Plant Pathology (2002) **51**, 13–23

The recent history of *Puccinia striiformis* f.sp. *tritici* in Denmark as revealed by disease incidence and AFLP markers

A. F. Justesen[a], C. J. Ridout[b] and M. S. Hovmøller[a]*†

[a]*Department of Crop Protection, Danish Institute of Agricultural Sciences, Research Centre Flakkebjerg, DK-4200 Slagelse, Denmark; and* [b]*Disease and Stress Biology Department and Sainsbury Laboratory, John Innes Centre, Norwich Research Park, Colney, Norwich, NR4 7UH, UK*

Disease observations and amplified fragment length polymorphism (AFLP) markers were used to study recent developments in the *Puccinia striiformis* f.sp. *tritici* population in Denmark. The fungus appeared spontaneously at 10 locations in Denmark in 1997 after it was not observed under natural conditions in 1996. The pattern of disease development and prevailing winds suggested that the fungus reappeared by airborne spores from the south or west. In 1998, disease incidence was more evenly distributed throughout the country. Forty-eight single lesion isolates were collected from most crops where the disease was observed in these years; all except one from 1997 belonged to two pathotypes that were not previously detected in the country, and both possessed the newly discovered *Yr17* virulence. The isolates were characterized with AFLP markers together with 28 isolates representing eight of 13 pathotypes observed prior to 1996. Initial screening of 240 *Pst*I/*Mse*I AFLP primer combinations on four isolates showed that a primer combination, on average, revealed 0·4 polymorphisms between any isolate pair. A selection of 21 primer combinations resulted in 28 AFLP markers, which revealed 16 AFLP phenotypes among all 76 isolates. The two *Yr17*-virulent pathotypes consisted of three AFLP phenotypes, which were observed in both 1997 and 1998; the two most frequent AFLP phenotypes occurred at most sampling locations and often within the same crop. AFLP diversity was larger among samples collected prior to 1996, and also in this period most AFLP phenotypes were observed at different sampling locations. These results are consistent with the features of an entirely asexually reproducing pathogen dispersed by aerial spores across large areas.

Keywords: DNA fingerprinting, molecular variation, stripe rust, virulence, wheat yellow rust, *Yr17* resistance

Introduction

Wheat yellow rust, or stripe rust, caused by the biotrophic fungus *Puccinia striiformis* f.sp. *tritici*, is a common disease in many areas around the world. The most severe epidemics are often observed in cool maritime or high altitude climates (Zadoks, 1961), but the fungus is also well adapted in countries where wheat is grown during relatively mild winter seasons with warm summers (Johnson, 1992). In Denmark, yellow rust occurs naturally in most years, but it may be absent in growing seasons following cold winters (Hovmøller, 2001). The disease spreads by means of urediniospores. A single infecting spore may result in a lesion consisting of numerous pustules on the leaf surface, each harbouring abundant urediniospores. During each generation, lesions may expand to some extent, but disease mostly spreads by wind dispersal of urediniospores to neighbouring leaves and plants, where new infections establish. Within the first few generations (generation time in general varying from 2 to 4 weeks depending on temperature and host cultivar) the disease appears as a focus of heavily diseased plants. A small fraction of urediniospores are dispersed over larger distances, and form new foci and, after some additional generations, the entire crop may be severely infected (Zadoks, 1961).

Pathotype diversity in *P. striiformis* f.sp. *tritici* populations is often low within the same wheat crop and also within different crops of the same cultivar in a certain agricultural area, but it may vary considerably between regions and years (Stubbs, 1985; Vallavieille-Pope & Line, 1990; Bayles *et al*., 2000; Hovmøller, 2001). In this context, pathotype denotes an array of virulence/avirulence alleles matching a set of known host resistance genes according to the pattern expected of gene-for-gene

*To whom correspondence should be addressed.

†E-mail: mogens.hovmoller@agrsci.dk

Accepted 25 July 2001