Variation of *Pithomyces chartarum*, causal agent of facial eczema

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The mycotoxic fungus *Pithomyces chartarum* lives saprophytically on decaying plant material in pasture. When its population builds to high levels, animals feeding on the pasture ingest large quantities of the fungus and, in the case of sheep, cattle and deer, may suffer from facial eczema. We are interested in developing a biocontrol for this disease based on the proposal to substantially replace the toxin-producing strains present in New Zealand pastures by non-producing strains of this species.

There are about 19 species in the genus *Pithomyces*. Most are readily distinguishable from *P. chartarum* by spore characters, but some less readily so. For example, *P. maydicus* is separated by having paler spores with fewer transverse septa (usually two rather than three). Sporidesmins, the toxins implicated in facial eczema, have not been identified in species other than *P. chartarum*.

The sexual stage of *P. chartarum*, *Leptosphaerulina chartarum*, has been identified in South Africa but not elsewhere. The genus *Leptosphaerulina* comprises about six species, most of which lack a conidial stage. The role of the sexual stage in controlling variation is as yet unclear.

When isolated from nature, *P. chartarum* is variable both in morphology and physiology. Additionally, variants arise readily during subculture. Sporidesmin production in particular is a variable character. Most New Zealand isolates produce significant quantities of toxin, but occasional isolates do not produce toxin. In South Africa, both toxin-producers and non-producers are common, and in America most strains are reportedly non-producers.

We are exploring the genetic basis of variation in two ways. First, we are examining whether or not vegetative compatibility (VC) genes exist controlling fusion between different strains. In other filamentous fungi, fusion is controlled by a series of genes which must be identical for fusion to occur. We have selected mutants in some strains and demonstrated heterokaryosis between different mutants derived from the one parent. However, to date we have not demonstrated heterokaryosis between mutants derived from different parents, indicating the existence of VC genes in this species. If VC genes are widespread, this would restrict the possible interchange of genetic material between toxin-producers and non-producers.

Second, we are examining the genetic differences between isolates using the RAPD (random amplification of polymorphic DNA) technique. Results to date indicate that toxin-producers from New Zealand strains differ genetically from non-producers from South Africa and America.

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