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YIELD LOSSES IN SUNFLOWER (*Helianthus annuus* L.) DUE TO HEAD ROT CAUSED BY *Sclerotinia sclerotiorum* (Lib.) de Bary

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SUMMARY

The objective of this work was to estimate yield losses in sunflower caused by sclerotinia head rot. Subplots with different levels of disease intensity (=percentage of plants with head rot) were generated by inoculations utilizing different environmental conditions and genetic materials (five commercial hybrids). Seed yield, seed oil content, weight per 1000 seeds, dockage percentage, and oil acidity were recorded in the inoculated subplots and in the healthy ones which were used as controls. Highly positive correlations between percentage of diseased plants and reduction in seed yield (r=0.76***), increase in dockage (r=0.67***) and oil acidity levels (r=0.58***) were found. Yield reduction estimates varied among hybrids; this indicates that they are genetically different for their tolerance to head rot. The obtained results permit to state that head rot causes direct (seed yield reduction) and indirect (increase in dockage and oil acidity levels) damages to the sunflower crop. An inverse relation would exist between the magnitude of both types of damages. That is, when capitula are completely disintegrated there would be no indirect damages, whereas if the development of the disease is slow (high genotypic tolerance, unfitted environmental conditions for the pathogen or both) indirect damages would be greater since more heads can be harvested. It is concluded that sunflower breeding for head rot control must be oriented to select those genotypes with better resistance levels (in terms of lower percentage of diseased plants) rather than to select genotypes with better tolerance levels (in terms of yield losses).

Keywords: Head rot, resistance breeding, sunflower, yield loss assessment.

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INTRODUCTION

Sunflower head rot incited by the ascomycete *Sclerotinia sclerotiorum* (Lib.) de Bary is one of the most important diseases of this crop in many countries (see Sackston, 1992; Maširević and Gulya, 1992, for reviews).

Many studies have been published concerning the distribution of this disease (Aćimović, 1988; Sackston, 1988), epidemiology (Lamarque et al., 1985), biochemistry of resistance (Hemery et al., 1988), genetic variability for resistance (Robert et al., 1985; Pereyra et al., 1991; see Miller, 1992, for a review), and cultural, biological and chemical methods of control (Maširević and Gulya, 1992). As far as we know, however, there are no reports of any research designed specifically to estimate yield losses caused by this disease. This is, precisely, the objective of this contribution.

MATERIALS AND METHODS

Five commercial hybrids (coded A, B, C, D and E) were used. Field trials were conducted in Ballenera (Buenos Aires, Argentina) in two sowing dates. In each of them, plots were arranged following a completely randomized block design with 3 replications. Each plot consisted of five rows, 6m long and 0,70m apart.

To quantify yield losses caused by head rot, different levels of disease intensity (=percentage of plants with head rot) were generated by inoculations in subplots (one row). Then, the yield of the inoculated row was related to the yield of the healthy subplot. Genetic materials with known differences in their resistance levels (unpublished previous results) and two different environmental conditions (sowing dates) were used to generate different levels of disease intensity (see Sah and Mac Kenzie, 1987, for a review).

The second row of each plot (ca. 20 plants) was inoculated with an ascospore suspension of the fungus, and the fourth was used as healthy control. Plants were inoculated at the beginning of the flowering stage using the "ascospore test" (Tourvielle et al., 1978). Flower surfaces were sprayed with an ascospore suspension (5000 ascospores/ml; 5ml per capitulum). Then, capitula were covered with paper bags which were removed two days after inoculation.

At harvest, data were recorded on the percentage of diseased capitula in the inoculated rows. Seed yield (11% moisture level), seed oil content (w/w), weight per 1000 seeds (grams), dockage percent (w/w), and oil acidity percent (w/w) were recorded in both subplots. Seed oil content was determined by nuclear magnetic resonance spectroscopy (Granlund and Zimmerman, 1975). Oil acidity percent (i.e.: free fatty acids - expressed as grams of oleic acid - per 100 grams of oil) was determined following the standard 5512 of IRAM (Instituto Argentino de Raciónalizacion de Materiales, 1988).

The following variables were calculated for each experimental unit:

$$\text{- Seed yield percent reduction} = 100 - \frac{\text{seed yield of inoculated subplot}}{\text{seed yield of control subplot}} \cdot 100$$

$$\text{- Dockage percent variation} = \text{dockage percent of inoculated subplot} - \text{dockage percent of control subplot}$$

$$\text{- Weight per 1000 seeds percent variation} = 100 - \frac{\text{weight per 1000 seeds of inoculated subplot}}{\text{Weight per 1000 seeds of control subplot}} \cdot 100$$

$$\text{- Oil percent variation} = \text{oil percent of inoculated subplot} - \text{oil percent of control subplot}$$

$$\text{- Oil acidity percent variation} = \text{oil acidity percent of inoculated subplot} - \text{oil acidity percent of control subplot}$$

Taking into account the Argentine marketing standards for sunflower (see below), the net yield (NY) for each row was calculated as:

$$NY = \text{seed yield} \cdot (0.002 \cdot \text{oil percent} - 0.009) - \text{dockage discount} - \text{oil acidity discount}$$

So, the net yield percent reduction of the inoculated subplots in relation to the healthy control can be expressed as:

$$100 - \frac{\text{NY of inoculated subplot}}{\text{NY of healthy control}} \cdot 100$$

Correlation and regression coefficients were calculated between plot data for each of these variables and the percentage of diseased plants of the same plot (N=30). Linear regressions were estimated using the model $y=b \cdot x$ (Steel & Torrie, 1980).

Commercialization standards for sunflower in Argentina are the following:

a.- Oil acidity: On the surplus of the basis (1.5%) 2.5% will be deducted on each percent or proportional fraction.

b.- Oil content: The commercialization basis is 44%. On the surplus of the basis, a bonus of 2% will be applied on every percent or proportional fraction. On values under the basis, a reduction of 2% will be applied on every percent or proportional fraction.

c.- Dockage: Up to tolerance (3%) a deduction of 1% will be applied on each percent or proportional fraction. Above the tolerance limit a deduction of 1.5% will be applied on each percent or proportional fraction.

RESULTS

The average percentage of diseased capitula in the inoculated subplots was 32.7 (± 27.5), with a minimum value of zero and a maximum value of 100 (Table 1).

Table 1: Means, standard deviations, minimum and maximum values for the percentage of plants with *Sclerotinia* head rot (PS), seed yield percent reduction (SYPR), dockage percent variation (DPV), weight per 1000 seeds percent variation (SWPV), oil percent variation (OPV), oil acidity percent variation (OAPV) and net yield percent reduction (NYPR) for 30 subplots inoculated with *Sclerotinia* with respect to their healthy control. Correlation coefficients between these variables and PS are also listed

Variable	Mean	Standard Deviation	Min.	Max.	Correlation Coefficient
PS	32.7	27.5	0.0	100.0	-
SYPR	17.2	16.1	-9.17	64.7	0.76**
DPV	1.8	1.5	0.09	7.9	0.67**
SWPV	2.5	7.6	-13.6	22.4	0.21ns
OPV	-0.7	1.6	-2.2	5.0	0.15ns
OAPV	1.8	1.7	0.0	6.63	0.58**
NYPR	25.8	17.3	-1.5	78.4	0.75**

Mean seed yield percent reduction of the inoculated subplots with respect to the controls was 17.2%. Such loss in yield presented a strong positive correlation ($r=0.76$; $P<0.0001$) with the percentage of diseased capitula (Table 1). Linear regression coefficient between both variables was 0.49 (±0.046, $P<0.0001$). This indicates that seed yield - taking the five hybrids as a group - decreases 5% for each 10% of increase of the percentage of diseased capitula (Table 2).

Table 2: Linear regression coefficients between the percentage of plants with *Sclerotinia* head rot and the variables: seed yield percent reduction (SYPR), dockage percent variation (DPV), oil acidity percent variation (OAPV) and net yield percent reduction (NYPR) for 30 subplots inoculated with *Sclerotinia* with respect to their healthy control

Variable	b	Standard error	Prob.	R^2
SYPR	0.49	0.046	0.0001	0.80
DPV	0.048	0.0051	0.0001	0.75
OAPV	0.046	0.0062	0.0001	0.79
NYPR	0.63	0.056	0.0001	0.83

Dockage percent variation among inoculated and healthy subplots presented a highly positive correlation ($r=0.67$; $P<0.0001$) with the percentage of diseased plants (Table 1). Regression coefficient between both variables was 0.048 (±0.0051, $P<0.0001$). Such estimate suggests that the percentage of seed impurities increases at a rate of 0.5 points over the control for each 10% of increase in the percentage of diseased plants.

It was found that the variation in oil acidity among inoculated and control subplots was correlated with the percentage of capitula affected with *Sclerotinia* (r=0.58, P<0.0001). The regression coefficient between both variables was 0.046 (±0.0062, P<0.0001) which suggests that oil acidity increases 0.5 percent over the control for each 10% of increase in the percentage of diseased plants (Table 1).

No association was found between the percentage of diseased plants and the variables oil content variation and weight per 1000 seeds percent variation.

Net yield percent reduction of inoculated subplots with respect to the healthy ones showed a strong positive correlation with the percentage of diseased plants (r=0.75, P<0.0001). Net yield percent reduction averaged 25.8% with a minimum value of zero and a maximum value of 78.4 (Table 1). These values were higher than those obtained for the reduction in seed yield because net yield includes not only seed yield losses but the discount for the increase in dockage and oil acidity levels, as well. As a matter of fact, 34% of the inoculated subplots exceeded the established maximum value for dockage percentage. Such samples showed an average of 49% of diseased capitula. On the other hand, 53% of the inoculated subplots surpassed the established commercialization limit for oil acidity. These subplots presented a mean percentage of disease plants of 46.5%. It has to be emphasized that none of the control subplots surpassed the maximum tolerance limits for dockage or oil acidity levels. The linear regression coefficient between

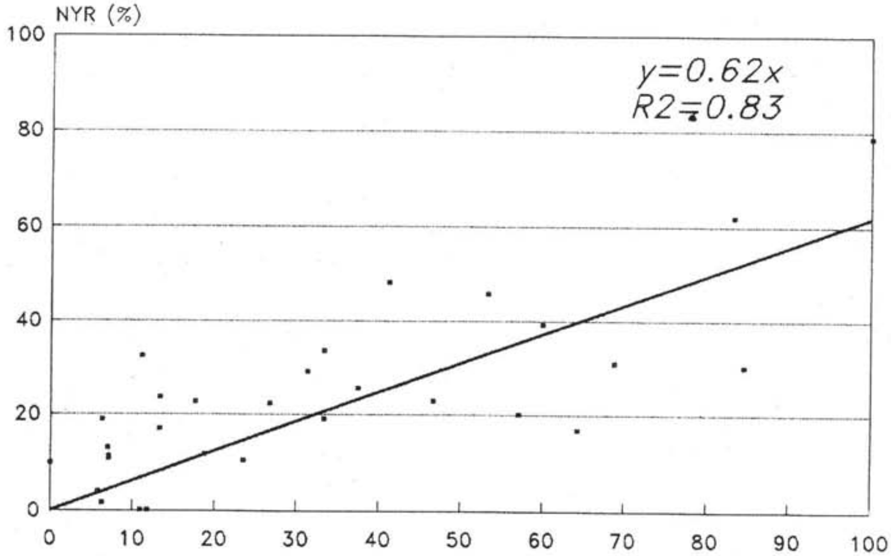

Figure 1. Net yield reduction of the inoculated subplots over the healthy ones (NYR) as a function of the percentage of plants with sclerotinia head rot

net yield percent reduction and the percentage of diseased plants was 0.63 (±0.056, $P<0.0001$). This value indicates that for each 10% increment in the percentage of capitula affected with head rot, the net yield percent reduction would be 6.3% (Table 2; Figure 1).

To determine if there exist genotypic differences for the pattern of yield reduction when the percentage of diseased plants increases, regression coefficients between both variables were estimated for each hybrid. The same was done for net yield percent reduction (Table 3). It was found that these coefficients were different among hybrids, with the minimum estimate of b=0.36 for the hybrid E till the maximum value of b=0.97 for the hybrid C. Yield losses in this last hybrid showed no differences with those obtained when all the affected capitula are destroyed. Similar results - with respect to genotypic variability - were obtained for the net yield percent reduction (Table 3).

Table 3: Linear regression coefficients between the percentage of plants with *Sclerotinia* head rot and the variables seed yield percent reduction (SYPR) and net yield percent reduction (NYPR) for commercial hybrids

Hybrid	b(1)	Standard error	Prob.	R^2
A	SYPR 0.58	0.18	0.026	0.66
	NYPR 0.75	0.19	0.010	0.71
B	SYPR 0.54**	0.10	0.003	0.85
	NYPR 0.57*	0.11	0.003	0.82
C	SYPR 0.97	0.19	0.003	0.84
	NYPR 1.03	0.207	0.004	0.80
D	SYPR 0.60**	0.08	0.0006	0.92
	NYPR 0.78	0.09	0.0005	0.93
E	SYPR 0.36**	0.047	0.0006	0.92
	NYPR 0.69**	0.075	0.0035	0.81

(1) *, ** signigicantly different from $\beta = 1$

DISCUSSION

The obtained results permit to state that head rot causes direct and indirect damages to the sunflower crop, as was previouly shown by Gulya et al. (1989).

Seed yield reduction is the direct damage. Such losses occur mainly by the reduction in the number of seeds per head since the 1000 seed weight is not modified by the disease (Table 1). The magnitude of yield losses seems to depend on genetic and environmental factors. With respect to the latter, two of the authors (A.B.R. & A.N.V., unpublished results) proved that when the environmental conditions are favorable to head rot developing, seed yield percent reduction in affected fields is equal to the percentage of diseased plants (i.e.: b=1). In such cases, diseased capitula are completely disintegrated, with the frayed vascular tissue resembling a broom head. On the other hand, when the environmental conditions subsequent to the fungus penetration in the host are not adequate for the development of the disease, the heads do not drop off and they can be partially or totally harvested. Yield reduction in these cases is lower (v.g.: b=0.49, see Table 2). In agreement with this estimate, yield losses of 4% were documented in North Dakota (U.S.A.) when an estimated 10% of the plants were infected (Gulya et al., 1989).

With respect to the genetic factors mentioned above, yield reduction estimates for each of the five hybrids utilized (Table 3) indicate that they are genetically different for their tolerance to head rot.

Head rot also causes indirect damages to the sunflower crop by increasing dockage and oil acidity levels. Both variables contribute to the reduction in economical profits. It is remarkable, in this sense, that an inverse relation between the magnitude of direct and indirect damages would exist. That is, when heads are completely desintegrated there would be no indirect damages, while if the development of the disease is slow (high genotypic tolerance, unfitted environmental conditions for the pathogen or both) indirect damages would be greater since more heads (and more sclerotia and impurities) can be harvested.

These results permit to asseverate that sunflower breeding for head rot control must be oriented to select those genotypes with better resistance levels (in terms of lower percentage of diseased plants) rather than to select genotypes with better tolerance levels (in terms of lower yield loss).

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PÉRDIDAS DE RENDIMIENTO DEL GIRASOL (*Helianthus annus* L.) DEBIDO A LA PODREDUMBRE DEL CAPÍTULO CAUSADA POR *Sclerotinia sclerotiorum* (Lib.) de Bary.

RESUMEN

El objetivo de este trabajo fue estimar las pérdidas de rendimiento en el cultivo de girasol ocasionadas por la podredumbre del capítulo causada por esclerotinia. Se generaron mediante inoculaciones, utilizando diferentes condiciones ambientales y materiales genéticos (cinco híbridos comerciales), sub-parcelas con distintos niveles de intensidad de ataque (=porcentaje de plantas con podredumbre del capitulo). En las sub-parcelas inoculadas y en las no inoculadas, las que fueron usadas como controles, se tomaron los datos de rendimiento de semillas, contenido de aceite de los aquenios, peso de 1000 aquenios, porcentaje de impurezas y acidez del aceite. Se detectó una correlación alta y positiva entre el porcentaje de plantas enfermas y la reducción del rendimiento de semillas (r=0.76***) y los incrementos en los niveles de impurezas (r=0.67***) y de acidez del aceite (r=0.58***). Las estimaciones de reducción del rendimiento variaron entre los híbridos, lo cual indica que los mismos son genéticamente diferentes para su tolerancia a la podredumbre del capítulo. Los resultados obtenidos permiten afirmar que la podredumbre del capítulo causa daños directos (reducción del rendimiento) e indirectos (incremento en los niveles de impurezas y de acidez del aceite) al cultivo de girasol. Existiréa una relacion inversa entre la magnitud de ambos tipos de daño. Esto es, cuando los capítulos son completamente desintegrados no habría da□os indirectos, mientras que si el desarrollo de la enfermedad es lento (alta tolerancia genotípica y/o condiciones ambientales adversas para el desarrollo del patogeno) los daños indirectos serían mayores ya que un mayor numero de capítulos pueden ser cosechados. Se concluye que el mejoramiento genetico del girasol para controlar esta enfermedad debe estar orientado a seleccionar aquellos genotipos con más altos niveles de resistencia (en terminos de menor porcentaje de plantas enfermas) más que a seleccionar genotipos con mejores niveles de tolerancia (en terminos de perdida de rendimiento).

PERTES DE RENDEMENT CHEZ LE TOURNESOL (*Helianthus annuus* L.) LIÉES À LA POURRITURE DU CAPITULE CAUSÉS PAR *Sclerotinia sclerotiorum* (Lib.) de Bary

RÉSUMÉ

L'objectif de ce travail était d'estimer les chutes de rendement chez le tournesol causées par le *Sclerotinia* sur capitule. Des parcelles comportant différentes intensités de maladie (=pourcentage de plantes avec pourriture du capitule) ont été générées par inoculation de matériel génétique (cinq hybrides commerciaux) soumis à diverses conditions environnementales. Le rendement en grains, la teneur en huile, le poids de 1000 grains, le pourcentage d'impuretés et l'acidité de l'huile ont été mesurés dans les parcelles inoculées et les parcelles saines utilisées comme témoins. Des corrélations positives hautement significatives ont été détectées entre le pourcentage de plantes malades et la réduction du rendement en grains (r=0.76***), l'augmentation du pourcentage d'impuretés (r=0.67***) et de l'acidité de l'huile (r=0.58***). Les estimations de réduction de rendement varient selon les hybrides; indiquant que ceux ci sont génétiquement différents pour leur tolérance aux attaques sur capitule. Les résultats obtenus permettent d'établir que le *Sclerotinia* sur capitule provoque des dommages directs (réduction du rendement en grains) ou indirects (augmentation du niveau d'impuretés et d'acidité de l'huile) à la culture de tournesol. Un relation inverse existerait entre l'importance des deux types de dommages. C'est à dire, que lorsque les capitules sont complètement désagrégés il n'y aurait pas de dommages indirects; alors que si le développement de la maladie est lent (par suite de forts niveaux de tolérance génotypiques, de conditions de milieu peu favorables au parasite ou les deux) les dommages indirects seraient plus importants car plus de capitules peuvent être récoltés. On en conclut que la sélection du tournesol pour le contrôle de la pourriture du capitule doit être orientée vers le choix de génotypes à meilleur niveau de résistance (pourcentage réduit de plantes attaquées) plutôt que vers la sélection des génotypes les plus tolérants (en termes de pertes de rendement).