NARAIN, U. and SAKSENA, H.K. 1973. Occurrence of Alternaria leaf spot of sunflower in India. *Indian Journal of Mycology and Plant Pathology,* 3:115 — 116.

REDDY, P.C. and GUPTA, B.M. 1976. Evaluation of fungicides *in vitro* against *Alternaria helianthi* (Hansf.) Tubaki and Nishihara, the incitant of leaf blight of sunflower *(Helianthus annuus* L.*). Hindustan Antibiotic Bulletin,* 18(3/4):94 — 95.

SHANE, W.W., BAUMER, J.S. and SEDERSTROM. S.G. 1981. *Alternaria helianthi:* A pathogen of sunflower new to Minnesota. *Plant Disease* 65:269 — 271.

SIDDIQUI, M.Q., BROWN, J.F. and ALLEN, S.J. 1975. Growth stages of sunflower and intensity indices for white blister and rust. *Plant Disease Reporter,* 59:7 — 11.

TAKANO, K. 1963. [A disease of sunflower caused by species of *Alternaria.*] *Annals of the phytopathological Society of Japan,* 28:300.

TUBAKI, K. and NISHIHARA, N. 1969. *Alternaria helianthi* (Hansf.) Comb. Nov. *Transactions of the British mycological Society,* 53:147 — 149.

WALLACE, G.B. and WALLACE, M.M. 1950. *Tanganyika Fungus List: Recent Records No. 14.* Mycological Circular, No. 19, Tanganyika Department of Agriculture.

WILLIAMSON, A.J.P. 1979. *In* Five states give us the current news on sunflower — Queensland. *Sunflower,* 3:19 (Australian Sunflower Association).

SOME FACTORS AFFECTING THE INCIDENCE AND IMPORTANCE OF SUNFLOWER RUST IN AUSTRALIA.

J.F. BROWN, S.J. ALLEN, P. KAJORNCHAIYAKUL and M.Q. SIDDIQUI
Department of Botany, University of New England, Armidale, N.S.W. 2351, Australia.

ABSTRACT

The incidence of rust (caused by *Puccinia helianthi* Schw.) in commercial sunflower crops in eastern Australia was monitored during the period 1972 to 1982. The incidence of rust has decreased in recent years due primarily to an increase in the use of rust resistant hybrid cultivars. Wholly or partially resistant hybrids became commercially available in Australia in about 1975 after it was found that yield could be increased by 70% when rust was controlled in susceptible, open pollinated cultivars grown in New South Wales and Queensland. Glasshouse studies showed that the effects of simulated rust epidemics on growth and yield parameters depended on the stage of plant growth when the epidemic occurred and on the duration and intensity of the epidemic. Laboratory studies designed to investigate the mechanisms by which rust infection reduced yield showed that healthy plants exported between 42 to 70% of the photosynthate they produced to other parts of the plant. In contrast to this, infected leaves retained about 97% of the photosynthate they produced. Only about 3% of the photosynthate produced was exported to other plant parts. Rust infection also increased the water requirements of plants that were subjected to conditions of moisture stress. Studies on the infection process showed that the fungus gained entry into its host through stomata. The optimal temperature for infection and disease development was 20°C. At this temperature a dew period of 8 h enabled maximal infection by the rust fungus. A longer dew period was required at temperatures above or below the optimum. Light inhibited development during the pre-penetration phases of infection. Under optimal environmental conditions, the time taken for pustules to erupt was 11 days.

INTRODUCTION

Rust caused by *P. helianthi* is a common disease of sunflower in most countries of the world. The disease has been shown to cause serious loss of yield in Russia (Eremeyeva and Karakulin, 1929), Kenya (Nattrass, 1950; Singh, 1975), Argentina (Muntanola, 1954), Canada (Putt and Sackston, 1955) and Hungary (Kurnik and Meszaros, 1962). Some workers in the United States however, consider rust to be of minor importance (Culp and Kinman, 1965; Robinson, Johnson and Soine, 1967) while others consider it to be important on susceptible cultivars (Cobia and Zimmer, 1975; Zimmer and Hoes, 1978).

Middleton (1971) and Stovold and Moore (1972) discussed the incidence and importance of diseases of sunflower in Queensland and New South Wales respectively. They considered that rust caused by *P. helianthi* was the most important disease present in commercial sunflower crops at that time. Middleton and Obst (1972) and Brown, Kajornchaiyakul, Siddiqui and Allen (1974) showed that when sunflower rust was controlled, yield increased by over 70% in open pollinated, rust susceptible cultivars in Australia. The significance of the losses resulted in rust resistant hybrid cultivars being released and used by sunflower farmers.

During the period 1971 to 1981 there has been a marked change in the relative importance of different diseases of sunflower. During the early to mid 1970's, when open pollinated rust susceptible cultivars were grown *P. helianthi* rust was observed in all crops examined and was regarded as being the most important disease in the sunflower areas of eastern Australia. In subsequent surveys the incidence of the disease was very variable as a result of genetic differences between commercial sunflower lines. The disease occurred in all open-pollinated cultivars examined with a usual level of infection of between 19 and 20%. Rust was also common in most inbred A lines ("female parent") which were usually susceptible to the disease. Most of the hybrid cultivars observed were resistant to rust although some showed some disease. However, the rust levels in these latter cultivars were usually about three times less than that observed in open pollinated cultivars. The relative importance of other diseases of sunflower in eastern Australia has been discussed by Allen, Brown and Kochman (1980).

The objective of this paper is to review the research that has been undertaken on sunflower rust in Australia during the past 10 years. Much of this work has been done by past and present members of the Botany Department's Plant Pathology Laboratory at the University of New England. Although a considerable amount of the work referred to in this paper has been published elsewhere, it is summarised here together with previously unpublished work in order to give an overall perspective of the sunflower rust situation in Australia.

Effect of natural rust epidemics on sunflower yield during the 1972 and 1973 seasons.

Field trials were conducted in the Gwydir Valley of N.S.W. to investigate the effects of rust infection on the yield of the rust susceptible sunflower cultivar Peredovic. (Brown *et al.,* 1974).

An irrigated trial (trial 1) was hand sown on 24 February 1972 in rows 15m long and 76cm apart at the rate of 74,000 plants/ha. Commencing at 5 weeks after sowing, the fungicide mancozeb (80% a.i. at 200g/100 l) was applied to the foliage of plants to control rust. Rust was allowed to develop on untreated control plots. The trial was designed

according to a randomized splitplot design consisting of three replicates. Each plot consisted of 3 rows and only the central 9m of the central row was assessed for rust incidence (using a pictorial key similar to that described by Siddiqui, Brown and Allen, 1975) and yield parameters. The outer two rows of each plot served as buffer rows. The trial was hand harvested on 18 May and the yield parameters listed in Table 1 were determined. The percentage oil content was determined by the nuclear magnetic resonance method.

A second trial, in a non-irrigated commercial sunflower crop, was commenced on 25 February 1973. Three pairs of plots, each consisting of 11 rows of 20m length were marked in a crop that had been sown with a row spacing of 36cm and at a density of about 90,000 plants/ha. Half of the plots were sprayed thoroughly with mancozeb (200g/100 1) at 14 day intervals commencing on 23 March. The unsprayed plots served as controls. Disease severity was assessed in the same way as for the 1972 trial. The plots were harvested on 15 June, and various yield parameters were measured.

The data presented in Table 1 show that mancozeb was effective in controlling rust and that a reduction in rust incidence was associated with estimated oil yield increases of over 70%.

Table 1. Effects of rust on yield parameters of the sunflower cultivar Peredovic (From Brown *et al.*, 1974)

	Rust Assessment (%)	Head Diameter (cm)	1000 Seed wt. (g)	Oil Content (%)	Est. Seed Yield (kg/ha)	Est. Oil Yield (kg/ha)	Oil Yield increase over control (%)
Trial 1 (1972)							
Untreated control	11.4	9.9	35.9	43.6	1112	486	
Mancozeb treated	3.4	11.4	42.4	45.0	1904	857	76
L.S.D. $P > 0.05$		0.7	5.6	2.3	439	224	
Trial 2 (1973)							
Untreated control	18.7	7.3	26.6	42.6	588	253	
Mancozeb treated	8.0	9.0	34.2	44.0	990	442	74.4
L.S.D. $P > 0.05$		1.6	7.0	0.4	520	269	
Trial 3 (1973)							
Untreated control	4.4	15.2	53.3	48.5	2017	977	
Mancozeb treated	3.5	16.7	61.1	48.8	1939	945	−3
L.S.D. > 0.05		3.1	4.3	2.1	314	139	

A third trial was sown on 23 January, 1973 under irrigated conditions. The results (Table 1) show that a rust epidemic failed to develop in the trial and the level of rust infection rarely exceeded 4 percent in either of the treatments. The application of mancozeb did not cause any significant ($P = 0.05$) differences in yield parameters relative to those observed in unsprayed controls. These results indicate that the effect of mancozeb on yield parameters was due to control of rust rather than to a direct effect of the fungicide on plant growth.

Effects of simulated rust epidemics on growth and yield of sunflower

Glasshouse experiments were made to determine the effects of simulated rust epidemics, which developed at different intensities throughout different stages of plant growth, on growth and yield of sunflowers. Sunflower plants (cv. Peredovic) were inoculated by spraying them with a suspension of *P. helianthi* urediospores contained in a light mineral oil. The inoculated plants were incubated for 16 h in a dew chamber kept at 20°C. This method of incubation resulted in rust infection which covered 10 — 15% of the area of the lower surfaces of leaves which were inoculated once only. Rust developed only on leaves which received inoculum. Leaves that were produced after inoculation did not show disease symptoms. Flowers were hand-pollinated during anthesis, and when plants had reached maturity they were harvested and various growth and yield parameters were determined.

The treatments included: (i) plants inoculated once at the vegetative stage of growth; (ii) plants inoculated at vegetative stage, inoculation repeated at 10 day intervals; (iii) plants inoculated at budding stage only; (iv) plants inoculated at budding stage, inoculation repeated at 10 day intervals; (v) plants inoculated at anthesis stage only; (vi) plants inoculated at anthesis stage, inoculation repeated at 10 day intervals; (vii) plants inoculated at seed development stage only; (viii) plants inoculated at seed development stage, inoculation repeated at 10 day intervals; and (ix) control plants, not inoculated.

The inoculation of plants at the vegetative, budding, anthesis or seed development stages of growth and thereafter at 10 day intervals resulted in oil yield reductions of 85, 73, 38 and 13 percent respectively relative to uninoculated control plants. In contrast, plants inoculated once only at each of these growth stages showed reductions in oil yield of 13, 42, 35 and 10 percent respectively. In plants initially inoculated at the vegetative stage and thereafter at 10 day intervals the dry weight of the stem and roots was decreased by 62 and 70% respectively relative to uninoculated control plants. Plants inoculated at the budding stage and thereafter at 10 day intervals showed a reduction in the stem and root dry weights of 37 and 40% respectively. The reduction in the dry weight of the secondary roots was greater than that of the tap root (Siddiqui and Brown, 1977).

Studies on the physiology of yield reduction caused by rust infection

Studies were made in a glasshouse to investigate the mechanisms by which rust infection reduced growth and yield parameters of sunflower. Rust infection did not significantly affect transpiration (as measured by a diffusion porometer) until flecks became visible. After flecking a sharp increase in dark or cuticular transpiration (or decrease in diffusive resistance) was observed due presumably to cuticular damage. Rust infection did not appear to affect light or stomatal transpiration of plants grown in an adequate moisture regime (Fig. 1). However, when plants were exposed to conditions of moisture stress, rust infection caused a marked decrease in light diffusive resistance (Table 2). One would expect this to be related to an increase in transpiration. It is likely that the water conserving mechanism (stomatal closure) that operates in healthy plants that are exposed to water stress failed to operate in the diseased plants.

Figure 1. Diffusive resistance of uninfected and rust infected leaves of sunflower. ▲ Uninfected leaves exposed to darkness; ● infected leaves exposed to darkness; △ uninfected leaves exposed to light; ○ infected leaves exposed to light (from Siddiqui, 1980)

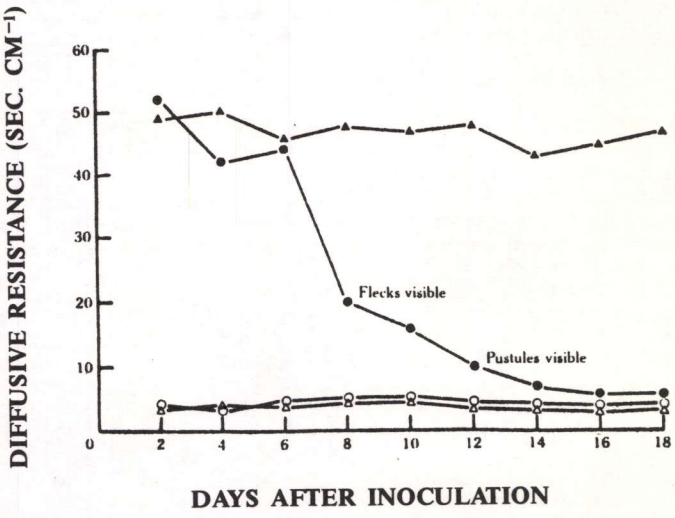

Table 2. Effect of rust infection at the sporulation stage of development and leaf water potential on the diffusive resistance of sunflower leaves (from Siddiqui, 1980).

Leaf water potential (bars)	Diffusive Resistance (sec.cm^{-1})*			
	Light		Darkness	
	Uninfected	Infected	Uninfected	Infected
− 4	3.9a	3.1a	43.6b	3.5a
− 7	3.0a	3.2a	41.2b	3.1a
−12	37.6b	3.6a	37.9b	3.5a

*Values followed by a different letter differ significantly (P = 0.05)

Severe rust infection on sunflower leaves (24% leaf area covered with pustules) caused reductions of up to 34% in the uptake of labelled carbon dioxide. In general infection resulted in a reduction in photosynthetic activity either by destroying photosynthetic tissue or by causing a reduction in the production of photosynthetic tissue.

The rate of translocation of photosynthate from diseased leaves was less than that from healthy leaves (Table 3). Severe rust infection resulted in about 96 to 98% of labelled assimilates being retained in leaves where they were produced; only 2 — 4% was exported. In uninfected leaves between 30 and 58% of assimilate was retained and the remaining 42 to 70% was exported to other parts of the plant. The distribution pattern of assimilate translocated from leaves fed with $^{14}CO_2$ was modified by rust infection.

Table 3. Percentage of the total carbon-14 assimilate exported from uninfected and rust-infected sunflower leaves in plants subjected to different levels of moisture stress (from Siddiqui, 1980).

	Percentage of assimilate exported at three degrees of moisture stress*		
	None	Mild	Moderate
Uninfected	64a	70a	42b
Uninfected (9% disease)	35c	44b	27d
Infected (24% disease)	2c	4c	4c

*Values followed by a different letter differ significantly (P = 0.05)

As pointed out by Siddiqui (1980) the results reported above show that rust infection caused disruption to a number of physiological processes in sunflower plants. In addition to destroying photosynthetic tissue and thus the amount of photosynthate produced, infected leaves retained photosynthate which was consequently unavailable for growth of other plant parts such as newly developing leaves, roots, stems and seeds. Moreover, rust infection appeared to cause an increase in the rate of transpiration in darkness and in light when leaves were subjected to moisture stress. Thus, under conditions of moisture stress, one would expect diseased plants to have greater water requirements than uninfected plants. It is likely therefore that rusted plants are more prone to water deficit than uninfected plants.

Factors influencing infection of sunflower by *Puccinia helianthi*

The experiments reported here were designed to investigate the effects of environment on infection of sunflower by *P. helianthi.* Quantitative studies of the infection process were made on sunflower seedlings (cv. Peredovic) that were kept in a glasshouse. When plants had produced the first pair of true leaves they were inoculated by depositing dry urediospores onto the upper leaf surface (1,650 spores/cm^2 of leaf surface) in a spore settling tower (Brown and Kochman, 1973). The inoculated seedlings were incubated in a dew chamber kept at various temperatures and light intensities for 24 h and were then transferred to a growth cabinet or a glasshouse. Inoculated leaves were sampled at various intervals after inoculation and were prepared for microscopic examination by the clearing and staining method of Shipton and Brown (1962).

Urediospores of *P. helianthi* germinated by producing a germ-tube from one of the two pores in the spore wall. The tip of the germ-tubes produced irregularly shaped appressoria over stomates or on other regions of the leaf surface. Penetration tubes developed from appressoria and penetrated the host through stomata to form H-shaped sub-stomatal vesicles. Intercellular infection hyphae developed from sub-stomatal vesicles and permeated host tissue to form haustoria in the mesophyll cells of the leaf.

Urediospores of *P. helianthi* required free water for germination to proceed. The dew period required to promote maximal infection was 8 h. The spores germinated well in darkness at temperatures of 10 to 25°C. Germination was inhibited at 30°C on agar media and on living leaves. Under optimal temperatures, germination occurred within 2 h of inoculation and reached a maximum of about 90 percent within 4 —6 h after inoculation. Germ-tube elongation occurred best at 20°C both on agar media and on the leaves of sunflower. Germ-tube length reached a maximum (about 250 μm) on leaf surfaces at 6 h after inoculation at 20°C in darkness. Light was found to reduce the percentage germination and the rate of elongation of germ-tubes. Light intensity of 16,500 lux completely inhibited urediospore germination.

Maximal development of appressoria occurred in darkness at 20°C and no appressoria were form at 4°C and 37°C. Light intensities of 5,500 lux and above decreased the number of appressoria produced. Maximal numbers of appressoria were formed at 12 h after inoculation at 20°C in darkness. The first sub-stomatal vesicles were observed at 8 h after inoculation. Temperatures of 20 to 30°C in total darkness or under light intensities of 2,200 lux and below were most favourable for maximal formation of sub-stomatal vesicles. Disease development was greatest at temperatures of 20 to 25°C and at a light intensity of at least 22,000 lux (12 h photoperiod). Under these conditions pustules erupted at about 11 days after inoculation.

The results reported above are in close agreement with those of Sood and Sackston (1970; 1972). Although Sood and Sackston (1970) reported that germ-tubes sometimes issued from two of the equatorial germ pores of urediospores of *P. helianthi,* none of the urediospores observed in this study produced more than one germ-tube. Development during the prepenetration and penetration phases of infection occurred more rapidly initially in the study reported here than in the one reported by Sood and Sackston (1970). Differences between the strains of rust used could have contributed to this difference between the two studies. Ogle and Brown (1971) found that germination, formation of appressoria and penetration occurred more rapidly in one strain of *Puccinia graminis* Pers.f.sp. *tritici* Eriks and Henn. than another. Furthermore, Sood and Sackston (1972) reported that different races of *P. helianthi* responded differently to temperature and light intensity.

ACKNOWLEGEMENTS

The authors thank the Rural Credits Fund of the Reserve Bank of Australia, Gwydir Valley Oilseeds Pty Ltd and the Australian Department of Primary Industry for providing financial support for this investigation. Thanks are also due to Pacific Seeds for determining the oil content of some of the seed and to David Bailey for his assistance for field aspects of the study.

LITERATURE CITED

ALLEN, S.J., BROWN, J.F. and KOCHMAN, J.K. 1980. The incidence of sunflower diseases in New South Wales and Queensland during the 1978 — 1979 season. *Proceedings of the Australian Sunflower Association's fourth Australian sunflower workshop* Shepparton pp 4 — 1 to 4 — 7.

BROWN, J.F., KAJORNCHAIYAKUL, P., SIDDIQUI, M.Q. and ALLEN, S.J. 1974. Effects of rust on growth and yield of sunflower in Australia. *Proceedings of the sixth international sunflower conference* Bucharest pp 639 — 646.

BROWN, J.F. and KOCHMAN, J.K. 1973. A spore settling tower for uniform inoculation of leaves with rust urediniospores. *Australian Plant Pathology Society Newsletter* 2, 26 — 27.

COBIA, D. and ZIMMER, D. 1975. Sunflowers: production, pests and marketing. *Extension Bulletin 25,* North Dakota State University of Agriculture and Applied Science. 59 pp.

CULP, T.W. and KINMAN, M.L. 1965. Rust on sunflower in the Mississippi delta. *Plant Disease Reporter* 49, 433 — 434.

EREMEYEVA, A.M. and KARAKULIN, B.P. 1929. [Observations on the sunflower rust at the lower Volga regional agricultural experiment station] In Russian. *Morbi Plantarum* (Leningrad) 18, 11 — 30. (Cited in *Review of applied Mycology* 8, 791 — 793).

KURNIK, E. and MESZAROS, L. 1962. Ergebnisse von Spritzversuchen gegen Sonnenblumenrost *(Puccinia helianthi* Schw. *) Pfleschber* 28, 47 — 60. (Cited in *Review of applied Mycology* 42, 27*)*.

MIDDLETON, K.J. 1971. Sunflower diseases in south Queensland. *Queensland Agricultural Journal.* 97, 597 — 600.

MIDDLETON, K.J. and OBST, N.R. 1972. Sunflower rust reduces yield. *Australian Plant Pathology Society Newsletter.* 1, 18.

MUNTANOLA, M. 1954. Sobre la presencia de la roya del Girasol *(Puccinia helianthi* Schw.*)* eu Tucuman. *Revista agronomica del Noreste Argentino.* 1, 2 pp 213 — 215 (cited in *Review of applied Mycology.* 34, 789).

NATTRASS, R.M. 1950. Annual Report of the senior plant pathologist. 1948. Report of the Department of Agriculture, Kenya. pp 95 — 98 (cited in *Review of applied Mycology.* 30, 308*)*.

OGLE, H.J. and BROWN, J.F. 1971. Some factors affecting the relative ability of two strains of *Puccinia graminis tritici* to survive when mixed. *Annals of applied Biology,* 67, 157 — 168.

PUTT, E.D. and SACKSTON, W.E. 1955. Rust resistance in sunflowers. *Nature* (London) 176, 77.

ROBINSON, R.G., JOHNSON, F.K. and SOINE, O.C. 1967. The sunflower crop in Minnesota. *University of Minnesota Extension Bulletin.* No. 299.

SHIPTON, W.A. and BROWN, J.F. 1962. A whole leaf clearing and staining technique to demonstrate host-pathogen relationships of wheat stem rust. *Phytopathology* 52, 1313.

SIDDIQUI, M.Q. 1980. Some effects of rust infection and moisture stress on growth, diffusive resistance and distribution pattern of labelled assimilates in sunflower. *Australian Journal of Agricultural Research.* 31, 719 — 726.

SIDDIQUI, M.Q. and BROWN, J.F. 1977. Effects of simulated rust epidemics on the growth and yield of sunflower. *Australian Journal of Agricultural Research.* 28, 389 — 393.

SIDDIQUI, M.Q., BROWN, J.F. and ALLEN, S.J. 1975. Growth stages of sunflower and intensity indices for white blister and rust. *Plant Disease Reporter.* 59, 7 — 11.

SINGH, J.F. 1975. Field evaluation of fungicides for the control of sunflower rust in Kenya. *Plant Disease Reporter.* 59, 200 — 202.

SOOD, P.M. and SACKSTON, W.E. 1970. Studies on sunflower rust VI: Penetration and infection of sunflowers susceptible and resistant to *Puccinia helianthi.* race I. *Canadian Journal of Botany.* 48, 2179 — 2181.

SOOD, P.N. and SACKSTON, W.E. 1972. Studies on sunflower rust XI: Effect of temperature and light on germination and infection of sunflowers by *Puccinia helianthi.* race I: *Canadian Journal of Botany.* 50, 1879 — 1886.

STOVOLD, G.E. and MOORE, K.J. 1972. Oilseed sunflowers: diseases. *New South Wales Agricultural Gazette.* 83, 262 — 264.

ZIMMER, D.E. and HOES, J.A. 1978. Diseases. In *Sunflower science and technology.* p 225 — 262. American Society of Agronomy, Madison.

INVESTIGATIONS INTO INCREASED RUST *(PUCCINIA HELIANTHI)* INTENSITY ON SOME HYBRID SUNFLOWER CULTIVARS GROWN IN QUEENSLAND.

J.K. KOCHMAN and K.C. GOULTER
Department of Primary Industries, P.O. Box 102, Toowoomba, Qld, 4350, Australia.

ABSTRACT

Rust *(Puccinia helianthi)* intensity measured in crops of a number of hybrid cultivars grown in Queensland during 1980 and 1981 ranged from 30 to 60% of leaf area. In previous years these cultivars had a good level of resistance with rust ratings of 2 — 5% being usual. A set of differential lines, a number of open-pollinated and inbred lines and a range of commercial hybrids were inoculated with uredospores collected during both seasons from these and crops of open-pollinated cultivars as well as with uredospores collected during previous seasons. Because of the resistant reactions obtained on differentials with the resistance gene R1, all the collections could be allocated to race group 1. However, a differential line (supposedly containing resistance genes R1 and R2) was more susceptible to the rust from hybrids in 1980 and 1981 than to the other collections. Data recorded from the other lines and cultivars were somewhat variable. On some lines and cultivars either no pustules were produced, or the pustules were very small in size and number. On cultivars where rust developed the isolate collected from hybrids produced equal generation times on the hybrids Hysun 31, Sunace and, Suncross 52, and on the open-pollinated cultivars tested, whereas generation time was significantly shorter on open-pollinated cultivars than on these hybrids for the isolate from collections prior to 1980. Both isolates generally produced more and larger pustules on the open-pollinated cultivars than on the hybrids, but the differences appeared to be smaller with the 1980 isolate from hybrids than the other isolate.

INTRODUCTION

Hybrid sunflower cultivars with resistance to rust *(Puccinia helianthi* Schw.*)* became available in Australia during the early nineteen seventies and have become widely accepted and grown by farmers. Because of the nature and selection of parental lines and the involvement of private seed companies in production of hybrid cultivars, there is some uncertainty as to which genes for rust resistance are present in the various hybrids available. The first cultivars released were immune to rust with resistance apparently conferred by the R1 gene. Subsequently other cultivars were released which took some rust under the local conditions, but typical ratings were in the order of 2 — 5% of leaf area. Resistance in some of these cultivars may have been conferred by the R2 gene.

During 1980 and 1981 several crops of the latter type hybrids in southern Queensland were found with 30 — 60% of their leaf area covered by rust pustules. The levels of rust in these hybrids were of great concern to the sunflower industry in Queensland because it had been shown that control of severe epidemics in open-pollinated cultivars increased yield by about 70% (Middleton and Obst, 1972; Brown *et al.,* 1974).

Studies were conducted to determine the reasons for the increased incidence of rust in some of these hybrids and the results are reported and discussed in this paper.

MATERIALS AND METHODS

Uredospores were collected from crops of open-pollinated and hybrid cultivars either with a large spore collector (Cherry and Peet, 1966) or by scraping uredospores from infected leaves in the laboratory. Single spores were taken from these collections and increased on a very susceptible cultivar (either cv. Polestar or cv. Sunfola 68-2). Portions of the bulk collections were stored in liquid nitrogen for future reference.

The race group of the isolates was determined on a set of seven differentials. These were: S37-388 ("universal suscept"), S37-388 RR (R1 gene), 69-17-8-1-1 (R1 gene), 29-3-1-3-2-1 (R2 gene), 953-102-1-1-41 (R1 and R2 genes), 953-88-3-1-54 (original source of rust resistance) and, Polestar (rust susceptible birdseed cultivar). With the exception of Polestar all the other lines were imported from Canada. Information from several sources (Sackston, pers. comm.; Sackston, 1962; Jabbar Miah and Sackston, 1970) was used to identify the genes for rust resistance supposedly carried by the various lines. Plants were inoculated as seedlings with four replicates of each being used to determine reaction type. If the reactions were not equivalent on all plants of a particular line, isolates were retyped on 10 replicates. Reaction types of each isolate were rated 14 days after inoculation on a 0 — 4 basis (Sackston, 1962) with 0, 1 and 2 considered resistant and 3, 4 susceptible. All rust typing was done under uniform conditions in controlled environment cabinets set at 18/22 $\pm$ 1°C night/day and 600 μ E m^{-2} sec^{-1} (measured by a Lambda Li-170 quantum/radiometer/photometer) supplied in 12 h photoperiods.

The macroscopic development of two rust isolates (one collected during 1979 and the other collected from hybrids during 1980) was observed on the following lines and cultivars: RHA 266, RHA 274, cms HA 89, Polestar (open-pollinated), Sunfola 68-2 (open-pollinated), Hysun 30 (hybrid), Hysun 31 (hybrid), Suncross 52 (hybrid) and, Sunace (hybrid). Seedlings of these lines were uniformly inoculated with uredospores, incubated and grown in the same controlled environment conditions used for rust typing. Data collected were; generation time (days from inoculation to pustule eruption), pustule number, pustule diameter and reaction type (the last three at 14 days after inoculation). Ten replicates with two plants per replicate were used in each experiment. Pustule diameter was determined by measuring 20 pustules in each replicate, but all pustules were measured