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Abnormal Ripening of Tomato Fruit

Field- and glasshouse-grown tomato (*Lycopersicon esculentum* Mill.) plants are frequently subject to a number of environmental stresses and microorganisms. These stresses may result in the production of various types of fruit abnormalities. With few exceptions, these fruit disorders have not been consistently reproduced experimentally. In addition, common names based on symptomatology were assigned to these abnormalities in an attempt to identify and classify them. The synonymy resulting from this practice resulted in confusion and controversy. Three terms—blotchy ripening, graywall, and internal browning—are in general use in the United States to describe the most commonly encountered fruit disorders of disputed etiology.

The purpose of this paper, in part, is to show that blotchy ripening may be a pathogen-incited complex that may result from either tobacco mosaic virus (TMV) infections or bacterial infections, alone or in combinations. An explanation for the frequent bacterial infections concomitant with the TMV-incited internal browning–graywall syndrome is also given. However, to induce the internal browning–graywall syndrome, healthy plants must be inoculated with tomato strains of TMV after full fruit set. The translocation of the virus into developing fruit results in a hypersensitive response and cell collapse. "The expression of internal browning is therefore interpreted as a shock reaction, coming as a direct result of late TMV infection, with virus accumulating in certain of the fruit where there is a hypersensitive response on the part of the host" (5,11). In addition, it is suggested that other disorders of unknown or uncertain etiology that have been reported in the trade magazines and refereed journals—e.g., cloud, vascular browning, streak, and tomato fruit bronzing—are due to TMV or possibly other virus infections after fruit set. That this may be true is suggested by a brief report in 1950 by Diachun and Valleau (23) on these viruses (TMV, potato virus X, and cucumber mosaic virus) recovered from green-wrap tomatoes shipped from California to Kentucky, which ripened abnormally. However, no attempts were made by these workers to use the virus isolates to reproduce the mottled fruit symptoms.

Pertaining to time of virus infection and disease symptomatology, Ford (25) correlated pea pod streak with time of virus infection. A brief consideration of literature pertinent to the blotchy ripening, graywall, and internal browning disorders follows.

Blotchy ripening, which derives its name from a nonuniform pigmentation of a mature tomato fruit, has long been a serious and unexplained problem in the glasshouse-grown tomato crops of the British Isles and Europe, and has been much studied and discussed by workers of those countries (4,18,19,29,30,35,36, 54–56). The practice of picking tomatoes at the pink or early red stage of maturity focused attention on blotchy ripening as an economically important production problem in that geographic area. In the United States, where a relatively smaller volume of fresh tomatoes for market is greenhouse-grown than is field-grown, blotchy ripening has been of less concern. During the off-season winter months, much of the fruit for the eastern seaboard fresh market is produced in south Florida, with some shipments also from Puerto Rico and Mexico. During the summer months, some southern-based producer-distributors produce fruit in northern states for shipment to southern states. Field-grown tomatoes for fresh market usually are picked mature green and taken in bulk to packing facilities where they are washed, polished, graded, often individually paper wrapped, packed, and shipped. Ripening of these green-wrap tomatoes is induced by exposure of the fruit to ethylene gas.

Graywall, a Production Problem for the Fresh Market

The report of vascular browning in Dade County, Florida, by Conover (17) in 1949 was the first to indicate a new problem in the production of fresh market green-wrap tomatoes. Conover noted that while the disorder, locally known as graywall, had been seen annually in the area since 1937, losses during the winter growing season of 1948–49 were serious. He estimated 10–20% of the fruit maturing in late January and February were affected, with losses in some fields ranging as high as 70%. Conover observed that "while vascular browning might appear at any picking, it was most common the third picking this season and that in many cases, plants which bore affected fruit subsequently set fruit that matured normally without any vascular browning." Although Conover had no explanation for this observation, it appears logical that pickers, looking for and harvesting first-ripening fruit, were also inadvertently inoculating healthy plants with TMV at the most opportune time for graywall development (5,9,11,45,51). Tomato plants are extremely susceptible to TMV, and the virus is readily moved from infected to healthy plants by pickers. Early observations were also made that affected fruit were commonly found in the rows traversed by the sprayers. This led to speculation that soil compaction might be a factor in the syndrome. It is now apparent that TMV was moved from diseased to healthy plants as the sprayers moved along the rows and brushed against plants (20). That Conover was also describing the shock reaction of TMV infection, which follows when plants with maturing fruit are inoculated (5,11), is revealed in the observation that "plants which bore affected fruit subsequently set fruit that matured normally without any vascular browning." This is also the sequence followed when internal browning is experimentally reproduced. In all experiments in which internal browning–graywall was reproduced by TMV inoculation (11), a small percentage (usually less than 25%) of fruit on each plant was affected within 3 weeks. Those fruit not affected, and fruit formed after the initial shock reaction, developed normally (11). While Conover's report was primarily of crop loss due to a ripening disorder, he concluded, "The only consistency in the whole picture has been that vascular browning is usually more severe when vines are luxuriant and the fruit heavily shaded. At present no explanation for

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the cause of this malady is available. Repeated microscopic examination and attempts at isolation have uniformly failed to reveal the presence of an organism. This combined with the failure of the fruit to break down after picking, indicates the cause is either physiological or virus in nature. Field observations do not consistently support either hypothesis."

The importance of the report by Conover became increasingly apparent in the decades that followed. While Conover named the disorder vascular browning, growers and buyers referred to it as graywall. The following detailed description by Conover seems to suggest graywall as a more appropriate common name for the disorder: "Vascular browning appears externally on the fruit as a grayish-brown discoloration seen through the somewhat translucent outer wall of the tomato. The margin is usually indistinct and the discolored areas vary from nearly circular to longitudinal sectors in that portion of the fruit wall covering the locules. This discoloration is more noticeable on the sides of the fruit, but has been seen extending from the stem scar to the blossom end. In milder cases, only one or two such areas may be seen in one fruit, in which case the surface of the fruit is smooth. In more severely affected fruit, the entire tomato may be discolored and the skin somewhat corrugated but not broken. In cross section the affected area is dark-reddish brown and is centered in and around the vascular bundles of the fruit wall." In apparent deference to this trade preference, the 1950 report of the disorder by Stoner and Hogan (50) was titled, "A Report of Graywall or Internal Browning of Tomato in South Florida." This report was based on field observations and an experiment involving six plants growing in a field "heavily affected with the graywall condition." Stoner and Hogan are vague about the tissue used to provide inoculum for their attempts to isolate TMV, stating only that "All inoculation material used in the investigation was taken from these six plants, and each time material was collected one or more of the plants were bearing graywall fruit." Using this inoculum, attempts to mechanically transmit a virus by juice-Carborundum rubbing of three plants each of tomato, Turkish tobacco, *Nicotiana glutinosa,* squash, and cucumber from each source plant on three separate occasions—30 January, 28 February, and 9 March 1950—were without success. Thirty days after inoculation, all plants were discarded except the tomatoes, which were held to see if fruit symptoms would develop. Of the 270 young, vigorously growing plants inoculated, the 54 saved tomato plants produced a total of 239 fruit, which were all harvested when fully red ripe, cut in cross section for examination, and found free of internal browning. Stoner and Hogan further reported that the six source plants were pruned, transplanted to 25-cm (10-in) pots, and maintained in the greenhouse for further observation and fruit production. "While these plants did not grow well, 15 fruit were harvested without symptoms and 59 normal fruit were produced on plants grown from rooted cuttings propagated from the source plants." At the time Stoner and Hogan made their observations, the shock reaction of TMV infection, which occurs only in fruit already set at the time of infection, was not recognized as a necessary requirement for disease development. Therefore, much credence was given to the fact that all fruit produced on the young, inoculated tomato plants, and all plants and cuttings from plants which had previously produced internally browned fruit, were free of symptoms, thus indicating freedom from a causal virus. While one cannot explain why TMV was not mechanically transmitted from the six tomato source plants to Turkish tobacco, it is possible that the Turkish tobacco cultivar used was TMV resistant, showed mild systemic symptoms overlooked in the 30 days of observation, or (most likely) the tissue used as inoculum was low in or free of virus. We must assume that TMV was absent or in extremely low concentration, since no local lesion response was elicited in *N. glutinosa.* Squash and cucumber are not hosts of TMV. Stoner and Hogan also stressed the fact that "no association of graywall in Florida could be made with *Plantago* species, no definite pattern of spread was observed, and no consistently recognized foliage symptoms were seen in plants bearing graywall fruit, findings which F. 0. Holmes (33,34) associated with internal browning in New Jersey."

This report has been widely cited to disclaim the role of TMV in the graywall–internal browning syndrome and is the basis for seeking other explanations for the etiology of graywall in Florida (8,21,46). Field and greenhouse observations by Dennison and Hall (22) of conditions under which vascular browning (graywall) of tomatoes occurs did not help determine the etiology of the disorder, nor did a subsequent report, "Environmental Factors Influencing Vascular Browning of Tomato Fruits," by Hall and Dennison (27).

In 1964, Beraha and Smith (1), working at the Agricultural Marketing Service, Chicago, Illinois, reported "A dry, firm decay of mature-green tomatoes caused by a bacterium," which they isolated and described from affected fruit in a shipment from Texas. They then used the bacterial isolate to reproduce the decay symptoms. They also note that "an examination of tomatoes from which the epidermal tissues had been removed showed brown to dark brown discolorations in the pericarp region of the fruit, usually immediately beneath the sunken, affected areas. At first this disorder was suspected to be caused by the tobacco mosaic virus which has been described as the cause of the internal brown wall disease (Ref. Boyle and Wharton, 1957)." In pathogenicity studies, Beraha and Smith reported, "Tomatoes were inoculated with each of the isolates in at least six different places around the stem scar and the shoulder of the fruit. Controls consisted of fruit inoculated with sterile water. Inoculated fruit were incubated at 25° C for 5 days. At the end of that period they showed characteristic, sunken, dry areas with internal brown discoloration of the pericarp. Infections were obtained from 8 of the 21 selected isolates." Beraha and Smith further noted that "Bacteria appeared to spread very slowly through the parenchyma tissue of the pericarp." They also reported a persistence of green in unripened fruit and a bleaching of the red pigment in ripe affected fruit, resulting in fruit that would be blotchy when ripened. Apparently influenced by this report, Hall and Stall (28) in 1967 reported the production of graywall-like symptoms in tomato fruit by bacteria, and Stall and Hall (48) published in *Phytopathology* "Graywall Symptom Development in Tomato Fruit After Injection of Extracts of Graywall-Affected Tissue." A subsequent and apparent last report by Stall and Hall (49) followed in 1969, entitled "Association of Bacteria with Graywall of Tomato."

A conference on graywall of tomatoes organized by C. B. Hall was held in early spring 1968 at Homestead, Florida. I was invited and attended. Conference members were shown valid demonstrations in which bacteria injected into green tomato fruit induced a restricted area of discoloration and browning of the pericarp. The typical syndrome of graywall (internal browning) was not reproduced in these demonstrations, nor has it ever been reproduced by bacteria alone. On field trips in connection with the conference, graywall-affected fruit were observed in production fields and in packing sheds. During these trips, I collected from a packing house 10 fruit I considered to be typical internally browned fruit, and which were considered typical graywall by the Florida workers. TMV was isolated from all fruit, and bacteria were isolated from five of the fruit. The TMV isolates were used in experiments to reproduce graywall–internal browning, and some of the bacterial isolates were injected into fruit and induced wall browning. These observations and the use of the Florida TMV isolates to reproduce the typical shock reaction of internal browning in inoculated tomato plants with full fruit set led me to conclude and report that internal browning and Florida graywall

were the same (6). Additional experiments with Florida TMV isolates obtained from graywall-affected fruit substantiated the role of TMV in the etiology of the Florida graywall syndrome, and scanning electron microscope observations revealed microscopic rifts in the cuticular and epidermal surfaces above the affected pericarp wall tissue (7). These rifts (Fig. 1) provide openings for the entrance of bacteria and account for the frequent association of bacteria in graywall–internal browned fruit. At the time, I considered the bacteria present to be secondary invaders and of no importance in the syndrome, except as possibly accentuating the browning of affected necrotic tissue. Their role as a factor in blotchy ripening was overlooked. Surprisingly, fruit which were injected with suspensions of bacteria isolated from internally browned fruit, in addition to developing brown areas in the pericarp wall beneath the areas infused with bacteria, also became blotchy ripened (Fig. 2).

Results of an experiment to discern the role of TMV as an incitant of graywall–internal browning were published by Stall et al (47) in an article entitled "Effect of Tobacco Mosaic Virus and Bacterial Infections on Occurrence of Graywall of Tomato." In the introduction to this report, Stall et al state "Graywall is one of the most economically important and perplexing diseases of tomato, *Lycopersicon esculentum* Mill. The term 'graywall' is considered to be synonymous with 'blotchy ripening,' 'cloud,' and possibly 'internal browning.'" While the materials and methods used appear to have been designed to fairly determine the role of TMV in the graywall syndrome, two important factors apparently were not considered in these experiments: strain of TMV and time of inoculation. Tomato strains of TMV are the most effective in inducing graywall, not the Ohio strain V used by Stall et al; and plants must be kept free of virus and inoculated after full fruit set, not when the second cluster of flowers appears as was done in this experiment. In retrospect, it appears that Stall et al were evaluating blotchy ripening, not the shock reaction of TMV infection that results in the graywall–internal browning syndrome. They did not distinguish these two disorders, and in their introduction they stated "the term graywall is considered synonymous with blotchy ripening." Retrospective analysis of their experiment confirms the accuracy of their conclusion that "Studies of graywall have been handicapped by the inconsistency of disease development."

Internal Browning, a Problem for Processed Tomato Packers

Internal browning was the name Haenseler (26) gave a malady first observed in field-ripened tomatoes grown for processing in New Jersey. When the disorder was first noticed, Haenseler reported, "approximately 25 percent of the fully developed fruits at the time of observation were so badly damaged that they were unfit for market." While Haenseler conceded the new disease "to be very similar and probably identical with that recently described by Robert A. Conover as Vascular Browning," he nonetheless chose to apply the name internal browning. Like Conover (17), Haenseler could not explain the etiology of the problem but reported that studies

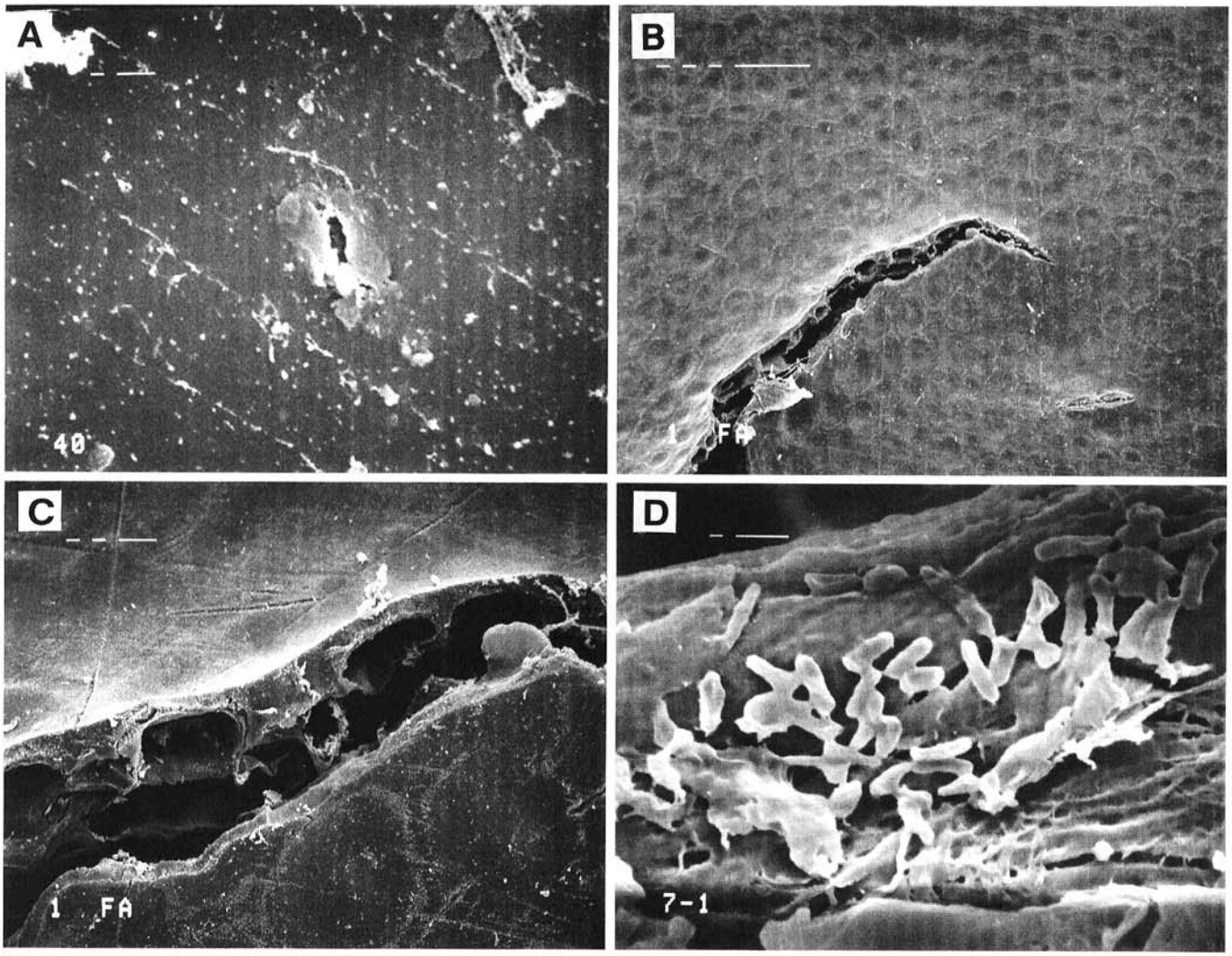


Fig. 1. Scanning electron micrographs of rifts in the epidermis above the necrotic areas of affected tomato fruit following the TMV shock response. These rifts provide points of entry for bacteria: (A) 7,200×, (B) 140×, (C) 720×, and (D) 10,000×.

to correlate the disorder with nutrient levels, minor element deficiencies, or toxic materials in the soil were inconclusive, and the possible role of virus was being investigated in cooperation with F. O. Holmes. Holmes, working at the Rockefeller Institute of Medical Research, New York, was considered the authority on studies involving TMV (32). Even so, the reports shortly thereafter by Holmes (33,34), that an unusually severe strain of tobacco mosaic virus was found in affected fruits, were considered inconclusive by most workers because TMV was ubiquitous in tomato fields, and Holmes was unable to use his virus isolate to reproduce the disease. Subsequently researchers, reasoning that environmental factors must be involved in symptom expression, conducted several greenhouse and field experiments to test factors thought to predispose fruit to disease development, including submerging the developing fruit in water (41,42). Working in the same laboratories with Holmes and using TMV to inoculate young tomato plants, Raychaudhuri (41) concludes from his experiment that, while the results reported are only preliminary observations, "they indicate, never-the-less, that high moisture content of the air which results possibly in water congestion of the tissue of the tomato fruit may be an important factor in the internal breakdown of the tomato tissue. The role of the virus in this disease remains uncertain." Rich (42) reports that "the results, although negative, are of value to show that the various combinations of fertilization, fruit shading and water-saturation used here will not produce symptoms of internal browning in the field."

In 1956, I reported on the nature of the internal browning disease (5); and in 1957, Boyle and Wharton (11) detailed the experimental reproduction of tomato internal browning by inoculation with strains of TMV. Subsequent papers by Smith et al (45), Boyle and Bergman (9), and Taylor et al (51) confirmed the role of TMV as the incitant of internal browning when plants were inoculated at the proper stage of maturity, i.e., with full fruit set. Boyle and Bergman (9) also noted that virus isolates, soil moisture, and cultivar susceptibility were factors that affected the incidence and severity of the disease. Studies by Wharton (52) and Bergman and Boyle (2,3) also showed significant changes in the mineral metabolism of tomato when infected with strains of TMV.

The Role of TMV in Blotchy Ripening

Beginning in 1961 with a review of the literature, Broadbent (12) singly and cooperatively published a series of papers (13–16) on the epidemiology of tomato mosaic in glasshouse tomato production. The comprehensive review of the literature by Broadbent (15) in 1976 detailed the epidemiology and control of tomato mosaic virus, e.g., strains of TMV found in association with infections of *L. esculentum* in the glasshouses of Great Britain. While Broadbent recognized that TMV infections of tomato plants may result in fruit abnormalities, he did not stress the conditions under which these abnormalities occurred. A conclusion he drew suggests that he did not understand the shock reaction proposed by Boyle and Wharton (11) to explain the internal browning syndrome, for in his 1976 review (15) he stated, "Hygiene measures necessary to prevent TMV introduction and spread are too uncertain and involve complications which make them commercially unacceptable. So the development of a mild-strain inoculation technique that minimizes fruit losses and protects the plants against invasion by more damaging strains has been of great importance." Rather than protect plants from infection, the inoculation of young plants before fruit set precludes the shock reaction that follows infection after fruit set. This is not a case of cross-protection, however, for infections with other strains may occur, although less frequently, and the main damage from chance infection at a critical time of fruit development is averted (personal observations).

The possible role of viruses in blotchy ripening was tentatively considered by Boyle and Bergman (10), who reported that when field- and greenhouse-grown tomato plants were inoculated with mild strains of TMV before fruit set, the fruit later produced were essentially free from blotchy ripening. The association of internal browning and blotchy ripening was noted earlier by Boyle and Bergman (9), who reported that internally brown fruit, when allowed to ripen, would be blotchy ripened.

The practice of inoculating young tomato plants with a mild strain of TMV to protect against later infections by more severe strains, and the adverse effects of these strains on fruit quality, are detailed by Rast (37–40). By 1975, the practice of cross-protection was widely accepted by growers in the British Isles and Europe, with the Rast strain MII-16 being used to inoculate the tomato seedlings (personal observations while on Sabbatic leave in Ireland).

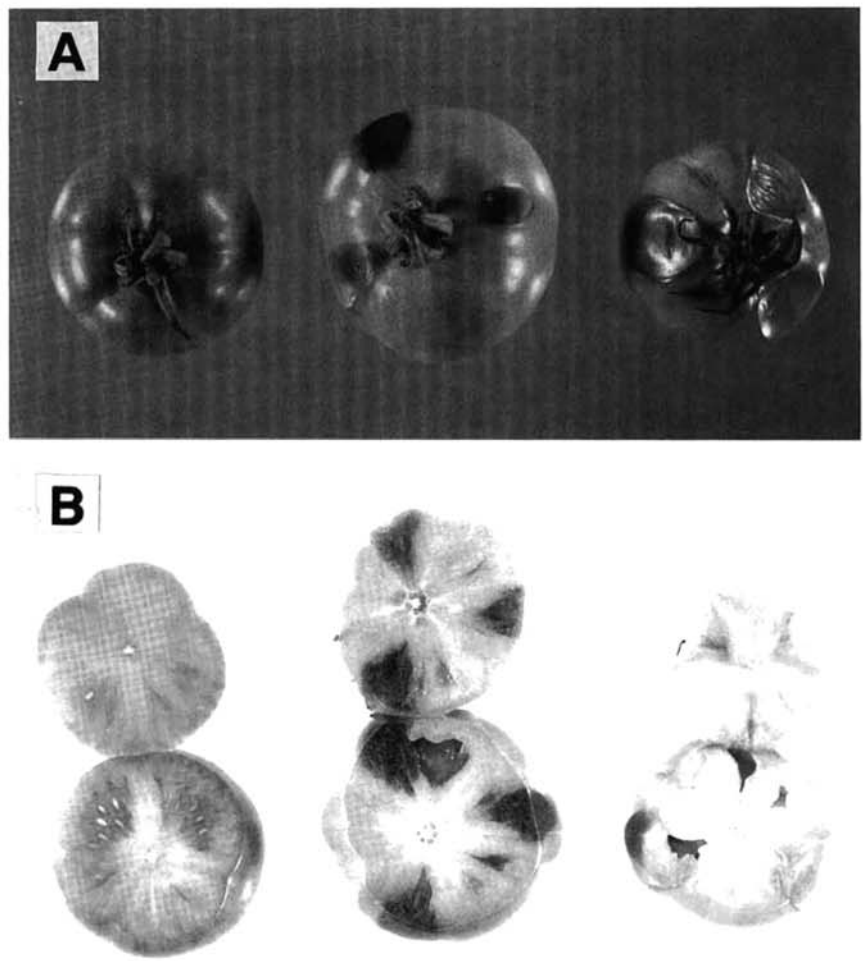


Fig. 2. Green tomato fruit hypodermically infused with bacteria isolated from internally browned fruit. (A) Whole fruit, and (B) fruit cut to show discoloration of tissue restricted to sites of infusion.

The description of tomato mosaic virus by Hollings and Huttinga (31) summarizes a great amount of literature dealing with diseases, host range, symptomatology, strains, morphology, structure, and chemical composition relevant to this virus. While not recognizing internal browning as a "uniquely different disease," Hollings and Huttinga note that disease symptoms are greatly influenced by temperature, day length, light intensity, age of plant, virus strain, and cultivar, and broadly group these symptoms into 1) green leaf mottle, distortion, and stunt; 2) yellow or "aucuba" mosaic leaf mottling; and 3) necrosis of stems, petioles, leaves, and/or fruit. While they recognized that pathogenesis is a complex phenomenon depending on environmental conditions and genetic variability of both pathogen and host, the response of the host at different stages of growth and development was not emphasized, especially as it affects fruit quality. Rather, it appears that they considered the symptoms observed in developing fruit to be dependent only on strain differences.

Blotchy Ripening as a Production Problem

Comprehensive literature appraisals of blotchy ripening and allied disorders of tomato were made by Cooper (18,19), and Hobson and Davies (29). The term blotchy ripening, first used in 1921, was considered by all workers to be a physiological disorder induced by nutritional imbalances or, more recently, environmental factors that affect the metabolism of developing fruit. The term blotchy ripened was also used extensively to identify fruit that are not uniformly red when mature ripe. In attempts to be more definitive, various workers noted hard green areas, discolored or necrotic vascular bundles, or clear glassy areas in association with blotchy ripened fruit. Hobson and Davies (29) state, "Little progress has been made in the last two decades towards an adequate definition of blotch, or even a convincing explanation of the cause of the disorder." In a later publication, Hobson et al (30) define blotchy ripening as follows: "Blotchy ripening occurs most frequently on the lower trusses (Cooper, 1960) and is characterized by green, greenish-yellow or waxy areas on apparently normal red fruit. Occasionally, the whole fruit is affected and never turns red at all, except perhaps at the blossom end. The surface of the fruit does not show any indentations or pits, but the discolored area remains firmer than the surrounding tissue which, on turning color, softens normally. Usually, there is no very sharp line of demarcation between the red and green areas, the colors merging gradually over a distance of 2 mm or so. More often than not, the region showing abnormal pigmentation is adjacent to the calyx area (Winsor, 1960; Sadik & Minges, 1966). The course of the vascular bundles can often be traced within the outer walls as a network of brownish-grey tissue, especially when the normal pigments have largely disappeared. The disorder is apparent only in mature fruit (Seaton, 1933; Seaton & Gray, 1936)." With reference to blotchy ripening, Hobson and Davies (29) also noted that "the basic cause is certainly physiological and does not involve tobacco mosaic virus (TMV) infection (Boyle and Bergman, 1967)." According to this statement, it seems Hobson and Davies consider internal browning different from blotchy ripening, at least as they recognize it. The blotchy ripening that follows when internally browned-graywall affected fruit are allowed to ripen is certainly different from the blotchy ripening that results from early plant infections with some strains of TMV, or that might result from bacterial infections alone or in combination with viral infections. The crux of the blotchy ripening dilemma is found only when the role played by TMV is fully understood. Experimental attempts to explain the etiology of blotchy ripening have been numerous but have never monitored plants or fruit tissue for virus

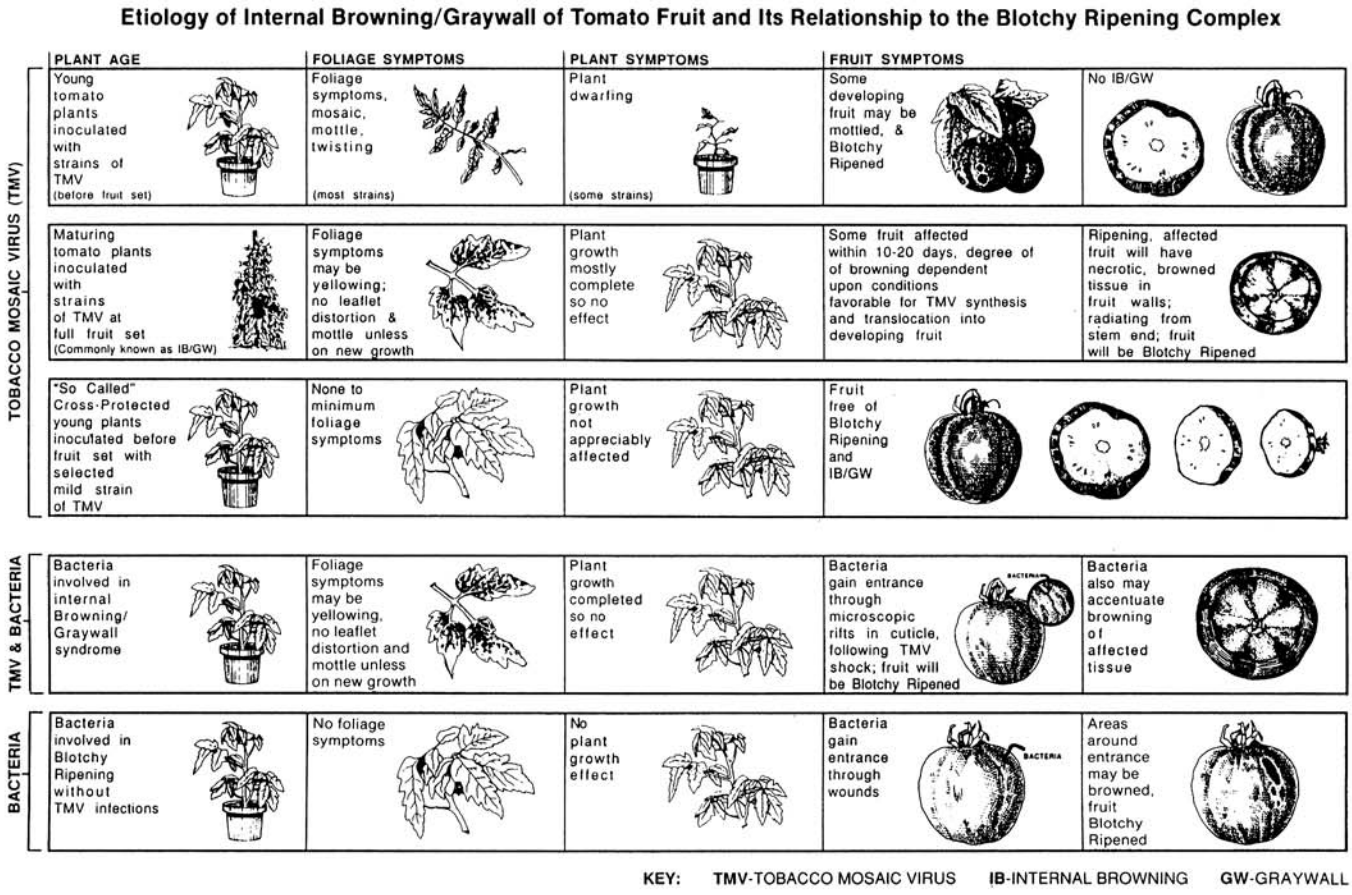


Fig. 3. Pictorial summary of tomato fruit and foliage symptoms associated with TMV and bacterial infections at different stages of plant growth.

infections or have done so without regard to plant development at the time infection occurred.

Detailed histological studies of affected tissues of blotchy ripened fruit were made by Sadik and Minges (43) and Wharton and Boyle (53), who studied the association between internal and external symptoms of fruit discoloration. Actually, Sadik and Minges were studying the pathological histology of internally browned fruit. That this is true is obvious from statements made in the introduction of the report, such as, "The disorder has been referred to by many terms such as 'blotchy ripening', 'graywall', 'vascular browning', 'cloud', 'waxy patch', 'green patch', 'piebald', and 'internal browning'." They also call attention to the discolored areas in the pericarp wall and note that "the location of discoloration is not restricted to any particular place, but is most common on the stem-end portion of the fruit." Naturally affected fruit were taken from both greenhouse- and field-grown plants. This study purports to show an association with a ligninlike substance in abnormal fruits but concludes the mechanism by which lignification of cell walls is triggered is unknown. Sadik and Minges also note that "necrosis takes place first in parenchyma cells immediately surrounding vascular bundles thus giving rise to brown strands." Anatomical studies by Fogleman (24) indicated that the vascular bundles of blotchy tissue, as well as being surrounded by disorganized parenchyma, were generally enlarged, with prominent lateral branching especially toward the stem end of the fruit. Blotchy ripening was also reported to bring about characteristic changes in the composition of affected areas as well as in the fruit as a whole (29). Compared with evenly ripened tomatoes, blotchy fruit contain lower amounts of dry matter, soluble solids, sugars, and nitrogen-containing compounds. The titratable acidity of the expressed juices of blotchy fruit was shown to be lower than that of evenly ripened fruit (29). Changes in enzyme activities in tissue showing blotchy ripening have received some attention, and the browning in the vicinity of the vascular system appears to be due to polyphenoloxidase (29; personal observation). In summary, Hobson and Davies (29) stated, "There is now abundant evidence that the metabolism of blotchy fruit is markedly abnormal. Many of the chemical and physiological changes accompanying normal ripening do not take place. Although adequate potassium nutrition ameliorates the condition, blotch cannot be dismissed merely as a symptom of potassium deficiency. The changing balance in protein constituents that accompanies the final phase in fruit development appears to be altered in a random manner in the regions of fruit affected by blotchy ripening. These haphazard disparities from normal behavior suggest either a physiological or even a physical isolation of blotchy tissue by loss of normal conducting links with the parent plant. Further investigation on blotchy ripening may define in more detail the underlying cause of the condition but we suspect that a certain proportion of fruit showing the disorder will always occur." Detailed histological and physiological

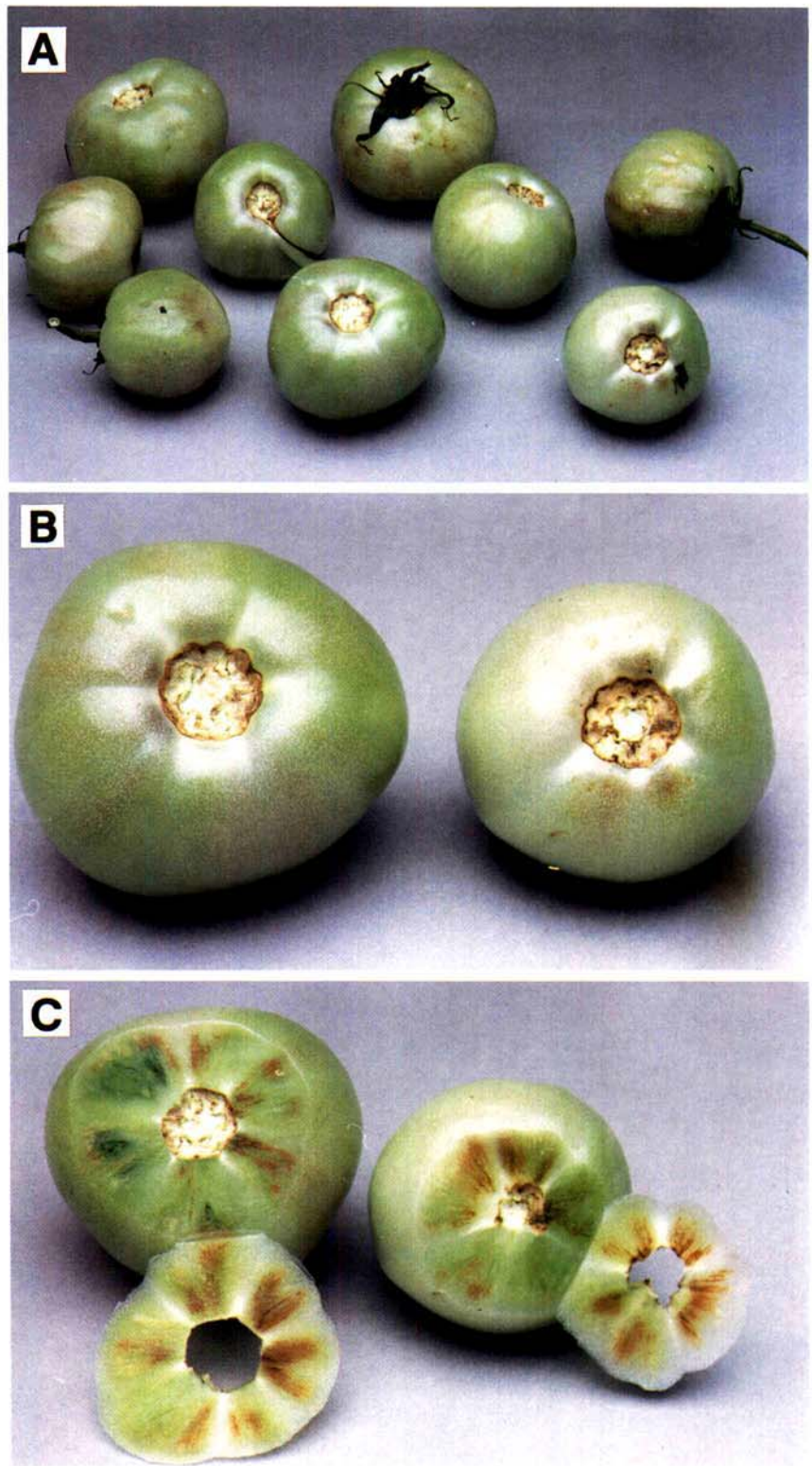


Fig. 4. (A) A group of graywall-affected tomato fruit received in the winter of 1993 from a grower in Florida. The fruit show varying degrees of browning. (B) Two affected fruit selected from (A) to show faintly browned tissue at the stem end. (C) Fruit in (B) cut to show internal brownish tissue radiating from the stem end where tobacco mosaic virus (TMV) was translocated into the developing fruit, and the resulting necrotic (shock) reaction. TMV was recovered from these fruit.

studies of TMV-induced internally browned tissue are similar in every respect with regard to blotchy ripened tissue (2,3,52,53). Infections by TMV were not monitored in any of these studies on blotchy ripening.

It is generally true that before a disease can be intelligently controlled, the causal organism or agent must be identified. It appears that strains of TMV, other viruses to which tomato is susceptible, bacteria alone, or virus-bacteria combinations, are responsible for or involved in most of the various forms of disorders known as blotch or blotchy ripening.

Summary

An understanding of the blotchy ripening complex and the pivotal roles played by TMV and bacteria (Fig. 3) has been slow in evolving because of many factors, primarily the following:

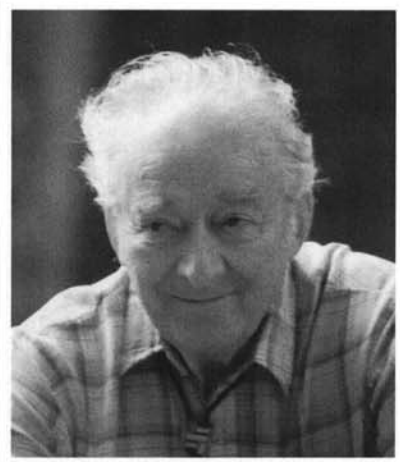
1. In either field or greenhouse environments, affected fruit may be found on large, healthy-appearing plants without typical TMV mosaic symptoms. Affected fruit are also free of signs indicating the presence of bacteria.
2. Tomato plants at all stages of growth are extremely susceptible to TMV, which is ubiquitous and persistent in fields and greenhouses where tomatoes are grown.
3. A wide range of symptoms is associated with infections of TMV strains and isolates, and environmental conditions.
4. Preventing TMV infection of healthy controls once the virus is introduced into an experiment is difficult.
5. Some researchers fail to recognize the need to inoculate virus-free plants at full fruit set and to carefully monitor the spread of TMV once introduced into an experiment.
6. Some strains of TMV, even when young plants are infected, will induce a blotchy ripening of the fruit as they set and develop. However, internal tissues are not affected.
7. Although different isolates or strains of TMV may influence the incidence or intensity of internal browning–graywall, the disorder is not a developmental disease. The fruit that will be affected are affected within 2–3 weeks after inoculation, and if allowed to ripen, will be blotchy with internal tissue discolored and corky.

Changes in the cultural practices of tomato production since the 1940s resulted in new problems. Internal browning became a problem in northern-grown processing tomato production when the source of field transplants shifted from local greenhouses and cold frames to field-grown, virus-free plants shipped north from southern states. Greenhouse-grown plants were usually TMV-infected before being set in the field. This preempted the shock reaction of later infections after fruit set. The development of direct seeding, determinate cultivars, and mechanical harvesting have resulted in the decline of TMV infestation, and spread in the field and internal browning–graywall is not a major problem for producers of tomatoes for processing. The incidence of graywall–internal browning as a production problem in Florida appears linked to increases in acreage and intensity of production, which may result in greater retention of TMV in plant debris and reservoirs. The production of tomato fruit for the fresh market requires a lot of hand labor and plant contact. As a consequence, blotchy ripening and graywall–internal browning still cause losses in the Florida green-wrap industry (20,21) (Fig. 4). Research has clearly showed that TMV infections can induce the graywall–internal browning syndrome (5,6,9,45,51), despite reports to the contrary (46,50).

While there have been no definite strategies developed for the control of blotchy ripening, it has been demonstrated that cultivars resistant to TMV or inoculated with mild strains before flowering will produce few, if any, abnormally ripened fruit. When TMV resistance or early plant inoculation does not solve the problem, other viral or bacterial causes should be investigated. Bacteria are frequently found in association with internally browned–graywall fruit, having gained entrance through the epidermis via microscopic rifts (Fig. 1) associated with the necrotic tissue resulting from the shock TMV reaction. These bacteria may accentuate the blotchy ripening caused by TMV or may even cause blotchy ripened fruit when alone. A paper by Samish and Etinger-Tulczynska (44) on distribution of bacteria within the tissue of healthy tomatoes suggests that this might be a fruitful area of research to explain some ripening disorders with unusual symptomatology.

Literature Cited

1. Beraha, L., and Smith, M. A. 1964. A bacterial necrosis of tomatoes. Plant Dis. Rep. 48:558-561.
2. Bergman, E. L., and Boyle, J. S. 1962. Effect of tobacco mosaic virus on the mineral content of tomato leaves. Phytopathology 52:956-957.
3. Bergman, E. L., and Boyle, J. S. 1966. Symptoms, yield and leaf mineral composition of greenhouse tomatoes affected by soil moisture and tobacco mosaic virus. Acta Hortic. Tech. Comm. I.S.H.S. 4:96-99.
4. Bewley, W. F., and White, H. L. 1926. Some nutritional disorders of the tomato. Ann. Appl. Biol. 13:323-338.
5. Boyle, J. S. 1956. The nature of the internal browning disease of tomato. (Abstr.) Phytopathology 46:7.
6. Boyle, J. S. 1970. Evidence that Pennsylvania tobacco mosaic virus-induced internal browning and Florida graywall are the same. (Abstr.) Phytopathology 60:571.
7. Boyle, J. S. 1982. The etiology of internal browning–graywall of tomato. (Abstr.) Phytopathology 72:258.
8. Boyle, J. S. 1992. Confusion over the etiology of graywall or internal browning of tomato. Plant Dis. 76:1084-1085.
9. Boyle, J. S., and Bergman, E. L. 1967. Factors affecting incidence and severity of internal browning of tomato induced by tobacco mosaic virus. Phytopathology 57:354-362.
10. Boyle, J. S., and Bergman, E. L. 1969. The prevention of blotchy ripening in tomato. (Abstr.) Phytopathology 57:397.



John S. Boyle

Dr. Boyle received a B.A. degree from Wichita State University in 1939 with a major in botany and minors in chemistry and zoology. He continued his education at the University of Iowa in mycology under the supervision of Professor G. W. Martin and was granted the M.S. degree in 1942 before enlisting in the United States Naval Reserve and serving in the Pacific Theater during World War II. Upon discharge in 1945 and a consultation with Professor Martin on opportunities available for the application of mycological knowledge, he was advised to pursue a career in plant pathology and was admitted to the phytopathological program at the University of Wisconsin. He worked as a research assistant in Fruit Disease Control under the direction of Drs. G. W. Keitt and J. D. Moore. Upon completion of requirements for the Ph.D. in 1949, Dr. Boyle joined the botany faculty of the then Pennsylvania State College as assistant professor of plant pathology, where he remains as professor emeritus. At Wisconsin, his research interests changed to the area of plant virus diseases, which resulted in pioneering work in the transmission of viruses from woody to herbaceous hosts. Dr. Boyle established the first course in plant virus diseases at Penn State, and his research focused on understanding and developing strategies for the control of virus diseases of fruit and vegetable crops.

11. Boyle, J. S., and Wharton, D. C. 1957. The experimental reproduction of tomato internal browning by inoculation with strains of tobacco mosaic virus. Phytopathology 47:199-207.
12. Broadbent, L. 1961. The epidemiology of tomato mosaic. 1. A review of the literature. Pages 96-116 in: Annu. Rep. Glasshouse Crops Res. Inst. 1960.
13. Broadbent, L. 1964. The epidemiology of tomato mosaic. 7. The effect of TMV on tomato fruit yield and quality under glass. Ann. Appl. Biol. 54:209-224.
14. Broadbent, L. 1965. The epidemiology of tomato mosaic. II. Seed transmission of TMV. Ann. Appl. Biol. 56:177-205.
15. Broadbent, L. 1976. Epidemiology and control of tomato mosaic virus. Annu. Rev. Plant Pathol. 14:75-96.
16. Broadbent, L., and Fletcher, I. T. 1966. The epidemiology of tomato mosaic. 12. Sources of TMV in commercial tomato crops under glass. Ann. Appl. Biol. 57:113-120.
17. Conover, R. A. 1949. Vascular browning in Dade County Florida greenwrap tomatoes. Plant Dis. Rep. 33:283-284.
18. Cooper, A. J. 1956. Blotchy ripening and allied disorders on the tomato: A critical review of the literature. Pages 39-49 in: Annu. Rep. Glasshouse Crops Res. Inst.
19. Cooper, A. J. 1958. The definition and classification of abnormalities of fruit pigmentation in the tomato variety 'Potentate'. Ann. Appl. Biol. 46:699-674.
20. Cox, R. S. 1970. Observations on the cause of graywall of tomato in Florida. Plant Dis. Rep. 54:678.
21. Dangler, J. M., and Locascio, S. J. 1990. External and internal blotchy ripening and fruit elemental content of trickle-irrigated tomatoes as affected by N and K application time. I. Am. Soc. Hortic. Sci. 115(4):547-549.
22. Dennison, R. A., and Hall, C. B. 1954. Field and greenhouse observations of conditions under which vascular browning (graywall) of tomatoes occurs. Proc. Fla. Hortic. Soc. 67:133.
23. Diachun, S., and Valleau, W. D. 1950. Viruses in green-wrap tomatoes shipped to Louisville, Kentucky from California. Plant Dis. Rep. 34:269.
24. Fogleman, M. E. 1966. A genetic and histological study of blotchy ripening in segregating generations of a tomato cross. Ph.D. thesis. Iowa State University of Science and Technology, Ames.
25. Ford, R. E. 1964. Pea pod streak symptoms correlated with time of virus infection. Plant Dis. Rep. 48:564-566.
26. Haenseler, C. M. 1949. Internal browning of tomatoes in New Jersey. Plant Dis. Rep. 33:336-337.
27. Hall, C. B., and Dennison, R. A. 1955. Environmental factors influencing vascular browning of tomato fruits. Proc. Am. Soc. Hortic. Sci. 65:353-356.
28. Hall, C. B., and Stall, R. E. 1967. Graywall-like symptoms produced in tomato fruit by bacteria. Proc. Am. Soc. Hortic. Sci. 91:573-578.
29. Hobson, G. E., and Davies, J. N. 1977. A review of blotchy ripening and allied disorders of the tomato, 1957-1976. Pages 139-147 in: Annu. Rep. Glasshouse Crops Res. Inst., Little Hampton, England.
30. Hobson, G. E., Davies, J. N., and Winsor, G. W. 1977. Ripening disorders of tomato fruit. Growers' Bull. No. 4. Glasshouse Crops Res. Inst., Little Hampton, England.
31. Hollings, M., and Huttinga, H. 1976. Tomato mosaic virus. No. 156 in: Descriptions of Plant Viruses. Commonw. Mycol. Inst./Assoc. Appl. Biol., Kew, England.
32. Holmes, F. O. 1929. Local lesions in tobacco mosaic. Bot. Gaz. 87:39-55.
33. Holmes, F. O. 1949. Association of strains of tobacco-mosaic virus and internal browning of tomatoes. Plant Dis. Rep. 33:338-339.
34. Holmes, F. O. 1950. Internal browning disease of tomato caused by strains of tobacco mosaic virus from *Plantago*. Phytopathology 40:487-492.
35. Jenkins, J. E. E., Wiggell, D., and Fletcher, J. T. 1965. Tomato fruit bronzing. Ann. Appl. Biol. 55:71-81.
36. Massey, D. M., and Winsor, G. W. 1956. "Blotchy ripening" of tomato fruit: A preliminary report on some chemical aspects of the disorder. Rep. Exp. Res. Stn. Cheshunt, 1954. 40:50-54.
37. Rast, A. T. B. 1967. Yield of glasshouse tomatoes as affected by strains of tobacco mosaic virus. Neth. J. Plant Pathol. 73:147-156.
38. Rast, A. T. B. 1972. MII-16, and artificial symptomless mutant of tobacco mosaic virus for seedling inoculation of tomato crops. Neth. J. Plant Pathol. 78:110-112.
39. Rast, A. T. B. 1973. Systemic infection of tomato plants with tobacco mosaic virus following inoculation of seedling roots. Neth. J. Plant Pathol. 79:5-8.
40. Rast, A. T. B. 1975. Variability of tobacco mosaic virus in relation to control of tomato mosaic in glasshouse tomato crops by resistance breeding and cross

protection. Neth. Agric. Res. Rep. No. 834. Wageningen, Netherlands.
41. Raychaudhuri, S. P. 1953. Reproduction of symptoms resembling internal browning under greenhouse conditions. Plant Dis. Rep. 37:220-221.
42. Rich, S. 1953. An attempt to induce internal browning of tomatoes in the field. Plant Dis. Rep. 37:615-617.
43. Sadik, S., and Minges, P. A. 1966. Symptoms and histology of tomato fruits affected by blotchy ripening. Proc. Am. Soc. Hortic. Sci. 86:532-543.
44. Samish, Z., and Etinger-Tulczynska, R. 1963. Distribution of bacteria within the tissue of healthy tomatoes. Appl. Microbiol. 11:7-10.
45. Smith, P. R., Stubbs, L. L., and Sutherland, J. L. 1965. Internal browning of tomato fruit induced by a late infection of tobacco mosaic virus. Aust. J. Exp. Agric. Anim. Husb. 5:75-79.
46. Stall, R. E. 1992. Confusion over the etiology of graywall or internal browning of tomato. Plant Dis. 76:1085.
47. Stall, R. E., Alexander, L. J., and Hall, C. B. 1969. Effect of tobacco mosaic virus and bacterial infections on occurrence of graywall of tomato. Proc. Fla. State Hortic. Soc. 81:157-161.
48. Stall, R. E., and Hall, C. B. 1967. Graywall symptom development in tomato fruit after injection of extracts of graywall-affected tissue. Phytopathology 57:1276-1277.
49. Stall, R. E., and Hall, C. B. 1969. Association of bacteria with graywall of tomato. Phytopathology 59:1650-1653.
50. Stoner, W. N., and Hogan, W. D. 1950. A report of graywall or internal browning of tomato in south Florida. Plant Dis. Rep. 34:379-390.
51. Taylor, G. A., Lewis, G. D., and Rubatzky, V. E. 1969. The influence of time of tobacco mosaic virus inoculation and stage of fruit maturity upon the incidence of tomato internal browning. Phytopathology 59:732-736.
52. Wharton , D. C. 1956. Tomato mineral metabolism and respiration as affected by two strains of tobacco mosaic virus. Ph.D. thesis. Pennsylvania State University, University Park.
53. Wharton, D. C., and Boyle, J. S. 1957. The pathological histology of the internal browning disease of the tomato. Phytopathology 47:208-212.
54. White, H. L. 1938. Further observations on the incidence of blotchy ripening of tomato. Ann. Appl. Biol. 25:544-557.
55. Winsor, G. W., and Massey, D. M. 1958. The composition of tomato fruit. I. The expressed sap of normal and blotchy tomatoes. J. Sci. Food Agric. 9:493-498.
56. Winsor, G. W., and Massey, D. M. 1959. The composition of tomato fruit. II. Sap expressed from fruit showing colorless areas in the walls. J. Sci. Food Agric. 10:304-307.