

EARLY LODGING, A NOVEL MANIFESTATION OF *Albugo tragopogonis* INFECTION ON SUNFLOWER IN SOUTH AFRICA

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SUMMARY

Albugo tragopogonis (DC) S. F. Grey causes white blister rust, a disease restricted to leaves of sunflower. Oospores of *A. tragopogonis* have, however, been associated with grey areas on stems, petioles and receptacles of sunflower in Australia and Argentina. During the past two growing seasons, sunflower plantings in South Africa were subjected to early lodging. Disease patterns and symptoms were studied in the field and in the laboratory. Bruiselike lesions occurred on stems of sunflower which usually resulted in stem-breakage. These lesions were heavily colonized by oospores of *A. tragopogonis* infection appeared to occur from the axillas of lower leaves, and resulted in stem-breakage during head expansion. Early lodging was recorded over a large geographical area, with losses as high as 80% in certain fields. Systemic infections with *A. tragopogonis* have also been observed, but are limited to a few plants.

Key words: Early lodging, sunflower, white rust, *Albugo tragopogonis*

INTRODUCTION

Albugo tragopogonis (DC) S. F. Grey is responsible for white blister rust of sunflower (*Helianthus annuus* L.) (Zimmer and Hoes, 1978). The disease has been reported from most countries where sunflower is grown as a commercial crop (Kajornchaiyakul and Brown, 1976; Novotelnova, 1962; Sackston, 1957; Sarasola, 1942; Wilson, 1907). Symptoms are noticeable as chlorotic areas of 5-8 mm in diameter on the leaf upper sides. White blister-like pustules develop on leaf undersides. These symptoms are restricted to leaves of sunflower (Zimmer and Hoes, 1978), and have been reported to be damaging (Sackston, 1957).

A disease tentatively referred to as "petiole greying" was detected in New South Wales, Australia, in 1972 (Allen and Brown, 1980). Microscopic examination revealed the presence of oogonia, antheridia and oospores of *A. tragopogonis*. Symptoms included the development of grey areas on the stems, receptacles, involucre bracts, and more particularly, on the petioles of sunflower plants (Allen and Brown, 1980). In the southeast of Buenos Aires, Argentina, dark greyish-green areas were observed on stems, petioles and receptacles of sunflower plants (Delhey and Kiehr-Delhey, 1985). The basal portions of the petioles were particularly affected. Numerous oospores of *A. tragopogonis* were found in the cortex of plant tissue. Breakage of petioles and defoliation were observed, but no breakage of stems was reported (Delhey and Kiehr-Delhey, 1985).

In South Africa *A. tragopogonis* was first reported from sunflower leaves in 1929 (Van der Bijl, 1929; Verwoerd, 1929). It has, however, always been considered of minor economic importance (Holtzhausen, 1981). During the 1992/93 and 1993/94 seasons sunflower plantings were subjected to large numbers of plants which lodged at the early flowering stage. The aim of this study was to describe the new disease of sunflower and to identify the responsible pathogen.

MATERIALS AND METHODS

Disease patterns and symptoms associated with lodging of sunflower were studied over a large geographical area in South Africa. Plants that broke over were taken to the laboratory for further examination. Stem lesions were sectioned and studied at the areas of breakage. Stems of plants were surface disinfected and small pieces of plant tissue at the margins of infected areas were aseptically transferred to water agar and incubated at 25°C.

Sunflower seedlings used in pathogenicity tests were grown to the first leaf stage in the greenhouse. Seedlings were then transferred to the field and placed among plants naturally infected with *A. tragopogonis*. Plants were removed at daily intervals to monitor disease development on stems. Infected tissue was prepared for viewing by means of free hand sectioning and viewing facilitated with the use of phase optics.

RESULTS AND DISCUSSION

Stem-breakage of sunflower occurred on stems at 5-6 cm above soil level at early flowering (Fig. 1). Deep-seated, bruise-like lesions were associated with these stem-breakage areas (Fig. 2). Similar lesions also occurred on other parts of the stem (Fig. 3). No organisms could be isolated from stem lesions when incubated on culture media. When bruise-like lesions and tissue were sectioned and studied



Figure 1. Stem-breakage of sunflower occurring at 5-6 cm above soil level



Figure 2. Bruise-like lesions on the lower stem of sunflower infected with *Albugo tragopogonis*

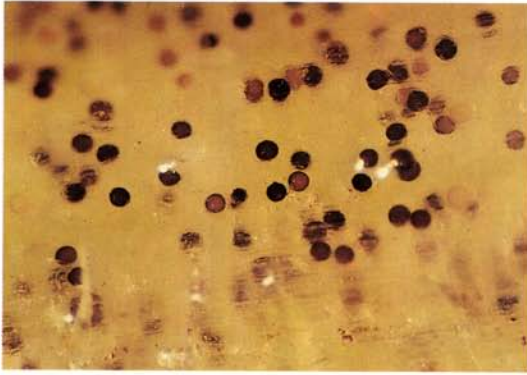


Figure 4. Oospores of *Albugo tragopogonis* in bruise-like lesions of stems of sunflower



Figure 5. Two zoospores of *Albugo tragopogonis* encysting the substromal cavity on the stem of a sunflower



Figure 6. Oogonia of *Albugo tragopogonis* produced within cells on the stem of sunflower



Figure 3. Bruise-like lesions on the higher stem of sunflower infected with *Albugo tragopogonis*

under the microscope, numerous dark-brown spherical bodies were observed (Fig. 4). These bodies matched the descriptions for oospores of *A. tragopogonis* with respect to size and epispore patterning (Makerji and Brown, 1975). This is the first report of *A. tragopogonis* causing lodging of sunflower.

Infection of sunflower seedlings demonstrated that sporangia of *A. tragopogonis* infect petioles and stems of sunflower seedlings through stomata. After germination, up to two zoospores encysted in the sub-stomatal cavity (Fig. 5). Germ tubes started colonizing the intercellular areas and haustoria penetrated the intercellular space. Oogonia (Fig. 6) were later produced within the cells, and dark coloured oospores in the deeper stem tissue gave rise to the

bruise-like symptoms on stems. Many stem lesions originated from leaf axillas (Fig. 7), presumably as a result of optimal conditions created for zoospore action following the catchment of free water.

During the 1992/93 and 1993/94 planting seasons, lodging of sunflower plants was recorded in many fields over a large geographical area. Severity ranged from a few to more than 80% of the plants lodged in a field (Van Wyk, unpublished data). Most plants appeared to be affected during the early flowering stage. Within a few days of lodging the open ends of the break-over area were rapidly colonized by *Alternaria alternata* Fr. (Kaissler) and became dark brown. This is consistent with the invasion of receptacles by other fungi, mainly *Alternaria* spp., following infection by *A. tragopogonis* (Delhey and



Figure 7. A stem lesion originating from the leaf axillas. The infection process was facilitated by catchment of free water



Figure 8. Petioles infected with *Albugo tragopogonis* led to necrosis of sunflower leaves

Kiehr-Delhey, 1985).

Early lodging of sunflower caused by *A. tragopogonis* contrast with the more commonly occurring lodging caused by *Phoma macdonaldii* Boerema. In the latter, the stem-breakage area is associated with black stem lesions and occurs much higher and at a different position on the stem. Late lodging also manifests much later than early lodging, often well after fruitset.

White blister rust occurs on leaves throughout the season. Plants, however, seemed to "outgrow" the disease in the early season. Early lodging was mostly restricted to plants sown in midseason or later (Van Wyk, data unpublished). It appeared that initial stem infections occurred from the axillas of lower leaves. This might explain why stem-breakage usually occurred on lower stems.



Figure 9. Defoliated sunflower following petiole infection by *Albugo tragopogonis*



Figure 11. Systemic infection of sunflower by *Albugo tragopogonis*



Figure 10. Stem-breakage of mature sunflower plants

Infected petioles dried out, became necrotic (Fig. 8) and eventually defoliated (Fig. 9). Sometimes lesions on petioles seemed to originate from leaf axillas, or even independent of leaf or stem infections. The colonized area only become visible after extensive oospore production. Lesion development appeared to coincide

with head expansion, resulting in stem-breakage of the plant when the slightest force was applied. Strong winds could cause break-over until very late, even near maturity (Fig. 10).

Another very unusual manifestation of white rust has been observed for the first time. A few plants were found to be systemically infected in the past season (Fig. 11). In these cases sporangia were not produced in localized blisters, but production was evenly spread over the lower leaf surface of infected areas. It is not clear at this stage whether systemically infected plants results from seed-borne and/or soil-borne inoculum.

Infection of sunflower by *A. tragopogonis* was regarded as economically unimportant in the past (Holtzhausen, 1981), and breeding for resistance was therefore non-existent (P. J. van der Merwe, breeder, Oil and Protein Seed Centre). From our observations it appeared that some cultivars are resistant to early lodging. The role of cultivar resistance, and the reason for this fundamental change in the manifestation of *Albugo* infections are being investigated.

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ENCAMADO TEMPRANO, UNA NUEVA MANIFESTACIÓN DE LA INFECCIÓN DE *Albugo tragopogonis* EN GIRASOL EN AFRICA DEL SUR.

RESUMEN

Albugo tragopogonis (DC) S.F. Grey causa la roya blanca, una enfermedad restringida a las hojas del girasol. Sin embargo las coosporas de *A. tragopogonis* han estado asociadas con las áreas grises de los tallos, peciolos y receptáculo del girasol en Australia y Argentina. Durante las dos campañas pasadas, las plantaciones de girasol en Africa del Sur estuvieron sujetas a encamado temprano. Las plantas y síntomas de la enfermedad fueron estudiadas en el campo y en el laboratorio. Lesiones similares a contusiones tuvieron lugar en tallos de girasol que normalmente se rompieron. Estas lesiones estuvieron colonizadas fuertemente por oosporas de *A. tragopogonis*. La infección tuvo lugar desde las hojas inferiores y resultó en ruptura durante la expansión. El encamado temprano fue detectado en una extensa área geográfica, con pérdidas de hasta el 80% en ciertos campos. Infecciones sistémicas con *A. tragopogonis* han sido observadas también pero limitadas a unas pocas plantas.

LA VERSE PRÉCOCE, NOUVELLE CONSÉQUENCE DE L'INFECTION DU
TOURNESOL PAR *Albugo tragopogonis* EN AFRIQUE DU SUD

RÉSUMÉ

Albugo tragopogonis (DC) S.F. Grey, agent de la rouille blanche, est une maladie dont l'expression se limite au feuillage du tournesol. Pourtant en Australie et en Argentine, des oospores d'*A. tragopogonis* ont été associés à la présence de taches grises sur tiges, pétioles et capitules de tournesol. Durant les deux cycles culturaux précédents, les cultures de tournesol en Afrique du Sud ont subi des dégâts de verse précoce. Les symptômes de la maladie ont été étudiés au champ et au laboratoire. Des lésions de type talure apparaissent sur les tiges de tournesol dont elles provoquent habituellement la cassure. Ces lésions sont fortement colonisées par les oospores d'*A. tragopogonis*. L'infection se développe à partir de l'axe des feuilles basses et conduit à une cassure durant la phase d'extension. La verse précoce se manifeste sur une vaste aire géographique, avec pour conséquence des pertes pouvant atteindre 80% dans certains champs. Des infections systémiques avec *A. tragopogonis* ont été aussi observées sur un nombre limité de plantes.