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Effect of Bhindi Yellow Vein Mosaic Virus on its Host

By

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Bhindi Yellow Vein Mosaic Virus causes a serious disease of bhindi (*Abelmoschus esculentus* Moench. = *Hibiscus esculentus* L.) in North India. The virus causes a 62.82% reduction in total chlorophyll, 46.70% in chlorophyll *a*, 89.09% in chlorophyll *b*, and 53.30% in carotenoids in infected leaves. Apparent rate of photosynthesis of the infected leaves decreases by 89.47%, while overall photosynthesis is decreased by 51.61%. Rate of respiration of the infected tissue is increased by 8.33%. It is concluded that carbohydrates are transported from healthy to diseased leaves in which they accumulate. This may partially explain the failure of fruit formation in the infected plants.

Bhindi Yellow Vein Mosaic Virus (BYVMV) causes the yellow vein mosaic disease of bhindi (*Abelmoschus esculentus* Moench. = *Hibiscus esculentus* L.). It is a systemic and a very serious disease of bhindi. All the leaves produced after infection are almost completely yellow in colour. In severely infected plants there is almost a complete absence of fruit formation. Since the fruits of this plant are used as a very important summer vegetable in North India, the crop is a complete failure in all such cases. The virus and its relationship with the vector have been studied and reported from India (CAPOOR and VARMA, 1950; VARMA, 1952). However, nobody seems to has yet studied the effect of BYVMV on the physiology of the host plant. Such a study attains significance in view of reports of the destruction of chlorophyll, (OWEN, 1957; JENSEN, 1968; GATES and GUDAUSKAS, 1969 and MANDAHAR and SINGH, 1970), decrease of photosynthetic rate (OWEN, 1957; ORLOB and ARNY, 1961; JENSEN, 1968), and increase in respiration (OWEN, 1956; ORLOB and ARNY, 1961; JENSEN, 1968; GATES and GUADAUSKAS, 1969) of the infected tissue. These factors will obviously decrease the yield of a crop or vegetable and the absence of fruit formation in bhindi plants infected with BYVMV may be explained, perhaps partially, on these altered physiological phenomena.

Materials and Methods

Discs of 1 cm^2 size were obtained from a number of healthy and fully chlorotic leaves of *Abelmoschus esculentus* Moench. (*Hibiscus esculentus* L.) collected in the early morning and were placed in two separate beakers containing distilled

water. Immediately afterwards, eight samples of chlorotic tissue, each containing 20 discs, were placed into Petri dishes fitted with wet cotton and filter paper. Four Petri dishes were placed in full sunlight for five hours and the other four were kept in dark for the same duration of time. Four more such samples, again each sample containing 20 discs, were placed immediately into an oven at 60 °C for recording initial dry weight (control) and another identical four samples were placed immediately in a referigerator for pigment determination. Exactly similar treatment was given to the discs obtained from the healthy leaves. After five hours the samples exposed to sunlight and those kept in the dark were placed into an oven at 60°C for recording the gain as a result of photosynthesis (net production) or loss (as a result of respiration) in dry weight in accordance with the method described by MISRA et al. (1968). Gross production was estimated by adding the value for respiration to the value of net production.

Pigments from each of the four samples (of healthy and chlorotic tissues, respectively) kept in the refrigerator were extracted separately by macerating thoroughly the 20 discs in 80% acetone with a pestle and mortar, filtering and then making the volume of each filtrate to 100 ml with acetone. Optical density of each such pigment extract was determined in a spectro-colorimeter. The various pigments were estimated by the method of DUXBURY and YENTSCH (1956) and ARNON (1949).

Results and Discussion

Table 1 indicates a considerable destruction of chlorophyll pigments in the infected leaves. PETERSON and MCKINNY (1938) found that virus infection in tobacco results in a loss of chlorophyll because of increased activity of the enzyme chlorophyllase. This may explain the loss of chlorophyll in the present case as well. Destruction of chlorophyll in the virus-infected tissues has also been reported by a number of other workers (DIENER, 1963; GOODMAN et al., 1967; JENSEN, 1968). GATES and GUDAUSKAS, (1969) found a 19% decrease in chlorophyll contents of corn leaves infected with maize dwarf mosaic virus; MANDAHAR and SINGH (1970) report on a 64.6% decrease in chlorophyll content of the leaves of

Table 1

Pigment content of healthy and virus-infected leaves of *Abelmoschus esculentus*

Pigment	In healthy leaves (mg/m²)	In diseased leaves (mg/m²)	Per cent decrease in diseased leaves
Chlorophyll *a* and *b*	173.50	64.50	62.80
Chlorophyll *a*	107.50	57.30	46.70
Chlorophyll *b*	66.00	7.20	89.09
Carotenoids	55.10	25.73	53.30

Luffa aegyptiaca infected with cucumber mosaic virus, while the present study indicates a 62.8% decrease in the chlorophyll content of the infected tissue.

Infected tissue shows a 46.70% reduction in chlorophyll *a*, 89.09% reduction in chlorophyll *b* and 53.30% reduction in carotenoids (Table 1). Destruction of primary (chlorophyll *a*) and accessory (chlorophyll *b* and carotenoids) pigments will effect both the photosystems of photosynthesis, since photosystem I is associated with chlorophyll *a* (VERNON et al., 1967; LEVINE, 1969) and photosystem II is associated with chlorophyll *b* (VERNON et al., 1967). Hill reaction, which is a measure of the integrity of photosystem II (SAN PIETRO, 1967), must also, therefore, be effected. SPIKES and STOUT (1965) and ZAITLIN and JAGENDORF (1960) report on an impairement of Hill reaction in the virus infected tissues. Photosynthetic phosphorylation is also decreased in such a tissue (ZAITLIN and JAGENDORF, 1960).

All these factors (decrease in chlorophyll content, impairement of photosystems I and II, Hill reaction and photosynthetic phosphorylation) cumulatively decrease the overall photosynthesis of the infected tissue. This is apparent from the differences in the net production (apparent photosynthesis) and gross production (overall photosynthesis) of the healthy (190 mg/m²/hr and 310 mg/m²/hr, respectively) and diseased (20mg/m²/hr and 150mg/m²/hr, respectively) leaves (Table 2). This is a 84.97% decrease in apparent photosynthesis and a 51.61% decrease in overall photosynthesis by infected leaves. Similar results have been obtained by other workers as well. For example OWEN (1957) found a 80% decrease in the apparent rate of photosynthesis by tobacco leaves infected by tobacco etch virus, ORLOB and ARNY (1961) report that the rate of photosynthesis in barley leaves infected by barley yellow dwarf virus is reduced to a little more than 50%, and the authors have already reported a 48% decrease in photosynthesis of

Table 2

Productivity of healthy and virus-infected leaves of *Abelmoschus esculentus*

	Healthy leaf	Diseased leaf	Remarks
Initial dry wt(control) (mg/20 cm²)	51.60±0.52	51.90±0.47	X
Five hours in full sunlight (mg/20 cm²)	53.50±0.50	52.10±0.47	X
Five hours in dark (mg/20 cm²)	50.40±0.47	50.60±0.22	X
Increase in dry wt due to apparent (net photosynthesis) net production (mg/m²/hr)	190	20	89.47% decrease
Respiration (amount of photosynthate respired (mg/m²/hr)	120	130	8.33% increase
Overall photosynthesis/gross production (mg/m²/hr)	310	150	51.61% decrease

the leaves of *Luffa aegyptiaca* infected with cucumber mosaic virus (MANDAHAR and SINGH, 1970).

Table 2 also indicates that the amount of photosynthate respired by the diseased leaves (130 mg/m²/hr) is more than that of the healthy leaves (120 mg/m²/hr). This means that the rate of respiration of the diseased leaves is more (8.33%) than that of the healthy leaves. Such a conclusion has also been reached by other workers (ORLOB and ARNY, 1961; GOODMAN et al., 1967; JENSEN, 1968; GATES and GUDAUSKAS, 1969).

The apparent rate of photosynthesis by healthy leaves is more than their rate of respiration, while in diseased leaves the apparent photosynthesis is far less than the amount of photosynthate respired during respiration (Table 2). This clearly leads one to conclude that to meet the demands of the diseased tissue carbohydrates must be transported from healthy leaves of the plant. The reports that with regard to rusts and mildews an extensive transport of nutrients does occur from distant leaves towards the infected ones (LIVNE, 1964; POZSÁR and KIRÁLY, 1964), support the above conclusion. This will obviously greatly diminish the flow of nutrients to the developing fruits which will, therefore, either not develop or develop poorly. This may partially explain the failure of fruit formation in severely infected plants. Another factor, which has still not been investigated by any worker, may be that this is a property of the virus *per se*.

The greater dry weight of the diseased tissues than that of the healthy ones can be readily explained on the basis of an abnormal transport of carbohydrates to, and their accumulation in the former (Table 2). ORLOB and ARNY (1961), and GOODMAN et al., (1965) actually report on the accumulation of soluble carbohydrates and starches in the virus-infected leaves. ORLOB and ARNY (1961) and JENSEN (1969) also found the dry weight of the infected tissue to be greater than that of the healthy tissue.

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