each controlled by a single gene (Brown *et al.*, 1992, 1996; Brown, 1996).

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