Recent shifts in pathogenesis of *Albugo tragopogonis* on sunflower in South Africa

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Abstract

Albugo tragopogonis is responsible for white blister rust of sunflower. Although white blister rust has been severe in some countries, the disease has never been regarded as a serious threat to the production of sunflower in South Africa. The typical symptoms, which resulted in the name of the disease, are yellow, raised chlorotic pustules on leaves. Since 1992, sexual structures of *A. tragopogonis* (oospores) have been responsible for severe early lodging of sunflower in some fields in South Africa. Other symptoms on sunflower now include petiole greying, infection of involucral bracts, and the colonization of veins on leaves. Systemically infected plants have also been observed. It therefore appears that *A. tragopogonis* is responsible for a disease complex on sunflower that may threaten this fast growing industry. The cause of the increasingly severe manifestations of the white blister rust fungus is a result of many contributing factors rather than a shift in virulence by the pathogen.

Introduction

White blister rust has been reported from most countries where sunflower is cultivated as a commercial crop (ZIMMER and HOES 1978). In South Africa, the disease was first reported in 1929 (VAN DER BIJL 1929). Although white blister rust has been severe in some countries (ZIMMER and HOES 1978), the disease has never been regarded as a serious threat to the production of sunflower locally. Recent losses of sunflower infected by *Albugo tragopogonis* (DC) S.F. Gray indicated that the white rust fungus is a more important pathogen than it was previously thought to be.

Symptoms

The typical symptoms of *A. tragopogonis* infections are yellow, raised chlorotic pustules on leaves. The underside of each pustule contains layers of white spores which are released as the pustules mature and rupture. With heavy infections, adjacent pustules merge to form large

diseased areas of irregular shape. Linear pustules are sometimes formed along the veins of apical leaves. The veins eventually turn greyish-green as they become invaded with numerous oospores, oogonia and antheridia. Systemically infected plants have been observed for the first time. In these plants, sporangia were not produced in localized blisters, but were evenly spread over the entire leaf surface.

Oospore infected tissue was also observed on petioles, receptacles and involucral bracts of sunflower. These symptoms were described on sunflower in Australia during the 1970's, and have tentatively been referred to as 'petiole greying' (ALLEN and BROWN 1980). Similar symptoms have also been observed on sunflower in Argentina and France (DELHY and KIEHR-DELHY 1985, PENAUD and PERNY 1995). Petioles of affected plants die prematurely, causing the subsequent defoliation of plants.

Since 1992, deep-seated, bruise-like lesions have been observed on stems of sunflower at early flowering. These lesions seemed to originate at the base of petioles or even independent of leaf or petiole infections and often lead to stem-breakage 5-6 cm above soil level. On closer inspection, numerous dark-brown oospores of *A. tragopogonis* were found in the cortical tissue beneath the epidermis. This invasion by oospores weakens the stems and results in stem-breakage when the slightest force is applied. Strong winds could cause break-over until maturity.

The Pathogen

Albugo tragopogonis is an obligate pathogen, and has been recorded on more than 300 genera of plants, always within the Compositae (BIGA 1955). The fungus is normally present in its asexual form on leaves of plants. Mycelia form intercellularly, and produce club-shaped sporangiophores that produce short cylindric to spherical-cuboid sporangiospores in chains (MUKERJI 1975). At the onset of cooler weather conditions late in the growing seasons, sexual structures (antheridia, oogonia and oospores) are produced (ALLEN and BROWN 1980). The existence of new, more virulent races of *A. tragopogonis* responsible for the new manifestations of the disease is currently under investigation. This possibility, however, is unlikely. The wide geographical distribution on all the different cultivars counters such a disposition. In addition, artificial inoculation with inoculum from white rust pustules resulted in stem infection in a commercial greenhouse.

Similarly, potted sunflower plants developed petiole greying following inoculation with a zoospore suspension of *A. tragopogonis* in Australia (ALLEN and BROWN 1980).

Impact

Damage to sunflower by *A. tragopogonis* includes white blister rust, leaf defoliation and early lodging. White blister rust often becomes severe, but has not been associated with losses of sunflower or considered to cause any significant yield reduction. Of far more concern is the effect of premature defoliation as a result of petiole infections. Certain cultivars are very susceptible to petiole infections and became almost completely defoliated.

Early lodging is currently the most serious threat to the cultivation of sunflower in South Africa. Severity of losses ranged from a few plants to more than 80% in certain sunflower fields. The disease occurred over a large geographical area, and caused tremendous concern among commercial farmers.

Causes

In South Africa, sunflower is normally planted in November. A second planting period from mid-January to the beginning of February, where planting of maize has failed, is often implemented. The climatic conditions at the end of this second planting period are very different from those at the end of early planting. Cooler temperatures, higher rainfall and a high humidity might play an important role in the development of the new symptoms caused by *A. tragopogonis* (VAN WYK and VILJOEN, these Proceedings).

Control

No specific program has been developed to reduce or eliminate the threat of *A. tragopogonis* to sunflower because of its limited economic importance in most countries. Initial trials with metalaxyl as a foliar spray have been successful in preventing infections. Two beetles, *Astylus atromaculatus* Blanchard and *Formicomus rubricollis* Laferte have potential as natural enemies for reducing disease incidence (VILJOEN et al. 1996). Variation in white rust incidence suggests that there may be sufficient genetic variation in host susceptibility to use resistance as a means of control (VAN DER MERWE, these Proceedings).

Discussion

The recent shifts in pathogenesis of *A. tragopogonis* to sunflower in South Africa are a result of many contributing factors rather than a single shift in virulence by the pathogen. Sunflower production in South Africa increased dramatically over the last decade (DU TOIT et al. 1994). The inoculum build-up of *A. tragopogonis*, susceptible cultivars, predisposition by herbicides, and favourable climatic conditions (VAN WYK and VILJOEN, these Proceedings) most probably resulted in the development of the new symptoms. The economic importance of these new Albugo diseases in South Africa has not been investigated. The new manifestations of the disease, and the worldwide occurrence of similar symptoms suggest that *A. tragopogonis* is likely to become an increasingly important pathogen of sunflower in the future.

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