Respiration and Nucleic Acid Metabolism Changes in Cowpea Mosaic Virus-Infected Etiolated Cowpea Seedlings

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ABSTRACT

Higher respiration rates and higher nucleic acid levels in cowpea mosaic virus-infected etiolated cowpea hypocotyls appear to result from a slower rate of decline during senescence rather than to a stimulatory effect accompanying virus infection. It is suggested that ethylene and 3-indoleacetic acid oxidase may be involved in the changes observed in infected tissue by interfering with the normal pattern of nucleic acid redistribution in the developing seedling. Phytopathology 60:1852-1853.

In a previous report (2) dealing with the effect of cowpea mosaic virus (CPMV) infection on peroxidase, indoleacetic acid (IAA) oxidase, and ethylene levels in etiolated cowpea seedlings, it was suggested that growth inhibition in virus-infected seedlings was due to an interference with auxin metabolism mediated by ethylene and IAA oxidase. The results reported below suggest another correlation, in this case between IAA oxidase and ethylene levels and differences in respiration rates and RNA content in healthy and CPMV-infected cowpea seedlings.

Cowpea (*Vigna unguiculata* [L.] Walp. 'Early Ramshorn') seedlings were grown in darkness and inoculated with CPMV as reported previously (2). Respiration rates (as oxygen uptake) were determined daily for slices of hypocotyl tissue 0.4-0.8 mm thick at 1-7 days after inoculation (days p.i.). All measurements were made at 27 C, using a Gilson Differential Respirometer. The results in Fig. 1 indicate that while the respiration rates of both healthy and infected tissue decline over the 7-day period, that of infected tissue declines less rapidly. The observed differences in respiration rate were not affected by the addition of 0.05 M glucose, nor by the presence of 10^{-2} M malonate or 10^{-2} M fluoride. The observed difference in respiration indicated that the higher oxygen uptake by virus-infected tissue was not due to differences in substrate level or degree of participation of the glycolytic pathway or citric acid cycle. The differences in oxygen uptake were also not altered by the presence of 2,4-dinitrophenol (5×10^{-5} M), cyanide (NaCN, 10^{-5} M), azide (NaN_3 10^{-3} M), or diethyldithiocarbamate (DIECA, 10^{-3} M) (3), suggesting that neither uncoupling nor extensive changes in the pathway of terminal oxidation could account for the higher O_2 uptake by infected hypocotyl tissue. That nonrespiratory uptake was not a component of the

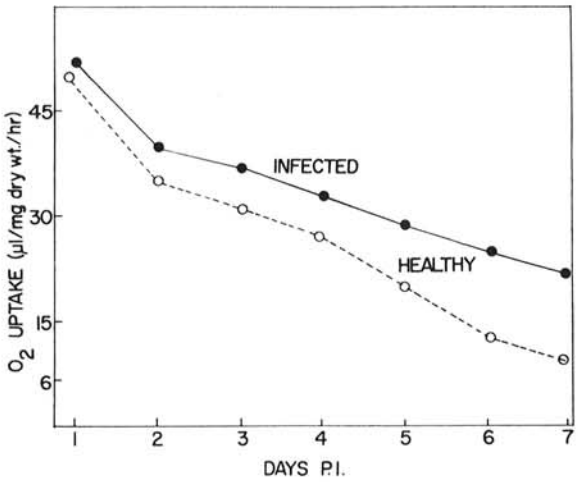

Fig. 1. Respiration rates (as O_2 uptake) of tissue slices of healthy and cowpea mosaic virus-infected etiolated cowpea hypocotyls at 1-7 days after inoculation (days P.I.).

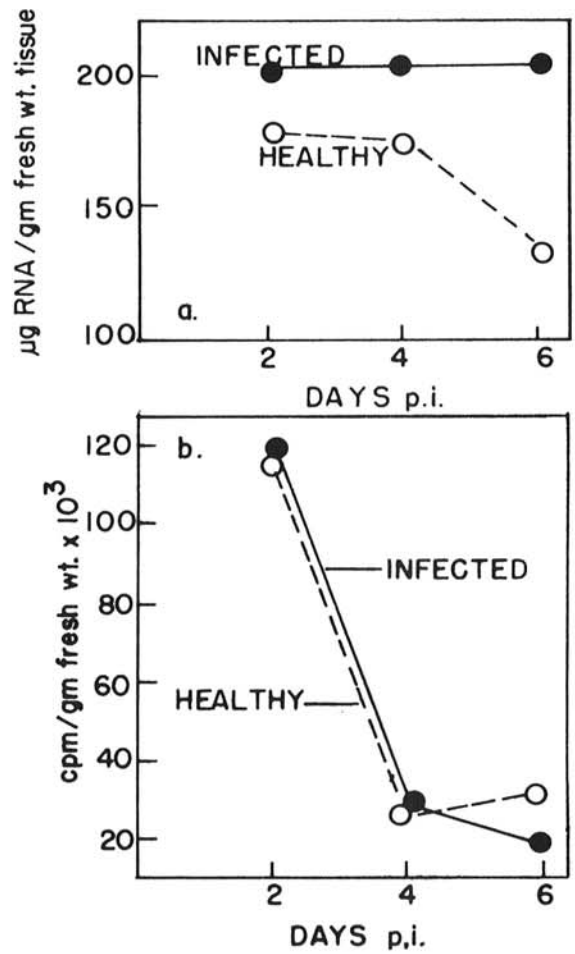

Fig. 2. **a)** Total RNA; **b)** ^{32}P incorporation into RNA, expressed on a unit fresh wt basis, in healthy and cowpea mosaic virus-infected etiolated cowpea hypocotyl tissue at 2, 4, and 6 days after inoculation (days P.I.).

higher respiration rate of infected tissue was further suggested by the observation that the RQ (Q_{CO_2}/Q_{O_2}) of infected tissue remained above that of healthy tissue. From these results, it was concluded that the difference in respiration rates between healthy and CPMV-infected tissue was a quantitative rather than a qualitative effect. Levels of total RNA (phenol extracted) were determined for healthy and CPMV-infected cowpea hypocotyl tissue at 2, 4, and 6 days after inoculation. The results (Fig. 2) show that while the RNA level declined quite markedly in healthy tissue, it was practically unchanged in the infected tissue. Rates of ^{32}P incorporation into RNA were determined using excised hypocotyl segments (1), and the results (Fig. 2) indicate that the differences in RNA levels could not be ascribed to differences in rate of de novo RNA synthesis by infected tissue. Furthermore, since there were no differences in the sedimentation profiles of the respective RNA's, pattern of ^{32}P incorporation into the three sedimenting species of RNA, or changes in ribonuclease level, it appeared that the difference in total RNA could also not be ascribed to extensive degradation of RNA in healthy tissue. From these data, therefore, it was concluded that the higher levels of both respiration and RNA observed in the virus-infected tissue were due to a failure to decline rather than to any stimulatory effect of virus infection.

In cowpea seedlings, RNA is mobilized from the senescing hypocotyl tissue into the elongating epicotyl, and the RNA level of the hypocotyl may be maintained by removal of the epicotyl (3). It appears, therefore, that CPMV infection has the effect of delaying senescence of the hypocotyl tissue, thereby resulting in the maintenance of respiration and RNA levels. It was previously suggested (2) that inhibition of epicotyl elongation was mediated through the action of peroxidase, IAA oxidase, and ethylene. From the data presented above, it now appears that perhaps a more immediate effect of these compounds is to inhibit transfer of RNA from hypocotyl to epicotyl, thereby delaying in the former those changes (decline in respiration, loss of RNA) associated with senescence. It is suggested that the result of virus multiplication is an effect on auxin metabolism, mediated through the action of ethylene and IAA oxidase, thereby preventing the normal redistribution of nucleic acid in the developing seedling.

LITERATURE CITED

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