

Physiological Races of *Venturia pirina* on Pear

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ABSTRACT

Five races of *Venturia pirina* were isolated from cultivated and wild pears in Israel. These races were found to be specific to five differential hosts of *Pyrus communis* and *P. syriaca* origin. Four of these differential hosts were immune to incompatible races; the fifth was sensitive to its compatible race, immune to one race, and resistant to three races. Environmental conditions such as

temperature, periods of free moisture on the leaves, and light did not change the specific pathogenic properties in two races on which the environmental influences were tested. In these tests, infection of *V. pirina* occurred at temperatures from 5 to 28 C with an optimum of ca. 20 C.

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Additional key words: pear scab, pear cultivars.

Observations in England (9), the USA (2), and Switzerland (12) on distribution of *Venturia pirina* Aderh. on pears indicate that various host cultivars are parasitized by different biotypes of the pathogen. No experimental work verified these observations, and no attempts were made to justify the use of the term "biotypes". In the absence of such proof, the described phenomenon could also be related to the presence of ecological or physiological races, and even to distinct species of *Venturia*, as found in Japan (10). Similar possibilities may explain the occurrence of pear scab in Israel (7). There has been commercial cultivation of pears in this country for about 40 years, and until the early 1960's, the introduced cultivars of *Pyrus communis* L. did not become infected by *V. pirina*. However, at the same time, scab was found in old and neglected remnants of cultivated orchards of unknown cultivars of *P. communis* (5). In the early 1960's, scab appeared on the Spadona cultivar, which occupies about 80% of the acreage in Israel, as well as on the Gentile, Costia, and Amanlis cultivars (4). Scab was also found on wild trees of *Pyrus syriaca* Boiss. growing in the hills of Galilee and Carmel (E. Shabi, unpublished data).

In the present work, we report attempts to determine whether the fungus infecting pears in Israel belongs to ecological or physiological races of *V. pirina* or to a different species of *Venturia*. This was done by (i) a taxonomic study of fungi attacking various hosts; (ii) establishing differential hosts by which races can be identified; and (iii) checking on whether the host-parasite interactions described in section (ii) are stable under a variety of ecological conditions.

METHODS AND RESULTS.—*Identification.*—More than 100 samples of scab-infected leaves were collected from commercial and unidentified cultivars of *P. communis* and from *P. syriaca* trees. Isolations of the pathogen were made on 1% Difco potato-dextrose agar mixed in equal proportions with

Difco-Bacto agar. From 10 isolates belonging to five groups, single-spore cultures were made, and measurements were made on conidia from these cultures, as well as on conidia of artificially inoculated leaves. In each case, the length and width of 100 conidia were measured.

The results showed that the dimensions of conidia of all our isolates were similar. The width-length averages ranged from 6.0 to 17.4 to 7.0 to 20.6 μ in conidia from infected leaves, and from 7.0 to 18.6 to 8.1 to 21.2 μ in conidia from cultures. Width-length averages of *V. pirina* conidia, as found in the literature, varied from 6.0 to 18.5 to 8.3 to 25.3 μ (6, 10, 11, 12). Our measurements indicate that our isolates belong to *V. pirina*.

Establishment of differentials and races.—Buds from eight cultivars of *P. communis* and five trees of *P. syriaca* were grafted on Quince A (*Cydonia oblonga* Mill.) cuttings and cultivated in a greenhouse in 1-kg pots (10 cm diam, 18 cm height). For preliminary identification of races, we sprayed emerging leaves of the test plants with a population of conidia found on infected leaves. Altogether, 103 isolates of the fungus were used in different inoculations. This procedure enabled us to make the first selection of isolates infecting different cultivars of pears but having a similar range of hosts, and hosts which showed a similar reaction to various isolates. For instance, a similar isolate was able to infect both Spadona and Gentile. Since the reaction of Spadona to infection was more marked, Spadona and not Gentile was chosen as a representative differential host to the suspected race. Other differential hosts consisted of Amanlis and two trees of an unknown cultivar of *P. communis*. These latter were named "Galilee" and "Judea" in accordance with the region in which they were found. The fifth differential was of a wild tree of *P. syriaca*.

In addition to these five differential hosts, the cultivar Succari of *P. communis* was found to be

TABLE 1. Physiological specialization of five races of *Venturia pirina*. The differential hosts were infected with two single-spore isolates of each race (A and B)

Race of <i>V. pirina</i>		Avg percentage of infected leaves on differential hosts ^a				
Race	Isolate	<i>Pyrus communis</i> cultivars				<i>P. syriaca</i>
		Amanlis	Spadona	Judea	Galilee	
1	A	90	0	0	0	(37) ^b
	B	80	0	0	0	(57)
2	A	0	85	0	0	(27)
	B	0	71	0	0	(11)
3	A	0	0	61	0	0
	B	0	0	65	0	0
4	A	0	0	0	68	(16)
	B	0	0	0	74	(11)
5	A	0	0	0	0	36
	B	0	0	0	0	53

^a The figures describe the average percentage of infected leaves out of inoculated leaves of five tests. In each test, two plants of each differential with a total of 20 to 40 leaves were inoculated per treatment.

^b Figures in parentheses indicate that the lesions were necrotic, almost nonsporulating.

incompatible with all of the tested isolates. Succari was immune to three races, whereas two others caused necrotic, almost nonsporulating lesions.

For cross-inoculations between races and hosts, single-spore cultures were used. However, the slow growth of the fungus in culture and the low rate of sporulation in some isolates prevented the use of culture-grown conidia. Therefore, conidia for inoculations were obtained from compatible plants previously infected by a given single-spore culture, grown in isolation in a greenhouse. The inoculations were made with 5-mm-diam discs of filter paper dipped in a suspension of ca. 280,000 conidia/ml and placed on both sides of the emerging leaves. The infected plants were kept for 24 hr in a moist chamber at 20 C, and for an additional 21 days in a growth chamber at 20 C. Descendants of two single-spore cultures from each suspected race were used. These cross-inoculations were repeated 5 times. In each test, two plants from each differential, with a total of 20 to 40 leaves, were infected.

Each of the four differentials of *P. communis* origin was susceptible to only one race (Table 1). The other three differential hosts of *P. communis* origin were immune to this race. This was confirmed by microscopic examination of the inoculated tissue, as no traces of necrosis were found. *Pyrus syriaca* became infected with its own specific race (race 5) as well as by the races 1, 2, and 4. However, infection of *P. syriaca* by its own race caused sporulating lesions, whereas infection by the races 1, 2, and 4 resulted in necrotic spots, with few, if any, conidia.

Effect of ecologic factors on infection.—This set of experiments was done to check whether variations of temperature, period of free moisture on the leaves, and light may change the specific reaction of the host to inoculation by a given race. For inoculum, race 1 or race 3 was used. The temperature and moisture period experiments were made on three hosts: one compatible to a given race; one incompatible; and the resistant *P. syriaca*, which reacts to these two races by necrotic, almost nonsporulating lesions. In the light experiments, the resistant Succari cultivar was used instead of *P. syriaca*. Degrees of infection were evaluated in similar fashion to those established for evaluating reaction of apples to *V. inaequalis* (8), and consisted of 0 = no microscopic evidence of infection; 1 = necrotic nonsporulating lesions; 2 = necrotic or chlorotic lesions with few conidia; 3 = sporulating lesions restricted by a chlorotic halo; and 4 = extensive and abundantly sporulating lesions.

In the temperature and moisture-period tests, the inoculated plants were kept in moist chambers for either 24 or 64 hr at 5, 10, 15, 20, 25, or 28 C, after which they were incubated in a growth chamber at 20 C.

The patterns of disease development remained constant under all the conditions tested and were similar to those described in the foregoing section (Fig. 1). At all but the highest temperature, sporulating lesions formed on the compatible hosts. No lesions developed on immune hosts, and the lesions on *P. syriaca* were necrotic, almost nonsporulating. The period of incubation was always shorter on the compatible hosts (Fig. 1-A, C) than on *P. syriaca* (Fig. 1-B, D). After incubation for 9 days, the maximum development of lesions occurred at 15 and 20 C. After incubation for 20 days, no distinct peak of optimum temperature was obtained (Fig. 1-A, C). In all cases, a wet period of free moisture on leaves of 64 hr compensated for the less favorable temperatures (Fig. 1).

In the last experiments, the Amanlis, Judea, and Succari test plants were infected with race 1 or race 3 under various conditions of light. The light treatments started 5 days before inoculation. Groups of the three differentials were kept under natural light in a greenhouse, under 30 ft-c of light, or in complete darkness, all at 20 C, then inoculated by either of the two tested races. After inoculation, each group was divided into three subgroups, each of which was then kept under continuous light of 300 ft-c, 30 ft-c, or complete darkness, in 20-C growth chambers. The first 24-hr period of this incubation was in a moist chamber. For every combination, three plants of each differential with ca. 25 leaves on each plant were tested. The results were evaluated after incubation of 23 days.

It was found that the 5-day period of high light, low light, or darkness prior to inoculation had no effect on disease development. The inoculated plants incubated in high light and low light reacted to infection by the two races according to their compatibility, as described before. Inoculated plants incubated in darkness became etiolated, and no

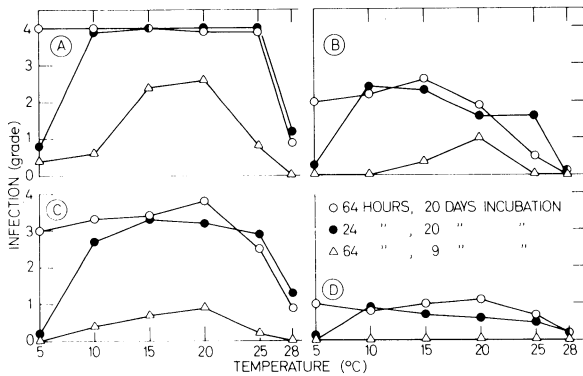


Fig. 1. The effect of temperatures during 24 or 64 hr of free moisture on the leaves on development of the races 1 or 3 on A, C) compatible hosts and on B, D) resistant hosts. After the periods of free moisture on the leaves, the test plants were incubated for 20 days at 20 ± 1 C. Average of four replicates (ca. 100 leaves per treatment).

lesions were seen even on the susceptible hosts. However, when these plants were transferred, after 16 days, from darkness to a greenhouse, the leaves became green and the disease spread on the compatible hosts only. Necrotic and almost nonsporulating lesions appeared on the leaves of the resistant cultivar Succari.

DISCUSSION.—The specific reaction of some cultivars and species of pears to *V. pirina* seems to be due to the occurrence of physiological races of the fungus. Verification of this hypothesis was determined by establishment of differential hosts, each of which reacted specifically to a given race (Table 1); and by retention of the pathogenic properties of a race under a variety of environmental conditions (Fig. 1).

Of the five differential hosts established in this study, the four cultivars of the *P. communis* were susceptible to one race and immune to other races. The fifth differential host was represented by the wild *P. syriaca*, which was sensitive to one race, immune to one race, and highly resistant to three races. The appearance of lesions on *P. syriaca* and on Succari differs from the hypersensitive reactions found in some cases of apple scab (8), because they appear after a long period of incubation. When compared with reactions of apples to *V. inaequalis*,

these lesions resemble the class 2 reactions, or the "fleck" type of host-parasite response (1, 8).

We do not know the genetic relationship between the differential hosts and their specific races of *V. pirina*. It is possible that there are more than five races of *V. pirina*. Considering the role played by ascospores in the creation of new races, this is a likely situation, and Langford & Keitt (3) succeeded in producing segregates differing in pathogenicity by crossing single ascospore cultures of this fungus.

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