RESISTANCE TO WHITE RUST (Albugo tragopogonis) and EVIDENCE OF MULTIPLE GENES

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ABSTRACT

USDA sunflower germplasm, including 1168 Plant Introductions and 100 released inbred lines, were evaluated for resistance to leaf pustules and other symptoms caused by *Albugo tragopogonis* in a three-year study in South Africa utilizing natural infection during late season plantings. Nearly 83% of the Plant Introductions were totally susceptible, with eight accessions rated as immune or highly resistant to both leaf, stem and petiole infection. Evaluation of released USDA inbred lines revealed some genotypes with resistance to one *Albugo* symptom and susceptibility to other symptoms. For example, leaf pustules were restricted to lower leaves and expressed as small lesions on some lines, while petiole and stem lesions were large and widespread on the plant. In other instances, *Albugo* may cause lesions on bracts or on the backs of heads, or both, and these infections may occurr in the presence or absence of leaf and stem susceptibility. The only accession identified to date with near immunity to leaf, stem, petiole, bract and head lesions is Ames-3430, a breeding line from Russian designated as VIR 107.

INTRODUCTION

White rust of sunflower, caused by *Albugo tragopogonis* (DC) S.F. Grey, is characterized by large (5-8 mm diameter) chlorotic, blister-like pustules on the upper side of leaves, with abundant white sporangia borne on the lower leaf surface. Additional symptoms of white rust, associated with the sexual stage of the fungus, have been observed in Australia (Allen and Brown, 1980), Argentina (Delhey and Kiehr-Delhey, 1985), and more recently in South Africa (Van Wyk *et al*., 1995). These bruise-like lesions on petioles, stems, and receptacles are caused by an abundance of black oospores just beneath the epidermis, and totally lack any asexual spores. Lesions on petioles and stems, referred to as petiole greying and grey stem spot, respectively, have resulted in severe defoliation and lodging in South Africa (Van Wyk et al., 1995). White rust has generally been considered as economically unimportant in most countries in the past, but is now considered to be one of the most important sunflower diseases in South Africa. White rust has also recently been found on sunflower in France (Penaud & Perny, 1995), Hungary (Zoltan, 1995), and the United States (Gulya, 1999). This report summarizes the resistance found in the 3-year study, and lists the various symptom types we have observed.

MATERIALS AND METHODS

USDA sunflower germplasm was planted in Potchefstroom, South Africa for three consecutive years, beginning in 1998, in late January to coincide with the rainy, summer weather that is conducive for infection and symptom development by *A. tragopogonis*. All trials had three replications of single row plots of 20 to 25 plants per row. The commercial hybrid Hysun 333 was used as a resistant check while the USDA inbred RHA 274, a multi-headed restorer, was included as a susceptible check. Multiple strips of both checks were planted throughout the field plots to verify uniformity of natural inoculum. Supplemental

overhead irrigation was applied to ensure conditions were optimum for continual infection and symptom development. Plants were evaluated for white rust symptoms at flowering, and again at physiological maturity. White rust severity was evaluated on a whole-row basis, using the percentage of leaf area covered on a '1-9' scale as follows: 1= immune, 3= resistant, with <5% leaf coverage, 5= moderately susceptible, with 5-40% leaf coverage, 7= highly susceptible, iwht 40-80% coverage, and 9= lethal). Genotypes with a mean leaf score <2 were further evaluated for petiole, stem and head lesions, and were also retested the following year.

RESULTS

Of the 1068 USDA Plant Introductions tested in the 3-year study, 83% were rated as susceptible (>5 on the 1-9 scale), with only 1.5% rated as immune or highly resistant. Entries rated as susceptible to leaf blisters were nearly always observed with numerous stem, petiole and head lesions also. Since many of the entries were segregating for leaf reaction, it was more practical to evaluate on a whole row basis. The frequency of *Albugo* resistance is summarized in Table 1. There were eight entries which had average Albugo ratings of 2 or less in two year's trials. These lines, with origin and background in parenthesis, were Ames-3285 (US, ornamental), Ames-3398 (Romania, Romsun C-5357), Ames-3430 (Russian, VIR 107), Ames-3437 (Argentina, Charata), Ames-18918 (Russia, VIR 425), PI 386230 (Russia, VIR 847), PI 431547 (Yugoslavia) and PI 490315 (Germany, ornamental).

During evaluation of the USDA inbred lines, it became obvious that there were some genotypes which were either immune or highly resistant to foliar infection, and yet had stem and petiole lesions. We also noticed that lesion size of foliar pustules varied, as did the location of infected leaves on the plants. The following is a listing of the various symptom types we have observed, in addition to the typical, large foliar pustules.

1. Foliar lesions confined either to the lower or to the upper portion of the plant:

. a). Large, fully susceptible leaf pustules confined to the lower foliage This symptom type is not common, but is seen in RHA 398 and HA 207.

b). Pinpoint lesions confined to the lower foliage with pustules in the 1-2 mm diameter range, as contrasted to 5-10 mm in fully susceptible genotypes . This symptom type is commonly expressed in RHA 362, HA 384, and Ames-3300.

c). Large, fully susceptible leaf pustules on the upper foliage, with little or no leaf symptoms on the lower foliage. This symptom is illustrated by HA 286, HA 290, and HA 305.

d). Pinpoint lesions only on the upper foliage. This symptom expression, which we interpret as a highly resistant reaction, is seen in HA 379, HA 402, and HA 407.

2. Petiole lesions may be found on all levels of leaves in totally susceptible genotypes, or may be limited to the lower leaves. Inbreds displaying petiole lesions only on lower leaves included RHA 340, RHA 377, RHA 379, HA 402 and HA 407. Genotypes with only pinpoint foliar lesions, but petiole lesions on all leaves include

RHA 276 and HA 380. Additionally, genotypes susceptible to petiole infection may have lesions limited to the petiole itself, or in cases of extreme petiole susceptibility, there may be large lesions in the axils, as exemplified by RHA 362, HA 207, and HA 384. The extreme case of petiole susceptibility is exemplified by HA 408, which shows systemic lesions along the entire petiole length.

3. Stem lesions are generally limited to the lower stem but in some cases we have observed lesions occurring along the entire stem, as seen in RHA 274, HA 290 and HA 302. Additionally, in some sunflowers, a galling due to hypertrophy has been noted on the lower stem, as seen in HA 290 and Ames-3300. There are a few rare genoytpes, such as the South African line AP 891/66, derived from an old Romanian composite, and the discontinued hybrid Carnia 1204, which have such severe stem lesions that lodging often results due to rind degradation associated with lesions at the base of the plant (Kruger et al., 1999).

4. On fully susceptible sunflower genotypes, it is common to observe bract lesions , with abundant asexual sporulation. HA 362, HA 379, and HA 384 are examples of genotypes which are highly resistant to foliar infection, but have severe bract infection, while RHA 276, RHA 408, HA 385 and HA 404 have both severe foliar and bract infection. The presence of bruise-like lesions (having only oospores) on the back of the receptacle may be independent of bract lesions, as seen in RHA 398 and HA 290, or both lesion types may be observed in RHA 276, RHA 408 and HA 207.

DISCUSSION

From observations made of a diverse group of sunflower genotypes under optimal conditions for natural infection by *A. tragopogonis*, it is apparent to us that sunflower genotypes can manifest infection by several, and oftentimes independent symptoms. The independence of these symptoms, where fully susceptible genotypes display all lesion types throughout the plant, suggests that genetics rather than environment are responsible for the different symptom manifestations. We hypothesize that several to many genes may be involved in conferring resistance to *A. tragopogonis*. First, there may be one gene group governing leaf pustule size, and another gene group governing resistance in the lower and upper foliage layers. Second, the gene(s) governing petiole lesions and distribution along the foliage appears to be independent of the genes governing foliar infection. Third, stem lesions appear to fall into three categories: (a) those primarily on the lower stem, (b) severe lower stem infections resulting in either galling or lodging, and (c) stem lesions occurring along the entire stem, oftentimes with lesions exceeding 10-15 cm. Finally, it appears that the incidence of bract and head lesions can occur independently of each other, and also independent of foliar symptoms. The exact number of genes involved, and the gene action will await inheritance studies, but the examples provided above could serve as the foundation for such studies.

Several sunflower genotypes were identified with high levels of resistance, or in one case, immunity. Those lines displaying pinpoint foliar lesions, petiole and stem lesions limited to the lower foliage, and no bract or head lesions could be used as excellent sources of resistance. Some of these lines also combine resistance to other diseases. For example, RHA 340 has multi-race immunity to *Plasmopara halstedii*, and HA 207 was selected for

resistance to *Macrophomina phaseolina*. The two entires most resistant to *Albugo* infection among the 1300 accessions tested were USDA line HA 380, a very early, reduced height inbred, and the Russian breeding line VIR 107 (catalogued as Ames-3430).
An additional question to be studied is whether the symptom types observed in these evaluations are due to a fungal biotype unique to South Africa, or if these symptoms are reproducible in other countries such as Argentina and Australia where *A. tragopogonis* is commonplace.

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Table 1. Frequency of *Albugo* leaf blister reaction classes of 1168 USDA Plant Introductions tested under natural infection in South Africa over a three-year period.

Leaf rating	**1-2***	**2-3**	**3-4**	**4-5**	**5-6**	**6-7**	**7-8**	**8-9**
% of entries	0.8	0.7	4.4	12	46	24	12	1
# entries	8	7	47	128	489	255	128	106

Leaf rating scale: ‘1'= immune, ‘3'=resistant, or <5% pustule coverage, ‘5'= moderatelysusceptible, or 5-40% leaf coverage, ‘7'= highly susceptible, or 40-80% leaf coverage, and ‘9'= lethal

Table 1. Severity and distribution of white rust (*Albugo tragopogonis*) symptoms on selected sunflower genotypes, illustrating the independence of occurrence of the individual symptoms.

Line	Pedigree	Leaf Pustules[1]		Petiole[2]	Stem[3]	Head[4]
		Size	Dist.			
RHA 276	T66006-2-2-11-3-2 cross	PP	U	A,AX	A	H
RHA 340	HA89/H. argophyllus	PP	L	L	L	0
RHA 377	RHA299/Sorem HT58 (Rom)	S	L	L	0	H
RHA 408	R-Line Pop. (Romania)	S	A	A,AX,S	0	B,H
HA 379	HA821/DDR (Germany)	PP	U	L	L	0
HA 380	USDA1858/HA89	0	0	L	0	0
Ames 3300	Funksinka 10 (Germany)	PP	L	L	L,G	0
Ames 3430	VIR 107 (Russia)	0	0	0	0	0

[1] Leaf pustule size designations: S= large, susceptible pustules; PP= pinpoint; 0=absent. Distribution (dist): L= lower leaves only; U= upper leaves only; A= all leaves;

[2] Petiole lesion designations: A= found on all leaves; L= limited to lower leaves; AX= axillary lesions also present; S=systemic infection of entire petiole; 0= petiole lesions absent.

[3] Stem lesion designations: L= lower stem only; G= galling present; A= along entire stem; 0= stem lesions absent;

[4] Head lesion designations: B= bract infection only; H= lesions on back of receptacle.