CUCUMBER MOSAIC IN PUERTO RICO

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During the winter of 1935–36 there was a severe outbreak of cucumber mosaic in Puerto Rico and in some fields all the plants were infected. The writer came to Puerto Rico in July 1923 and this is the first severe outbreak of the disease during the period beginning with that date. In fact, mosaic plants have been very rare. The symptoms of the 1935–36 outbreak were the same as those of the common mosaic in the eastern part of the United States. Diseased plants were rare in 1936–37. There was an abundance of *Aphis gossypii* Glover in the fields in 1935–36 but no experiments were conducted to determine if they were the vectors.

The source of this infection is an unsolved problem at this time. The seed came from the United States as in previous years, but it appears very unlikely that the disease came from that source, as the United States records show that it is rarely if ever transmitted by seeds of the cucumber but that it is transmitted by the seeds of the wild cucumber (*Micrampelis lobata*). Doolittle and Gilbert (1918) reported that one cucumber seed in 5500 produced a mosaic plant. The native wild cucurbits in the immediate vicinity of the fields in Puerto Rico did not show any symptoms of the disease but it is possible that they may be symptomless carriers. *M. lobata* does not occur in Puerto Rico. It is possible that the disease may be due to an entirely different virus from the common mosaic of cucumber in the United States. Bewley and Corbett (1930) reported a mosaic disease of cucumbers which appears to be entirely different from the mosaic cucumbers in the United States in that it was transmitted by the seeds.

The disease was so abundant that the writer decided to continue his studies on the effects of virus diseases on the histology of the host plants. In 1925, 1927, 1930 and again in 1931 the writer published papers showing the effects of viruses on the structure of the leaves and stated that some of the viruses caused an inhibition of the development of the chloroplasts. Several species of host plants were used in these studies. This idea was contradictory to the idea of destruction of the chloroplasts by viruses which had been held up to this time but has been supported by the studies of later workers.

A brief review of the more important work on this phase of the subject since that time is as follows:

Holmes (1931) published a paper on local lesions of tobacco mosaic which indicated changes or destruction at points of inoculation but he did not make histological studies.

Dufrénoy (1928) reported that in some cases chloroplasts did not reach the normal size while in other cases there was a disintegration.

Nelson (1932) said:

"In some cases, no disintegration of the chloroplasts is associated with the chlorosis but in others destruction of the chloroplasts is one of the contributing causes of the development and extension of the chlorotic areas."

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"In the chlorotic areas, the chloroplasts may be fewer in number and smaller than those in the green portions, but, in some cases, there is no deficiency in number and the chloroplasts are normal in size. The affected cells are pale yellow in color and structural changes in the plastids are frequently to be observed. The stroma becomes flattened and larger in diameter and, as a result of dissolution processes, the chloroplasts eventually collapse into a coherent mass of viscous, pale yellow or colorless material with a more highly diffractive surface membrane at the surface."

Clinch (1932) said that her "observations on mosaic-infected leaves also show that inhibited developed is a feature of chlorosis in certain diseases, but the pathological effect, in potato at any rate, is considered to be more complex than Cook has suggested".

In the summary she said:

"The chloroplasts in the cells of the chlorotic areas also display inhibited development, as they are frequently smaller and fewer in number than in corresponding healthy cell. They may, however, be almost normal in size, but differ in other respects from the plastids of a healthy leaf."

Sheffield (1933) published a paper on the aucuba mosaic of tomato in which the results agreed with the writer. This may be summarized by the following extracts:

"As aucuba mosaic does not affect the chloroplasts of leaves which are fully developed at the time of infection, it seems unlikely that mature plastids are destroyed by the virus. The mottling of only young growing leaves suggests that the virus prevents the formation of plastids, and this has been found to be the case".

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"If the virus reaches the cell before plastid development has commenced, then the development of the plastids is inhibited and usually they are destroyed."

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"It should be emphasized that no evidence whatever was found of the destruction of mature plastids. Very occasionally the development of proplastids is inhibited during its course, but usually if a primordium is not destroyed or its development inhibited in a very early stage, then it will give rise to a perfectly normal chloroplast."

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"The young cell responds in several ways to the virus attack. Cell growth is inhibited, simultaneously the proplastids are destroyed or their development prevented."

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"In plants infected with aucuba mosaic certain of the leaf tissues are devoid of plastids and the cells may be undifferentiated. The absence of chlorophyll is brought about by the inhibition by the virus of the development of the plastid primordia. Usually the primordia are destroyed. If plastid development is not prevented in a very early stage, perfectly normal plastids are formed. Mature plastids are never affected by the virus but occasionally intermediate stages may be."

Esau (1933) reported disintegration of the chloroplasts in the leaves of curly top sugar beets.

Bawden (1934) published a paper on a foliar necrosis of potato in which he said:

"The plastids rapidly degenerate, their breakdown often being the first obvious sign of disease. Cells some distance from demonstrably necrotic ones frequently show plastids in all stages of degeneration."

A study of sections of mosaic and normal leaves and of the mosaic and normal parts of leaves of the cucumber in Puerto Rico showed that in most cases the mosaic leaves and mosaic parts of leaves were thinner than the normal leaves and normal parts of leaves.

The palisade cells were always longer in the normal than in the mosaic areas. In some cases the palisade in the mosaic had remained undeveloped and were cuboidal in shape (See figs. 1 to 6).

The chloroplasts were always more numerous and usually larger in the normal than in the mosaic (figs. 1–6).

All the above facts are in harmony with the previous work of the author and with the work of Clinch and Sheffield. It would be presuming entirely too much to say that all viruses have this effect on all species of host plants until the effects of more of them have been studied but it opens a field of research.

Studies were also made of the chlorotic and green areas of the fruits (Figs. 7 & 8). In general it may be said that there is very slight if any differences in the size of the palisade cells but number of chloroplasts in the chlorotic cells was much less than in the green areas.

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Explanation of Plates

1. Cross section of normal part leaf near tip.
2. Cross section of mosaic part leaf near tip.
3. Cross section of normal part leaf six inches back.
4. Cross section of mosaic part leaf six inches back.
5. Cross section of normal part.
6. Cross section of mosaic part leaf same section.
7. Section of epidermis of normal fruit.
8. Section of epidermis of mosaic fruit.

Note that the sections of normal leaves 1, 3 and 5 are thicker than the sections of the mosaic leaves 2, 4 and 6 and that the chloroplasts in the normal leaves are larger and more numerous than in the corresponding leaves of the mosaic plants.

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PLATE XII