The Fruit Pox and Gold Fleck Syndromes of Tomato

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ABSTRACT

The disease formerly known as tomato fruit pox was shown to consist of two diseases each with distinct symptom patterns. The two diseases were found to be separate and distinct when studied in the field and laboratory and were named fruit pox and gold fleck. Fruit pox was shown to be controlled by a recessive gene designated (*fp*) and gold fleck by a dominant gene designated as (*Gdf*). Maternal effects on expression of both characters were absent and dominance apparently complete.

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The disease known as tomato fruit pox was first noted by Ivanoff in 1937 in scattered fields in southwest Texas (5). In 1940, Ivanoff & Young (6) published a detailed description of the disease, and stated that it has caused serious economic losses in 1938 in Texas. In 1940, Locke (7) reported the disease was occurring in Arkansas tomato fields. In summary, Walker (8) described pox as a disease which occurred on outdoor tomatoes, particularly in Texas and Arkansas but also in the middle western and southeastern states to some extent. The next report of the occurrence of the disease was by Butler et al. (1) in 1958 describing an outbreak of pox in California.

Ivanoff (5) and Ivanoff & Young (6) concluded that the disease was not caused by any fungus or bacterium. They were unable to culture any pathogen using standard isolation techniques and were unable to observe any microorganisms when diseased tissues were studied microscopically. Locke (7) and Butler et al. (1) reached a similar conclusion which was based upon the failure to isolate any microorganisms from the diseased tissue. Based upon three observations, Ivanoff & Young (6) concluded the fruit pox disease was not hereditary in nature: (i) pox occurred on all observed varieties; (ii) pox occurred in most tomato-growing areas of the U.S. as well as in Mexico, Cuba, and Puerto Rico; and (iii) seed from pox-affected fruits often produced plants that bore only normal, nonpoxed fruit.

Ivanoff & Young (6) described the tomato fruit pox disease as follows: The first symptom on green fruit consisted of many conspicuous, abnormally dark green dots scattered over the fruit surface. Spots varied in size from a very small speck to about 3-mm in diam and were round, elongated or irregular. Later, many of the dark green spots became sunken as pits or pox with ruptured surface tissues. The change from unbroken, dark green spots to the pitted, pox stage occurred in a few days and presumably occurred during transit and storage. As the green fruits turned pink and red, the dark green spots remained green or turned yellow while the pox corked over and appeared as an abnormally large lenticel on the surface of the fruit. Butler et al. (1) described similar symptoms. Their report conflicted with that of Ivanoff & Young (6), however, in that they described the pox lesions as being tan-colored. These lesions generally were ruptured and the tissue of older lesions became corky.

Since 1966 this disease has increased in importance on Florida fresh market tomatoes (2). The fruit are unsightly and tomato buyers discriminate and often reject fruit afflicted with this disease. Many tomato growers in Florida have complained about fruit pox and consider it to be one of their more serious production problems. The indeterminate fresh market cultivar 'Floradel' is especially damaged by this disease and, because of this, the number of acres devoted to this otherwise outstanding cultivar have decreased significantly. The objective of this study was to define and elucidate the tomato fruit pox disease. Preliminary reports of this research have been published elsewhere (2, 4).

MATERIALS AND METHODS.— *Genetic study 1.*—Analysis of observations of progenies from the University of Florida tomato breeding program and results from several years dating back to 1950 led to the formation of the hypothesis that fruit pox is genetically controlled. To investigate this hypothesis, near-isogenic lines of 'Tropi-Red' were selected as parents and preliminary crosses were made with "fruit pox Tropi-Red" by "normal Tropi-Red". Selected progeny from this cross were evaluated in the F_2, F_3, and F_4 and numbers of plants producing poxed and normal fruit were recorded.

Symptomological Studies.—During the critical evaluation of various populations from the Tropi-Red crosses it became obvious that studies on symptom production and expression were required. Several cultivars including Floradel, 'Manapal', Tropi-Red, 'Walter' and the breeding line which was later released as 'Florida MH-1' were grown in the field during the spring 1969 season, using standard cultural practices employed for stake-tomatoes. Fruit development was observed periodically and as soon as early symptoms were noted, fruit were marked by attaching a shipping label to the fruit with a string tied loosely around the pedicel. These early symptoms on each fruit were noted and described at the time of marking. Fruit from 71 plants were studied in the field from early symptom development through the red-ripe stage. Another group of symptomatic fruit was harvested at the mature-green stage from 148 plants and observed in the laboratory through the red-ripe stage. In both studies, individual incipient lesions were marked

with a fine-point, felt-tipped marker and designated as either a fruit pox or gold fleck incipient lesion.

Genetic study 2.—A second genetic study was initiated using parents selected on the basis of symptomological studies. Reciprocal crosses were made using the breeding lines "normal Floradel" and "pox and fleck Floradel". Limited observations were made in the F_1 generation but detailed observations were recorded in the F_2. A very few F_3 families were observed because of limited facilities available for growing large populations. The study was terminated when the F_4's were killed by an unseasonal freeze. One to 25 fruit from each F_2 plant were observed for the presence of fruit pox or gold fleck. As soon as symptoms of either disease were noted on fruit from a particular cross, disease readings were made. If symptoms of either disease had not been observed after 20-25 fruit were examined, it was assumed that that particular plant was normal for both fruit pox and gold fleck. Each of the plants in the F_2 population were noted as producing fruit of one of four types; normal, gold fleck, fruit pox, or both gold fleck and fruit pox. The F_2 data were subjected to chi-square analysis for interpretation.

RESULTS AND DISCUSSION.—*Symptomology.* It was not possible to observe or isolate any pathogen from incipient pox or fleck lesions using standard plating, staining, and light microscopy techniques. From older pox lesions which had ruptured, secondary-invading microorganisms were frequently isolated.

From marked fruit studies in the field and laboratory it was obvious that two distinct sets of symptoms were involved in what was formerly referred to as pox. This possibility was suggested by the similar, but conflicting, reports of Butler et al. (1) and Ivanoff & Young (6) concerning symptom development of tomato fruit pox. These two syndromes have been termed (i) fruit pox and (ii) gold fleck. This designation is based on the fact that, of the fruit from 71 plants studied in the field, 93 percent of the incipient lesions were properly identified as either fruit pox or gold fleck. When more mature fruit were harvested and observed in the laboratory where inspections were much more convenient and easier to conduct, 100 percent of the incipient lesions on fruit from 148 plants were properly identified. Most of the fruit studied were from the cultivar Floradel. No symptoms of either fruit pox or gold fleck ever occurred on fruit of Florida MH-1.

Symptoms of fruit pox are initiated with the appearance of incipient lesions on immature green fruit. The incipient lesion is usually slightly elongate to oval and white. As the green fruit enlarges and progresses to maturity, the lesion enlarges and becomes more elongate. Eventually the lesion ruptures in the center along its long axis. Such ruptured lesions become necrotic within a period of a few hours to a few days. Speed of necrosis is apparently dependent in a large degree upon environmental conditions. If the lesion does not rupture before the fruit begins to turn pink, it may become tan and extremely difficult to distinguish from a gold fleck lesion unless it has been previously marked. This fact alone will explain the confusion which has existed in past reports referring to pox. Fruit pox lesions always appear to rupture and become necrotic before fruit turn red if the fruit are allowed to continue enlarging on the vine. It is only in the pink-fruit stage that lesion appearance is similar to gold fleck. If fruit having incipient lesions of pox are harvested very immature, these lesions never rupture and become necrotic. This suggests that the rupture is caused by decreased growth rate in the pox lesions, while surrounding tissues continue to grow and enlarge at a normal rate. This results in a stretching and eventual pulling apart or rupture in the pox lesion. Necrosis does not occur except after rupture of the lesion, and is apparently due to a desiccation.

Symptoms of gold fleck are also initiated with the appearance of small incipient lesions on immature green fruit. These lesions appear round (rather than oval) and are a dark green. As the green fruit enlarges, the lesion also enlarges proportionately. It does not rupture, and when epidermal strips are viewed with a light microscope, the only detectable difference between the lesion and surrounding tissue is an apparent excess of green pigment; whereas, pox lesions are morphologically distinguishable. As the fruit begins to turn pink, the gold fleck lesion begins to lose the dark green pigment, and becomes progressively lighter until it is tan. As the fruit becomes more mature and more red the lesion progresses from the light tan to a golden yellow on red-ripe fruit.

Development of these two distinctive syndromes does not appear to be affected by the various seasonal and environmental conditions to which tomatoes are subjected. Symptoms of both diseases were observed on Florida varieties and breeding lines in several of the tomato-producing areas of the United States including Florida, California, Oregon, Oklahoma, Texas, New Jersey, Ohio, Illinois, Wisconsin, Pennsylvania, South Carolina, and North Carolina as well as Mexico, Canada, Haiti, Puerto Rico, Jamaica, Antigua, and Montserrat. In several of these locations it was possible to compare fruit from plants involved in a spray program with fruit from unsprayed plants, and no symptomological differences were observed. Those lines known to be susceptible were consistently afflicted with the diseases; whereas, those lines which were expected to be free from disease remained healthy or normal. Several experiments with methods of culture and spray programs were unsuccessful in inducing symptoms in normal lines or preventing symptoms from occurring in susceptible lines. The general conclusion from this study is that symptom expression is evidently little influenced by environment. Environmental conditions probably do have an effect on the amount of secondary infection which occurs through ruptured pox lesions, and are in this sense important. The disease is particularly evident, and of significant economic importance, when infection by fruit-rotting secondary invading pathogens occurs.

Genetic studies.—Eleven plants were selected in the F_2 generation of the first genetic study and evaluated in two succeeding generations for pox and fleck. Because of the limited space available for such studies, plants were chosen at random for further evaluation in the next generation. Results indicated both fruit pox and gold fleck were inherited with both conditions being transmitted to the progeny through four generations.

Results of the second genetic study were much more conclusive. Gold fleck appeared to be controlled by a dominant gene and fruit pox by a recessive gene with dominance being complete (Table 1). The genetic system envisioned is one in which the presence or absence of gold

TABLE 1. F_2 segregation patterns for normal:pox (3:1) and fleck:normal (3:1) obtained from the 'Floradel' × 'Pox and Fleck Floradel' cross and its reciprocal

Class	'Floradel' × 'Pox & Fleck Floradel'			'Pox & Fleck Floradel' × 'Floradel'		
	Observed	Expected	*P*	Observed	Expected	*P*
Normal	65	70		54	60	
Pox	28	23	.05-.30	26	20	.05-.30
Fleck	73	70		69	60	
Normal	20	23	.30-.50	11	20	.01-.05

fleck or fruit pox is controlled by single genes. The number of fleck or pox lesions which actually occur on fruit possessing such single genes is in turn controlled by some other gene or genes. This hypothesis is based on the fact that the number of lesions of either pox or fleck is more or less uniform on all the fruit from a single plant. The minimum number of gold fleck lesions varies from 25-50 while the maximum number approaches 2,500-3,000 per fruit. The range for fruit pox lesions varies from a minimum of 10-15 to a maximum of 500-600 per fruit. When any of the fruit from a single plant are afflicted with either fruit pox or gold fleck, all the other fruit from that single plant will have a similar number of lesions. The symbols *Gdf* and *fp* have been suggested by C. M. Rick and C. D. Clayberg of the Coordinating Committee of the Tomato Genetics Cooperative to designate, respectively, the genes controlling the syndromes described herein as gold fleck and fruit pox.

The application of the genetic facts uncovered by this study has resulted in the nearly complete eradication of fruit pox and gold fleck from the University of Florida tomato-breeding program. The results of this study make it possible for the plant breeder to eliminate fruit pox and gold fleck in segregating populations within two generations if progeny tests are conducted. The release of Florida MH-1 as a cultivar free from fruit pox and gold fleck (3) was based directly upon application of the results of this study.

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