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# UPDATE ON INHERITANCE OF SUNFLOWER CHARACTERISTICS

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## SUMMARY

Genetic inheritance of important characteristics in sunflower (Helianthus annuus L.) is important to guide researchers in selecting the most efficient breeding methodology to improve selected characteristics. Without information on inheritance, a breeder will not be able to understand the control of a trait or how to proceed in transferring that trait to adapted lines. The purpose of this paper is to summarize available research on the inheritance and control of several important characteristics of sunflower. Altering plant architecture by changing leaf orientation or reduced height may lead to increased yields by increasing photosynthetic efficiency or standability of the plant. Great strides have been made in identification of genes for resistance to downy mildew, rust, and Phomopsis. Genetic control of tolerance to Sclerotinia is better understood and will enhance future research. Presently, there have been 28 sources of cytoplasmic male sterility reported in sunflower. Genetic control of male fertility restoration appears to be complex with variable interactions between nuclear genes and cytoplasms. New techniques to distinguish genes utilizing RFLP analyses of mitochondrial DNA appear to be positive. Several studies have contributed toward understanding factors affecting drought tolerance. Development of sunflower oil with high oleic fatty acid concentration has provided distinctive new interest in growing sunflower worldwide.

## INTRODUCTION

Determination of genetic inheritance, the number of genes controlling the expression of a trait, factors influencing heritability, and contributions of genetic effects are all important to breeders of sunflower, Helianthus annuus L. Knowledge of inheritance will guide the breeders in selecting the most efficient breeding methodology to improve selected characteristics. These characteristics vary with specific programs and are determined by needs of

each country or production area. All programs generally emphasize improving seed yield and oil percentage in sunflower. However, there are many characteristics which are associated with improvement of yield, e.g., plant vigor, head size, seed weight, and physiological factors affecting efficiency of photosynthesis. Conversely, other characteristics prevent losses in yield, such as standability or resistance to diseases and other pests. Equally important may be factors affecting quality of the oil or seed.

It is the objective of each breeder to combine as many traits together providing the highest yielding and highest quality product possible. Without information on inheritance, a breeder will not be able to understand the control of the trait or how to proceed in transferring that trait to adapted lines.

The purpose of this paper is to summarize research presented on the inheritance and control of several important characteristics of sunflower, so that researchers or breeders will be able to better utilize this information to make further progress in their programs. Also, an update on genes controlling traits is important for biotechnology projects using RFLP and other analyses.

## MORPHOLOGICAL CHARACTERISTICS

### Petiole Length and Upright Leaf

Altering plant architecture may be useful in increasing photosynthetic activity, decreasing transpiration, or increasing the efficiency of light or photosynthetic conductivity. These characteristics may lead to increases in yield or increases in one of the yield components, such as number of achenes per head or achene weight.

One characteristic of interest in the past few years has been petiole length (Table 1). Luczewicz (1975) reported plants with short, thick petioles that were derived from Karlik 68. Genetic inheritance was determined to be by two complementary recessive genes. Vranceanu et al. (1988) reported that a short petiole characteristic derived from Orizont was controlled by two dominant genes with cumulative gene action, designated $Ps_1$ and $Ps_2$. Leaves with short petiole were characterized by a rough, uneven surface, with smaller than normal leaf area. Skoric (1988) also reported a short petiole plant type derived from an interspecific cross with H. mollis Lam. The leaves from this source are smooth and normal in appearance.

Table 1. Inheritance factors controlling several morphological characteristics in sunflower.

| Character | Inheritance factor | Reference |
|---|---|---|
| Short Petiole | Two complementary dominant genes | Luczlewicz, 1975 |
| | Two cumulative dominant genes $Ps_1$, $Ps_2$ | Vranceanu et al., 1988 |
| Upright Leaf | Single, recessive gene, $ul_1$ | Miller, 1991 |
| Conventional Height | Dominant genetic effects<br>Dominant genetic effects | Marinkovic, 1982<br>Putt, 1966 |
| | 57% dominance effects<br>30% additive effects | Lay & Khan, 1985 |
| | Dominance to overdominance | Kovacik & Skaloud, 1990 |
| Reduced Height | Recessive | Fick, 1978 |
| | Recessive | Vranceanu, 1974 |
| | Recessive | Berger & Miller, 1985 |
| | Additive 48-71%<br>Dominance 3-16% | Miller & Hammond, 1991 |
| Stem Diameter | Additive 12-50%<br>Dominance 34-61% | Miller & Hammond, 1991 |
| Head Inclination | Four genes, additive<br>Three genes, dominance | Kovacik & Skaloud, 1990 |

Soldatov and Kalaidzhyan (1987) reported finding a short petiole plant with petiole length of 5 cm compared with 25-30 cm on a conventional plant. The short petiole was derived through mutagenesis, producing plants with normal height, thin but strong stem, and a more compact growth habit.

Another leaf characteristic of interest is termed the upright leaf. An introduction of sunflower derived from Spain was identified as having distinctly more upright petiole structure, particularly at the V6 to V12 stage (Schneiter and Miller, 1981). The genetic inheritance of the upright leaf utilizing $F_1$, $F_2$, and $BC_1$ generations was determined to be controlled by a single recessive gene, $ul_1$ (Miller, 1991).

Both the short petiole and upright leaf characteristics may enhance yield through more efficient light receptivity. Planting sunflower with these characteristics in narrow rows of 60 cm would allow plants to be spaced more evenly. Therefore, plant population increases of 20 to 30% may be feasible without causing decreases in head size or seed weight.

Plant Height

Plant height in conventional sunflower has been regarded as a quantitatively inherited character, with dominant genetic effects most important for conditioning conventional height (Marinkovic, 1982; Putt, 1966). Lay and Kahn (1985), utilizing a generation means analysis averaged over six crosses, reported that approximately 57% of the total genetic variation controlling conventional height was due to dominance and 30% due to additive gene action (Table 1).

Decreasing plant height and increasing stem diameter may be useful in increasing standability of sunflower. Lodging or stem breakage due to adverse growing conditions can reduce yields significantly in some years. Strong winds in conjunction with excessive precipitation and saturated soil conditions can cause up to 100% loss. However, progress in improving the standability of conventional height sunflower has been slow. Therefore, the potential of reduced-height germplasm to increase stem strength was recognized.

Reduced plant height controlled by recessive genes in lines with a reduced number of leaves has been reported by Fick (1978), Vranceanu (1974), and Beretta de Berger and Miller (1985). A single recessive gene was involved in controlling this type of reduced plant height. Three sources of reduced-height sunflower, DDR, Donsky, and Donskoi 47, had reduced height and an equal or similar number of leaves as conventional height sunflower (Miller and Hammond, 1991). The internode length was reduced in these lines. Estimates of additive, dominance, and epistatic genetic effects controlling reduced height indicated that the additive component ranged from 48 to 71%, while the dominance component ranged from 3 to 16%. The epistatic component was important in one cross. The dominance component of genetic effects controlling stem diameter ranged from 34 to 61%, while the additive component ranged from 12 to 50%. Epistasis was present, but minor, for controlling stem diameter.

America (Gulya et al., 1991), Races 3, 4, and 6 in Europe (Gulya et al., 1991; Tourvieille et al., 1988), and Races 3 and 7 in Argentina. It is imperative that genetic inheritance to each individual race be known and identified for breeding resistance into new commercial cultivars.

The $Pl_5$ gene derived from RF-5566-74, Novinka, and Progress (Vranceanu et al., 1981; Fick and Auwarter, 1982; Miller and Gulya, 1987) was determined to be effective against Race 3 downy mildew (Table 3). Novinka and Progress were derived from interspecific crosses of H. tuberosus L. with open-pollinated cultivars of the USSR (Pustovoit et al., 1976). Pustovoit and Krokhin (1977) reported that resistance to downy mildew derived from H. tuberosus was controlled by one dominant gene; therefore, the gene imparting resistance to Race 3 in the two selections probably was derived from H. tuberosus.

The $Pl_6$, $Pl_7$, and $Pl_8$ genes were derived from interspecific crosses of cultivated sunflower with wild H. annuus L., H. praecox Engelm & Gray ssp. runyonii, and H. argophyllus Torrey & Gray, respectively (Miller and Gulya, 1991). These genes have been found to impart resistance to all seven races (Gulya et al., 1991).

RHA 274 and DM-2 both have resistance to Races 1 and 2. However, DM-2 was susceptible to Race 6, and RHA 274 was resistant. The additional gene conferring resistance in RHA 274 to Race 6 was tentatively designated $Pl_9$. Since both RHA 274 and RHA 325 are resistant to Race 7 and RHA 325 is susceptible to Race 6, RHA 274 and RHA 325 both carry an additional gene, designated $Pl_{10}$, conferring resistance to Race 7.

A single dominant gene, $Pl_t$, was reported from interspecific crosses with H. tuberosus and cultivated sunflower (Burlov and Artemenko, 1983). The $Pl_t$ gene and the gene $Pl_2$ were found to be allelic. An interesting observation is that H. tuberosus could possibly contribute three different genes for resistance to downy mildew, $Pl_4$ (Vear, 1974), $Pl_5$ (Pustovoit & Krokhin, 1977), and $Pl_t$ ($Pl_2$) (Burlov and Artemenko, 1983).

Tan et al. (1991) found resistance to Race 4 was controlled by a single dominant gene in four sources of wild H. annuus accessions, PI 413047, PI 413131, PI 413157, and PI 413161. Since $Pl_6$ was also derived from wild H. annuus , the allelic relationship between these new genes and $Pl_6$ was determined. All were found to be nonallelic to $Pl_6$. Allelic relationships determined among the four sources indicated that each is contributing a new gene for resistance to Race 4.

Table 3. Inheritance factors, race resistance, and source of resistance to downy mildew of sunflower.

| Gene | Resistant to races | Source | Reference |
|---|---|---|---|
| $Pl_1$ (Pl) | 1 | AD 66 | Vranceanu & Stoenescu, 1970 |
| $Pl_2$ (H1) | 1,2 | HA 61 | Zimmer & Kinman, 1972<br>Vear & Leclercq, 1971 |
| $Pl_3$ (H2) | 1,2 | HA 61 | Zimmer & Kinman, 1972<br>Vear & Leclercq, 1971 |
| $Pl_4$ | 1,2 | HIR 34 | Vear, 1974 |
| $Pl_5$ | 3 | RF-5566-74 | Vranceanu et al., 1981 |
| $Pl_6$ | 1,2,3,4,5,6,7 | HA 335, HA 336 | Miller & Gulya, 1991 |
| $Pl_7$ | 1,2,3,4,5,6,7 | HA 337-339 | Miller & Gulya, 1991 |
| $Pl_8$ | 1,2,3,4,5,6,7 | RHA 340 | Miller & Gulya, 1991 |
| $Pl_9$ | 6 | RHA 274 | Gulya et al., 1991 |
| $Pl_{10}$ | 7 (2) | RHA 325, RHA 274 | Garcia, 1991<br>Gulya et al., 1991 |
| $Pl_t$ ($Pl_5$) | | H. tuberosus | Pustovoit & Krokhin, 1977 |
| $Pl_t$ ($Pl_2$) | | H. tuberosus | Burlov & Artemenko, 1983 |
| $Pl_{4a1-4}$ | 4 | H. annuus<br>PI 413047<br>PI 413131<br>PI 413157<br>PI 413161 | Tan et al., 1991 |
| $Pl_{2a1}$ | 2 | H. annuus<br>PI 413078 | Jan et al., 1991 |

Two additional sources of wild H. annuus, PI 413078 and PI 413087, were found to have resistance to Race 2 (Jan et al., 1991). The resistance in each source was determined to be controlled by a single dominant gene. Allelic studies confirmed that PI 413087 possesses the $Pl_2$ gene. However, PI 413078, has a different resistance gene than $Pl_2$, and is a new source of resistance to Race 2 downy mildew.

Rust, incited by Puccinia helianthi Schw.

Sunflower rust is a significant disease problem in production areas of North America, Australia, and Argentina, and is a potential disease on sunflower grown in all regions of the world. The $R_1$ gene was derived from 953-102 (MC 69) and imparts resistance to the North American (N.A.) Races 1 and 2 (Table 4). The $R_2$ gene was derived from 953-88 (MC 29) and imparts resistance to N.A. Races 1 and 3. These two genes were effective against prevalent races in North American since 1970. However, recent surveys of rust indicate that the prevalence of new races is increasing in North America, and rust incidence in Australia has caused severe yield losses.

Inheritance studies of rust resistance to N.A. Race 4 indicated that single dominant genes conferred resistance in the lines HA-R1, HA-R2, HA-R3, HA-R4, and HA-R5 (Miller et al., 1988). The lines were derived from the Argentine sunflower cultivars Pergamino 71/538, Saenz Pena 74-1-2, Impira INTA, Charata, and Guayacan INTA, respectively. Lines HA-R1, HA-R3, HA-R4, and HA-R5 had the same gene, $R_4$, while HA-R2 had a different gene, $R_5$, conferring resistance to N.A. Race 4.

Yang et al. (1989) reported a single gene from the Argentine line, P386, was conferring rust resistance to N.A. Races 1, 2, 3, and 4. The gene in P386 was designated $Pu_6$ and was nonallelic to the genes $R_1$, $R_2$, $R_4$, and $R_5$.

Inheritance studies using an Argentine rust isolate, clone 340, showed single dominant genes conferring resistance in the inbred lines LC74/75-20620, MP557, and MP555 (Senetiner et al., 1985; Antonelli, 1985). A dominant and a recessive gene conferring resistance were suggested to be present in the inbred line Pergamino 71-538. The genes were named $Ph_1$ from line LC74/75-20620, $Ph_2$ from lines MP557 and MP555, and $Ph_{2a}$ and $ph_3$ from line Pergamino 71-538. $Ph_{2a}$ is allelic to $Ph_2$, and $ph_3$ is recessive. The relationship among these genes and $R_1$, $R_2$, and $R_3$ has not been determined.

Goulter (1990) investigated the inheritance of resistance in the commercial sunflower hybrid Hysun 33 to Australian rust races 0 and 1. The genetic analysis indicated that a single dominant gene conferred resistance to both races. The source of this gene was named PhRR3.

Recent studies with wild accessions of sunflower have indicated that inheritance of resistance to N.A. races is more complicated than single dominant gene action (Quresh and Jan, 1992). Seedlings of 78 wild accessions from H. annuus, H. argophyllus, and H. petiolaris were screened for resistance to N.A. Races 1, 2, 3, and 4, using a multiple race inoculation technique.

Table 4. Genes identified conferring resistance to sunflower rust and source of resistance. N.A.=North American.

| Gene | Resistant to races | Source | Reference |
|---|---|---|---|
| $R_1$ | N.A. 1,2 | 953-102(MC69, CM90) | Putt & Sackston, 1963 |
| $R_2$ | N.A. 1,3 | 953-88(MC29-USDA) | Putt & Sackston, 1963 |
| $R_3$ | | CM 403-4 | Research Report, Morden Manitoba, CAN (1967) |
| $R_4$ | N.A. 4 | HA-R1, HA-R3, HA-R4, & HA-R5 | Miller et al., 1988 |
| $R_5$ | N.A. 4 | HA-R2 | Miller et al., 1988 |
| $R_6$ | N.A. 1 | PI 413037 | Quresh & Jan, 1992 |
| $R_7$ | N.A. 2 | PI 413037 | Quresh & Jan, 1992 |
| $R_8$ | N.A. 3 | PI 413037 | Quresh & Jan, 1992 |
| $R_9$ | N.A. 4 | PI 413037 | Quresh & Jan, 1992 |
| $R_{10}$ | N.A. 1,6 | MC 29 (AUS) | Lambrides & Miller, 1992 |
| $Pu_6$ | N.A. 1,2,3,4,5 | P386 (Charata) | Yang et al, 1989 |
| $Ph_1$ | ARG 340 (N.A. 2) | LC74/75-20620 | Antonelli, 1985 |
| $Ph_2$ | ARG 340 (N.A. 2) | MP 555, MP 557 | Antonelli, 1985 |
| $Ph_{2a}$, $ph_3$ | ARG 340 (N.A. 2) | Pergamino 71-538 | Antonelli, 1985 |
| ($R_3$) | AUS 0&1 (N.A. 1&3) | phRR3 | Goulter, 1990 |

Seven of the 78 accessions showed high levels of resistance to all four races of rust. In all seven accessions, one gene appeared to give resistance to both Race 1 and Race 2. Three accessions possessed a gene conditioning resistance to Race 1 linked at 17.8 map units to another gene imparting resistance to Race 2. PI 413171, tracing to H. petiolaris had a gene resistant to Race 3 linked at 28 map units to a gene imparting resistance to Race 4.

Most interesting was a plant derived from PI 413037 which had all four resistance genes linked in the coupling phase. The genes in this linkage

group were named $R_6$, $R_7$, $R_8$, and $R_9$ that confer resistance to N.A. Races 1, 2, 3, and 4, respectively. The gene order on the chromosome was determined to be $R_8$-$R_9$-$R_6$-$R_7$.

The linkage relationships of the types found in these studies would be very useful to plant breeders. Selection for resistance to one race of rust in segregating generations of crosses would give a high probability for selecting genes conferring resistance to other races. Therefore, resistance to several races can be transferred easily to a single genotype.

**Verticillium Wilt**, incited by Verticillium dahliae Kleb.

Breeding for Verticillium wilt resistance in sunflower appears to be the most effective means of control. Putt (1964b) determined resistance to the prevalent North American race of Verticillium was controlled by a single dominant gene named $V_1$ (Table 5). Fick and Zimmer (1974a) studied the inheritance to Verticillium wilt in three oilseed inbred lines, HA 89, HA 124, and P-21VR1. These three lines showed high resistance to Verticillium under field conditions in North America. Control was determined to be by a single dominant gene and this one gene conditioned resistance in the three lines. However, the relationship between $V_1$ and this gene has not been determined.

Hoes et al. (1973) tested 43 accessions of wild sunflower for resistance to Verticillium wilt. Resistance was dominant and controlled by single genes. Interestingly, accessions collected in Manitoba, Saskatchewan, and North Dakota were generally less resistant than accessions collected in more southern latitudes. They postulated that resistance coincided closely with the center of origin of H. annuus and H. petiolaris.

Table 5. Genes identified conferring resistance to Verticillium wilt in sunflower and source of resistance. N.A.=North American.

| Gene | Resistant to races | Source | Reference |
|---|---|---|---|
| $V_1$ | N.A. | CM 144 | Putt, 1964 |
| $V_a$ | N.A. | HA 89, HA 124, P21VR1 | Fick & Zimmer, 1974 |
| $V_{t1}$,$V_{t2}$ | Russia | H. tuberosus | Pustovoit & Krokin, 1978 |
| | Argentine | | Bertero & Vasquez, 1982 |

Pustovoit and Krokhin (1978, summarized in Skoric, 1988) reported that resistance to Verticillium in material derived through interspecific crosses with H. tuberosus was controlled by two recessive genes or by two complementary, dominant genes. In one of the genotypes, the resistance was controlled by three recessive genes.

In 1982, Bertero and Vazques (1982) reported a new race of Verticillium in Argentina and the resistance gene in HA 89 was not effective.

Sclerotinia wilt and head rot

Sclerotinia wilt and head rot, incited by Sclerotinia sclerotiorum (Lib.) de Bary, has been noted to be the most serious disease in sunflower. The reasons for this distinction are several, but is most likely due to its persistence in soil for many years and its wide host range, making rotations with other crops difficult. Sclerotinia causes multiple infections including basal stalk rot and wilt, midstem rotting and breaking, head rot due to ascospore infection, and occasional leaf and cotyledon infection. Losses may be severe, approaching 100% in some fields, and decreases in oil content and quality may be associated with severity of infection. Genetic control for resistance has been lacking, but evidence now indicates that a degree of tolerance may be obtained through sophisticated breeding techniques.

Several studies investigating resistance to Sclerotinia indicated that little relationship exists between genetic control of resistance to mycelium infection of roots and genetic control of ascospore infection of the head (Vranceanu et al., 1984; Vear and Tourvieille, 1984; Pirvu et al., 1985; Robert et al., 1987; Shevelukha and Zaichuk, 1987; Skoric, 1988; Tourvieille and Vear, 1990). These conclusions were based on development of lines for resistance to one type of infection and then testing for a second type of infection, or the reaction of their hybrids to the two types of infection. The following discussion summarizes the research on each infection.

Early studies regarding tolerance to Sclerotinia reported large specific combining ability (SCA) effects compared with general combining ability (GCA) effects, and low levels of heritability of resistance (Fick and Gulya, 1980; Vranceanu et al., 1984). However, due to the development of better inoculation techniques and more reliable testing methods, recent research has generally indicated that resistance is quantitatively controlled, with additive effects more important than dominance effects and higher levels of heritability.

Tourvieille and Vear (1984) studied ascospore infection of heads in nine

F1 hybrids known to have a wide range of field reactions to Sclerotinia. Resistance to Sclerotinia was determined to be partial and polygenic (Table 6). Robert et al. (1987) used parental inbred lines and $F_1$ hybrids in a factorial cross and found resistance to ascospore infection was polygenic, with resistance to the rate of infection heritable and largely additive. The correlation coefficient between parents and the mean of their hybrids was r=0.93, and the midparent-hybrid regression coefficient b=1.11. These

Table 6. Inheritance factors controlling Sclerotinia head and wilt resistance in sunflower.

| Infection type | Inheritance factor | Reference |
|---|---|---|
| Ascospore Infection | | |
| | Quantitative inheritance<br>Polygenic nature of resistance | Tourvieille & Vear, 1984 |
| | Polygenic, largely additive<br>b=1.11, r=0.93 | Robert et al., 1987 |
| | Largely additive<br>GCA/SCA ratio=4.49-2.74<br>r=0.83(Art.), r=0.49 (Nat.) | Vear & Tourvieille, 1988 |
| | Polygenic, Additivity=31%, SCA | Vranceanu et al., 1981<br>Pirvu et al., 1985 |
| Root Infection | | |
| | Low level of heritability b=.31 | Fick & Gulya, 1980 |
| | Additive greater than dominance or epistasis, H = 59% | Fick et al., 1983 |
| | Large SCA | Vranceanu et al., 1984 |
| | Polygenic, Additivity=25%<br>Single gene, recessive, $s_1$ | Pirvu et al, 1985 |
| | GCA greater than SCA<br>b=0.58, r=0.79 | Tourvieille & Vear, 1990 |
| | Recurrent Selection | Vear & Tourvieille, 1984<br>Shevelukha & Zaichuk, 1987 |

conclusions indicated that breeding materials could be tested in early generations and their results used to predict the values of hybrids based on performance of their inbred lines. An additional study (Vear and Tourvieille, 1988a) on resistance to ascospore infection of heads of parental inbred lines and $F_1$ hybrids found the GCA/SCA ratio was 4.49-2.74 for the ascospore test. The correlation between inbreds and hybrids was r=0.83 in the ascospore test. They concluded that additive effects were generally more important than dominance, although some hybrid combinations showed significant SCA effects.

In Romania, the resistance of 30 $F_1$ hybrids and their parents was investigated (Vranceanu et al., 1981; Pirvu et al., 1985). Inheritance of resistance was considered complex and controlled by several genes. Additive gene action was determined to be 31% conditioning resistance to ascospore infection of the stalk and head, and 25% for resistance of mycelium to the root. They concluded that selection for this complex character should be feasible. However, they also noted presence of SCA in some crosses where resistant hybrids appeared when susceptible parents were crossed.

Fick and Gulya (1980) evaluated resistance to mycelium infection of roots in 20 hybrids produced using 11 female parents and 6 male parents. The parent-offspring regression was calculated to be b=0.31 with differences in resistance determined to be heritable at a relatively low level. However, in further studies investigating 90 hybrids, 15 female parents, and 6 male parents over 2 years, additive gene effects rather than dominant or epistatic gene effects were of primary importance in controlling wilt resistance (Fick et al., 1983). Narrow sense heritability was 59% indicating that selection for resistance within this group of genotypes should be effective.

Vranceanu et al. (1984) evaluated 3 varieties, 7 inbred lines, 10 $F_1$ hybrids and one synthetic under natural and inoculated conditions for resistance to Sclerotinia wilt. In general, plants in the varieties showed the highest proportion of resistance and inbred lines the lowest proportion of resistance. They concluded there was sufficient variability among genotypes to warrant selection. However, large SCA effects existed as hybrids could not be predicted reliably from the resistance shown by their parents. In additional studies (Pirvu et al., 1985) additive gene action was determined to be important and control of resistance polygenic. Two inbreds, CS-77-999-1 and CS-77-1081, were determined to have a single recessive gene, $\underline{s}_1$, controlling mechanical resistance to the initial penetration of the stem surface tissues.

Tourvieille and Vear (1990) studied $F_1$ hybrids and their six female and six male lines for resistance to mycelium infection of the roots. A factorial analysis showed highly significant parental effects, with the ratio of general to specific combining ability variances 1.57. They concluded that additive effects were more important than dominance effects in controlling resistance. A high regression coefficient (b=0.58) and correlation (r=0.79) between inbred lines and their hybrids indicated that resistance had high heritability. A negative correlation between oil content and head rot resistance was observed in studies by Vear and Tourvieille (1988b), but this correlation was not present between oil content and wilt resistance. Additional studies indicated that it was possible to obtain recombination, resulting in high oil lines with head rot resistance.

Due to the polygenic control of resistance to Sclerotinia, breeding techniques such as recurrent phenotypic selection has been implemented (Vear and Tourvieille, 1984). Rapid improvement in resistance was found in the first cycle, followed by three generation of no marked improvement. In the fifth cycle, increases in resistance were again measured (Vear and Tourvieille, 1985). Shevelukha and Zaichuk (1987) used cycles of selection to increase resistance in an open-pollinated variety, Peredovik. Repeated infections of heads and seedlings were utilized. The proportion of plants in the population with resistance was increased to 30-50%. However, it was discovered that increasing resistance to head infection did not increase resistance to root infection. Therefore, recurrent phenotypic selection utilizing root as well as head infection was applied. A reliable increase in the proportion of plants with complex resistance to both forms of infection was reported.

Phomopsis, incited by Phomopsis helianthi Munt.-Cvet. et al.

Phomopsis is considered a major disease in Yugoslavia and Romania, and is a disease of moderate level in France, Bulgaria, Hungary, and Turkey. Phomopsis, however, is widely distributed, and has been found in Australia, the United States, Brazil, Argentina, and Iran.

Preliminary genetic studies reported by Vranceanu et al. (1983) indicated resistance was controlled by a small number of genes with partial dominance (Table 7). The high mean square value for genotypes suggested that selection for resistance could be successful, but in both male and female parents of hybrids. The expression of resistance was associated with the "stay green" character of sunflower stems.

Table 7. Inheritance factors controlling Phomopsis resistance in sunflower.

| Inheritance factor | Reference |
|---|---|
| Partial dominance | Vranceanu et al., 1983 |
| Two or more complementary genes | Skoric, 1985 |
| Recessive genes; quantitative interaction | Tourvieille et al., 1988 |
| Resistance correlated with resistance to Macrophomina phaseoli, Phoma macdonaldii | Skoric, 1988 |
| Drought tolerance<br>"Stay green" character of stems | Vranceanu et al., 1983 |

Skoric (1985) found resistance to be partially dominant, in that crosses of a highly tolerant line with a susceptible line resulted in a hybrid with an intermediate level of resistance. He concluded that resistance is most probably controlled by at least two or more complementary genes.

Tourvieille et al. (1988) used mycelium tests on leaves and petioles to investigate $F_2$ progenies of a cross between French inbred lines and resistant cultivars. The results indicated that resistance is partly under control of recessive genes, but it also depends on the interaction between a number of genes.

An interesting observation by researchers is that resistance to Phomopsis is positively correlated with resistance to Macrophomina phaseoli and Phoma macdonaldii (Skoric, 1988). Additional associations between resistance to drought tolerance and the "stay green" character of sunflower stems have been reported.

## CYTOPLASMIC MALE STERILITY - MALE FERTILITY RESTORATION

After discovery of the PET1 cytoplasmic male sterility (Table 8), several geneticists initiated studies to find effective male fertility restoration genes. Kinman (1970) reported a male fertility restorer gene in the sunflower

Table 8. The FAO code, common name, species of origin, and reference of known cytoplasmic male sterility (CMS) sources in sunflower.

| FAO code | Common name | Species of origin | Reference |
|---|---|---|---|
| PET1 | French, Leclercq | H. petiolaris | Leclercq, 1969 |
| ANL1 | Kouban, Anashenko | H. ann. lenticularis | Anaschenko, 1974 |
| ANL2 | Indiana 1 | H. ann. lenticularis | Heiser, 1982 |
| PET2 | CMS 89(PET2), CMG1 | H. petiolaris | Whelen, 1980;Miller & Wolf, 1991 |
| PET3 | Petiolaris BIS | H. petiolaris | Leclercq, 1983 |
| PEF1 | Fallax | H. petiolaris fallax | Serieys, 1984 |
| PEP1 | PET/PET | H. petiolaris petiol. | Serieys, 1987 |
| ANN1 | H. annuus 397 | wild H. annuus | Serieys, 1984 |
| ANN2 | H. annuus 517 | wild H. annuus | Serieys, 1984; Jan, 1988 |
| ANN3 | H. annuus 519 | wild H. annuus | Serieys, 1984; Jan, 1988 |
| ANN4 | H. annuus 521 | wild H. annuus | Serieys, 1984 |
| ANN5 | NS-ANN 81 | wild H. annuus | Skoric, 1988 |
| ANN6 | NS-ANN 1344 | wild H. annuus | Skoric, 1988 |
| ANT1 | Fundulea 1 | H. annuus texanus | Vranceanu et al., 1986 |
| ARG1 | Argophyllus 1 | H. argophyllus | Christov, 1990 |
| ARG2 | Argophyllus 2 | H. argophyllus | Christov, 1990 |
| ARG4 | Argophyllus 4 | H. argophyllus | Vannozzi, 1987 |
| ANO1 | Anomalus | H. anomalus | Serieys, 1987 |
| BOL1 | H. bolanderi | H. bolanderi | Serieys, 1984 |
| BOL2 | Bolanderi 2 | H. bolanderi | Vannozzi, 1987 |
| EXI1 | Exilis 1 | H. exilis | Serieys, 1987 |
| EXI2 | Exilis 2 | H. exilis | Serieys, 1991 |
| GIG1 | CMS 89(GIG1), CMG2 | H. giganteus | Whelen, 1981;Miller & Wolf, 1991 |
| MAX1 | CMS 89(MAX1), CMG3 | H. maximiliani | Whelen&Dorrel;Miller& Wolf, 1991 |
| PRP1 | Praecox | H. praecox praecox | Serieys, 1991 |
| NIC1 | Canesens | H. niveus canesens | Serieys, 1991 |
| RIG1 | Vulpe | H. rigidus | Vulpe, 1972 |
| GRO1 | Grossesseratus | H. grosseserratus | Spirova, 1990 |

(Serieys, 1991; Miller et al. 1992)

line T66006-2-1, and determined that a single dominant gene, $\underline{Rf_1}$, conditioned male fertility restoration (Table 9). Several agronomically desirable restorer lines utilized in the industry for hybrid seed production have been derived from the T66006-2-1 source (Fick and Zimmer, 1975). Vranceanu and Stoenescu (1971) initiated studies to determine if inbred lines of varied genetic and geographic backgrounds had factors for male fertility restoration. Only one source, MZ-1398, derived from the local population Mezehedeshy, had restoration. They determined that a single dominant gene controlled

Table 9. Inheritance of male fertility restoration of various cytoplasmic male sterility sources in sunflower.

| Cytoplasm | Genes or number of genes | Source or gene action | Reference |
|---|---|---|---|
| PET1 | $Rf_1$ | T66006-2-1 | Kinman, 1970 |
| | Single, dom. | Acc(MO) 1338 | Enns, 1970 |
| | $Rf_2$ | MZ-1398 | Vranceanu & Stoenescu, 1971 |
| | Msc 1 | H. petiolaris | Leclercq, 1971 |
| | $Rf_2$ | Present in female parents | Kinman;Fick & Zimmer, 1974 |
| | $Rf_1$,$Rf_2$ | Two complementary genes | Fick & Zimmer, 1974 |
| | Four genes | Two complementary, dominant genes necessary | Gimenez & Fick, 1975 |
| | Two genes | Complementary, dominant | Vranceanu & Stoenescu, 1978 |
| | Three genes | Complementary, dominant | Vranceanu & Stoenescu, 1978 |
| | Two genes | Cumulative, nonallelic | Vranceanu & Stoenescu, 1978 |
| | Single, dom. | Progress(H. tuberosus X H. annuus) | Artemenko, 1987 |
| | One gene | Single dominant | Anashchenko & Duka, 1985a |
| | Two genes | Duplicate dominant | Anashchenko & Duka, 1985a |
| | | Two complementary genes | Anashchenko & Duka, 1985a |
| ANL1 | One gene | Single dominant | Kukosh, 1984 |
| | Two genes | Two complementary genes | Anashchenko & Duka, 1985b |
| | Three genes | Three, independent, & complementary | Anashchenko & Duka, 1985b |
| PET2 | Two dominant | RPET2, CMG1 | Kural & Miller, 1990 |
| | Two complementary | CMG1 | Whelen, 1980 |
| GIG1 | One dominant | RGIG1, CMG2 | Kural & Miller, 1990 |
| MAX1 | Two complementary | RMAX1, CMG3 | Kural & Miller, 1990 |
| | One dominant | RHA 274 ($Rf_1$) | Kural & Miller, 1990 |
| ANT1 | One dominant | Rf-ANT | Iuoras et al., 1989 |
| ANN2 | One dominant | P21, RMAX1, PI413178 | Jan, 1991 |
| ANN3 | One dominant | P21, RPET2, PI413180, & RHA 280 | Jan, 1991 |

restoration and tentatively named the gene $Rf_2$. Enns et al. (1970) working with Canadian lines derived from wild H. annuus and cultivated sunflower crosses, also reported control of fertility restoration by a single, dominant gene. Leclercq (1971) derived restoration from progeny of a H. annuus and H.

petiolaris cross and reported that restoration was controlled by a single dominant gene, Msc 1. Fick et al. (1974) crossed the fertility restorer lines Acc (Mo) 1338 from Canada and bc1-17-2-2 from France with a line derived from T66006-2. Their results suggested that the Canadian line carried the $Rf_1$ gene, but that the French source of restoration carried a gene or genes different from $Rf_1$.

Kinman (personal communication in Fick and Zimmer, 1974) reported that a second dominant gene, complementary to $Rf_1$, was necessary for full expression of restoration. He found a 9:7 (fertile:sterile) ratio from the $F_2$ population cross between CMS PET1 and a 1970 R composite line, and designated this gene $Rf_2$. He indicated that several cytoplasmic male-sterile female parents with the PET1 cytoplasm were homozygous $Rf_2Rf_2$. Since only one gene, $Rf_2$, was present, the female parents still expressed complete sterility. Fick and Zimmer (1974b) confirmed the presence of two complementary dominant genes in crosses of the nonoilseed lines CMS HA 267 and RHA 280.

Vranceanu and Stoenescu (1978) determined allelic relationships between several sources of male fertility restoration genes. Crosses between MZ-1398 and T66006-2-1-B indicated that MZ-1398 has a different restoration gene than the $Rf_1$ gene in T66006-2-1-B. They confirmed that T66006-2-1-B and MO-1338 from Canada both have the $Rf_1$ gene. In additional crosses, pollen fertility resulted from the complementary action of more than two genes in three lines, and three complementary genes in four lines. Genetic control of pollen fertility restoration appeared to be by the cumulative action of two nonallelic dominant genes, giving a ratio of 9:6:1 (fertile:partially fertile:sterile) in the $F_2$ segregating generation. This study and the data reported by Fick et al. (1974) indicate that the restoration factor derived from the lines developed by Leclercq was different than $Rf_1$ and $Rf_2$. Since most lines with $Rf_1$ and $Rf_2$ could be traced to wild H. annuus and the French restorer to wild H. petiolaris, this assumption appears valid.

Dominguez-Gimenez and Fick (1975) reported four fertility restoration genes among several wild accessions of H. annuus and H. petiolaris, and proposed that at least two dominant alleles would need to be present at any of the four loci for complete fertility restoration to be expressed. One of the genes appeared to be present in the female parent, CMS HA 89(PET1), confirming the conclusions of Kinman and Fick and Zimmer (1974), that many of the cytoplasmic male sterile lines have a restoration factor.

Artemenko (1987) reported that a single dominant gene controlled restoration of the cytoplasmically male-sterile line OD2625. The origin of the restorer gene was from Progress, derived from the interspecific cross H. tuberosus and cultivated H. annuus. This gene, and the restorer gene found in H. petiolaris, were located on homologous chromosomes, and no crossing over between them occurred.

Kukosh (1984) reported a single dominant gene, $Rf_L$, which restored male fertility to the cytoplasm derived from H. annuus spp. lenticularis, or CMS ANL1. $Rf_L$ was ineffective in restoring CMS PET1. However, several maintainer lines of PET1 did restore ANL1. The gene is functionally distinguished from $Rf_1$ or $Rf_2$ in that it produces normal, but sterile, anthers when present in the genome with PET1 cytoplasm. This research confirmed the presence of restoration (Rf) factors in the CMS PET1 parents.

Extensive research by Anashchenko and Kukosh (1984) and Anashchenko and Duka (1985a, 1985b) confirmed the complex genetic control of pollen fertility restoration in sunflower. Fourteen restorer lines were investigated for genetic control of the PET1 cytoplasm. Five were found to control restoration by a single dominant gene. Five other lines controlled restoration by two independent dominant genes and four lines controlled restoration by two complementary dominant genes. CMS ANL1 was restored by 11 of 27 lines by a single dominant gene. However, other lines controlled restoration by three nonallelic, dominant genes, two complementary dominant genes, and three complementary dominant genes. One line controlled fertility by two nonallelic genes with cumulative effects, with three phenotypic classes observed in the $F_2$ generation in the ratio 1:2:1 (fertile:partially fertile:sterile). They suggested that restoration of CMS ANL1 is more complex than CMS PET1, with up to three pairs of restorer genes involved. Working with restorers for both CMS ANL1 and CMS PET1, Anashchenko and Duka (1986) found lines which have two nonallelic restorer genes which restore male fertility to both CMS ANL1 and CMS PET1. In addition, several restorer lines were identified with allelic restorer genes for CMS PET1.

$BC_8$ lines CMS HA 89(PET2), CMS HA 89(GIG1), and CMS HA 89(MAX1) were developed by substituting the genome of HA 89 into the cytoplasm from sterile plants identified in the CMG-1, CMG-2, and CMG-3 composites, respectively (Miller and Wolf, 1991; Whelen and Dedio, 1980). Fertile plants within the three composites were utilized in crosses to produce the restorer

lines, RPET2, RGIG1, and RMAX1 (Miller and Wolf, 1991). The three CMS sources were crossed with their respective restorers to study inheritance of restoration (Kural and Miller, 1990). The analysis of $F_2$ and $BC_1F_1$ progenies indicated that two dominant genes in RPET2 were necessary for full restoration. This conclusion differed from Whelen's, who in preliminary studies indicated two complementary dominant genes were necessary for full restoration. One dominant gene in RGIG1, and two independent complementary dominant genes in RMAX1 controlled restoration to their respective cytoplasms. RHA 274 ($\underline{Rf_1}$) was not effective in restoring the PET2 and GIG1 cytoplasms; however, one dominant gene in RHA 274 controlled restoration of CMS HA 89(MAX1).

A new CMS source derived from H. annuus spp. texanus was found to be stable under different environmental conditions and was named CMS ANT1 (Iuoras et al., 1989). In studies of fertile plants found in crosses of CMS ANT1 with H. annuus spp. texanus, a single dominant gene was found to control pollen fertility restoration. This restorer was named Rf-ANT.

Cytoplasmically male sterile plants were found in the H. annuus accessions PI 413178 and PI 413180 (Jan, 1988). These stable CMS sources were named ANN2 and ANN3, respectively. The CMS sources were crossed with 11 restoration testers and fertile plants derived from PI 413178 and PI 413180 (Jan, 1991). P21, RMAX1, and fertile 413178 restored CMS ANN2 by a single dominant gene. P21, RPET2, and fertile 413180 restored CMS ANN3 by a single dominant gene, and RHA 280 restored CMS ANN3 by two complementary dominant genes.

Restoration of cytoplasmic male sterility in sunflower is controlled by a complex array of genes, with variable reactions between the genes and cytoplasms. Inheritance studies are complicated by complementary genes existing in several female lines, as well as genes present in the restorer lines. Complex allelism studies will be necessary to distinguish among genes and to determine relationships between those genes and several cytoplasms.

A new technique may be useful in distinguishing genes utilizing Restriction Fragment Length Polymorphism (RFLP) tests of mitochondrial DNA. Crouzillat et al. (1991) reported that three restriction enzymes and 12 probes permitted distinction of 13 cytotypes of sunflower. Each CMS differentiated

by the pattern of restoration using several lines closely corresponded to the cytotypes differentiated by RFLP of mtDNA. The identification of the mitochondrial genes responsible for cytoplasmic male sterility should make it possible to identify some nuclear genes necessary for restoration. For example, since the same mitochondrial gene appeared to produce CMS in PET1 and ANO1, the genes found to restore CMS PET1 should also restore CMS ANO1. Results indicated that 80% of the lines that restored PET1 CMS also restored ANO1. An additional technique of crossing several CMS sources with a number of lines and comparing fertility of $F_1$ hybrids differentiated groups of cytotypes with little time and resources (Havekes et al., 1991). This technique may give researchers information about which lines may be effective restorers for a CMS grouped similarly with another CMS.

## DROUGHT TOLERANCE

Studies on drought tolerance in sunflower have determined that this characteristic is a complex trait, controlled by factors of the leaf and root, many affecting physiological aspects of the plant. The following discussion is a summary of research conducted on several of these traits.

Fereres et al. (1983) determined that the only true reliable method in determining whether a line or hybrid has drought tolerance is to measure the ratio of drought stress yield to irrigated yield (Table 10). The ratio would be a measure of drought stress, if the irrigated yield accurately determines the ultimate yield potential of that cultivar. In a study of several genotypes, Fereres et al. (1983) determined that soil water extraction capability differs considerably among genotypes, and rooting depth varied from 180 to 270 cm.

Tuberosa et al. (1985) studied stomatal density and size on sunflower leaves. They determined that stomatal frequency among genotypes was consistent. Yield or agronomic benefits might be obtained by selection for reduced stomatal frequency and size on leaves of genotypes used in breeding for drought tolerance. Turner and Begg (1981) determined that maintenance of turgor pressure or dehydration tolerance would maintain high rates of photosynthesis. The measurement of turgor pressure could identify genotypes with mechanisms for drought escape.

Table 10. Inheritance of characteristics, or relationships of various characteristics with drought tolerance in sunflower.

| Inheritance or relationship of characteristic | Reference |
|---|---|
| Ratio of dryland yield to irrigated = overall measure of drought tolerance. Soil water extraction 180-270 cm | Ferres et al., 1983 |
| Low water use efficiency = low stomatal resistance, reduced stomatal freq. & size | Tuberosa et al., 1985 |
| Maintenance of turgor pressure, dehydration tolerance=maintenance of photosynthesis | Turner & Begg, 1981 |
| LAI and LAD correlated with yield under drought conditions | Merrien et al., 1982 |
| Seed germination and seedling growth under high osmotic pressure | Terbea, 1979 |
| High osmotic potentialwas controlled by partial dominance and over-dominance gene action. Tolerance to heat was controlled by partial dominance gene action. | Kamali & Miller, 1982 |
| Trichome length, density, and stiffness was controlled by several recessive genes in H. argophyllus. | Harada & Miller, 1982 |
| Inheritance of pubescence was polygenic and rate of water loss 39% less. | Iuoras & Voinescu, 1984 |
| Lower leaf water potential and higher photosynthetic activity for a given leaf water potential in H. argophyllus. | Morizet et al., 1984 |
| GCA controlling canopy temperature was significant in female parent and high in heritability. Additive gene effects were important. | Alza et al., 1990 |
| Salt tolerance may be an indication of drought tolerance - GCA and SCA significant, with high additive and domiance genetic effects | Hardwick & Ferguson, 1978 |

Leaf area index (LAI) and leaf area duration (LAD) were highly correlated with yield under drought conditions (Merrien et al., 1982). Therefore, genotypes with increased leaf area and the ability to maintain the established leaf area during seed filling growth stages are important characteristics for drought tolerance.

Terbea (1979) found good correlations of seedling growth and seed germination under high osmotic pressure with drought tolerance. Preliminary results indicated that crossing two inbred lines with tolerance to high osmotic pressure may produce drought and high temperature tolerant F1 hybrids. Hybrid tests also indicated that osmotic pressure was under intermediate inheritance and lacked full dominance when comparing crosses of two inbred lines with high osmotic pressure with crosses of inbreds with high and low osmotic pressure. High osmotic pressure was induced by a mannitol solution.

Kamali and Miller (1982) studied the inheritance of heat tolerance and tolerance to high osmotic potential of inbreds and their hybrids. Tolerance to heat and ability of seedlings to recover after stress was controlled by partial dominance gene action. High osmotic pressure, induced by a mannitol solution, was controlled by partial dominance and over-dominance gene action. However, the two screening methods had little similarity in identifying the same genotypes. Only genotypes which had tolerance to both tests were suggested for use in a breeding program.

Harada and Miller (1982) suggested that density and type of trichomes on leaves were an important characteristic contributing to drought tolerance. Helianthus argophyllus has been suggested as a potential source for trichome variability. Genetic inheritance studies of trichome length, density, and stiffness indicated that each characteristic was simply inherited and controlled by one to two recessive genes. Heritability estimates calculated by using variance of parents and progenies of crosses indicated high degrees of heritability. Additive genetic variance accounted for a major portion of the observed genetic variation.

Helianthus argophyllus trichome characteristics were selected in $F_6$ plants derived from crosses between H. argophyllus and cultivated sunflower (Morizet et al., 1984). Plants with the H. argophyllus trichome type wilted more rapidly and at higher leaf water potential than the H. annuus type.

These plants also had lower leaf water potential and photosynthetic activity was higher for a given leaf water potential.

Iuoras and Voinescu (1984) crossed H. argophyllus with H. annuus and studied inheritance of pubescence. Segregation patterns of progeny suggested that inheritance of pubescence was polygenic. Moisture loss was less rapid in plants exhibiting dense pubescence, compared with moisture loss of H. annuus trichome types. The rate of water loss was 39% less in the most resistant hybrid population. They concluded that this wild species represents a useful source of drought tolerance.

Canopy temperature may be considered an indirect measure of water stress, with a genotype cooler in canopy temperature considered more tolerant to stress (Alza et al., 1990). Inbred lines and hybrids were tested for canopy temperature under irrigation and dryland conditions in southern Spain. A significant negative correlation was found between canopy temperature and yield, verifying this hypothesis. Biomass was found to be negatively correlated with canopy temperature, as genotypes with higher biomass and leaves have greater evaporative surface. GCA controlling canopy temperature of the female parents was significant whereas GCA of the male parent was nonsignificant, as was SCA. Therefore, additive genetic effects were more important than non-additive effects in controlling canopy temperature. High heritability among the female parents indicates breeding and selection may be more effective in female genotypes.

A study was conducted to ascertain if cultivars, inbred lines, and their $F_1$ hybrids exhibited differential responses to salt levels during germination and hypocotyl growth periods (Hardwick and Ferguson, 1978). Ability to grow in high salt conditions showed high heritability. GCA of the males and GCA of females were significant, as well as the SCA, indicating high additive and dominance genetic effects were present.

## ALTERNATIVE FATTY ACID CONTENT IN SUNFLOWER OIL

The quality of sunflower oil generally is associated with the relative content of linoleic fatty acid. However, the development of alternative fatty acid contents in the oil has stimulated new markets, thus providing new interest in growing sunflower. The most successful of these alternatives has been high oleic fatty acid sunflower oil, with levels higher than 800 g $kg^{-1}$ of oil.

The development of a sunflower with a high oleic acid content was first reported by Soldatov (1976), after seed treatment with dimethyl sulfate. Crosses with plants derived from the high oleic cultivar, Pervenets, with low oleic lines resulted in $F_2$ progenies having three classes: high, intermediate, and low oleic percentage (Fick, 1984). By combining the high and intermediate classes, a satisfactory fit for a 3:1 ratio was obtained. Thus, high oleic acid content was determined to be controlled by a single partially dominant gene, designated Ol (Table 11). This genetic control has been confirmed by researchers in the USA, Spain, and Germany (Urie, 1985; Miller et al., 1987; Fernandez-Martinez et al., 1989; Popov et al., 1988; Schmidt et al., 1989).

Miller et al. (1987) determined that a modifier gene, acting recessively, influenced oleic contents, particularly in the intermediate and high oleic classes. When the recessive gene, ml, was present in homozygous condition, and combined with the gene, Ol, oleic levels in seed were elevated to 820 g

Table 11. Inheritance of fatty acid and tocopherol content in sunflower oil.

| Fatty acid | Gene | Gene action | Reference |
|---|---|---|---|
| High Oleic | Ol | Single, partially dominant | Fick, 1984 |
| | Ol | Single, dominant | Urie, 1985 |
| | Ol, ml | Single, dominant and recessive modifier | Miller et al., 1987 |
| | Ol | Single, dominant | Schmidt et al., 1989 |
| | $Ol_1$,$Ol_2$,$Ol_3$ | Complementary dominant | Fernandez-Martinez et al, 1989 |
| Tocopherol | $tph_1$,$tph_2$ | Two recessive | Popov et al., 1988 |
| High, stable Linoleic | | Partially recessive, with maternal influence | Simpson et al., 1989 |
| High Palmitic | | Low palmitic controlled by partial and incomplete dominance | Ivanov et al., 1988 |

$kg^{-1}$ of oil or higher. The action of this gene was particularly evident when intermediate $F_2$ plants were self-pollinated to attempt to find high oleic segregants. The number of $F_3$ segregants in the high oleic class was lower than would be expected if a single dominant gene was controlling high oleic expression.

Fernandez-Martinez et al. (1989) also studied the genetic control of plants in the intermediate range. $F_2$ ratios of 9:3:4 (high:intermediate:low) indicated the presence of two complementary dominant genes, $\underline{Ol}_1$ and $\underline{Ol}_2$. Two $F_3$ populations were found to segregate 27:9:28, leading the researchers to postulate that a third gene, $\underline{Ol}_3$, was also controlling oleic acid.

Differences in the studies could be explained by two factors. Fick, Urie, and Miller et al. utilized plants self-pollinated directly from Pervenets, whereas Fernandez-Martinez et al. used high oleic segregants after crossing and backcrossing to low oleic inbred lines. Fernandez-Martinez et al. suggested that in the low oleic acid parents, variability may exist in $\underline{Ol}$ alleles at some of the three loci, leading to different conclusions when different lines are utilized in genetic studies. Also, it has been found that environments substantially influence the expression of the oleic content of the seed (Robertson et al., 1979). This is particularly critical in the evaluation of the intermediate class. Miller et al. (1987) evaluated $F_2$ seeds from plants grown in a cool maturation environment, whereas Fernandez-Martinez et al. (1989) and Urie (1985) evaluated seed from plants grown in a relative warmer environment. Different genetic interpretations can be made depending on the number of seeds classified as having intermediate oleic levels. One point should be emphasized; there may be many genes involved in modifying the oleic content of sunflower.

Popov et al. (1988) determined that tocopherol biosynthesis was controlled by recessive alleles, $\underline{tph}_1$ and $\underline{tph}_2$. The $\beta$ and $\gamma$ forms in the tocopherol complex reached 60% and 95%, respectively, in lines homozygous for the two alleles. Oils of these lines are more stable to oxidation in comparison with conventional lines since the $\beta$ and $\gamma$-tocopherol possess a powerful anti-oxidative effect. Combining these two alleles with the $\underline{Ol}_1$ gene controlling high oleic could provide even higher stability of sunflower oil to oxidation.

Inbred sunflower lines with higher than normal and stable levels of linoleic acid were tested in a wide range of maturation temperatures in

Australia (Simpson et al., 1989). The inheritance of high linoleic acid content was controlled mainly by a partially recessive gene with maternal influence. To maximize linoleic acid levels in hybrids, it will be necessary for both parents to have germplasm high in linoleic acid.

Ivanov et al. (1988) utilized gamma-rays on dry seeds to produce sunflower with palmitic fatty acid contents 4-5 times higher than in present sunflower varieties and hybrids. Inbred lines averaging palmitic contents of 402 g $kg^{-1}$ of oil have been produced. Genetic analysis of progeny of crosses between the high palmitic inbred lines and low palmitic parents indicated partial and incomplete dominance controlled low palmitic fatty acid. Hybrids produced by crossing low and high parents were 104 to 131 g $kg^{-1}$ in palmitic acid compared to 70 and 306 g $kg^{-1}$ for the low and high parents, respectively. These results indicate that it will be necessary for both parents to have high palmitic acid to produce a high palmitic hybrid. Oil high in palmitic acid will be stable under high temperatures, and may be a substitute for palm oil in production of soap products.

Interest in producing sunflower oil with very short chain fatty acids for utilization as industrial oil may stimulate additional specialized quality markets for sunflower.

## CONCLUSION

Genetic control of some characteristics, such as resistance to downy mildew and rust, appear to be relatively straight forward and controlled by single dominant genes. However, a number of characteristics, such as height, resistance to Sclerotinia, and drought tolerance are quantitatively controlled. Head inclination, male fertility restoration, and resistance to Phomopsis and Verticillium appear to have mixed inheritance. Modifier genes are important in controlling high oleic fatty acid. These arrays of inheritance make it more important for a breeder to understand the control of a trait. Certainly, molecular techniques could be a great asset to our knowledge of inheritance in the future. At the present time, though, we have a tremendous amount of knowledge available to utilize in making significant improvements in sunflower.

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