MAIN ADDRESS

MANAGING THE MAJOR SUNFLOWER DISEASES: FROM CULTURAL PRACTICES TO BREEDING FOR RESISTANCE

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SUMMARY

Sunflowers, native to North America, were first developed as a crop in eastern Europe. Their putative parents and closely related species still occur wild in North America. Pathogens of most major sunflower diseases, some of them also native to North America, now occur almost worldwide with the crop, wherever climatic conditions and soil types are favorable. Cultural practices such as crop rotation, and in some instances choosing the sowing date to avoid conditions favoring specific diseases, for many years were the only practical control measures. The first real success in managing a major sunflower disease was achieved by selection for resistance to broomrape. The first real success in breeding sunflowers for resistance using specific genes for resistance was achieved with rust. At least some level of resistance is now known to exist even for the most generalized pathogens with extremely wide host ranges. Resistance to many of the pathogens is controlled by one or several dominant genes; in other cases it is additive. The pathogens appear to have much the same range of genetic possibilities as the sunflower host, with new races appearing fairly soon after lines or cultivars are released which are resistant to the prevailing pathogen populations. Although some of the diseases can be controlled by using chemicals, such treatments are usually uneconomic. The major exception is seed treatment with a specific systemic fungicide to control downy mildew.

INTRODUCTION

Surviving in spite of poor crops has been a problem for humanity since the beginning of agriculture. It has been recognized at least since the earliest times that promising crops could fail, because of disastrous weather, outbreaks of insects, or mysterious blasts and blights. To avoid such disasters, early farmers sought to buy off malicious deities by ceremonies and sacrifices, and to invoke assistance from benevolent deities by prayers, ceremonies, and sacrifices.

Observant farmers realized that certain problems tended to be absent or minor the first year or two that a particular crop was grown in a given plot or field, but appeared and then worsened if the crop was grown there year after year. This led to the concept of "soil sickness", the search for new land, and to the practice of crop rotation to avoid or lessen losses. With increasing knowledge of the causes of plant disease came new ideas and technologies for managing them. Some proved very effective, at least for a while. The pathogens also proved flexible, in many cases surviving and even thriving after various control measures were generally adopted. Sometimes the concept of malevolent deities seems almost reasonable!

CULTURAL PRACTICES

Crop Rotation

Good cultural practices were the only measures available to control sunflower diseases when I started to work with this new crop in 1948 in Manitoba, Canada. The conspicuous diseases were sclerotinia wilt and stalk rot (*Sclerotinia sclerotiorum* (Lib.) de Bary), rust (*Puccinia helianthi* Schw.), and downy mildew (*Plasmopara halstedii* (Farl.) Berl. & De Toni). Verticillium wilt (*Verticillium dahliae* Kleb.) was discovered soon after. (For descriptions of the diseases discussed see Zimmer and Hoes (1978) and Mihaljcevic et al (1982)). The most obvious practice to recommend was crop rotation.

S. sclerotiorum, P. halstedii, and V. dahliae are soilborne organisms, so the logic of crop rotation is clear. Because S. sclerotiorum has an extremely wide host range, including common weeds, success depends on weed control as well as the length of the rotation. V. dahliae has a more restricted range, but also affects or survives on some weeds. Oospores of P. halstedii are long-lived in soil, so effectiveness of rotations depends on their length. Few sunflower growers are willing or able to follow even the four-year rotation which seems to be helpful in reducing disease severity, let alone the ten-year rotations that were recommended for the huge state and collective farms of the former USSR. The rust pathogen is disseminated over great distances by wind-blown spores, but even with it rotation could be helpful, by reducing the risk of early infection of seedlings by sporidia from overwintered teliospores on overwintered infected debris. Crop rotation may also reduce or delay initial infection of seedlings by spores from debris of the preceding crop infected by various stem and leaf-spotting pathogens.

Timing The Crop To Escape Or Minimize Disease

Weather conditions may be critical for infection by and development of various sunflower pathogens. The stage of the host when the disease occurs may determine its severity and effects on yield and quality of the crop.

In areas with continental climates, long and severe winters, and relatively short frost-free growing seasons, there may be little latitude in choosing date of seeding. Where growing seasons are long, however, or where sunflowers can be grown at almost any time during the year, damage by some diseases may be minimized by appropriate choice of seeding date.

An interesting example is charcoal rot (Macrophomina phaseolina (Tassi) Goidanich) in Spain. This disease is most conspicuous and damaging on plants maturing during hot dry weather, and stressed by drought (Jimenez-Diaz et al 1983). Much of the sunflower area of Spain has a Mediterranean climate, with most of the rain falling during a cool winter, and very little or no rain between May and October. Sowing sunflowers is feasible in

late January or early February in many regions. The plants can use winter rainfall to make their vegetative growth and to bloom, and utilize moisture stored in the soil to fill the seed during early summer before the heat becomes excessive. Where irrigation is available, one well-timed application of water during the critical stage of early flowering may be sufficient to minimize the effects of both drought and charcoal rot.

Other interesting examples are Alternaria leaf blight and stem spot (Alternaria helianthi Tubaki & Nishihara), Sclerotinia stem rot (Sclerotinia minor Jagger), and Sclerotinia head rot (Sclerotinia sclerotiorum). Alternaria blight disease occurs in eastern Australia, but is significant mainly in a part of Queensland. Sunflowers sown in the spring matured in the summer when conditions were optimum for development of the disease. It was suggested that sowing in the summer would let the crops mature in the cooler, drier autumn when conditions are less likely to favor the development of the disease (Allen et al 1982). Kolte and Tewari (1977) (cited in Allen et al 1982) indicated the possibility of decreasing incidence and effect of the disease in India by changing the seasons in which sunflowers were sown and developed.

Stem rot caused by S. minor is a limiting factor in production of sunflower seed in the northern part of Victoria in Australia. Plants sown early (mid October to mid November) had fewer plants infected and gave greater yields than crops sown late (mid December) (Clarke et al 1992).

Headrot caused by S. sclerotiorum resulted in significant yield losses for the first time in northern New South Wales and in Queensland in the 1988/89 season. This was attributed to the adoption of late maturing, higher yielding cultivars, which flowered when weather favored infection. The appropriate sowing date for each cultivar should be planned so that flowering occurs before or after the late summer period when temperatures and wet periods suitable for the pathogen are most likely to occur (Slatter 1992).

Even where climate limits the choice and length of growing season for sunflowers, there may be some possibility of escaping

the most severe damage by disease by varying the date of seeding and choosing cultivars of the most appropriate maturity group. For example, where rust inoculum originates locally from overwintered debris, sowing as early as possible may permit the seeds to fill before rust infection becomes heavy on the upper leaves, which have the greatest influence on seed weight.

DISEASE CONTROL BY CHEMICALS

Ecological considerations nowadays may affect the decision whether or not to use chemicals to prevent or lessen effects of sunflower diseases, but the economic factor has always affected that decision. Sunflowers are a relatively low-value crop. Treatments may be expensive because of the costs of the chemical and its application, therefore may not be justified by the probable increase in yield and/or quality of the crop. Cost may ignored when the crop to be treated is an important seed stock, experimental material, or in other special circumstances.

Protectant Chemicals

Most efforts to control sunflower diseases by chemicals have involved spraying or dusting plants with fungicides to prevent or inhibit spore germination and/or infection of leaves or stems. Rust was reported to be controlled by various protectant chemicals and some systemic fungicides in various countries, in work cited by Kolte (1985). Protectant and systemic fungicides were also shown to be effective against Alternaria blight and Phomopsis brown stem canker (Phomopsis helianthi Munt.-Cvet. et al) by Iliescu and Baicu (1984), in Romania. Sunflower yields in Romania tend to be very high. Under their conditions, well-timed applications of fungicides, integrated with use of appropriate varieties and fertilizers, gave good economic returns. In most other cases, however, even where fairly effective control was achieved, the use of fungicides on sunflowers in the field could not be justified on economic grounds.

Where seed treatment is effective, the situation is different. Even though the chemical may be expensive, the amount required to treat enough seed for a given area of crop is much

less than that required to treat plants in the field, only one application is required, and applying the chemical is much cheaper than field applications. Metalaxyl and other systemic fungicides, effective against Oomycetes when applied to the seed, protect sunflowers against downy mildew not only during emergence of the seedlings, but throughout the period of susceptibility (Melero-Vara et al 1982). Metalaxyl is now used routinely to treat sunflower seed in various countries, particularly since the recent appearance of a whole series of new races of downy mildew (Gulya et al 1991). Such wide use carries with it the danger that the pathogen may develop resistance to the fungicide; resistance to metalaxyl has been discovered in various other pathogens (Cohen and Coffey 1986).

Broomrape is a parasitic higher plant, therefore herbicides used against weeds were tried in efforts to control it (Acimovic 1978; Melero-Vara and Garcia-Baudin 1985). They proved relatively ineffective, or injurious to sunflowers at effective dosages.

SELECTION TO MINIMIZE DISEASE

Sunflower is a cross-pollinated species, with from 700 to 3000 individual flowers per head in oilseed cultivars, and up to 8000 flowers in the nonoilseed cultivars (Knowles 1978), each theoretically capable of pollination from a different source. Variability was consequently great within and between sunflower populations in various localities in Russia, where it was first developed as a crop, and in the seeds brought from there to North America and other areas by immigrants. Russian peasants must have selected for large plants. Some of the early cultivars in North America, growing from two to three metres tall under good conditions, had names such as "Mammoth Russian". An early success in development of sunflowers as an oil crop in North America was the selection in Canada of short cultivars suitable for combine harvesting, starting from lines selected by Mennonite farmers in Saskatchewan, and dwarf lines obtained from Russia (Putt 1978).

Farmers selecting the sources of the next year's seed normally choose the healthiest looking and highest yielding

plants or fields. In some instances at least they may also succeed in selecting for disease resistance.

Broomrape

Broomrape (Orobanche cumana) Wallwr. is a major disease in parts of Europe, the Near East, and China (Acimovic 1984, 1988). Apparently it has not yet been recorded on sunflower in North America, although other species of the parasite occur there on other crops. Broomrape was at one time "the most dangerous enemy of sunflower" in the USSR; there were years when the sunflower crop on tens of thousands of hectares was almost entirely destroyed (V.S.Pustovoit 1967).

Peasant farmers in pre-revolutionary Russia selected local strains of sunflower resistant to broomrape. The best of these strains, up to 80% resistant, were employed in an official program of sunflower breeding started by V.S. Pustovoit in 1912 at Krasnodar, at what became VNIIMK, the All Union Institute for Oil Crops Research. By 1927 Pustovoit had selected strains with 97 to 99% of the plants resistant to both broomrape and head moth, a major insect pest. By that year, however, previously resistant sunflowers were succumbing to what turned out to be a complex of new races of broomrape. Pustovoit's labor-intensive version of recurrent selection produced lines resistant to the new race complex (Pustovoit 1967).

Verticillium wilt

Although I have not encountered any record of deliberate early selection for resistance to Verticillium wilt (Verticillium dahliae Kleb.) in Russia, some resistant materials must have been selected and incorporated into later breeding programs. The first Russian high-oil-content cultivar released internationally, Peredovik, showed considerable field resistance to Verticillium wilt in North America, although older Russian lines were susceptible.

BREEDING FOR DISEASE RESISTANCE

Selection within available populations may serve to improve specific characteristics, but it is usually a slow process. To

introduce new characters such as resistance to various diseases into adapted cultivars it is necessary to make crosses with appropriate sources of resistance.

Rust

Workers in various countries had reported differences in rust (Puccinia helianthi Schw.) reaction in sunflowers, but the first rust resistant variety to be released to growers anywhere in the world apparently was in 1954 in Manitoba, where it was widely grown in 1955 (Putt and Sackston 1957).

The Canadian rust resistant material arose accidentally. One resistant plant was found in 1949, another in 1950, in experimental plots at Altona and at Morden, Manitoba, grown from seed of Canadian lines increased during a winter generation near Renner, Texas. Characteristics of the resistant plants were typical of the wild H. annuus form found near Renner. Five additional resistant wild-type plants were observed in 1952 and 1953, all grown from seed produced in the Renner area (Putt and Sackston 1957). The original resistant plants in the various accessions were open pollinated; self pollination to develop inbred rust resistant lines was begun in subsequent generations, and these were used at Morden in breeding resistant cultivars, and in genetic studies (Putt and Sackston 1963).

Some seedlings from progenies of the various resistant plants proved resistant when inoculated with rust in the greenhouse at Winnipeg (Putt and Sackston 1957). This first demonstration of seedling resistance in cultivated H. annuus made it possible to distinguish pathogenically distinct rust cultures among collections from various sources (Sackston 1962). These distinct cultures made it possible to identify two non-allelic dominant genes for resistance, R 1 and R 2, which occurred singly in some lines and together in others (Putt and Sackston 1963).

All collections of rust from various sources fell into the four race groups (hereafter called "races") which could be distinguished by the two genes. All four races were identified from sunflower fields and experimental plots before cultivars carrying any of the resistance genes were grown to any extent. It

was therefore expected that such races would increase rapidly in the rust population soon after resistant sunflowers were widely grown (Sackston 1962).

The first rust resistant cultivar was a "synthetic" combination of lines carrying gene R 2. Subsequent breeding for resistance in various countries in addition to Canada was based largely on gene R 1. The expected shift in race populations apparently did not occur until rust damaged crops in the Red River Valley of the United States and Canada in 1979 and 1980 (J.A. Hoes 1981, personal communication), in 1988 and 1989 (Gulya 1990) because of a buildup of race 3, and 1988 to 1990 (Rashid 1991) because of race 4 and new races; in Texas (Yang 1986) because of races 3 and 4; and in Australia in 1983 (Goulter et al 1984) because of change from race 1 to race 3. R 1 gave good resistance in Argentina prior to 1967, but in subsequent years most rust collections attacked it. Using differentials carrying R 1 or R 2, as well as a series of lines derived from outcrosses with various wild annual Helianthus spp., Antonelli (1985) identified 10 distinct races of rust in Argentina.

Rust races were originally assigned sequential numbers. As new races were discovered, a more informative system of nomenclature, based on the resistance genes which the various races could overcome, was adopted for international use (Ad hoc committee 1988). The North American designations which will be used in this review) and their international equivalents [in brackets] are: 1 [0], 2 [2]), 3 [1]), and 4 [1,2].

Results of genetic studies with some of the races in Argentina indicated the presence of three apparently distinct dominant genes for resistance; inoculations with a new race showed that two of the genes were alleles at one locus, the first proof of multiple allelism for rust resistance in sunflowers (Senetiner et al 1985). Although rust resistance from most of the wild annual Helianthus spp. appeared to be dominant, resistance from H. argophyllus Torrey & Gray appeared to be recessive in Russian work (Putt and Sackston 1957), as was resistance derived from the perennial H. tuberosus to rust in the region of Odessa (Pogorletsky and Geshele 1976).

Resistance to rust race 4 was dominant and controlled by one locus, R 4, in five of six lines studied in North Dakota, possibly with different alleles in some of them. Resistance in the sixth line appeared to be controlled by a single gene at a different locus, named R 5 (Miller et al 1988). The lines were selected (Gulya 1985b) from materials derived in Argentina from cultivated sunflowers in nurseries some of which were exposed to open pollination by various wild annual Helianthus spp.

Resistance to the four North American races was encountered in individual plants of seven accessions of wild annual Helianthus spp.(Quresh et al 1990). Crosses and backcrosses were made with a universal suscept. The results indicated dominance of resistance controlled by two allelic genes for each race considered independently, but close linkage in the coupling phase on the same chromosome for genes controlling resistance to races 1 and 2 jointly. Quresh (1991; Z. Quresh, personal communication 1991) concluded from extensive studies that in some lines resistance to each of the four races was controlled by two different pairs of dominant genes for each race.

The rust race situation is obviously complicated and is likely to become more so with time. It would be helpful to have a series of near isogenic lines each carrying a single, distinct, identified gene for rust resistance. One such line based on an agronomically acceptable universal suscept as the recurrent parent, and a commercial hybrid as the source of a new gene for resistance, was developed in Australia (Goulter 1990). It could be expected to give a clear-cut susceptible reaction to any rust collections virulent to the resistance gene employed. As only one such line was produced, however, and it was developed in a private seed company program, sunflower pathologists can only keep hoping that differential series will eventually be produced for rust, and for other pathogens as well.

Kochman and Kong (1990) reported that in 22 three- and four-way crosses among various sources of resistance, all plants of several lines were resistant to five races of rust. Segregation ratios obtained indicated the presence of 1 or 2 dominant genes and possibly 2 recessive genes. Other ratios

obtained did not fit any of the patterns. Their preliminary results led them to believe that resistance gene pyramiding may confer the slow-rusting type of resistance. It may allow the re-use of some 'spent' resistance genes, prolonging their useful life. Mundt (1991) pointed out that although durable resistance to cereal rusts has been achieved by combinations of resistance genes, the durability does not necessarily derive from a low probability of a pathogen mutating to virulence simultaneously at multiple loci, the usual explanation. Such apparent multipoint mutations in fungi have occurred under laboratory conditions, and presumably could occur in nature.

Downy Mildew

Downy mildew of sunflowers (Plasmopara halstedii (Farl.) Berl. & De Toni) occurs in most countries where sunflowers are grown commercially, with the apparent exception of Australia, South Africa, and possibly North Africa It has caused heavy losses in wet years, and is considered of major importance in many countries (Acimovic 1984, 1988, Sackston 1981). The pathogen was first described on Eupatorium in 1882. The name P. halstedii is commonly used in North and South America, P. helianthi Novot. in Europe.

The only available control for downy mildew until the 1960's was crop rotation, which was only partially effective because of the longevity of the oospores in the soil. Pustovoit (1966) reported that 11 of 24 perennial species of Helianthus were not affected by downy mildew in seedling tests at VNIIMK, and that interspecific crosses between one of them, the hexaploid H. tuberosus, and diploid annual sunflower, gave hybrids immune to mildew. Putt (Putt 1964) mentioned that resistance to downy mildew was present in his material in Manitoba, but that a controlled breeding program depending on erratic natural infection was not practical. Tests using a seedling inoculation method showed that a Canadian sunflower line carrying the R 1 gene for rust resistance appeared to be resistant to the downy mildew occurring in Quebec, Canada (Sackston & Goossen 1966, Goossen & Sackston 1968).

Lines carrying the R 1 gene were found to be completely free

from downy mildew in Romania; their resistance was attributed to a gene Pl closely linked to R 1, and was exploited in developing downy mildew resistant cultivars (Vranceanu 1970). Leclercq et al 1970) found a single diploid (aneuploid; 2n=35) plant resistant to mildew from a cross between sunflower and H. tuberosus. The line derived from this was subsequently labelled HIR 34. Vear and Leclercq (1971) reported that the US line HA 61 carried two genes for resistance to downy mildew at Clermont Ferrand, and that these were distinct from the gene associated with R 1.

Zimmer (1974) showed that sunflowers resistant to mildew in the Red River Valley of the United States and Canada were all resistant in European trials, but European lines with resistance based on Pl were susceptible in the U.S. Zimmer and Kinman (1972) found resistance to the Red River race in HA 61 (derived from a Canadian line carrying the R 2 gene for rust resistance, but inherited independently of it), and also in HIR 34. Their studies at Fargo indicated that HA 61 and HIR 34 had a gene in common, conditioning resistance to the Red River race, which they designated Pl 2. They designated the gene associated with R 1 by Vranceanu, Pl 1, and the other gene reported in HA 1 by Vear and Leclercq, Pl 3.

Selecting locally adapted lines from the open pollinated Russian cultivars proved relatively easy. Incorporating resistance to specific diseases into open-pollinated cultivars, however, was a long and difficult task. Diaz de la Guardia et al (1981) released an open-pollinated cultivar based on the Russian Peredovik, and deriving resistance to downy mildew from HA 61, after four generations of back crossing, four of selfing, and one of poly-crossing. The process is too slow to keep up with changes in pathogen race populations. Leclercq (1969) in France discovered cytoplasmic male sterility in sunflowers, and Kinman (1970) in U.S.A. and Enns et al (1970) in Canada discovered the necessary restorer genes, making possible the production of hybrid sunflowers and the relatively easy and rapid incorporation of disease resistance using dominant genes in the male parent.

Zimmer and Kinman (1972) pointed out that the downy mildew pathogen completes the sexual cycle annually, and that

undescribed races might develop through mutation and hybridization to render the Pl 2 gene ineffective. Even the most resistant lines and hybrids are not immune from infection by mildew; mycelium of the pathogen can occur in inoculated seedlings in the hypocotyl, sometimes as high as the cotyledonary node, and oospores can be formed in the basal tissues (Delanoe 1972, Montes and Sackston 1974, Virany and Dobrovolszky 1980). The pathogen present in tissues of resistant plants could be particularly dangerous, as a mutation for extended parasitism would be ideally located (Sackston et al 1976).

The danger of depending on a single gene to provide protection against downy mildew in many countries is obvious. The need to incorporate several distinct effective genes for mildew resistance simultaneously into varieties or hybrids before they were released was pointed out by Sackston (1974) and by Vear (1974). It was based on the assumption that a mutation at two or more loci simultaneously in the pathogen is much less probable than a single mutation and that such hybrids would therefore be likely to remain resistant for significantly longer periods. This assumption has been questioned recently (Mundt 1991). Although the assumption was accepted by Vranceanu et al (1981), they decided against pyramiding genes. They felt that combining two or more genes for resistance into hybrids would give higher mildew resistance, but would also put greater selection pressure for virulence on the pathogen, and could lead to the rapid development of some extremely virulent forms difficult to control with the existing genes. For this reason they proposed to continue releasing hybrids with only the Pl 1 gene for mildew resistance, and to develop isogenic lines carrying the Pl 2 or Pl 5 genes, to keep in reserve until such time as the widespread occurrence of new races made their release necessary.

Pustovoit and Krasnokutskaya (1976) reported that annual wild species of Helianthus inoculated under controlled conditions in the laboratory were severely affected by all diseases. They concluded that because there was no mildew resistance in the wild annuals, and several of the perennial spp. were susceptible in some degree to downy mildew in laboratory tests, races of downy

mildew in the Soviet Union were widely virulent and perhaps similar to the Red River race. My explanation is different. The collections of wild Helianthus species in the USSR, including H. annuus, although outstanding, were of necessity limited for each species, and might very well have come from individuals not resistant to any given pathogen. Similarly, those sunflower pathogens introduced to the U.S.S.R. indirectly with host materials from North America, were likely to be of a much narrower range of pathotypes than occur in their regions of origin.

Zimmer and Fick (1974) tested Russian interspecific hybrids obtained from Yugoslavia and found most of them to be highly susceptible to downy mildew. The demonstration that sunflowers deriving resistance to downy mildew from H. tuberosus are susceptible to some races of the pathogen from cultivated sunflower put into question the basic assumption of earlier workers at VNIIMK; that perennial polyploid Helianthus spp., particularly H. tuberosus, were immune to most sunflower diseases, and that interspecific hybrids would also be immune. Later work at Odessa (Pogorletsky and Geshele, 1976) and at VNIIMK (Pustovoit and Krokhin 1977, in Miller and Gulya 1987) using conventional genetic procedures and analyses, attributed the resistance of H. tuberosus to downy mildew to one pair of dominant genes.

The situation with races of downy mildew and resistance genes remained fairly stable for about ten years, from the release of Romanian cultivars resistant to race 1 (Vranceanu 1970), and the discovery of the Red River race and resistance to it (Zimmer and Kinman 1972), until the discovery of the new race 3 and resistance to it (Carson 1981, Fick and Auwarter 1981, 1982). Changes came rapidly thereafter. Races 4 and 5 were identified in North America (Gulya and Urs 1985, Ljubich et al 1988). By 1991 races 3 and 7 were identified from Argentina, race 4 from France, Bulgaria, and Hungary, and 6 from France and Canada (Gulya et al 1991). New genes for resistance to some of the races were discovered in lines previously thought to carry only one of the "older" resistance genes, in lines derived from

interspecific hybrids with H. tuberosus, and hybrids with a series of wild annual species of Helianthus (Gulya et al 1991). The increasing complexity of the race situation prompted a proposal to designate downy mildew races internationally by the host genes they can overcome (Sackston et al 1990): 1 (0), 2 (1), 3 (1,2), 4 (1,2,5CL), 5 (1,2,5), 6 (1,9), with the North American designations first, and the international ones in parentheses. The North American designations are used in this presentation.

Most of the genetic studies have been interpreted to show that resistance to one or a number of downy mildew races was governed by individual dominant genes, and that in some cases two or more such genes could be present in individual genotypes. Three different independent dominant genes conditioning resistance to race 4 were derived from three different wild species of Helianthus (Miller and Gulya 1991). Resistance to race 4 from four accessions of wild H. annuus was found to be monogenic and dominant; the genes governing the resistance were non-allelic and were different from those previously identified (Tan et al 1991). Garcia (thesis 1991) found that the inbred line RHA 274 had two complementary genes conferring resistance to races 2 and 7; it had in addition either a single gene, Pl 2 , or two genes, Pl 2 and an unidentified one, closely linked, conferring resistance to both races 2 and 7. Mouzeyar et al (1991) suggested that resistance to a number of races governed by a single gene is attributable, in some cases at least, to the presence of two or more allelic genes at a single locus. Some data suggest that resistance to each race may be controlled by a separate and distinct gene. It may be impossible to determine which hypothesis is correct unless appropriate differential lines can be produced, or chromosome mapping permits precise localization of the genes (F. Vear, 1991, personal communication).

Broomrape

Selection was used very successfully in Russia to develop sunflowers resistant to broomrape. At least some early Soviet sunflower breeders did study genetic ratios, however. Meister (1936, in Vranceanu et al 1980) reported that the resistance to

broomrape was inherited as a dominant character, and referred to simple segregation ratios. More recent Soviet breeders employing modern methods and concepts found that resistance to broomrape races A and B derived from the perennial H. tuberosus L. was controlled by a single simply inherited dominant gene (Burlov and Kotyuk 1976; Pogorletsky and Geshele 1976). Burlov and Artmenko (1983) found by hybrid analysis of a large collection of sunflowers resistant to broomrape that the genes for resistance are located on homologous chromosomes. Recombinant analysis of the genes in hexaploid H. tuberosus and diploid H. annuus showed crossing over, indicating that the germplasm for broomrape resistance may be more limited than assumed earlier. Tolmachev (1988) studying similar material also found that resistance in all forms was controlled by one dominant gene, that the genes were located on homologous chromosomes, and concluded that they were either identical or closely linked. The resistance mechanisms controlled by such genes have received relatively little study. Apparently penetration of parasite haustoria induces lignification of cells of the cortical parenchyma of the resistant host and those of the parasite, and the formation of an additional layer of lignin on host vessel walls at the point of contact with the haustoria (Antonova 1978).

Vranceanu et al (1980) reported on five virulence groups (races or groups of races) of broomrape encountered in Romania, and five types of resistance effective against the respective groups. They set up a series of differentials permitting identification of the five virulence groups, although not the individual races of the pathogen, as each resistance type was effective against a specific race group. The results of complex crossing studies demonstrated a gene-for-gene relationship between virulence in the broomrape and resistance in sunflower. They succeeded in introducing gene Or 5, which gives resistance to all five race groups, into inbred lines with high combining ability which are parents of existing and prospective hybrids, and released new

hybrids with this gene (Vranceanu et al 1986).

The pathogenic composition of broomrape populations has changed over the years, slowly at first, then rapidly in the Soviet Union, elsewhere in Eastern Europe, in Turkey (Indelen et al 1983), and recently in Spain (Melero-Vara et al 1989). Broomrape, known for many years on non-oilseed sunflowers in Spain, was first found there on oilseed sunflower by the author on a few plants in one locality in 1976. By 1988 it was potentially one of the most dangerous sunflower parasites in Spain, and apparently included populations that differed from the five virulence groups described by Vranceanu et al (1980).

The occurrence of individual host genes which confer resistance against two or more different races of a pathogen has been reported in various systems. What is intriguing to one who is not a plant breeder is that four of the host genes in the sunflower:broomrape system each control resistance to a progressively increasing number of race groups, each of which behaves as though it depended on a single gene for virulence. There seems to be no published work on the genetics of pathogenicity in Orobanche. It may be that the various components of each virulence group have in common mainly the absence of a gene effective against the resistance gene or genes in the sunflower lines they cannot parasitize effectively, or that they carry some sort of modifier gene or genes which prevent their virulence genes from operating.

Sclerotinia Wilt And Head Rot.

According to a recent compilation, " Sclerotinia sclerotiorum (Lib.) de Bary is distributed "worldwide, most common in cool moist regions", and occurs on about 150 genera of plants and their products in the United States (Farr et al 1989). On sunflower it can cause a basal stalk rot and wilt, midstem rotting and breaking, head rot, and occasionally leaf infection and rotting. Losses may be severe (Acimovic 1984, 1988, Sackston 1978). In Australia the closely related S. minor Jagger was of major concern causing root and basal stalk rot (Clarke et al

1992). Gulya (1985a) stated that stalkrot/wilt was the most important disease of sunflowers in the U.S. and one of the major diseases affecting sunflower production around the world.

It does not seem reasonable to expect to find resistance within one of its host species, or even a genus, to a fungus with such a wide host range. Kurnik et al (1978) in Hungary concluded that reaction of sunflower hybrids and lines to Sclerotinia head rot was affected primarily by weather conditions during the ripening and harvest periods, and that apparent resistance in some seasons was attributable to conditions unfavorable to infection.

Apparently, seeming reasonable is not a prerequisite for breeding sunflowers successfully. Early Russian investigators reported that H. tuberosus showed resistance to Sclerotinia after reaching the flowering stage, although seedlings and annual wild species were susceptible (Pustovoit and Krasnokutskaya 1976, Pustovoit et al 1976). A later Russian worker found accessions resistant to wilt in an irrigated nursery, in a large collection of hybrids with wild H. annuus (Platonov 1984). Resistance to Sclerotinia stalk rot and wilt of 90 sunflower hybrids, 15 female inbred lines, and six male parent lines was evaluated for 2 years at two naturally infested locations in North Dakota (Fick et al 1983). Additive gene effects rather than dominant or epistatic genes were important. Relatively high heritability indicated that selection for resistance within the group of sunflower genotypes should be very effective; lines and hybrids could be developed with better resistance to Sclerotinia than those currently available.

Robert et al (1987) compared reactions of six female and six male lines of sunflower in two inoculation tests and in natural infection by S. sclerotiorum. Effect of male parent was noted in reactions of crosses. Resistance appeared polygenic, with different genes involved for each organ; additivity appeared more important than dominance.

Gulya (1985a) tested about 750 open pollinated cultivars from 35 countries, wild H. annuus accessions, 42 USDA inbred lines, and 87 U.S. and European hybrids for 2 years in naturally

infested field soil with inoculum added. No genotypes were immune to Sclerotinia wilt, and no commercial hybrids were resistant. Some genotypes had high levels of resistance, including 10 collections of wild H. annuus. Eight USDA inbreds had significant resistance; HA 61 was one of them. Relative ratings did not differ much over 2 years of testing, especially for the most resistant and most resistant groups.

HA 61, among the most resistant lines in Gulya's work, appeared in inoculated field tests to have partial resistance to basal stalk rot which was transferred to some of its progeny (Dueck and Campbell 1978). They suggested that recurrent selection within HA 61 might increase its resistance, and that complete immunity might not be necessary for satisfactory control of wilt under field conditions. HA 61 is of particular interest; it was derived originally from one of the first Canadian rust-resistant lines as a source of a gene for rust resistance, derived originally from a wild H. annuus in Texas. HA 61 has also been a source of resistance to downy mildew and several other diseases, indicating the riches which may be available in the gene bank of wild annual Helianthus spp.

Much work has been done on several continents to develop and compare various methods of inoculating sunflowers with Sclerotinia to obtain consistent infection without obscuring relatively minor differences in reaction. Castano et al (1989) in France tried six different kinds of inoculation, by mycelium vs ascospores, in different parts of the plant, to evaluate different kinds of resistance to attack. There is no complete resistance to the pathogen. Resistance was horizontal, partial, polygenic, and additive. They concluded that there may be genes which control the reaction to infection by mycelium at various locations on the plant, by giving resistance to toxins produced by the mycelium. They further concluded that it would be necessary to make all six tests to determine the reaction of different genotypes to different kinds of attack.

Other methods for identifying potential sources of resistance to Sclerotinia have been tested. Huang and Dorrell (1978) were encouraged by the results of immersing seedlings in

culture filtrates of the fungus; they believed oxalic acid was involved. Hartman et al (1988) studied growth responses of callus cultures on media containing culture filtrates of three isolates of the fungus differing in virulence and in oxalic acid production. Goulter and Brown (1992) did not observe significant correlations between tests of seedlings with oxalate solutions and estimates of their resistance to S. minor in field and greenhouse experiments. Hemery-Tardin (1990) correlated responses of susceptible, intermediate, and resistant genotypes to their production of certain phenolic substances, although other factors were also involved. The development and release to growers of hybrids with effective levels of resistance to the various diseases caused by S. sclerotiorum in favorable environments will be a lengthy process.

Verticillium Wilt

Verticillium wilt (Verticillium dahliae Kleb.) has been reported from most major sunflower production areas in North and South America, Europe, and Australia (Acimovic 1984, 1988; Zimmer and Hoes 1978). It has been a major disease in the U.S.S.R., was considered a limiting factor in sunflower production in Canada in the 1950's and 1960's, and has caused major reductions in yield in heavily affected crops in Argentina and U.S.A. (Bertero and Vazquez 1985, Fick and Zimmer 1974, Putt 1964, Sackston et al 1957, Zimmer and Hoes 1978). In recent years the disease has been considered of major importance only in Argentina, and relatively minor in North America, Europe, and Australia (Acimovic 1984, 1988).

Resistance to Verticillium wilt was reported by Putt (1958). It was controlled in some lines by a single dominant gene designated V 1, in other lines appeared to be recessive, and in still others it was additive (Putt 1964). Resistance found in three inbred U.S. lines, including HA 89 (Zimmer et al 1973), was controlled by a single dominant gene; it was not determined if it was the same as the V 1 gene reported by Putt (Fick and Zimmer 1974). Gulya (1985) registered five sunflower germplasms, derived from Argentine open-pollinated cultivars, which were resistant to several diseases, including Verticillium wilt. It was not

indicated if they carried resistance to the new race attacking cultivars with the V 1 gene in Argentina (Bertero and Vazquez 1982).

Pustovoit et al (1976) reported that all wild species of Helianthus except the tetraploid H. tomentosus were susceptible to Verticillium wilt; resistance in interspecific hybrids was inherited from cultivars bred at VNIIMK, and was comparable to that in Peredovik. [Heiser et al(1969) in their monograph of North American sunflowers include H. tomentosus Michx. as a synonym of H. tuberosus L. and also of H. resinosus Small, both of which are hexaploid species].

Hoes et al (1973) tested wild annual sunflower species, 40 samples of H. annuus and six of H. petiolaris Nutt. from six locations in Manitoba and Saskatchewan in Canada, and 40 locations in 12 states of USA. Resistant plants were found in each of the collections. Genetic studies of crosses with a susceptible line gave variable results; resistance appeared to be dominant in some cases, recessive in others. The collections were obviously heterozygous as well as heterogeneous for wilt reaction. There seemed to be a greater frequency of resistance in collections from more southerly sources, suggesting the possible existence of a center for resistance to Verticillium wilt in the general area postulated to be the center of origin of the most common wild form of H. annuus (Heiser 1951). The results of Hoes et al (1973) indicate either that the collections of wild annual species at VNIIMK did not happen to include plants resistant to Verticillium wilt, or that the virulence of V. dahliae populations in the plots at Krasnodar was significantly different from that at Morden.

Phomopsis Brown Stem Canker

Phomopsis disease of sunflowers (Phomopsis helianthi Munt.-Cvet. et al), first found and described in Yugoslavia in 1980 (Mihaljcevic et al 1982), spread quickly. By 1983 it was considered a major disease in Yugoslavia and Romania and was present in Australia and U.S.A. (Acimovic 1984). Mihaljcevic et al (1982) found that the pathogen was distinct from similar fungi occurring naturally on some wild annual Helianthus spp.

Two wild annual species were infected by the sunflower pathogen in uninoculated experimental field plots and in inoculation experiments, but perennial species were not, nor were the many hosts of other related fungi. Inbred lines of hybrids between cultivated sunflowers and H. tuberosus and of hybrids between H. annuus and H. argophyllus showed greatest ability to survive after inoculation. Complete immunity may not be essential for control of the disease under field conditions.

Vranceanu et al (1983) demonstrated a high degree of resistance in three hybrids of a collection tested in the field. Two of the hybrids were released for general cultivation in 1982 and 1983. They found that some inbred lines could be used in a breeding program for resistance to Phomopsis. Preliminary observations indicated that a small number of genes was involved and that they exerted partial dominance. Resistance seemed to be of general or horizontal type. In order to obtain the highest resistance in F 1 hybrids, selection for resistance should be applied to both parents. Phenotypical resistance was associated with the "stay green" character of sunflower stems, which could make seed cleaning during and after harvesting more difficult.

Skoric (1885) stated that this disease is one of the most widely distributed; he included Brazil and Argentina in the range. His data show that the yield in regions in Yugoslavia with the disease was 45 to 50% less than in its absence. He reported on field data on over 5,000 inbred lines, 2,000 experimental hybrids, and more than 509 varieties tested each year during periods of high natural infection 1980-1984. Only four lines out of all the breeding material showed high tolerance to stem canker. Two of them derived from interspecific crosses with H. tuberosus, one with H. argophyllus, and one was from a local population from Morocco. Earlier work had shown a high degree of susceptibility in all hybrids and most open-pollinated cultivars except the Soviet Yubileynaya and Progress (hybrids derived from H. tuberosus), which had up to 5 % of plants in which infection did not reach the stem. Some wild species were free of infection, and were assumed to be potential sources of resistance. Resistance was not monogenic; in crosses between a

field resistant and a susceptible line the hybrid was intermediate in reaction. A cross between two highly tolerant lines gave the same or greater levels of tolerance than the parents. Skoric, like Vranceanu et al (1983), found that resistance was associated with the "stay-green" stem character. Resistance to Phomopsis was correlated with resistance to Macrophomina and to drought. He recommended a more intensive inclusion of wild sunflower species in the breeding program for disease resistance. Kurnik and Walcz (1985) reported finding resistance in H. argophyllus, tolerance in two other wild species, and, differing from most other investigators, susceptibility in two local forms of H. tuberosus.

Masirevic et al (1988) were concerned that very few sources of resistance, mostly from wild species, were available against Phomopsis. Screening in the greenhouse was difficult because ascospores were required, and those were produced only on infected stems, (although Yang et al(1984) reported obtaining them in culture). Masirevic et al (1988) tried screening for resistance by growing calli on agar medium containing culture filtrates of the pathogen. Differences between resistant and susceptible calli were apparent within six days, but the reactions were all alike after three weeks, indicating that concentrations of filtrate may have been too high. Prospects were encouraging, because sunflower calli could be regenerated after prolonged exposure on screening systems.

CONCLUDING REMARKS

Managing disease on sunflowers covers a wide range of methods, from trying to avoid or escape disease by changing locations or times of sowing, through protection or therapy by chemicals, to selection of resistant individuals or lines from heterogeneous populations, to highly developed crossing programs using genes for resistance from cultivated sunflowers or from their close annual diploid or more distant perennial polyploid wild relatives.

It is fortunate that some of the crossing programs have

yielded unexpected benefits. One was the discovery of resistance to downy mildew and other diseases from the same sources, and in one instance from apparently the same locus, as resistance to rust. Another is the correlation of the non-dominant polygenic resistance to Phomopsis brown stem canker, which develops best under relatively humid conditions, with apparent resistance to charcoal rot (Macrophomina phaseolina (Tassi) Goidanich), which is associated with drought and heat stress.

Biological control of S. sclerotiorum has been observed in nature, and there may be some hope of exploiting it in farm fields (Gulya et al 1992, Huang 1980). Preliminary studies are starting in some laboratories on possible biological control of downy mildew. Most genetic engineering work is being done in large private laboratories which do not publish their research. It is quite possible that some of them are attempting to incorporate genes from unrelated organisms into sunflowers to modify their products and to protect them from diseases.

Managing diseases of sunflowers, and of most other crops, is a perpetual struggle. Individual 'battles' may be won, and in some instances the 'peace' may last for years. The 'war' usually breaks out again, however. We can only trust that research will keep developing new technologies to help us in the struggle.

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