Environmental Influences on the Development of *Puccinia helianthi* on Sunflower

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ABSTRACT

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Nine epidemics of rust, caused by *Puccinia helianthi,* on sunflower (*Helianthus annuus*) were analyzed concurrently. Although the epidemics occurred over a period of 3 years (1991 to 1993) and in three growing regions, the apparent infection rates were relatively uniform (average rate ± standard error = 0.237 ± 0.007). The possibility of using this rate to predict disease progress at an early stage of an epidemic was evaluated in four fields during 1994. In general, the extrapolated apparent infection rate was very close to the observed. To understand the biological basis for relatively uniform rates of disease increase, effects of environmental parameters on components of the life cycle of *P. helianthi* were examined. Urediniospore germination occurred at temperatures of 4 to 20°C, lasted 4 to 6 h, and required 6 to 10 h of leaf wetness. At 15 to 25°C, the time from inoculation until the appearance of the first pustule was 8 to 10 days. The minimum, optimum, and maximum temperatures for infection were 4, 10 to 24, and 30°C, respectively. Sporulation occurred over a wide range of temperatures (4 to 39°C); the optimum was between 20 and 35°C. On the basis of these findings, it was concluded that during the period of rust epidemics in Israel (May through June), the environment is rarely nonconducive to *P. helianthi* development.

Additional keywords: disease forecasting.

Sunflower rust, caused by *Puccinia helianthi* Schwein., occurs almost everywhere in the world where its host (*Helianthus annuus* L.) is grown. Rust usually appears first on lower leaves and then on the upper leaves, petioles, stems, and the back of the flower head. Rust severity varies with the environment, host age, and cultivar resistance. When infections are severe, leaves senesce prematurely, and yields may be reduced to as little as 15% of the attainable yield (4,12,13,18–21). Rust reduces sunflower yield, oil content, seed size, and kernel-to-hull ratio (15). The most effective method of preventing losses from rust is the use of resistant cultivars. In general, sunflower cultivars grown for direct consumption of seed are more susceptible to rust than those grown for oilseed (5). Destruction of volunteers and avoidance of high rates of nitrogen fertilizer and large plant populations also can minimize the risk of losses in a susceptible host (14). With the sunflower cultivars grown for direct seed consumption, the higher value of the yield may justify the use of fungicides to control rust (18,19).

Studies were recently conducted in Israel to measure the yield losses caused by rust (20) and to develop an action threshold for fungicidal disease suppression (18). Nine different epidemics of rust were followed. Disease progress curves revealed that disease onset varied substantially among the epidemics; however, the rate of disease increase was relatively uniform. This was not expected, because the nine epidemics were recorded over 3 years and in three different growing regions. In this study, the nine epidemics were analyzed concurrently, and an average apparent infection rate was calculated. The possibility of using this average rate to predict disease progress at an early stage of an epidemic was examined. Prediction of disease progress is important for disease management (18). To understand the biological basis for the uniform rate of disease increase, experiments were conducted in a controlled environment in which the effects of temperature and leaf wetness duration on the components of the *P. helianthi* life cycle were studied. A preliminary report, including a portion of the results, has been published elsewhere (17).

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MATERIALS AND METHODS

Rate of disease increase. Data from nine rust epidemics were recorded in field experiments during 1991, 1992, and 1993 in the Lakhish, northern Negev, and Hadera regions of Israel. The local cultivar DY-3 was sown in all experiments. This cultivar is grown for confectionery use (achenes are sold unhulled) and is highly susceptible to *P. helianthi.* Seed was sown on raised beds during the second half of March each year; plants were spaced at 0.4 m within rows and 1 m between rows. The crop was irrigated with a surface drip irrigation system and cultivated according to the recommendations for these regions, but fungicides and insecticides were not applied. The experiments were conducted to determine the yield loss induced by rust (19) and to develop an action threshold for fungicidal disease suppression (18). Each experiment included four to eight treatments (spray schedules) against *P. helianthi.* The experiments were laid out in a randomized block design with four replicates. The size of each experimental plot was four to six beds 12 m in length. In this report, disease progress recorded in the untreated plots will be presented; details about the other treatments are given elsewhere (18,19).

Disease was assessed visually every 7 to 10 days starting in mid- to late May and ending in early to mid-July. Flowering (i.e., the point at which 90% of the plants had flowered) occurred in late May to early June and physiological maturity in early to mid-

July. Ten arbitrarily selected plants located in the inner rows of each experimental plot were evaluated for disease development. On each plant, disease severity (i.e., the percentage of leaf area with rust) on the upper four leaves was assessed according to a scale developed for this purpose (6). The upper leaves were chosen because they are the main source of carbohydrates for the developing achenes (16). Disease estimates of the individual plants were averaged, and plot means were used for data analysis. Assuming a logistic increase of the epidemic, changes in disease severity over time were used to calculate an apparent infection rate, r (26). The apparent infection rate was calculated after transformation of disease severity values, x (in percentages), to logit values (logit = ln $[x/(100 - x)]$) and regression of the logit values over time (26). Differences in rates among the epidemics recorded from 1991 to 1993 were examined statistically for each pair of epidemics by using a t test ($P = 0.05$), and then an average apparent infection rate for the nine epidemics was calculated.

The possibility of using the average apparent infection rate to predict disease progress at an early stage of an epidemic was examined during 1994. Disease development was recorded in four commercial fields in the Lakhish region that had not been treated with fungicides. In each field, disease severity was recorded at 5- to 8-day intervals, beginning soon after disease onset (early May) and continuing until crop maturity (late July). One hundred plants were sampled arbitrarily in an area of 100 × 100 m; the upper four leaves were inspected and the severity of rust was assessed as indicated above. Disease estimates of the individual plants were averaged, and field means were used for data analysis. When disease severity was approximately 1%, future development of the disease was predicted by fitting a logistic equation to that point and assuming the average apparent infection rate observed in the epidemics recorded during 1991 to 1993. By the end of the season, predicted and observed progress of the disease were compared visually. In addition, the apparent infection rate was calculated for the observed data and compared by a paired t test ($P = 0.05$) with the predicted value.

Environmental effects on components of the *P. helianthi* life cycle. An isolate of *P. helianthi* race 3 sampled from a commercial field in 1993 was used for inoculation in all controlled environment experiments. Sunflower seed (cultivar DY-3) was sown in 1-liter pots, four plants per pot, and grown in a greenhouse at 25°C under natural light. When plants were at the two- or three-leaf stage, they were inoculated by spraying a solution of *P. helianthi* urediniospores (2×10^4/ml) to runoff with an atomized sprayer. Tween 20 (0.05%) was added to the urediniospore solution. After inoculation, plants were covered with plastic bags to maintain leaf wetness and placed in a growth chamber at 20°C in the dark. After 24 h, the plastic bags were removed, and the plants were returned to the greenhouse. Urediniospores were collected 14 to 21 days later and used for further studies.

Germination studies were conducted with fresh urediniospores on water agar. Urediniospores were placed in petri dishes containing water agar (2%) and spread with a glass rod. The dishes were then placed in incubators at seven different temperatures (4, 10, 15, 20, 23, 27, and 36°C) in the dark. After 24 h of incubation, urediniospore germination was determined by means of a microscope (100×). Urediniospores were considered to have germinated if the length of the germ tube was at least twice that of the urediniospore itself. Since urediniospore aggregation may reduce germination, only separate urediniospores were considered. There were six replicates (petri dishes) per treatment (temperatures) and 25 urediniospores were inspected per replicate.

In another set of experiments, the effect of temperature on the rate of urediniospore germination was examined. Urediniospores were planted as described above and incubated at 4, 12, 20, and 25°C. Germination was determined hourly during the first 6 h of incubation; at 25°C, however, it was recorded also after 24 and 30 h of incubation. There were three replicates for each combination of incubation time and temperature. The germination experiments were repeated twice.

Effects of temperature and leaf wetness duration on infection, latent period, and sporulation were determined on sunflower seedlings. Seeds were planted, grown, and inoculated as described above. Leaf wetness duration and temperature were varied in the different experiments according to the objective. Two weeks after inoculation, disease severity on true leaves was assessed visually according to a disease assessment scale (6). Each experiment had six replicates (pots with three or four plants), and experiments were conducted at least twice.

To determine the effects of leaf wetness duration and temperature during the germination and penetration processes, leaf wetness was maintained for 0, 3, 6, or 21 h after inoculation by keeping the plants covered with plastic bags for the appropriate times. When the plastic bags were removed, leaves were dried by exposing them to gentle air ventilation for approximately 10 min. Plants of all leaf wetness treatments were kept at temperatures of 5, 8, 20, or 25°C. Twenty-four hours after inoculation, plants were returned to the greenhouse and maintained at 25°C.

Effects of temperature during the latent period on the resultant infections were determined by placing the plants, 24 h after inoculation, in growth chambers at temperatures of 6, 15, 20, 25, and 30°C. The plants were inspected daily, and the appearance of chlorotic flecks or rust pustules was recorded. After the appearance of the first pustule at each temperature, the pustules per leaf were counted daily, and the time at which 95% of the final number of pustules had appeared was noted. Two weeks after inoculation, disease severity on true leaves was recorded.

In the last set of experiments, the effect of temperature on sporulation was determined. Plants were inoculated and maintained as described until the appearance of the first chlorotic flecks on leaves (7 days after inoculation). At that time, leaves were detached and cut into 2- × 2-cm segments. These segments were placed in petri dishes on water agar amended with 0.5 g of benzimidazole per liter, which postpones the senescence of the leaf segments. (A preliminary experiment indicated that benzimidazole at the concentration used does not affect sporulation.) The petri dishes were placed in incubators at temperatures of 4, 7, 12, 20, 25, 29, and 37°C. Five days later, the pustules on the leaf segments were counted, and then the leaf segments were placed into 10-ml tubes. Water (1 to 2 ml) with Tween 20 (0.05%) was added to the tubes, and they were vigorously shaken for 1 h to detach most of the spores. The number of spores in the suspension was determined with a hemacytometer, and the number of urediniospores per pustule was calculated. There were six replicates (tubes) of each treatment (temperature).

TABLE 1. Regression of the logit of sunflower rust severity on time for nine epidemics observed in Israeli fields during 1991 to 1993[a]

Year Region	Apparent infection rate	df[b]	r^2	P[c]	Time of 1% severity[d]
1991					
Lakhish-A	0.259 (0.043)[e]	2	0.924	0.039	−8
Lakhish-B	0.242 (0.040)	2	0.922	0.040	−4
Lakhish-C	0.245 (0.011)	2	0.992	0.004	−3
Lakhish-D	0.259 (0.012)	3	0.995	<0.001	2
1992					
Hadera	0.204 (0.014)	4	0.993	<0.001	2
Northern Negev	0.211 (0.010)	4	0.995	<0.001	18
Lakhish	0.229 (0.009)	2	0.995	0.002	25
1993					
Northern Negev	0.253 (0.016)	3	0.988	0.001	21
Lakhish	0.226 (0.007)	8	0.992	<0.001	16

[a] A logistic model was fit to the disease progress curves presented in Figure 1.
[b] Degrees of freedom.
[c] Probability associated with the regression equation parameter.
[d] Days after flowering at which disease severity was interpolated to be 1%. Minus sign indicates the days before flowering.
[e] Number in parentheses is the standard error.

Results of the controlled environment trials were analyzed by fitting regression equations to the data. Independent variables were the environmental parameters (e.g., temperature), and dependent variables were the components of the *P. helianthi* life cycle (e.g., percentage of urediniospore germination).

RESULTS

Rate of disease increase. Disease onset varied among the nine epidemics studied during 1991 to 1993. In some, disease appeared relatively early (e.g., in Lakhish-A, 1991), and in others it appeared late (e.g., in northern Negev, 1993). A severity level of 1% was reached from 8 days before flowering to 25 days after flowering (Table 1). The nine epidemics observed over 3 years and in three growing regions had two common traits (Fig. 1): i) disease development was rapid (the time from a severity of 5% to a severity of 95% averaged about 3 weeks), and ii) the rate of disease increase was relatively uniform, as illustrated in Figure 1 by the similarity of slopes among disease progress curves. The logit transformation made it possible to linearize the curves and calculate apparent infection rates (Table 1). The values of estimated apparent infection rates of the nine epidemics ranged from 0.204 to 0.259. Differences among rates of the epidemics, determined by the t test ($P = 0.05$) and calculated for all possible combinations of pairs of rates, were insignificant.

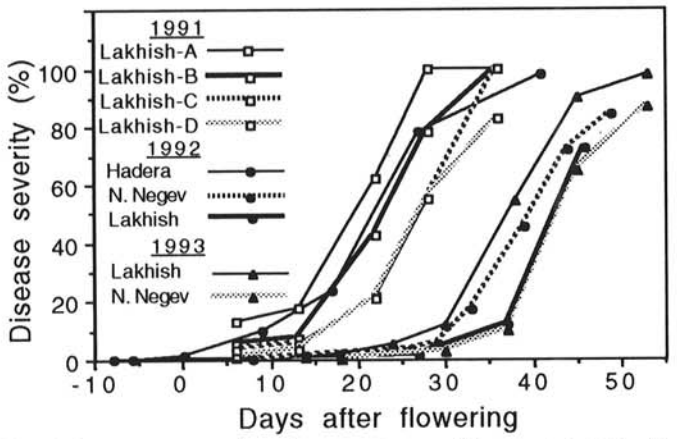

Fig. 1. Progress curves of sunflower rust, caused by *Puccinia helianthi*, recorded in three growing regions in Israel during 1991 to 1993. Parameters from the logistic model fitted to curves are presented in Table 1.

The average value of the apparent infection rate (± standard error) for the nine epidemics was 0.237 ± 0.007. This value was used in 1994 to predict disease progress in four fields. Predictions of disease progress curves were very close to the actual observed progress of the disease (Fig. 2). Values of observed apparent infection rates were calculated and compared, by means of a t test, with the predicted value ($r = 0.237$). In three of the four epidemics, differences between predicted and observed rates were not significant ($P = 0.05$) (Table 2). The average value of the apparent infection rate for the four epidemics observed in 1994 ($r = 0.232$) was very similar to the average rate determined from the nine epidemics observed previously.

Environmental effects on components of the *P. helianthi* life cycle. Temperature affected urediniospore germination significantly. Germination was >75% at temperatures below 16°C; the optimum temperature for urediniospore germination was between 4 and 14°C; and the maximum temperature for the process was 29°C (Fig. 3). At temperatures of 4 to 20°C, germination lasted less than 6 h, after which spores did not germinate (Fig. 4). At 25°C, germination continued for approximately 24 h, but final germination percentage was <15% (Fig. 4). Another parameter that is essential for germination and penetration is leaf wetness, the effects of which were examined in vivo. In general, the longer the duration of leaf wetness, the more severe was the eventual rust severity. However, 6 to 10 h of leaf wetness was sufficient to

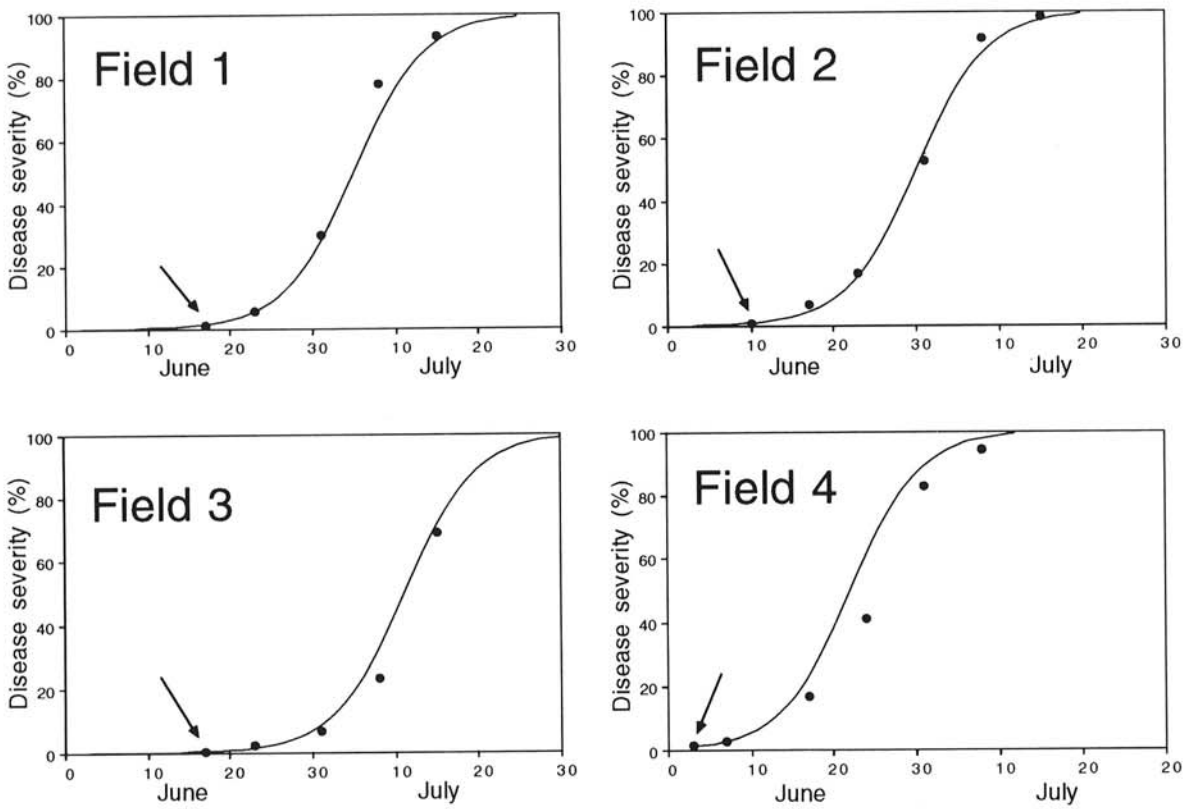

Fig. 2. Predicted (—) and observed (•) progress curves of sunflower rust, caused by *Puccinia helianthi*, in four Israeli fields during 1994. Predictions were made by computing a logistic curve based on an apparent infection rate of 0.237 and the data point in each field marked by an arrow. Statistics from the observed disease progress curves fitted to the logistic model are presented in Table 2.

produce infections. Some pustules developed after only 3 h of leaf wetness, but none occurred on leaves that did not remain wet after inoculation (i.e., the 0-h treatment) (Fig. 5).

Effects of temperature on the latent period and the severity of the resulting infections were examined in another set of experiments. Temperatures of 15 to 25°C did not affect the latent period. The first chlorotic flecks were observed 6 to 7 days after inoculation; and by 8 to 10 days, the first pustules were observed. Nearly all the pustules appeared within 10 (at 25°C) to 13 (at 15°C) days after inoculation. Effects of temperature on the severity of the resulting infections were significant: the minimum, optimum, and maximum temperatures were 4, 10 to 24, and 30°C, respectively (Fig. 6).

TABLE 2. Regression of the logit of sunflower rust severity on time for four epidemics observed in Israeli fields during 1994[a]

Field	Apparent infection rate	df[b]	r^2	P[c]	t[d]
1	0.252 (0.007)[e]	4	0.996	<0.001	2.14
2	0.267 (0.014)	6	0.994	<0.001	2.14
3	0.231 (0.015)	4	0.984	<0.001	0.40
4	0.177 (0.020)	4	0.997	<0.001	3.01*

[a] A logistic model was fit to the data points presented in Figure 2.
[b] Degrees of freedom.
[c] Probability associated with the regression equation parameter.
[d] Values of t from a t test conducted to examine the hypothesis that the apparent infection rate calculated for each epidemic differs from the value of 0.237. * = t significant at $P = 0.05$.
[e] Number in parentheses is the standard error.

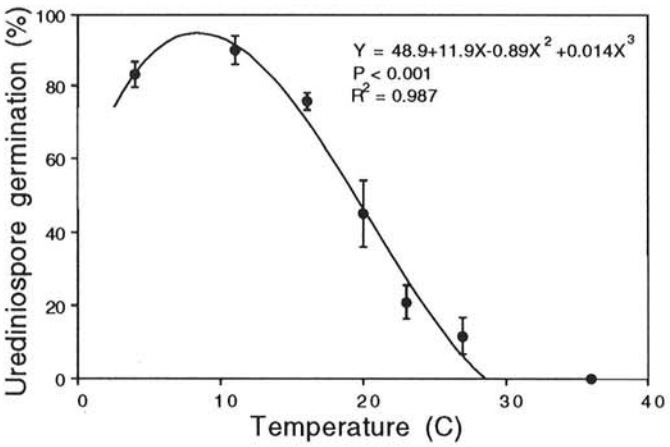

Fig. 3. Effect of temperature on the germination of *Puccinia helianthi* urediniospores on water agar. Bars indicate the standard errors.

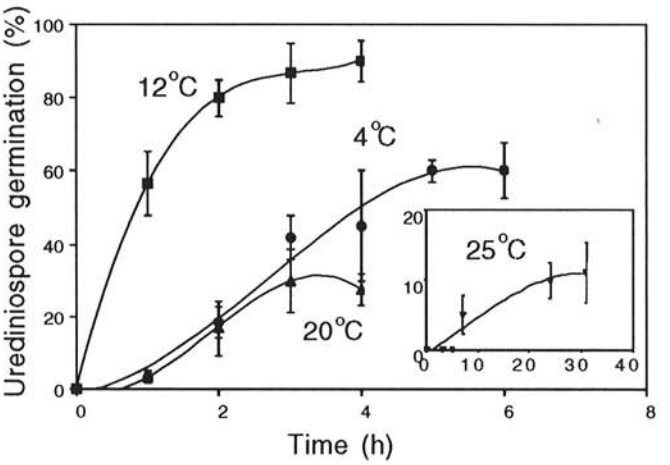

Fig. 4. Effect of temperature on the rate of germination of *Puccinia helianthi* urediniospores on water agar. Bars indicate the standard errors.

After the formation of uredia, urediospores formed. Sporulation occurred over a relatively wide range of temperatures (4 to 39°C). Within the optimum range of 20 to 35°C, 5,000 to 7,000 urediniospores were formed per pustule (Fig. 7).

DISCUSSION

The onset time of the rust epidemics varied substantially among sunflower fields during the 4 years of study. The differ-

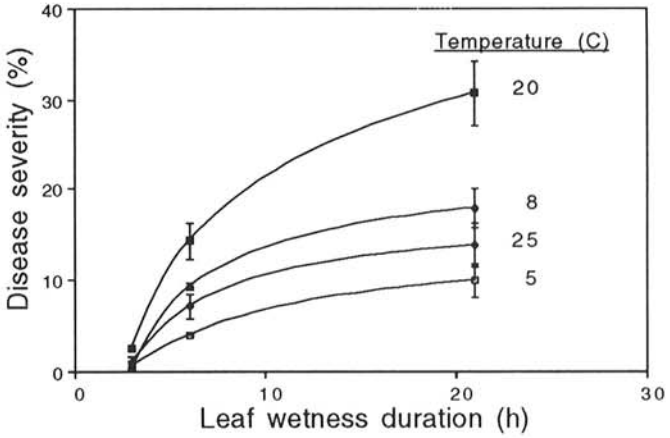

Fig. 5. Effects of leaf wetness duration and temperature during the first 24 h after inoculation with *Puccinia helianthi* on the severity of sunflower rust. Bars indicate the standard errors.

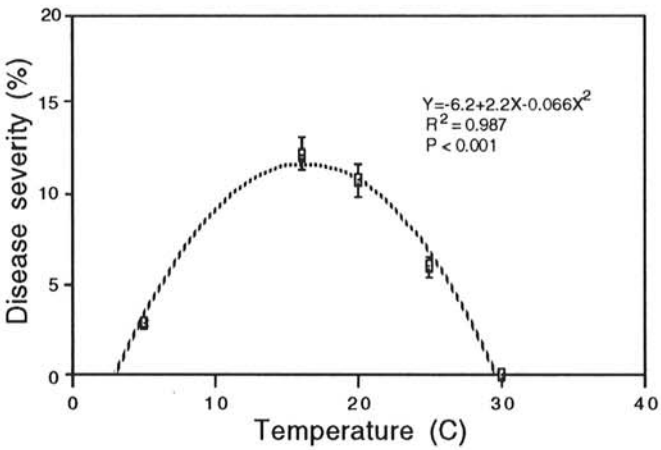

Fig. 6. Effect of temperature during the latent period of *Puccinia helianthi* on the severity of sunflower rust. Bars indicate the standard errors.

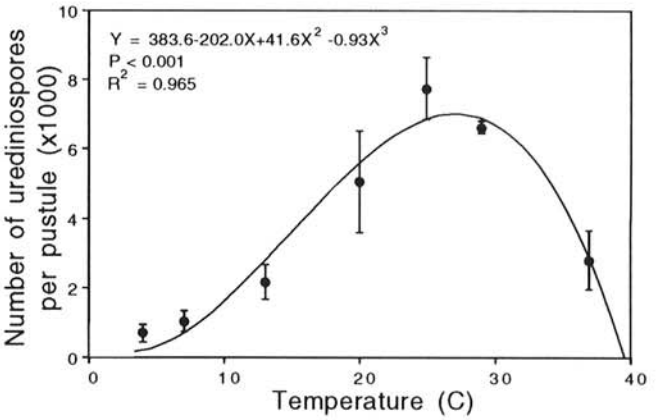

Fig. 7. Effect of temperature on the production of urediniospores of *Puccinia helianthi*, the causal agent of sunflower rust. Bars indicate the standard errors.

ences in time of epidemic onset probably resulted from differences in the amount or time of arrival of inoculum that initiated disease in each field. However, after the onset of disease, epidemics developed rapidly in all fields observed (Table 1). The fact that the rate of disease increase was relatively uniform over several years of the study in different regions is important from both the practical (planning disease-management strategies) and theoretical (increasing our knowledge of the epidemiology of *P. helianthi*) points of view.

Guidelines for the management of sunflower rust by fungicide applications were developed recently (18). The injury threshold, i.e., the level of disease intensity at which fungicides should be applied to achieve adequate disease suppression, was found to be an average severity of 3% on the upper four leaves. The relationship between the time of the season at which disease reached the injury threshold and the resulting yield loss was linear and negative. When the injury threshold was reached at or later than 27 days after flowering, the resulting damage was insignificant. Consequently, the action threshold for management of sunflower rust was defined as the occurrence of 3% disease severity prior to the 27th day after flowering. Results of the current study enabled researchers to predict, at an early stage of the epidemic, when disease severity was expected to reach the injury threshold and thus to know in advance whether fungicidal sprays would be needed. On the basis of these findings, a decision support system named Sunflower Disease Control Advisory (SDCA) was developed (17). The SDCA system was evaluated commercially during the 1995 season, and the results were promising (Shtienberg, *unpublished*).

To understand the epidemiology of *P. helianthi*, it is essential to know how it is affected by various factors of the environment. Effects of environmental variables on components of the life cycle of *P. helianthi* were studied thoroughly during the late 1960s and early 1970s by Sackston and coworkers (9,22,23) on sunflower oilseed hybrids and Canadian isolates of *P. helianthi*. Comparison of their results with ours provides an interesting insight into sunflower rust epidemiology.

Germination of urediniospores occurred at temperatures of 4 to 20°C, with the optimum at 4 to 14°C (Fig. 3); at 5 to 20°C, the process lasted 4 to 6 h (Fig. 4); moisture was required for infection, and 6 to 10 h of leaf wetness was sufficient to induce infections (Fig. 5). These findings are in close agreement with those reported for *P. helianthi* (9,22,23), *P. recondita* (2,3,11, 23,24), and *P. sorghi* (7). During the period of rust development in Israel, there is usually no rainfall, and sufficient moisture for germination of urediniospores on sunflower leaves is not likely to occur during the daytime. Moreover, urediniospore germination is inhibited, to some extent, by light (3,25). Therefore, germination occurs primarily at night, and the infection process may be completed within one night. The long-term average minimum temperature in the sunflower-production areas in Israel during May and June is 13.5 to 20.0°C. Dew is abundant, and leaves are wet each night for at least 6 h. Consequently, conditions at night are optimal for urediniospore germination and penetration, processes that are not likely to be limited by the environment in Israel.

Once a rust urediniospore has penetrated the leaf, only temperature affects its subsequent growth (6). At 15 to 25°C, approximately 8 to 10 days elapsed from inoculation until the appearance of the first pustule; the optimal temperature was 10 to 24°C. The minimum and maximum temperatures for completing the infection process were 4 and 30°C, respectively (Fig. 6). These findings are in close agreement with those reported previously for *P. helianthi* (9,22) and *P. recondita* (5,10,25). During the period of rust development in Israel, the long-term daily average temperature in the sunflower-production areas is 18.1 to 25.5°C. Thus, in Israel, infection is not likely to be limited by the environment.

After uredia formation, urediniospores are produced. During the initial stages of sporulation, high relative humidity is required, but for urediniospore dissemination and dispersal, dryness is preferable. Formation of individual urediniospores requires 10 to 14 h. Thus, if formation is initiated at night, urediniospores may be discharged in the following morning. Since sporulation occurs over a relatively wide range of temperatures (4 to 39°C) and the leaves are wet during the night and dry during the day, sporulation is probably not limited by the environment in Israel.

On the basis of these findings, one can understand why the rate of disease increase was so rapid and uniform over the 4 years of the study. It seems that the spring environment in Israel is usually conducive to *P. helianthi* development. The host, *H. annuus*, is not native to Israel and was introduced into the country only about 40 years ago (1). The geographical area of origin of *H. annuus* is the southern United States (8), and the conditions in that area, at least during the spring and early summer, are very similar to those prevalent in Israel. Another interesting finding was that, in general, the temperature requirements of *P. helianthi* isolates in Israel match very closely those in Canada (9,22,23). Since the environmental conditions in Canada and Israel during the time of sunflower growth differ markedly, it was expected that the ecological requirements of *P. helianthi* isolates would differ as well. The relatively broad range of conditions to which *P. helianthi* has adapted (2) probably account for the similar requirements of the Israeli and Canadian isolates.

LITERATURE CITED

1. Anikster, Y., and Dinoor, A. 1967. The biology and control of sunflower rust in Israel. Pages 48-49 in: Proc. Isr. Congr. Plant Pathol., 1st. Israeli Phytopathological Society, Rehovot, Israel.
2. de Vallavieille-Pope, C., Huber, L., Leconte, M., and Goyeau, H. 1995. Comparative effects of temperature and interrupted wet periods on germination, penetration, and infection of *Puccinia recondita* f. sp. *tritici* and *P. striiformis* on wheat seedlings. Phytopathology 85:409-415.
3. Eversmeyer, M. G., Kramer, C. L., and Hassan, Z. M. 1988. Environmental influences on the establishment of *Puccinia recondita* infection structures. Plant Dis. 72:409-412.
4. Fick, G. N., and Zimmer, D. E. 1975. Influence of rust on performance of near-isogenic sunflower hybrids. Plant Dis. Rep. 59:737-739.
5. Gulya, T. J., Miller, J., and Rashid, K. Y. 1989. Rust races occurring in North America in 1988 and resistance of sunflower hybrids to races 1 and 3. Pages 19-20 in: Proc. Sunflower Res. Workshop.
6. Gulya, T. J., Venette, R., Venette, J. R., and Lamey, H. A. 1990. Sunflower Rust. N.D. State Univ. Ext. Bull. PP-998.
7. Headrick, J. M., and Pataky, J. K. 1986. Effects of night temperature and mist period on infection of sweet corn by *Puccinia sorghi*. Plant Dis. 70:950-953.
8. Heiser, C. B., Jr. 1978. Taxonomy of *Helianthus* and origin of domesticated sunflower. Pages 31-53 in: Sunflower Science and Technology. J. F. Carter, ed. American Society of Agronomy, Crop Science Society of America, and Soil Science Society of America, Madison, WI.
9. Hennessy, C. M. R., and Sackston, W. E. 1970. Studies on sunflower rust. V. Culture of *Puccinia helianthi* throughout its complete life cycle on detached leaves of sunflower (*Helianthus annuus*). Can. J. Bot. 48:1811-1813.
10. Kramer, C. L., and Eversmeyer, M. G. 1989. Effect of temperature on the latent period of *Puccinia recondita* f. sp. *tritici* on adult wheat plants. Mem. N.Y. Bot. Gard. 49:62-65.
11. Kramer, C. L., and Eversmeyer, M. G. 1992. Effects of temperature on germination and germ-tube development of *Puccinia recondita* and *P. graminis* urediospores. Mycol. Res. 96:696-693.
12. Lal, B. B., Mathur, S., Chakravarti, B. P., Singh, R. B., and Singh, R. D. 1980. Increase in sunflower yield by controlling rust with systemic and non-systemic fungicides. J. Turk. Phytopathol. 9:89-96.
13. Lal, B. B., and Singh, R. D. 1977. Control of sunflower rust by pre- and post-infection spraying of systemic fungicides. Pesticides 11:24-25.
14. Lilleboe, D. 1990. Roping in rust: Problems and answers. Sunflower 16:18-21.
15. McMullen, M. P., ed. 1985. Sunflower Production and Pest Management. N.D. State Univ. Ext. Bull. 25.
16. Sackston, W. E. 1959. Effects of artificial defoliation on sunflowers. Can. J. Plant Sci. 39:108-118.
17. Shtienberg, D. 1994. Sunflower rust disease control advisory (SDCA): A decision support system for sunflower rust. (Abstr.) Phytoparasitica 22:78.
18. Shtienberg, D. 1995. Rational suppression of sunflower rust: Develop-

ment and evaluation of an action threshold. Plant Dis. 79:506-510.
19. Shtienberg, D., and Zohar, D. 1992. Fungicidal disease suppression and yield losses associated with sunflower rust in Israel. Crop Prot. 11:529-534.
20. Siddiqui, M. Q. 1980. Some effects of rust infection and moisture stress on growth, diffusive resistance and distribution pattern of labeled assimilates in sunflower. Aust. J. Agric. Res. 31:719-726.
21. Siddiqui, M. Q., and Brown, J. F. 1977. Effects of simulated rust epidemics on growth and yield of sunflower. Aust. J. Agric. Res. 28:389-393.
22. Sood, P. N., and Sackston, W. E. 1969. Studies on sunflower rust. VI. Penetration and infection of sunflowers susceptible and resistant to *Puccinia helianthi* race 1. Can. J. Bot. 48:2179-2181.
23. Sood, P. N., and Sackston, W. E. 1972. Studies on sunflower rust. XI. Effects of temperature and light on germination and infection of sunflower by *Puccinia helianthi*. Can. J. Bot. 50:1879-1886.
24. Stukey, R. E., and Zadoks, J. C. 1989. Effect of interrupted leaf wetness on pustule development of *Puccinia recondita* f. sp. *tritici* on wheat. Neth. J. Plant Pathol. 95(suppl. 1):175-185.
25. Tollenaar, H. 1985. Urediospore germination and development of some cereal rusts from south central Chile at constant temperatures. Phytopathol. Z. 114:118-125.
26. Van der Plank, J. E. 1963. Plant Diseases: Epidemics and Control. Academic Press, New York.