ATTACK OF SUNFLOWER TERMINAL BUDS BY *SCLEROTINIA SCLEROTIORUM*, SYMPTOMS AND RESISTANCE.

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SUMMARY

In 1980, widespread early attacks of sunflower by *Sclerotinia sclerotiorum* were observed in France, the symptoms being destruction of the terminal bud and young leaves. These symptoms were observed again in 1984 and more particularly in 1987, 1990 and 1991 on large areas and many varieties, with significant yield losses.

These attacks are caused by ascospores and plants are susceptible from the 6-8 leaf stage. However the climatic conditions necessary for infection are not yet known precisely.

Observations of natural attack have shown that significant differences in genetic resistance are available and results are highly repeatable between locations and years. In contrast with capitulum attack the heredity of resistance appears quite straightforward. Certain inbreds are completely resistant, others are highly susceptible. Crosses between resistant lines give resistant hybrids, between susceptibles, susceptible hybrids. However, it is not yet possible to predict exactly the reaction of hybrids between resistant and susceptible lines.

INTRODUCTION

Sclerotinia sclerotiorum causes several different forms of disease on sunflowers. LAMARQUE (1985), and REGNAULT *et al* (1991) describe :

* White head rot, which may cause complete destruction of capitula before maturity.
* Wilting of the whole plant due to invasion of the root system by *Sclerotinia*
* Wilting of part of the shoot, following destruction of the stem after infection of leaves
* Destruction of the terminal bud.

In France, this last form of attack was first identified in 1980, when certain fields showed significant yield losses. At first often mistaken for attacks by *Botrytis cinerea*, this type of attack is not specific to France, but, probably for climatic reasons, economic losses are greatest in this country.

Genotypic reaction is very different from that concerning other forms of attack by *Sclerotinia*. Certain varieties which show good levels of resistance to head or root attack appear very susceptible to infection of terminal buds.

Observations of natural attacks have been made since 1984. Based on experience of other forms of attack (VEAR and TOURVIEILLE, 1985), this paper defines the symptoms of terminal bud attack, describes methods to determine resistance levels and first ideas concerning the heredity of resistance.

MATERIALS AND METHODS

Sunflower genotypes : These were all hybrids, either commercial varieties or experimental combinations.

Trials : All the observations presented are of natural attacks in normal sunflower cultivation conditions, in yield trials for combining ability or varietal registration. The trials were randomized block or lattice designs, with 2 to 4 replications and plots of 50 to 100 plants. Observations were taken into consideration if at least one hybrid showed at least 10% attack.

In order to make comparisons between hybrids present in different trials, an index was calculated :

$$\text{Index} = \frac{\text{Percentage attack of the hybrid}}{\text{Percentage attack on a control variety MIRASOL}}$$

For studies of heredity, hybrids obtained from a certain number of known inbred lines were examined.

SYMPTOMS AND EPIDEMIOLOGY

Infections are caused by the sexual form of *Sclerotinia* : ascospores, produced by apothecia at the soil surface (ACHBANI *et al*, 1992). Sunflower plants are susceptible to this form of attack from the 6 leaves to 2cm flower bud stages. Contamination starts at the extremities of the young leaves which form a rosette around the terminal bud. The funnel-like form of this rosette maintains a certain humidity at the penetration sites.

If a humid period exceeds 40h, the young leaves rapidly show grey-brown zones at their tips. *Sclerotinia* can be isolated from these lesions. Three types of development of the disease are possible :

* the infection progresses rapidly to the terminal bud, which is destroyed. This occurs if a mild (>20°C), humid period follows infection.
* The terminal bud continues to grow, but the leaf rot spreads to

the stem, causing wilting and loss of the apical part of the plant. These symptoms are observed when the period after infection is cool and humid.

* Spread of the disease is stopped by a dry period, which allows healing and drying of lesions.

Losses due to disease thus depend on climatic conditions. If only leaves are attacked, a dry period can stop the disease, so that there are no yield losses. In contrast, if humid conditions continue, spread of the disease will destroy the terminal bud and the plant may produce no seed at all.

GENOTYPIC REACTIONS

Definition of varietal reactions requires a valid observation method. Observations of natural attack can only be used if reactions are repeatable in space and time. Fourty-nine pairs of observations of natural attacks of *Sclerotinia* on terminal buds, made between 1984 and 1991, were compared. Correlation analyses are presented in Table 1. They show that more than 90% of genotype classifications are correlated significantly. Natural attacks can therefore be used to determine varietal behaviour and to study the heredity of resistance.

Table 1. Study of the repeatability of observations of natural terminal bud attacks by *Sclerotinia* on sunflower yield trials.
49 trials in 5 years
79 possible comparisons = at least 6 common hybrids.

nb. pairs	correlation coefficients H.S.	S	N.S.	total nb. comparison
6	2	0	0	2
8	7	2	1	10
9	6	3	1	10
10	7	4	1	12
12	2	2	0	4
13	4	0	0	4
14	6	3	1	10
15	1	0	0	1
24	10	0	0	10
25	11	2	3	16
total	56	16	7	79

H.S. : highly significant ($P < 0.01$)
S. : significant ($P < 0.05$)
N.S. : non significant
nb. : Number

The behavoiur of some of the varieties most commonly grown in France is given in Table 2.

Table 2 Sclerotinia terminal bud susceptibility index for some sunflower varieties widely grown in France (index defined in Methods).

Variety	Nb. trials	Index	Groups
ALBENA	11	0.02	a
FRANKASOL	40	0.04	a
TOPFLOR	22	0.07	a
VIKI	39	0.89	b
MIRASOL	49	1.00	b
OSCAR	10	1.17	b
RODEO	11	1.34	c
VIDOC	11	1.69	c

HEREDITY OF RESISTANCE

In severe natural attacks on trials, three levels of resistance are generally found, illustrated by the results for 6 hybrids in Table 3 :

* almost complete resistance : HA821 * PSC6
* slight susceptibility : HA821 * PHU6, 2603 * PSC6, HA821 * PRH3
* susceptibility : 2603 * PHU6, 2603 * PRH3.

Table 3. Percentage *Sclerotinia* terminal bud attack on 6 experimental hybrids (1 trial, 1990).

females / males	HA821	2603
PHU6	17.4	82.2
PSC6	1.1	13.6
PRH3	19.6	48.9

Our hypothesis at present is that a hybrid between 2 resistant lines is resistant, one between 2 susceptible lines is susceptible, and one between a resistant line and a susceptible line is intermediate, generally

with a low level of infection. However, some combinations of resistant and susceptible lines are either highly resistant or highly susceptible (data not shown).

DISCUSSION

Early identification of *Sclerotinia* symptoms on terminal buds is essential if chemical control is to be undertaken. Some efficient compounds are known (PERES et al, 1991), but only curative chemical control would be economic and this would need to be at a very early stage.

In the absence of a satisfactory method of artificial infections, breeders can use observations of natural attack on yield trials.This situation is very different from that concerning capitulum attack, for which it has been shown that special trials with a specific methodology are neccessary (TOURVIEILLE and VEAR, 1984).

Genotypic reaction to *Sclerotinia* terminal bud attack shows clear differences. Some genotypes do not appear susceptible, and farmers can use them without risk, whilst other genotypes show levels of susceptibility such that attacks may be catastrophic for the farmer.

Heredity may be quite different from that of resistance to other forms of *Sclerotinia* attack (ROBERT *et al*, 1985; VEAR and TOURVIEILLE, 1988; TOURVIEILLE and VEAR, 1991). Heritability appears to be high, for inbred lines show the same behaviour as predicted from their hybrids. The great differences between levels of attack suggest that major genes could be involved. However, as for the other resistances, additive effects are apparent and it is not yet clear which combinations of resistant and susceptible lines will provide satisfactory hybrids. One of the most important differences compared with other forms of Sclerotinia attack is that a large proportion of modern cultivated sunflower genotypes show complete or almost complete resistance.

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REFERENCES

ACHBANI El H. & TOURVIEILLE D., 1992. Tournesol : revue bibliographique sur certaines de ses maladies cryptogamiques; 1er partie : La pourriture blanche. Al Awania (in press).

LAMARQUE C., 1985. Eléments de reconnaissance des maladies et accidents du tournesol rencontrés en France. Ed. I.N.R.A, France, 119 p.

PERES A., ALLARD L.M. & REGNAULT Y., 1991. *Sclerotinia sclerotiorum* du bourgeon terminal du tournesol : étude de la protection fongicide. A.N.P.P., IIIème Conf. Int. Maladies des Plantes, Bordeaux (France), 3-5 Déc., 401-405.

REGNAULT Y., DAVET P., PERES A., PENAUD A. & TOURVIEILLE D., 1991. Les maladies du tournesol. Les points techniques du CETIOM, ed. CETIOM - Paris, 72 p.

ROBERT N., VEAR F. & TOURVIEILLE D., 1987. L'hérédité de la résistance à *Sclerotinia sclerotiorum* chez le tournesol. I. Etude des réactions à deux tests mycéliens. Agronomie, 7, 423-429.

TOURVIEILLE D. & VEAR F., 1984. La sélection du tournesol pour une meilleure résistance au *Sclerotinia sclerotiorum.* Inf. Tech. CETIOM, 88, 5-23.

TOURVIEILLE D. & VEAR F., 1990. L'hérédité de la résistance au *Sclerotinia sclerotiorum* chez le tournesol. III. Etude des réactions à des infections artificielles sur racines et cotylédons. Agronomie, 10, 323-330.

VEAR F. & TOURVIEILLE D., 1985. Resistance to Sclerotinia sclerotiorum in sunflowers. Proc. XIe Int. Sunflower Conf. Mar del Plata, Argentine, 357-362.

VEAR F. & TOURVIEILLE D., 1988. Heredity of resistance to Sclerotinia sclerotiorum in sunflowers. II. Study of capitulum resistance to natural and artificial ascospore infections. Agronomie, 8, 503-508.