DOWNY MILDEW (PLASMOPARA) OF SUNFLOWERS: A POLICY FOR SEED IMPORTATION AND PLANT BREEDING

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Sunflowers (*Helianthus annuus* L.) have been grown in Spain on a small scale for confectionery use for many years. Small-seeded high oil varieties originating in the U.S.S.R. were introduced in 1963 on about 500 hectares. The area sown has expanded from about 5,000 ha in 1969 to about 370,000 ha in 1973, practically all of it Russian varieties grown from imported seed, much of it from France.

Various diseases, and pests such as insects and birds, have been recorded in Spain, but none of them has yet become a major problem. In 1972, downy mildew (*Plasmopara halstedii* (Farl.) Berl. et de Toni) was recognized for the first time (R. Jimenez-Diaz, unpublished) in Spain. This disease has been a limiting factor to sunflower production in many countries (Cohen and Sackston 1973; Leppik 1962; Zimmer 1972). Its occurrence at a number of locations in the provinces of Córdoba and Sevilla in 1972, and apparent rapid spread in Andalucia in 1973, indicate that it may become a limiting factor in Spain, as elsewhere.

As sunflowers have a fairly low value per unit area of land, it is not economically feasible to apply expensive methods for control of their diseases and pests. The only disease controls which farmers can easily and profitably use on a large scale for such low unit-value crops are modified cultural practices, including crop rotations; seed treatments; and sowing resistant varieties.

Resistance to downy mildew has been found in sunflower lines originating in Canada and the United States, by investigators in North America, France, and Romania (Goossen and Sackston 1968; Leclercq et al 1970; Vranceanu and Stoenescu 1970; Zimmer and Kinman 1972).

Work in France and the United States (Vear and Leclercq 1971; Zimmer and Kinman 1972) indicated that there are at least three different genes for resistance to downy mildew, and that these

are inherited independently. All three were effective against the collections of mildew studied in France (Vear and Leclercq 1971) Only one of the three genes was effective against the Red River race of mildew (Zimmer and Kinnman 1972).

Evidence obtained by North American investigators (Zimmer 1972; Zimmer and Kinman 1972) indicated that there are at least two races or race groups of the mildew pathogen. As far as is known at present, the mildew occurring throughout most of Europe seems to belong to one race or race group, whereas that found in the Red River Valley of the north central United States, belongs to another race or race group.

Sunflowers are native to North America, and were taken from there to Europe by early conquerors and explorers (Heiser 1951). As downy mildew of sunflowers is also a native of North America, circumstantial evidence indicated that it attained its present world wide distribution with sunflower seed (Delanoe 1972; Leppik 1962; Novotelnova 1963; Young and Morris 1972; Zimmer 1972). Because only a very small proportion, usually less than 1:1000, of plants grown from infected seed develop typical symptoms of systemic infection, the significance of seed dissemination of the pathogen has not always been realized (Delanoe 1972; Novotelnova 1963).

It has been shown that symptomless plants from infected seeds may carry latent infection, and that the fungus may sporulate on their roots (Novotelnova 1963; Cohen and Sackston 1974). Typical systemic symptoms may develop on plants of a subsequent crop grown in soil infested in this way. Spores produced on the rare plants with systemic symptoms from infected seeds may infect other plants in the field. Although these usually remain free of systemic symptoms, the fungus may sporulate on their roots, providing inoculum for future crops, and may also infect the seeds, giving rise to a new generation of symptomless plants with latent infection, to infect still other fields (Cohen and Sackston 1974).

Apparently because of its insidious type of dissemination by seed, downy mildew has spread unchecked from its native North America to most areas where sunflowers are grown commercially, and now including Spain. The race or race group of mildew introduced into Europe in this way has caused serious losses in sunflower yields, but resistance to it is being incorporated into sunflower varieties and hybrids in various countries. If the Red River race of mildew were to be introduced into Europe two of three factors for resistance would be ineffective against it.

Secondary infection on sunflowers by mildew spores has been documented (Cohen and Sackston 1973; Novotelnova 1966; Zimmer 1972). Zoosporangia have been shown to remain viable after being borne short distances by wind (Delanoe 1972), but they are delicate, and it is not probable that they could survive long distance aerial dissemination. The most obvious danger is that the Red River race, or others of similar or extended pathogenicity, may be introduced with sunflower seed.

To prevent, or at least delay, the introduction of such dangerous races, it is necessary to prohibit the introduction of commercial quantities of sunflower seed from any area where such a race is known to occur. Even small lots of fifty or a hundred seeds of experimental lines or varieties from potentially dangerous areas should be imported only if they can be grown to maturity under very careful supervision by competent pathologists in quarantine greenhouses or growth rooms, as recommended by Leppik (1962). Any diseased plants should be removed immediately, and if mildew occurs on them, differential varieties should be inocluated to determine if it is a dangerous new race. Seed harvested from the healthy plants should be sown for a second generation, again under supervision in an isolation greenhouse. Only if this second generation remains free of disease should the seed from it be released for growing in the field by plant breeders or others.

Successful exclusion of the Red River race of mildew from Europe or other regions where it is not yet known, does not guarantee that this or similarly dangerous races will not occur here. The mildew pathogen goes through a sexual cycle every season. It is therefore possible, and in fact probable, that new pathogenic races may arise by mutation and recombination, as they have done in many other plant pathogens. Until such new races occur, European sunflower breeders have at least three different genes for mildew resistance available to them.

If sunflower varieties with only one gene for resistance are released and are widely grown, mutation on only one locus in the mildew pathogen may enable it to overacome this resistance. If new varieties have two different effective genes for mildew resistance, the appropriate mutation would have to occur at two different loci controlling pathogenicity in the fungus before it could attack such varieties. The probability of such simultaneous mutation is very much less than of a single mutation. The usefulness of varieties with two different resistance genes would therefore probably last for many more years than varieties with only one resistance gene.

American plant breeders no longer have this possibility, as only one of the three genes for resistance to mildew originating in North American sunflower lines is of any use against the Red River race (Zimmer and Kinman 1972). Vear and Leclercq (1971) pointed out the wisdom of using two or three different genes in producing mildew-resistant hybrids. Other European plant breeders should take this same approach. To support their efforts, the appropriate authorities in each country should license for distribution only those mildew-resistant varieties or hybrids with at least two different effective genes.

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