Waldemar E. Sackston,
Fernando Romero-Muñoz,
Carlos Garcia-Baudin,
Spain

MILDEW RESISTANT SUNFLOWERS AND THEIR ROLE IN EPIDEMIOLOGY OF DOWNY MILDEW

Downy mildew (Plasmopara halstedii Berl. et de Toni) of sunflower (Helianthus annuus L.) was first recognized in the south of Spain in 1972 (R. Jimener-Diar, 1973). Although commercial production of oilseed sunflower began recently in Spain, large-seeded types for confectionery use have been grown for many years (R. Lopez de Haro, 1975). It is probable that downy mildew was introduced many years ago but was not noticed in the small-scale production of the old varieties. Judging by its distribution in 1972, mildew had been present in the south of Spain for at least two or three years before being recognized. The pathogen originated in North America, the centre of sunflower evolution, and spread from there with the seed (E.E. Leppik, 1962). Its ability to persist in "latent" infections in symptomless plants from infected seed for one and sometimes two generations, meanwhile infesting the soil with oospores produced on and in the roots, made it difficult to recognize and prevent this seed-borne dissemination to most countries where sunflower is grown (V. Cohen, W.E. Sackston, 1974; D. Delanoe, 1972; N.S. Novotelnova, 1963; O.I. Tikhonov, 1973). The disease is of major importance in many countries where temperature and moisture conditions are favourable, and has become a limiting factor in some areas.

The only effective control is the breeding of mildew-resistant varieties and hybrids. Plant breeders in most sunflower-growing countries are using sources of mildew resistance derived

from wild annual sunflower species from North America, and in a few instances, from interspecific crosses with H. tuberosus L. (P. Leclercq, V. Cauderon, M. Dauge, 1970; E.V. Pustovoit, I.A. Gubin, 1975; V. Vranceanu, F. Stoenescu, 1970; D.E. Zimmer, M.L. Kinman, 1972). At least three, and probably four genes have been shown to govern resistance, which appears to be inherited as a simple dominant factor (F. Vear, 1975; F. Vear, P. Leclercq, 1971; D.E. Zimmer, G.N. Fick, 1975). "Horizontal" or non-specific resistance may be present in some selections (F. Vear, 1975). It would be highly desirable to incorporate such resistance, in view of the occurrence of the "Red River" race of the pathogen in North America, which attacks two of the resistance genes effective in Europe (D.E. Zimmer, G.N. Fick, 1975), the apparent appearance of a similar race in the south of the USSR (B.K. Pogorletsky, E.E. Geshele, 1975), and its probable future appearance elsewhere (W.E. Sackston, 1975). Incorporation of two or more distinct and previously unused resistance genes in any new variety of hybrid should reduce greatly the probability of new, more virulent races arising by mutation, and should thus extend the useful "life" of such resistant varieties (W.E. Sackston, 1975; F. Vear, 1975).

The most resistant lines and hybrids produced are not immune from infection, although the term "immunity" is used in some countries to signify resistance. Mycelium of the pathogen was observed in inoculated seedlings of resistant lines in the lower hypocotyl (D. Delanoe, 1972), and as high as the cotyledonary node (F. Montes, W.E. Sackston, 1975). Seedlings in which the pathogen sporulates to a limited extent on the cotyledons, but not on the true leaves, may be classified as resistant in genetic studies (Vear, personal communication). Oospores of the fungus are formed in the basal tissues of inoculated resistant as well as sus-

ceptible seedlings within six days (Sackston, unpublished).

Although sporulation of the pathogen on and within resistant seedlings is less than in susceptible plants, it may serve to maintain and disperse inoculum in the field and in the soil. This inoculum may be particularly dangerous. As it is produced on resistant plants, a mutant for pathogenicity at the appropriate loci would be ideally situated to infect more tissues and to spread. This makes it even more important that any sunflower varieties or hybrids released for widespread sowing, possess at least two genes for resistance not previously widely used in the area. The identification and incorporation of genes for generalized or "horizontal" resistance would provide additional assurance that the mildew resistance in such varieties would remain effective for a longer period of years.

It would also be extremely useful to be able to protect sunflower from infection, or to destroy the pathogen in the host after infection occurred, by treating with fungicides. Given the relatively low value per unit area of field crops such as sunflower, the ideal treatment would be one applied to the seed. It should be effective against inoculum on and in the seed, should be systemic, and should be effective against subsequent infections at least until the seedlings reach the six leaf stage.

Various standard seed treatment fungicides have been tried at Córdoba, and in earlier work in Canada (Sackston, unpublished). None gave significant protection against soil-borne inoculum, or against later secondary infection. Several systemic fungicides submitted for test by chemical companies, and applied as seed treatments, also failed to protect the seedlings against subsequent infection by the pathogen.

Some of our current work with new fungicides is giving interesting results. Two systemic fungicides, Nurelle and Prothiocarb, which proved ineffective when applied as seed treat-

ments, appeared to eradicate the pathogen in inoculated pre-germinated seedlings if they were applied within four days after inoculation. Nurel-e, a product of Dow Chemical, is 2-chloro-6 methoxy-4-(trichloromethyl) pyridine. Prothiocarb, produced by Schering AG, is 1-(prophyldimethyl amino), 2-ethyl-thiocarbamate. Sporulation was observed on 25% of seedlings treated with Prothiocarb on the 5th day after inoculation, on 50% of seedlings treated on the 6th day, and on 95% of seedlings treated on the 7th day after inoculation. Sporulation was observed on 5 to 10% of the seedlings treated with Nurelle 5 to 7 days after inoculation.

We are now investigating the method of action of these fungicides, the duration of any possible protective effect, the concentration required to control the pathogen, phytotoxicity, etc. As far as we are aware, this is the first report of control of Plasmopara in infected sunflowers by a systemic fungicide. Although it may not prove feasible to control the disease in the field with either of these products, the fact that two dissimilar chemicals are effective against it indicates that products combining the necessary characteristics may be discovered in the near future.

References

1. Cohen, Y. and W.E. Sackston. 1974. Seed infection and latent infection of sunflowers by Plasmopara halstedii, Can. J. Bot. 52: 231-238.
2. Delanoe, D. 1972. Biologie et epidemiologie du mildiou du tournesol. Informations Techniques C.E.T.I.O.M., 29 (4): 1-49.
3. Jiménez-Diaz, R. 1973. Notas sobre la presencia en España del mildiu del girasol (Plasmopara halstedii (Farlow) Berl. et de Toni). An. I.N.I.A. Ser. Prot. Veg. n.3: 95-105.
4. Leclercq, P., Y. Cauderon and M. Dauge. 1970. Selection pour la résistance au mildiou de tournesol à partir d'hybrides topinambour x tournesol. Ann. Amélior. Plantes 20: 363-373.
5. Leppik, E.E. 1962. Distribution of downy mildew and some other seed-borne pathogens on sunflowers. F.A.O. Plant Prot. Bull. 10: 126-129.
6. Lopez de Haro, R. 1975. Sunflower seed production development in Spain. Proc. 6th Internat. Sunflower Conf., Bucharest, Romania, 1974: 439-442.
7. Montes, F. and W.E. Sackston. 1975. Growth of Plasmopara within susceptible and resistant sunflowers. Proc. 6th Internat. Sunflower Conf., Bucharest, Romania, 1974: 623-629.
8. Novotelnova, N.S. 1963. Nature of parasitism of the pathogen of downy mildew of sunflowers at seed infection. (In Russian). Bot. Zh. 48: 845-860.
9. Pogorletsky, B.K. and E.E. Geshele. 1975. On inheritance of immunity to downy mildew in sunflower. (In Russian). Genetica 11(3): 21-28.
10. Pustovoit, G.V. and I.A. Gubin. 1975. Results and prospects in sunflower breeding for group immunity by using the interspecific hybridization method. Proc. 6th Internat. Sunflower Conf., Bucharest, Romania, 1974: 373-381

11. Sackston, W.E. 1975. Downy mildew (Plasmopara) of sunflowers: a policy for seed importation and plant breeding. Proc. 6th Internat. Sunflower Conf., Bucharest, Romania, 1974: 619-622.
12. Tikhonov, O.I. 1973. Peculiarities of infection and forms of sunflower mildew. Proc. 5th Internat. Sunflower Conf., Clermont-Ferrand, France, 1972: 156-158.
13. Vear, F. 1975. Studies on resistance to downy mildew in sunflowers. Proc. 6th Internat. Sunflower Conf., Bucharest, Romania, 1974: 297-302.
14. Vear, F. and P. Leclercq. 1971. Deux nouveaux gènes de résistance au mildiou du tournesol. Ann. Amélior. Plantes 21: 251-255.
15. Vranceanu, V. and F. Stoenescu. 1970 Immunity to sunflower downy mildew due to a single dominant gene. (In Romanian). Probleme agric., 22: 34-40.
16. Zimmer, D.E. and G.N. Fick. 1975. Some diseases of sunflowers in the United States - their occurrence, biology, and control. Proc. 6th Internat. Sunflower Conf., Bucharest, Romania, 1974: 673-680.
17. Zimmer, D.E. and M.L. Kinman. 1972. Downy mildew resistance in sunflower and its inheritance. Crop Sci. 12: 749-751.