NOTE FROM THE EDITOR

The following Letters to the Editor by A. P. Roelfs and J. W. Martens and by D. R. Knott were submitted independently at nearly the same time. Since they relate to the same topic but approach it in different ways, they are published together here.

Letter to the Editor

The Virulence Associations in *Puccinia graminis* f. sp. *tritici* in North America

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Accepted for publication 21 May 1984.

In the recent book, *Host-Pathogen Interactions in Plant Disease*, Vanderplank (6, page 11) described two distinct populations of *Puccinia graminis* Pers. f. sp. *tritici* on the North American continent that are sharply separated at the 49th parallel. He indicated that in the United States and Mexico, virulence for gene *Sr*6 is associated with virulence on genotypes carrying *Sr*9d, whereas in Canada it is dissociated from it.

Comprehensive surveys and virulence typing of the cereal rusts are done annually in Canada and the United States and, indeed, the results of surveys in the two countries are not always, or even usually, identical. There are many reasons why the sampled populations would not be entirely homogeneous, even within the Great Plains region of the continent. For example, in 1982 with *P. graminis* f. sp. *avenae*, a large sample size from the United States and Mexico indicated that the population comprised about 90% NA 27 and 10% NA 16 (4). By the time the epidemic had run its continental course the NA 16 had all but disappeared (3). It is widely accepted, however, and the evidence is overwhelming, that the Great Plains population is one epidemiological unit consisting of several asexually reproducing clones. There is little, if any, evidence to suggest that the rust pathogens overwinter in the Great Plains region of Canada where most crops are spring planted; the primary inoculum of the rust pathogen must come from the south each year. There is also evidence that selection pressure on the northern phase of the population can influence the virulence genotype of the southern phase (5).

Therefore, the concept of a sharply distinct population of the pathogen, counting in the hundreds of billions, in Canada is somewhat difficult to grasp. What mechanism could possibly bring about such sudden change on so massive a scale? Initial infections often occur on the same date throughout Minnesota, North Dakota, and adjacent Manitoba. The temperature factor invoked by Vanderplank (6) does not change abruptly at the 49th parallel and could not possibly be the explanation. Although sampling is not simultaneous over the entire area, the time of sampling never differs more than a few days to a week between the northern area of the United States and southern Canada. In our view, *P. graminis* f. sp. *tritici* is incapable of the kind of change that Vanderplank postulated.

The late G. J. Green addressed the problems associated with *Sr*9d in 1981 (2) "Canadian and American results [used in their tests] with *Sr*9d differed and a Canadian genetic study showed that the line with *Sr*9d from H-44-24 was resistant to several races that were virulent on wheat lines with *Sr*9d from Hope [used in the United States]. The extra resistance of the line with *Sr*9d from H-44-24 and of H-44-24 was conferred by a resistance gene not present in Hope. The new gene was tentatively called gene H. [Race] formulas were changed to agree with the reactions of the line with *Sr*9d from Hope." The ISr9d-Ra line used in the United States surveys was derived from Hope and did not include *Sr*H.

When the virulence formulae for Canadian races C25, C35, C41, C57, C58, and C63 (2) are corrected for the presence of *Sr*H, all are virulent on both *Sr*6 and *Sr*9d, and the case for disassociation in Canada disappears.

Vanderplank's (6) concept of matching genes in the same chapter may have some merit. In the case of the *Avena-P. graminis* system in which few resistance genes are involved and the genotypes in the host population on the continent are well known, helpful inference might be drawn. The basis for the conclusions about *Triticum-P. graminis* in this chapter are, however, very tenuous, and the analytical approach simplistic. The *Sr*6-*Sr*9d match for durability is simply not valid and the chapter itself contains evidence against the *Sr*6-*Sr*7d and the *Sr*6-Sr15 matches. Any analysis of this type must take into account the very complex resistance genotype of the host population on which these races have evolved.

Associations of patterns of virulence, avirulence, or virulence and virulence may exist due to the predominantly asexual reproduction of *P. graminis* in North America. Asexual reproduction results in a complete linkage (association) of the entire pathogen genome. These associations may last for long periods of time or may even reverse themselves (1). In the North American populations of the cereal rust pathogens, most of the phenotypes that occur at low frequencies are lost during periods of low population numbers (eg, periods when host tissue is limited or when environmental conditions are very unfavorable). For rare mutant phenotypes to survive to the next season, they must be strongly selected for, or if they are selectively neutral they must occur in a genome that has a selective advantage. Thus, when considering effects of selection on an asexual population it is often necessary to consider the effect of the entire genome and not only a few selected genes.

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