NECROSIS, HYPERPLASIA, AND ADHESIONS IN MOSAIC TOMATO FRUITS

By Max W. Gardner

Associate in Botany, Purdue University Agricultural Experiment Station

INTRODUCTION

The extremely severe streak or winter blight type of mosaic occurred in a greenhouse crop of Bonny Best tomatoes at La Fayette, Ind., during November and December, 1923, and an opportunity was afforded to make a preliminary study of the structural abnormalities exhibited by many of the diseased fruits. The disease was characterized by the destructive necrotic streaking and spotting of leaves, stems, and fruits described in a previous account (12, p. 8). Every one of the more than 800 plants in the house also evinced on the young leaflets the typical dark green puffy areas on a lighter background, a reliable diagnostic feature of the mosaic type of disease. The mosaic nature of the disease was further verified by successful inoculation of healthy tomato seedlings with the juice of a young fruit by J. B. Kendrick in another house and by numerous cultural tests which proved the internal fruit lesions to be free from bacteria.

Hundreds of fruits of all sizes and ages showing a variety of responses to the disease were available for study, and unstained free-hand sections of fresh material, mostly from very young green fruits, revealed most peculiar abnormalities in the tissues. The sections were mounted in water and preserved by adding lacto-phenol (equal parts of phenol, lactic acid, glycerin, and water). Material was also embedded in paraffin, sectioned on the microtome, and stained with gentian violet, safranin and licht green, Haidenhain's iron-alum haematoxylin, and Ziehl's carbol fuchsin.

NORMAL ANATOMY OF THE FRUIT

An acquaintance with certain phases of the normal anatomy and development of the fruit is a necessary preliminary to a consideration of the abnormal. The mature tomato with its several-celled ovary and fleshy walls and its bulky axile placenta bearing the seeds embedded in a pulpy matrix of thin-walled parenchymatous tissue is familiar to all. A very young ovary about 4 mm. in diameter as shown in Figure 1, A, differs mainly in that the ovules more or less completely fill the locular cavity and are not embedded in a cellular matrix. During the early stages of enlargement of the fruit the placental tissue grows out between the ovules (fig. 1, B) to form the gelatinous matrix of thin-walled parenchyma which separates and finally engulfs the ovules and completely fills the locular cavity (fig. 1, C and D) by the time the fruit is about 10 mm. in diameter.

The placental matrix touches the inner surface of the fruit wall or pericarp and the surfaces of the radial locular partitions, but remains free from these surfaces as well as from the epidermis of each ovule (fig. 1, B, C, and D). The young fruit enlarges very rapidly under greenhouse conditions and hence is composed of very active meristematic tissue. Figure 1 shows that growth must progress with extreme rapidity in the placental matrix in the short period during which the ovary enlarges to 1 cm. in diameter. Later the epidermal cells of the seed coat elongate enormously to form a palisade layer, which touches but remains free from the placental matrix.

GROSS ABNORMALITIES IN MOSAIC FRUITS

One of the most conspicuous symptoms on mosaic fruits is the eruption of brownish, translucent, rather flat-topped blisters of various shapes and sizes as illustrated in Plate 1, B, and in a previous paper (12, p. 8). These may later be bordered by a shallow peripheral fissure. Large lesions of this type may eventually contain numerous cuticular fissures and form...
Roughened buckskin areas on the fruit as described and illustrated by McKay (18, p. 181). Mosaic fruits are also characterized by more deep-seated lesions, producing an irregular pattern of hard, sunken, brown or black pock-marks, as shown in Plate 2, E and F.

Ripening fruits often show rather extensive sunken granular areas which fail to color properly and under which the seed pulp or placental matrix tends to remain greenish.

Ovaries and very young fruits may show brown epidermal or deep-seated areas on the fruit.

**EXPLANATORY LEGEND FOR PLATE 1**

A.—Section of pericarp through a surface blister such as is illustrated in B, showing the hyperplastic character of the blister tissue. The subepidermal cells have elongated and divided, pushing up the epidermis. If this tissue collapses the blister sinks and the epidermis usually cracks where it is sharply curved upward at the edge of the blister, thus forming the marginal fissure noted about the older lesions. Photomicrograph × 33. Unstained.

B.—Surface blisters on a mosaic tomato

C.—Section of a pericarp lesion resulting from necrosis and collapse of an area of subepidermal cells and hypertrophy of the cells immediately beneath the necrotic area. The necrotic surface of the underlying placental matrix, to which an ovule is adhering, is in turn adhering to the inner surface of the pericarp. Photomicrograph × 33. Stained with Haidenhain’s iron-alum haematoxylin. Fruit 12 mm. in diameter

D.—Section of pericarp showing at the right a transverse necrotic plane accompanied by elongation of the near-by cells. At the left is a small necrotic spot in the pericarp. Photomicrograph × 33. Stained with carbol fuchsin. Fruit 12 mm. in diameter

E.—Intumescences from a radial locule partition extending into an abnormal cavity which has resulted from the collapse of the necrotic placental tissue. Photomicrograph × 46. Unstained. Fruit 7 mm. in diameter
Necrosis, Hyperplasia, and Adhesions in Mosaic Tomato Fruits

Plate 1

(For explanatory legend see p. 872)
Necrosis, Hyperplasia, and Adhesions in Mosaic Tomato Fruits

Plate 2

A.—Four very young tomato fruits showing necrotic lesions. Two show rupture of the pericarp and would probably develop into such fruits as those shown in Plate 3, C.

B.—Longitudinal rupture of the pericarp, as a result of an earlier necrotic lesion, exposing a considerable area of the seed pulp or placental matrix.

C.—Scabby lesions stunting growth and causing deformity and pericarp rupture in young fruit.

D.—Transverse rupture of the pericarp as a result of an earlier necrotic lesion.

E.—Scattered, slightly sunken surface lesions or pockmarks.

F.—Coalescence of lesions producing a brownish pattern with a hard, sunken, granular surface. Such fruits fail to color properly.
markings without the blister effect (pl. 2, A) and in some cases the entire surface of the fruit is involved, a condition associated with early abscession or invasion by rot-producing organisms. The discoloration may sometimes be internal and deep seated so as to show through the outer pericarp layers as an evident darkening of the underlying tissues (pl. 3, B).

The surface lesions may become hard, dark brown scabs which inhibit the symmetrical growth of the fruit and as a result produce marked malformation, as is shown in Plate 2, C. Often the brown necrotic lesions involve the pericarp so deeply that, after the affected tissues collapse, the weakened areas are ruptured by the growth pressure of the interior tissues, and large irregular openings are produced in the pericarp exposing the seeds and placental matrix to the drying effects of the air (pl. 2, B and D). In fruits thus affected when very young, as shown in Plate 2, A, mere shreds of the pericarp may remain as is shown in Plate 3, C, or deep cracks may result as shown in Plate 3, D.

When the fruit is cut across it is found that brown necrotic regions usually occur throughout the interior tissues if there are any external lesions on the specimen (pl. 3, A) and in cases of extensive surface discoloration all or considerable portions of the interior may also be similarly discolored. Ordinarily, however, the internal necrosis occurs in the form of scattered strips, pockets, or thin layers, not continuous or connected with each other nor, as a rule, sufficiently extensive to completely inhibit the enlargement of the fruit, although its normal development may be seriously interfered with. The necrotic regions occur most abundantly in the placental matrix that fills the locules, and the most prevalent type consists of rather extensive necrotic planes parallel to and in rather close proximity to the locule walls. These necrotic regions are usually but not always surrounded or bounded by zones of rather firm, glassy or translucent tissue. In young ovaries many of the ovules are entirely necrotic and atrophied. This occurs usually in only part of the locules and often involves only part of the ovules in the locule. Many mature seeds show brown spots.

Examination of razor sections of the young fruits with a hand lens reveals very clearly the necrotic regions and the translucent zones associated with them and also shows rather extensive cavities associated with the necrosis of the distal portions of the placental matrix. These cavities may be entirely surrounded by a brown necrotic lining as is the small one in Plate 4, A, or may occur between the necrotic placental surface and the locule wall as in Plate 1, E. Furthermore many of the seeds may be displaced or abnormally located and oriented in the placenta, and the normal arrangement of the placental matrix as shown in Figure 1 is very generally interfered with to a greater or less extent. It is plainly evident also that necrosis often occurs within the seeds, that some ovules are affected more than others, and that various degrees of retardation in the development of the seeds and the placental matrix occur in the same fruit and even in the same locule. For example, certain locules in fruits 5 mm. and 7 mm. in diameter showed necrotic atrophy of the ovules and complete suppression of any development of the placental matrix beyond the stage shown in Figure 1, A.

Necrosis, Hyperplasia, and Adhesions in Tomatoes

Microscopic examination of unstained sections shows that the surface blisters are produced by a compact muriform cushion of meristematic cells which have pushed up the epidermis (pl. 1, A). Epidermal pustules or intumescences of all sizes are found, the smaller ones being papillate rather than flat-topped. In the incipient stage of a blister, necrosis of the epidermal cells is visible and the hyperplasia is seen to originate in the first and second subepidermal cell layers (pl. 5, A). The translucent quality of the blisters is very evidently attributable to the lack of intercellular spaces in this hyperplastic tissue (pl. 5, B).

Zones of brown necrotic cells of varying shape, extent, and orientation are of common occurrence at varying depths in the pericarp, producing, as a result of collapse of the necrotic cells (pl. 6, B and C), dark sunken pockmarks on the exterior of the fruit. Such zones often involve the entire thickness of the pericarp and, as a result of early atrophy of the tissues (pl. 6, A) or collapse of the necrotic cells (pl. 3, E; pl. 6, C), produce weakened places in the pericarp which are easily ruptured by growth pressure. This results in such fruits as are shown in Plate 2, B and D, and Plate 3, C and D. Similar necrotic regions occur scattered promiscuously through the locule walls and the placental tissue,
Necrosis, Hyperplasia, and Adhesions in Mosaic Tomato Fruits

Plate 3

(For explanatory legend see p. 877)
and atrophied or necrotic weakened places occur commonly in the locule walls. Necrotic regions are found within the endosperm of the ovule and occasionally in the embryo, and complete necrotoysis and atrophy of certain ovules is of common occurrence. The brown spots on the seeds previously mentioned are due to necrotic areas in the outer part of the endosperm just under the seed coat. The occurrence of these symptoms within the seed is of peculiar interest in view of the fact that the disease apparently is not seed-transmitted.

Necrotic tissue is most abundant in the peripheral regions of the placental matrix (pl. 4, A; pl. 6, C). The more extensive necrotic areas seem to originate very frequently in epithelial tissues such as the lining of the locule and the surfaces of the placental matrix. In almost all cases except within the endosperm of the seed the necrotic cells soon collapse (pl. 6, D) and the necrotic tissue frequently surrounds or borders upon cavities of varying extent resulting from the collapse and shrinkage of the cells (pl. 1, E; pl. 3, F).

The collapsed necrotic regions are practically always accompanied and bordered or more or less surrounded by zones of closely packed hypertrophied cells elongated toward the necrotic region or by zones of hyperplastic tissue composed of more or less parallel columns of somewhat rectangular, closely packed, meristematic cells (pl. 1, C and D; pl. 6, C and D). This tissue apparently arises from the living parenchyma cells bordering on the necrotic region and is characterized by reduced intercellular space, a condition which accounts in part for the translucent appearance of these zones (pl. 4, A). These cells by radial elongation and frequently by transverse division produce the columnar tissue which grows and pushes in toward the necrotic region (pl. 7, B).

Numerous instances of marked spherical hypertrophy of individual cells and radial hypertrophy of groups of adjacent cells in the epithelium lining the locular cavity have been noted opposite necrotic ovules in young ovaries. The latter condition is interpreted as the incipient stage of hyperplastic growth.

When the necrosis occurs in the interior tissues of the pericarp, locule wall, or placenta, the response of the surrounding tissue is largely hypertrophy alone, the cells enlarging and elongating in toward the necrotic region as the latter collapses and shrinks (pl. 1, C and D; pl. 6, C and D). Elongation of endosperm cells also occurs. On the other hand, when the necrosis occurs in the epithelial tissues, such as the epidermis, the epithelium lining the locules, and the epithelium of the placental matrix, the response of the underlying and adjacent cells, particularly those of the pericarp and locule wall, is elongation followed by transverse division with the resultant production of intumescences (pl. 1, E; pl. 3, F; pls. 4, 5, 7, and 8).

At the bases of the hyperplastic growths, or intumescences, hypertrophied cells are very conspicuous, while the cells in the distal portions or advancing faces of these hyperplastic growths, the part nearest the necrotic tissue, are much smaller than normal cells (pl. 5, B; pl. 7, A and B; pl. 8, A). In the larger growths, or intumescences, the parallel nature of the cell columns may become deranged by the pressure from basal groups showing renewed or more rapid growth, possibly in response to secondary necrotic areas within the intumescence (pl. 7, B). In the basal region of hyperplastic zones originating in the placental matrix, the cell contents are dense and brownish (pl. 4, A and B).

The most extensive development of these hyperplastic zones is on the inner surface of the pericarp and on the locule walls, particularly at the outer angles of the locule, in response, it would appear, to peripheral necrosis of the adjacent placental matrix or to necrotic

EXPLANATORY LEGEND FOR PLATE 3

A.—Two young fruits cut across to show internal necrotic regions and translucent zones about the lining of the locules and in the placenta and pericarp.

B.—Badly affected young fruit showing rupture of pericarp and deep-seated internal brown discoloration along the sutures. Such fruits contain an abundance of internal necrosis and hyperplasia.

C.—Two ruptured placentae with remnants of pericarp as a result of necrotic or atrophic lesions in the young ovary wall, such as are shown in Plate 2, A.

D.—Deep cracking of a fruit that was affected with necrotic lesions when very young.

E.—Section of a pericarp lesion showing the collapse of a subepidermal necrotic region involving the entire thickness of the pericarp. At either side hypertrophied cells may be seen, and the placental matrix bounded by a necrotic plane has pushed up under the pericarp lesion. Such a lesion would be ruptured by the subsequent growth of the fruit. Photomicrograph × 33. Stained with carbol fuchsin. Fruit 12 mm. in diameter.

F.—Section of an intumescence on a radial locule wall extending in toward the necrotic surface of the placental matrix and adhering to the latter at one point. The hyperplasia originates in the subepithelial layers. Photomicrograph × 33 from longitudinal section of a fruit 12 mm. in diameter, stained with Haidenhain's iron-alum haematoxylin.
Necrosis, Hyperplasia, and Adhesions in Mosaic Tomato Fruits

(For explanatory legend see p. 879)
areas in the epithelium lining the locule. Internal intumescences of considerable size were found in the larger green fruits. Early stages in the growth of such intumescences are shown in Plate 1, E, and Plate 3, F, and larger ones are shown in Plate 7 and Plate 8, A. Plate 7, A, represents a very large intumescence, a double outgrowth which measured about 2 mm. in depth. Most of them are not much over 1 to 1.5 mm. in depth. Where these internal intumescences impinge upon the placental matrix there are thin plates of crushed brown cells, the necrotic planes mentioned above (pl. 5, C1; pls. 4, 7, and 8). Very often a reciprocal and opposite hyperplastic zone has arisen in the placental tissue meeting the invading intumescence (pl. 4, A) or pushing up against the pericarp (pl. 3, E; pl. 6, B) on the inner side of the necrotic separation plane.

The impression is gained from the examination of the sections that the intumescences from the inner wall surface actually invade and crush the placental parenchyma, exerting considerable pressure thereon (pl. 7, A; pl. 8, A), and the same condition would appear to exist at the centers of the whorls of hypertrophied cells surrounding necrotic pockets (pl. 6, D). The thin flattened condition of the necrotic separation planes may be due to the pressure of the opposing hyperplastic growths. This pressure might be one cause of the greater firmness of these regions noted when the tissues are cut. In many cases hyperplastic growths from the locule wall impinge upon ovules (pl. 5, C; pl. 7; and pl. 8, B), and in one instance noted the side of a seed was indented, apparently by the pressure of one of these growths. In a few cases, evidence of hypertrophy and hyperplasia was noted in the endosperm, and abnormal transverse walls in the palisade epidermal cells of the seed coat were noted in seeds lying near a necrotic zone in the placental tissue (pl. 8, B).

Hyperplastic tissue similar to that found in fruits has also been found in the cortical tissue of peduncles and petioles, immediately underlying necrotic surface stripes.

In the stained preparations it was found that the necrotic tissues tended to retain safranin and the living cells, light green, gentian violet, or Ziehl's carbol fuchsin. Both retained Haiden-hain's iron-alum haematoxylin.

A few cases of rounded dark green external projections or knobs on the older fruits due to hypertrophy of the interior cells of the pericarp have been found. These seem to represent a feature somewhat similar in nature to the abnormal projections on the fruit in the case of cucumber mosaic.

**ADHESIONS**

Perhaps the most interesting feature of the internal abnormalities in these mosaic tomatoes is the fact that the hyperplastic invasions result in actual and firm adhesions between the pericarp or locule wall and the adjacent placental matrix and ovules (pl. 1, C; pl. 3, F; pl. 4, A; pl. 6, B and C; and pl. 8, B) and also between the placental matrix and the epidermis of the seed coat. The intervening necrotic plane is not usually a true separation plane, but seems rather to represent a plane of adhesion. Furthermore it is not always continuous, in which case the opposing tissues appear actually to fuse or grow together by infiltration into each other (pl. 4, B).

This phenomenon of abnormal adhesions is readily demonstrated by peeling a segment of the pericarp from a locule in which case single seeds or groups of seeds surrounded by the placental matrix will pull loose from their normal funicular attachment and remain firmly adherent to the inner surface of the pericarp at many of the points where necrotic planes are located. Furthermore, these adhesions are readily recognizable in the manipulation of the razor sections. The abnormal fusions between interior surfaces add to the unnatural firmness of affected tissues, to the disarrangement of the ovules, and to the external malformations evinced by the growing fruits. In one case an unnatural groove in the fruit was attributable to an underlying adhesion at the corner of a locule.

**EXPLANATORY LEGEND FOR PLATE 4**

A.—Section through a fusion in the angle of a locule in a mosaic tomato 11 X 17 mm. in diameter. The normal thickness of the pericarp is shown at the top. The hyperplastic growth from the placental tissue begins near the base of the ovule and far exceeds in extent the reciprocal inward growth from the pericarp and locule wall. The diffuse cell contents at the base of the placental hyperplasia are a common companion of placental hyperplasia. The necrotic plane parallels the carpel or pericarp and ovules (pl. 1, C; pl. 4, A; pl. 6, B, and C; and pl. 8, B) and also between the placental matrix and the epidermis of the seed coat. The intervening necrotic plane is not usually a true separation plane, but seems rather to represent a plane of adhesion. Furthermore it is not always continuous, in which case the opposing tissues appear actually to fuse or grow together by infiltration into each other (pl. 4, B).

B.—Showing the fusion of the placental matrix and the epidermis of the seed coat. Photomicrograph X 33. Unstained.
Necrosis, Hyperplasia, and Adhesions in Mosaic Tomato Fruits

Plate 5

(For explanatory legend see p. 881)
DISCUSSION

From a study of these abnormal tissues the impression is gained that the hypertrophy and hyperplasia are responses to the necrosis, possibly an effort on the part of the host to repair and replace or to isolate and occlude the necrotic tissue, possibly a response to a stimulus emanating from the necrotic tissue. In the surface blisters this explanation is tenable, inasmuch as the epidermal layer is necrotic. The well-developed intumescences on the inner walls of the locules occur only opposite necrotic regions in the placental matrix or under necrotic areas of the inner epithelium of the pericarp. It may be stated as a general rule that the hypertrophy and hyperplasia occur only in close association with the necrosis.

Structurally the type of growth resembles that involved in wound tissue or cork formation. The fact that the smaller cells in the hyperplasias occur in proximity to the necrotic tissue would indicate that the latter may supply the growth stimulus. Riker (28, p. 427) found that the smaller cell size and the most rapid cell division in tomato stems inoculated with the crown-gall organism occurred in the cells adjacent to the intercellular spaces containing the bacteria and hence nearest to the source of stimulation. However, in the larger intumescences in the mosaic fruits there is evidence of renewed waves of growth in the basal portions quite distant from the necrotic region.

The destructive invasion of tissues by these proliferations and the abnormal tissue fusions may be interpreted as incidental consequences of the growth response to the necrotic condition. However, it may be possible that hyperplasia, as well as necrosis, is a direct effect of the mosaic virus. The flattened necrotic plates are doubtless the result of the pressure of the hyperplastic tissue.

That the epithelium lining the locules in a young tomato is readily reactive to a growth stimulus, or to a removal of growth inhibition, is shown by the ease with which Smith (31, p. 174) was able to produce surface proliferations by exposing this epithelium to the vapor from injections of 20 per cent solutions of ammonium carbonate, acetate, and tartrate. The greater prevalence of necrosis in the tissues of the placental matrix may possibly be associated with their extremely rapid growth.

A partial review of the literature on mosaic and related diseases shows that certain of these histological phenomena have been previously noted. With respect to the malformation and rupture of fruits, the bizarre effect of the mosaic disease on cucumber fruits has been described by Doolittle (11, p. 13), who also noted that the same disease caused a bursting or rupture of the pericarp of the wild cucumber fruit. Allard (1, p. 255) noticed malformation of the blossoms in tobacco mosaic which interfered with the normal development of the pistil and reported that mosaic reduced the number and the viability of the seeds. The malformation effect of mosaic on tomato fruit has been noted by Dickson (10, p. 18) and has been reported in connection with winter blight by Howitt and Stone (14, p. 164).

Both internal and external malformation of the tomato fruit were noted by Cobb (9, p. 412) in a disease called rosette. The necrotic surface-spotting of tomato fruits associated with mosaic or related diseases has been described by a number of observers including Selby (30, p. 238), Orton and McKinney (23, p. 242), Howitt and Stone (14, p. 163), Brittlebank (8, p. 132), Paine and Bewley (24, p. 187), McKay (18, p. 181), Gardner and Kendrick (12, p. 8), and Poole (25, p. 5).

Of particular interest in connection with the surface blister lesions on tomatoes and pericarp-rupture are the observations of Atanasoff (4, p. 8) upon the occurrence of flat translucent blisters on very young potato tubers of the Schotsche Muis variety affected with stipple streak, in which case the blisters sink and produce hardened

EXPLANATORY LEGEND FOR PLATE 5

A.—Section of an early stage of a fruit blister showing the brown necrosis of the epidermal cells and the hyperplasia of the subepidermal cells. Photomicrograph X 126. Stained with gentian violet.

B.—Cross section of a portion of a surface blister showing radial elongation and transverse division of the subepidermal cells to form the hyperplastic tissue of the blister. The necrotic tissue is in the epidermal region of the blister. Photomicrograph X 82. Unstained.

C.—Cross section near center of a fruit 1 cm. in diameter showing portions of septa, placenta, and seeds, with necrotic areas and a rather marked displacement or abnormal arrangement of the ovules. (1) Thin plate of necrotic tissue at the advancing face of a hyperplastic outgrowth from the locule wall. (2) Necrotic pocket near an ovule. (3) Necrotic ovule in contact with a plate of necrotic tissue and an outgrowth from the locule wall. The ovule is somewhat changed from its normal location. Photomicrograph X 25. Unstained.
Necrosis, Hyperplasia, and Adhesions in Mosaic Tomato Fruits

(For explanatory legend see p. 883)
areas which may result in severe and extensive cracking. Cracking of the tubers has also been reported by Barrus and Chupp (6, p. 126). Murphy and McKay (22, p. 351) have recently reported similar blisters on tubers of the President variety in which they found evidence of cell division and a later necrosis and collapse of the cells.

The occurrence of translucent tissue in mosaic and related troubles has been recorded by many observers. Bailey (5, p. 150), in 1892, noted dark translucent spots on tobacco leaves which were followed by longitudinal cracks, and Kunkel (17, p. 3) noted translucent spots in the very young leaves and translucent strips within the stalks of mosaic corn plants. Atanasoff (4, p. 6) noted translucent margins about the leaf and stem lesions of potato stipple streak, and Hungerford (16, p. 136) working with a similar disease, noted that the lesions were first watersoaked.

Dickson (10, p. 42) found that the lighter leaf areas were more translucent in a number of mosaic diseases including tomato, tobacco, potato, legumes, and raspberry and attributes this to the presence of less chlorophyll and to a reduction of intercellular space owing to the smaller, more isodiametric and closely packed cells, a condition which would facilitate the passage of light. This condition was described by Woods (34, p. 10) in the light areas of mosaic tobacco leaves and has also been observed by Doollittle (11, p. 17) in cucumber mosaic, by Rand (28, p. 14) in pecan rosette, and by Matsumoto (19, p. 295) in azuki-bean mosaic. Rand also found decreased intercellular space in the thickened, dark green leaf areas. It would seem, therefore, that the formation of translucent tissue with reduced intercellular space is rather common in the mosaic types of disease.

Necrotic spotting of the leaves and necrotic streaking of the stems seem to be of rather general occurrence among mosaic diseases. Internal necrosis, however, is not so generally recorded but has been noted by Smith and Bonequet (33, p. 104) in the phloem in beet curly top, by Artschwager (2, p. 669) in the phloem tissue in potato leaf-roll, by Robbins (29, p. 355) in the midrib phloem in sugar beet mosaic, and by Rankin (27, p. 32) in the phloem and pericycle in raspberry leaf-curl. Isolated, scattered, internal pockets or strips of necrotic tissue have been found by Matz (20, p. 75) in the stem parenchyma in mosaic sugar cane, by Kunkel (17, p. 5) in the stem parenchyma in corn mosaic, and by Güssow (13, p. 493), Murphy (21, p. 79), Barrus and Chupp (6, p. 126), Atanasoff (4, pp. 7 and 11), Artschwager (8, p. 248), and Murphy and McKay (22, p. 351) in the case of potato streak and related diseases. In sugar cane and corn, cavities were found associated with the necrotic strips and pockets.

In the tomato, Orton and McKinney (23, p. 244) noted internal necrotic areas in the stem, and Howitt and Stone (14, p. 163) state that the brown "discoloration extends deeply into the flesh of the fruit and can be traced from the epidermis along the septa to the center." Paine and Bewley (34, p. 188) found necrotic patches in the pith and cortex of the stem and in the petiole. Brittlebank (8, p. 232) also found the brownish lesions in the case of spotted wilt extending deeply into the fruit. The latter observers intimate, however, that the internal necrosis extends inward from the surface lesions, whereas in our material there was ordinarily no such direct connection between external and internal lesions.

Hypertrophy is reported by Dickson (10, p. 34) in the palisade parenchyma of the thickened, dark green areas in the

EXPLANATORY LEGEND FOR PLATE 6

A.—Cross section of pericarp of mosaic tomato ovary, 4 mm. in diameter, showing atrophy of entire thickness of wall due to early necrosis. Such areas rupture early and result in fruits such as are shown in Plate 5.

B.—Somewhat similar atrophy has been found in the locule walls. Photomicrograph X 85. Unstained.

C.—Section through a granular fruit lesion (pl. 2, F) showing variation in thickness of pericarp due to internal necrotic planes and cell hypertrophy. At the right, inward hyperplastic growth is visible. The placentral matrix bounded by a dense necrotic surface plate is pushing up against and adhering to the pericarp as a result of cell elongation; such internal pressure probably accounts for the greater firmness of the granular areas on the fruit. Photomicrograph X 33. Stained with carbol fuchsin. Fruit 12 mm. in diameter.

D.—A necrotic region in the axial tissue of the fruit showing the collapse of the necrotic cells and the hypertrophy of the surrounding cells forming the translucent zone visible under the hand lens. Photomicrograph X 33. Stained with carbol fuchsin. Fruit 12 mm. in diameter.
Necrosis, Hyperplasia, and Adhesions in Mosaic Tomato Fruits

(For explanatory legend see p. 885)
leaves of mosaic tomato, petunia, legumes, and raspberry, and a similar condition was found by Doolittle (11, p. 17) in cucumber leaves and by Rand (26, p. 19) in pecan leaves. Townsend (33, pl. 4) showed that the spines on the leaf veins of the lower epidermis characteristic of beet curly top were due to elongation of cortical cells. Dickson (10, p. 36) found hypertrophied chlorenchyma cells associated with the raised green areas on mottled mosaic tomato fruits, a condition similar to the pericarp hypertrophy occasionally met with in this study.

Extensive hyperplasia or proliferation of cells was found by Allard (1, p. 255) in the anthers of mosaic tobacco plants. The hyperplastic development of a double layer of palisade tissue in the dark green leaf areas is reported by Hunger (15, p. 272) and by Dickson (10, p. 26) in tobacco mosaic, and Doolittle (11, p. 18) reports hyperplasia in connection with the projecting knobs on mosaic cucumber fruits.

Of special significance are the observations of Smith and Boncquet (32, p. 104) on the formation of wound-healing cells about the phloem necrosis in beet curly top, the observations of Artschwager (2, p. 569) upon the radial stretching or hypertrophy of the cells surrounding the necrotic areas in potato leaf-roll and the observations of Kunkel (17, pl. 12) upon the elongation of the parenchyma cells around the necrotic pockets or cavities in the mosaic corn stalk, leading, as he says, to incipient gall formation. These areas, Kunkel (17, p. 11) states, first "appear water soaked and are more turgid than the surrounding tissues," later (17, p. 5) "take on a slightly yellow or brown color, and "in a still more advanced stage all or a part of the cells in the pockets collapse and elongated cavities are left within the stalk." He further states (17, p. 9) that "the disease usually causes the host cell to enlarge . . . Many diseased cells die and collapse. This may happen even when little or no abnormal growth has taken place. However, cells that make considerable growth die and break down earlier than cells that respond more slowly." These phenomena exhibit striking similarities with some of those observed in mosaic tomatoes, except for the unmistakable hyperplasia in the latter.

It would seem, therefore, that under certain conditions the young tomato fruit may express a range of rather striking symptoms, of which, however, some trace has been recorded in the mosaic and related diseases of other hosts. Malformation and bursting of the fruit, external and internal necrotic regions, cavities, surface blisters, translucent tissue with reduced intercellular space, elongation of cells around necrotic tissues, and hyperplasia have been previously noted. In view of the profound alterations and derangements in the normal hereditary course of development of the embryonic leaf tissues brought about by mosaic diseases in general, it is not surprising to find strikingly aberrant histological conditions in the tissues of the young, rapidly growing tomato fruit.

**SUMMARY**

The histological abnormalities in the young fruits of greenhouse tomato plants affected with the severe type of mosaic (streak or winter blight) were studied by means of unstained free-hand sections and stained microtome sections.

Normally the tissue of the axile placenta grows out between and around the ovules, engulfs them, and fills the locular cavity with a cellular matrix as the ovary enlarges. This placental matrix touches, but remains free from the carpellar walls and the seed coats.

Many of the mosaic fruits are characterized by brown necrotic surface dis-
Necrosis, Hyperplasia, and Adhesions in Mosaic Tomato Fruits

Plate 8

(For explanatory legend see p. 887)
coloration, by raised brownish translucent blisters, or by sunken necrotic lesions exhibiting a great variety of shapes and sizes and arranged in peculiar patterns. Such lesions on very young fruits result in great malformation.

Deep-seated necrosis in the pericarp and subsequent atrophy or collapse of the affected tissues result in extensive rupture of the fruit-wall and cracking of the fruit. Hypertrophic thickening of the pericarp was found.

Internal necrotic regions, surrounded by zones of translucent tissue, occur throughout the fruit, particularly about the periphery of the placental matrix. Cavities are often associated with these necrotic regions.

Abnormal adhesions between the ovules and the placental matrix and between the latter and the lining of the locular cavity are of rather frequent occurrence at the necrotic planes.

Ovules may be disarranged or abnormally oriented and may be retarded or atrophied. Seeds may show brown spots under the seed coat. The placental matrix may develop abnormally.

The epidermal blisters are caused by cushions of muriform hyperplastic tissue which push up under the necrotic epidermis. The translucent appearance of this tissue is caused by the reduced intercellular space.

The internal necrotic regions are usually isolated strips, pockets, or plates of crushed brown tissue surrounded or accompanied by zones of radially elongated cells or zones of hyperplastic tissue composed of parallel columns of meristematic cells growing in toward the necrotic region. Hyperplasia is most marked in cases where epithelial tissues are involved.

Rather well developed intumescences grow inward from the locular walls and invade the placental matrix, resulting in abnormal tissue fusions or adhesions.

Necrosis and cell hypertrophy within the seed and abnormal cross walls in the palisade epidermal cells of the seed coat were found.

Apparent hypertrophy and hyperplasia occur only in association with necrosis and are responses to the latter.

A review of the literature on mosaic and related diseases shows that certain of these phenomena have been previously observed.

**LITERATURE CITED**


(15) Gossow, H. T. 1918. OBSERVATIONS ON OBSCURE POTATO TROUBLES. Phytopathology 8: 401-405, illus.


**EXPLANATORY LEGEND FOR PLATE 8**

A.—Cross section through outer angle of a locale showing a large intumescence from the locale wall and a smaller one from the inner surface of the pericarp pushing a plate of crushed necrotic tissue down into the placental parenchyma. The normal thickness of the pericarp is shown on the upper right corner and the normal smooth inner surface at either side of the smaller intumescence. In the lower right corner may be seen the normal outer surface of the placental parenchyma or matrix that fills the locular cavity and in the lower left corner is an ovule. Photomicrograph X 27. Unstained

B.—Section through a hyperplastic growth from a locale wall (above) which has pressed a plate of crushed, necrotic tissue against a seed with the resultant production of a few abnormal cross walls in the palisade epidermal cells of the latter. This constitutes an adhesion between the locule wall and the seed coat. Photomicrograph X 26. Unstained
MCKAY, M. B.

MATSUMOTO, T.

MACE, J.

MURPHY, P. A.

MURPHY, P. A., and MCKAY, R.

ORTON, C. R., and MCKINNEY, W. N.

PAINE, S. G., and BEWLEY, W. F.

POOLE, R. F.
1924. Tomato Crop Losses May Be Reduced. Streak and Filiform Diseases Checked by Destroying Weeds and Insects. N. J. Agr. 6 (1): 5, 8, illus.

RAND, F. V.

RANKIN, W. H., and HOCKEY, J. F.

RIKER, A. J.

ROBBINS, W. W.

SELBY, A. D.

SMITH, E. F.

SMITH, R. E., and BONCQUET, A.

TOWNSEND, C. O.

WOODS, A. F.