Technical Papers

Interpretation of Resistance to Fusarium Wilt in Tomato

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It has been established (*6*) that the Pan America tomato is highly, but not completely resistant to the Fusarium wilt disease, caused by *F. oxysporum* f. *lycopersici* Snyder and Hansen, under conditions fatal to the susceptible variety, Bonnie Best. In endeavoring to determine the nature of this resistance Fisher (*2*), and more recently Gottlieb (*3*), and Irving, Fontaine, and Doolittle (*5*) have given evidence that the expressed sap from resistant Pan America plants is inhibitory to the growth of the pathogen *in vitro* when compared to that from susceptible Bonnie Best plants. Heinze and Andrus (*4*) have concluded from their study of reciprocal grafts of these two varieties that "resistance to the wilt fungus in tomatoes appears to be localized entirely in the root systems of the resistant varieties and is not transportable." This implies that the tops of Pan America are susceptible. However, only infection through roots was considered, and no evidence was presented on susceptibility or resistance of tops. Moreover, the work of Irving, *et al.* and Gottlieb indicates that the substance responsible for resistance is found in all parts of the plant.

The writers have approached this question with still a different technique. Our results, added to what has already been done on the subject, seem to justify the following conclusions: First, resistance is not localized in the root system. Second, the substance responsible for the resistance is not present in inhibitory amounts in the xylem stream. Third, resistance originates in the living tissues of the plant, and the material causing resistance does not migrate or diffuse into the xylem vessels. Fourth, field resistance to infection functions in living cells of the roots through which the fungus, a soil organism, must pass to become a vascular pathogen.

Bonnie Best and Pan America plants were grown in sterilized soil and inoculated when six weeks old by cutting through the lower taproot under a spore suspension of the fungus. The roots were washed and the plants repotted singly in six-inch pots in the greenhouse. In every case the fungus spores began growth in the vascular elements of the root into which they had been drawn when the root was cut, and the mycelium developed upward through the root into the above-ground stem, producing therein vascular discoloration. The discoloration extended higher in the stems of Bonnie Best than in those of Pan America. Foliage symptoms appeared on Bonnie Best rapidly under these conditions, but only a few individuals of Pan America showed disease symptoms. The failure of symptoms to appear in all Pan America plants containing the fungus in the xylem of the stem indicates the presence of resistance in the stem as well as the roots.

The presence of the fungus in the xylem was demonstrated by cultures, and by microscopic examination of stained sections it was shown to have developed in the vascular elements of Pan America about as robustly and abundantly per infected element as in Bonnie Best. Therefore, if there were any fungus inhibitor present in the tracheal fluid of the Pan America plants, it must have been negligible in its effect. The number of vessels infected in Pan America was much lower than in Bonnie Best (perhaps owing to the inoculation method), and this fact may explain in part why the former shows symptoms slowly, if at all, even though the fungus be present in root and above-ground stem. An explanation for this phenomenon may lie in multiple infection of the xylem elements of Bonnie Best following surface contamination of lateral roots during inoculation, whereas infection of Pan America by this technique allows invasion only of those elements into which the spores actually were drawn. If this interpretation is correct, the wilt symptoms obtained in Bonnie Best, but not in invaded Pan America plants, are an expression of fungus mass action in toxin formation.

The comparable robust growth of the fungus *in vivo* in the infected vascular elements of both varieties of plants also argues against a mobile fungus inhibitor in one of them. In the resistant plant, growth of the fungus in the xylem shows that resistance is not a property of the entire root system.

Additional evidence that an inhibitory substance is lacking in xylem fluid was shown by the comparable growth of the fungus *in vitro* in such liquid obtained from resistant as well as susceptible plants. The fluid forced by root pressure from the cut ends of plants truncated at the soil level was used for this test. It appears, therefore, that xylem fluid is noninhibitory to the fungus *in vivo* and *in vitro*. This would indicate the absence or high dilution of the resistant factor therein.

If disease resistance in Pan America is physiological in nature, conceivably it may consist of resistance to invasion, resistance to yellowing and wilting, or to both. The former, which is under consideration in this paper, is concerned with the entrance of the fungus into the xylem; the latter, with production of toxins in the tissue.

If it is true that under natural conditions favorable for the disease *F. oxysporum* f. *lycopersici* enters the susceptible plant through the undifferentiated tissue just back of the root tip and that the resistant plant does not become invaded, at least not to the extent that vascular elements are entered, the above observations would indicate that resistance is a quality of the living cells only of the Pan America plant. It is this quality which appears to bar the fungus from the lumen of the xylem tubes.

It may be concluded from all evidence so far presented that resistance here in the Pan America tomato to invasion by the wilt Fusarium is a direct function of the cellular protoplasm of the plant similar to that of cabbage (*1, 7*). Apparently it is present but not localized in the root system, and does not operate in the xylem.

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The Blood Parasites of the Blue Grouse

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In a recent paper Herman (*1*) listed the avian blood protozoa of North America and indicated the hosts in which they had been found. In view of the fact that the blue grouse (*Dendragapus obscurus*) is not included in his host list it would seem to be of value to record a few observations made recently upon the blood of this species.

Blood samples were collected during the summers of 1943 and 1944 from 44 specimens of blue grouse (Subspecies: *Dendragapus o. fuliginosus*) taken on Vancouver Island, British Columbia. In 1943 smears were made from 28 birds taken at Campbell River during June, July, and August. Four more were made in August of the following year. One smear was made from a bird collected at Cowichan Lake in 1943 and from 11 additional specimens collected from the same vicinity during September 1944.

Four blood parasites, namely, *Trypanosoma, Haemoproteus, Leucocytozoon,* and *Microfilaria,* were observed in smears after they had been stained in Giemsa. The incidence of infection with these parasites is shown in Table 1.

The incidence of infection was higher in Campbell River birds examined in 1943 (see Table 2) than in the combined sample from Campbell River and Cowichan Lake (see Table 1).

TABLE 1

Incidence of Parasites in Per Cent (Campbell River and Cowichan Lake, 1943 and 1944)

No. of birds	44 Whole sample	23 Adults	21 Juveniles
Trypanosoma	5	4	5
Haemoproteus	52	57	48
Leucocytozoon	18	22	14
Microfilaria	12	22	..
Negative	41	43	38

In Table 2 it will be noted that while the incidence of infection among juveniles approximates that shown in Table 1 the figures for adults are considerably higher. Unfortunately, no comparison can be made between the two collecting stations because there is insufficient material from Cowichan Lake for 1943.

TABLE 2

Incidence of Parasites in Per Cent (Campbell River Only, June, July, August, 1943)

No. of birds	28 Whole sample	12 Adults	16 Juveniles
Trypanosoma	7	10	6
Haemoproteus	64	83	50
Leucocytozoon	29	42	19
Microfilaria	15	40	..
Negative	25	17	31

The degree of infection observed for these four parasites varied, but in no case were they very great. Trypanosomes were found in extremely small numbers in only two birds. *Leucocytozoon* seldom exceeded two parasites per thousand blood cells. In the case of *Haemoproteus,* the commonest and most numerous form noted, the infection in adults at Campbell River averaged 12 parasites per thousand cells during the months of June, July, and August. Infections ranging from 1 to 27 organisms per thousand erythrocytes were noted. The figures for juvenile birds collected during the same period were, on the average, somewhat lower. All birds collected at Cowichan Lake during September 1944 showed light infections ranging around two to three organisms per thousand cells. Moreover, 7 out of 10 smears collected