# Estimated Relation Between Numbers of Urediniospores of *Puccinia graminis* f. sp. *tritici* and Rates of Occurrence of Virulence

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No significant stem rust epidemic has occurred in the spring wheat region of North America since 1955 (17). Control of stem rust (causal agent: *Puccinia graminis* Pers. f. sp. *tritici*) in this region appears to be due to a combination of barberry eradication (18) and host plant resistance (6). In spite of this 29 yr of continuous successful control of stem rust, "specific" or "vertical" resistance to plant rusts has a generally poor reputation for its durability. Limited effort has been made to explain this success of apparently race-specific resistance. Watson (26) reported that effective stem rust resistance depends on multiple genes for specific resistance. Vanderplank (23,24) stated that certain combinations of resistance genes may provide durability. Green and Campbell (6) suggested that both the larger number and certain combinations of resistance genes provided enduring resistance. A Hegelian change, passing of a threshold into a new magnitude of resistance, was proposed by Robinson (16), but without explanation of a mechanistic basis. Johnson (8,9) indicated that unidentified resistance genes might be present.

The stem rust resistance of recent North American spring wheat cultivars has lasted much longer than that of earlier resistant cultivars such as Kanred and Ceres (21). One obvious difference is that single resistance genes, for which virulence in the pathogen was already recognized, were added in those early cutlivars, whereas current cultivars have up to six pairs of resistance genes (6,9,12,26). Nevertheless, some cultivars with multiple resistance genes have not been as durable (8,24). The purpose of this letter is to suggest one reason why some cultivars with multiple genes for stem rust resistance could maintain that resistance over a longer period.

Dyck and Kerber (4) recognized that rate of mutation to virulence and size of rust populations influence the duration of effectiveness of genetically complex resistance. Using calculations from estimates of population size, mutation rates, and relative fitness, Person et al (15) proposed that single- and double-mutant pathogenic genotypes would likely be maintained in asexual or sexual pathogen populations. We believe that using the available, published information to quantify this relationship further would provide insights useful to the development of rust control strategies.

**Pathogen populations.** The pathogen population in this region is now asexually reproducing, so the effect of sexual recombination can be ignored. We derived an estimate of 634,000 spores produced per uredinium over a 26-day season on adult wheat plants from the data of Broyles (2), Mont (13), and Rowell and Roelfs (20). Kingsolver et al (10) equated 10 uredinia per wheat tiller to a 1% rust severity estimate. For an average 10% rust severity through a 26-day sporulating season, approximately $6.3 \times 10^7$ spores are produced per tiller, with about $3.7 \times 10^6$ tillers per hectare (ha) (19,20). Some 32,783,940 ha (80,948,000 acres) of wheat were harvested in the United States in 1981 (22). Assuming a 10% stem rust severity for 26 days during the season for the entire wheat crop, there would be about $7.5 \times 10^{21}$ urediniospores produced ($2.3 \times 10^{14}$ per ha). This level of severity would approximate the mean nationwide severity a week before maturity for the worst historic North American epidemic.

**Mutation rates.** Rates of mutation to virulence at various loci in *P. graminis* f. sp. *tritici* were reported to differ, ranging from those that mutate readily in the field to those that are not known to have mutated (11). However, actual mutation rates have seldom been calculated. With three isolates of *P. graminis* f. sp. *tritici*, Watson (25) found an average of one mutation for greater virulence (probably to the *Sr*11 resistance gene) on about 4,000 seedling plants. There were about 30 infections per plant in these tests (Watson, *personal communication*). We derived an estimated mutation rate of $8.3 \times 10^{-6}$ per uredinial generation from these data.

Such a rate of mutation is believed to be indicative of a population in which the fungal locus was heterozygous for dominant avirulence. If avirulence was homozygous dominant, mutations would be needed at both alleles of the locus. The calculated rate would be $6.9 \times 10^{-11}$ for the comparable homozygous locus, for a much decreased occurrence of variants. If avirulence was homozygous recessive, rates of production of virulent mutants would be twice those from heterozygous dominant loci, $1.7 \times 10^{-5}$, because an effective mutation could occur at either allele. The effect would be relatively minor in the context of other sources of variation.

**Equilibrium frequency.** Mutations may accumulate in a population until an equilibrium is reached. This frequency is approximately equal to the mutation rate divided by the selection coefficient (15), at least for lower levels of frequency. Grant and Archer (5) recently estimated the selection coefficient for virulence to *Sr*6 as from 4.00 to 5.24% less fit than the avirulent counterpart. We are concerned here with virulences that have not previously built up to major elements of the population, nor for which there has previously been selective pressure by cultivar resistance. Thus, these are rare specific virulences in the base population (7). Applying the average selection coefficient (5) to the effective mutation rate calculated from Watson's data (25) provides an equilibrium frequency of $1.8 \times 10^{-4}$.

**Single-gene resistance.** At this frequency, a single hectare of susceptible wheat with 10% stem rust for a 26-day period (one season), would produce about $4.2 \times 10^{10}$ mutant spores virulent to a monogenic resistance, for an average of over 11,000 such spores per tiller. If this mutation rate and selection coefficient are representative of many loci for virulence in *P. graminis* f. sp. *tritici*, it is understandable that corresponding single resistance genes have not provided durable resistance. Day (3) proposed that natural mutation rates are sufficient to generate mutants virulent on any nearby host protected by a single resistance gene.

**Multiple-gene resistance.** Hosts protected by two or even three genes are suggested to delay such occurrence, since virulent mutants that arise independently must now be combined, or else they must arise simultaneously or sequentially in the same line before infection can occur (3). For several virulence loci each with a frequency of $1.8 \times 10^{-4}$ for mutations in the pathogen population, occurrence of mutations together at two, three, four, or five loci would happen at frequencies of $3.2 \times 10^{-8}$, $5.8 \times 10^{-12}$, $1.0 \times 10^{-15}$,

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and $1.9 \times 10^{-19}$, respectively. At 10% infection for 26 days, these frequencies would provide about $7.4 \times 10^{6}$, $1.3 \times 10^{3}$, $2.3 \times 10^{-1}$, and $4.4 \times 10^{-5}$ spores with two, three, four, or five combined mutations, respectively, per hectare of susceptible wheat.

To overcome six host resistance gene pairs, the combined frequency of $(1.8 \times 1.0^{-4})^{6}$ would be $3.4 \times 10^{-23}$. This would require about four times the annual U.S. wheat area to provide a single virulent spore under the conditions outlined here. The occurrence or availability of such a spore to infect such a resistant cultivar appears unlikely in the absence of large intermediate or preexisting populations possessing one or more of the genes for virulence. The population available would not be cumulative between years as in North America it is drastically reduced each winter and early spring. Wheeler and Diachun (27) suggested that a cultivar with four or five resistance genes which conditioned resistance to all races might remain resistant for years or perhaps centuries if the gene-for-gene concept is valid.

**Other epidemiological factors.** Not all spores are viable or land on a host. Environmental conditions favorable for infection seldom occur each day. Not all infections produce uredinia (28). Thus, effective population size (inoculum potential) and functioning mutants may be reduced by 10- to 100-fold. Because of the annual cyclic development and reduction of the pathogen populations, it also seems unlikely that the equilibrium frequency calculated here could be reached. As a result, the figures that were used appear to be generous in representing the epidemiological capacity of the fungus as were several of the initial assumptions, such as 10% infection on the total crop for a 26-day period and choice of an apparently high mutation rate. Thus, fewer than six resistance genes might be effective in practice, if they were not also used separately in cultivars or multilines (4,14) in source areas for rust inoculum.

**Conclusions.** Although this discussion is based on calculations from very limited available information, it illustrates relations between the size of a pathogen population and its estimated capacity for variation within the constraints imposed by a genetically manipulated agricultural host crop. The vulnerability of certain single host gene pairs to the occurrence of pathogen virulence is evident in contrast to the possible durability of such resistance when combined with a few additional functioning resistance gene pairs. This phenomenon may provide one possible retrospective explanation for the durability of stem rust resistance of the North American spring wheats known to possess as many as six resistance gene pairs.

Differences in the durability of resistance to the various cereal rusts appear explainable by a combination of the differences in pathogen mutation rates (1,11,28), selection coefficients, population sizes, cyclic fluctuations in population size, and sexual and asexual recombination, as well as the numbers of initially effective resistance gene pairs incorporated into commercial cultivars. Research on mutation rates at virulence loci, selection coefficients, and annual minimum populations is needed to obtain more precise predictions of the durability of various corresponding resistance genes and their combinations (15).

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