THE LATE BLIGHT SITUATION IN THE USA

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The late blight situation in the USA is changing rapidly. In order to place these changes in perspective, some historical considerations are necessary. Potatoes and *Phytophthora infestans* probably were introduced to each other in the 1840s. Late blight appeared in the northeastern USA as early as 1842, and it was detected in Europe in 1845 where the damage it caused led to the Irish Potato Famine. Prior to the 1840s the fungus was apparently restricted to central Mexico. The mechanism by which the fungus was introduced to the USA and Europe is not known. Subsequently, the fungus was distributed with tubers to most locations where potatoes were grown. Apparently, worldwide populations were quite homogeneous and most were dominated by the same clonal lineage (all A1 mating type, so populations were asexual) (3).

Changes in Europe

Things changed dramatically in Europe during the 1980s. In 1980, resistance to Ridomil was detected. Resistance was unexpected and caught growers and scientists unawares, and there were serious control failures. Resistance was so serious that Ridomil was removed from several western European markets for several years. In 1984 there were reports that the second mating type of *P. infestans* (A2) was present in Europe. (There are only two mating types, A1 and A2.) Subsequently, A2 mating types were detected throughout Europe, although typically in frequencies less that 15%. A2 mating types were then detected throughout the world (3). It became clear that there had been a major migration into Europe, and that the previous population was being replaced by a new immigrant population that was genetically diverse and contained both mating types (A1 and A2) (3,5).

The occurrence of both mating types was significant. Sexual reproduction was now possible, whereas previously only asexual reproduction (via sporangia) was possible. When restricted to asexual reproduction, the fungus is essentially an obligate parasiste and can only exist with a living host (living foliage, stems, tubers). However, sexual reproduction leads to an oospore that not only produces recombinant individuals, but also serves as a survival structure. Oospores can survive extremes of temperature and moisture in the absence of a living host. Thus, oospores enable the fungus to survive in soil and plant debris in the absence of a living host for extended periods (months and years?). It now appears that sexual reproduction is occurring in Europe (6), and may have contributed to the relatively more severe disease there during the previous decade.

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Changes in the USA and Canada

Changes are now occurring in *P. infestans* populations in the USA and Canada. The first suggestions of changes were indicated by Deahl et al (1), with the report of an A2 individual detected in Pennsylvania in 1987, and an A2 individual in British Columbia in 1989. Subsequently, populations of the fungus were shown to be resistant to Ridomil in western Washington (2). It is now clear that there are several widely distributed immigrant forms of the fungus in the USA. We had detected these same forms in northwest Mexico prior to detecting them here (4), so the source of recent strains seems likely to be northwest Mexico. The three forms with the widest distributions are all resistant to Ridomil (Table 1); two are A2 and one is A1. Two of them are especially pathogenic on potatoes and tomatoes, whereas one is pathogenic primarily potatoes. Tomato pathogenicity adds a new dimension to late blight management, because tomatoes may now serve as a source of isolates especially pathogenic to potatoes. In addition to the three widely distributed recent immigrant forms, the previous forms are also widely distributed. One of these forms still dominates populations in the middle part of North America (Table 1). This form (US-1) is still sensitive to Ridomil.

Table 1. Characteristics of *Phytophthora infestans* isolates from the USA and Canada in 1992\93 (data of S. B. Goodwin, et al, unpublished).

Genotype name	Mating Type	Allozyme Genotype		Ridomil Reaction	Location (and sample size)
		Gpi	*Pep*		
US-1	A1	86/100	92/100	S	AL, FL, ME, MN, NJ, NY, ND, OR, PEI, WA, WI(100)
US-6	A1	100/100	92/100	R	BC, FL, OR, WA (33)
US-7	A2	100/111	100/100	R	CA, FL, KY, ME, MI, NC, NY, TN, WI (129)
US-8	A2	100/111/122	100/100	R	ME, NY (54)
US-9	A1	100/100	83/100	-	ID (1)
BC-1	A2	100/111	100/100	-	BC (1)
BC-2	A2	100/100	100/100	-	BC (1)
BC-3	A2	100/100	100/100	-	BC (1)
BC-4	A2	100/100	100/100	-	BC (1)
--	A2	111/122	100/100	R	WI (1)

Until late in 1993, Wisconsin populations of *P. infestans* were composed only of the historical forms (A1 and sensitive to Ridomil). However, two individuals collected in late 1993 were found to be recent immigrants. One was US-7 (A2 and resistant to Ridomil, Table 1), which is widely distributed. The other, however, was a rare individual that hasn't been widely distributed north of Mexico. Thus, if weather is again favorable to late blight in 1994, there could be problems associated with the new

immigrant forms.

Management of the new strains

The immigrant strains pose new challenges in late blight management. They differ from the previous population in several respects. They have a broader host range (tomatoes as well as potatoes), so that disease on one crop may serve as a source of inoculum for the other. They are largely resistant to Ridomil, and we need to manage this resistance knowledgeably. Although Ridomil alone has no effect on resistant forms, it appears to have some effect when applied in combination with a protectant. Individual growers need to determine whether the effect is sufficiently large to justify additional cost. The immigrant strains may be more aggressive (cause more severe disease) than the previous forms. Although this possibility is not yet confirmed, the implication is that growers will have greater need for resistant varieties, and will have greater need for scouting information, so that small focal epidemics aren't allowed to get out of control. The new immigrants may interact with other strains to produce oospores that might change the epidemiology of the disease in the USA and Canada. Oospores would enable the fungus to overwinter in soil or plant debris -- two potential locations where the previous population did not overwinter. It's even possible that survival might be for longer than one year. Additionally, sexual reproduction might enable the occurrence of especially troublesome traits in background genotypes that are sufficiently fit to survive in our agroecosystem; thus fungicide resistance, or other pathogen variation might become even more troublesome.

Many of the solutions to the potential problems are not yet known. It will be some years before we are fully aware of the implications of the population changes. During the interim, we will be learning about the population changes, and about how to deal with them. At this time the most important point is that these changes are happening, and that they could cause additional problems in late blight management. Thus, during the next several years, growers should approach late blight management with additional caution.

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