THE CHEMOTHERAPY OF CARNATION MOSAIC VIRUS

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There has been a recent trend to increase the quality of commercial carnations by selecting disease-free plants for the establishment of mother blocks. Some progress has been made in attaining this goal. Because the carnation-mosaic virus is easily transmitted by contact, some difficulty has been encountered in maintaining the selected stocks in a virus-free condition and in the selection of plants within certain varieties in which the incidence of mosaic infection approaches 100 percent. Because of this, it became necessary to test the possibility of inactivating the virus within infected plants.

White Patrician was used as a susceptable carnation variety (Dianthus caryophyllus L.) while sweet William (D. barbatus L.) was used as a test plant. Due to masking and physiological disorders, symptoms in carnations were never relied upon as the sole criterion for virus infection. In every case when a carnation was to be tested for the presence of the virus, juice from a young lateral shoot was rubbed on the leaves of four D. barbatus plants. The development of symptoms on any of the four inoculated test plants was considered as evidence of virus activity in the original carnation.

An effort was made to determine whether the following chemicals could inactivate the virus in plant juice: proliferol, sulfamerizine, malachite green, potassium phosphate, potassium chlorate, calcium chloride, sodium diethyl dithiocarbamate, sulfasuxidine, trypan blue, sulfaguanidine, sulfathalidine, sodium selenate, zinc sulphate, and Dithane Z78. One percent solutions of these chemicals in infectious plant juice were rubbed on the leaves of D. barbatus plants to test their effect on the infectivity of the virus.

Two methods were used in attempting to inactivate the virus in plant juice. In the first, D. barbatus plants were grown in steamed soil in flats containing 96 plants divided into four sets. The following chemicals in solution were applied: calcium chloride, zinc sulphate, sodium selenate, proliferol, malachite green, trypan blue, Dithane Z78, p-aminobenesulfonylamide, and sulfamerizine. One liter of each solution was applied daily for eight days. Two sets of plants in each flat were inoculated five days before and after the beginning of soil treatment, respectively, with centrifuged juice extracted from frozen infected plants. Another set was inoculated ten days after treatments were begun. The fourth set was left as an untreated check.

In the second method, diseased carnations were grown in glazed crocks filled with sand and were watered with a nutrient solution. Each test chemical was applied with the nutrient solution to the sand, and the excess solution collected by drainage tubes. At the end of the test D. barbatus plants were inoculated with extracts from these carnations to determine the virus activity.

Preliminary tests indicated that treatment with proliferol, malachite green, and sulfaguanidine reduced the infectivity of virus-containing plant juice. Further experiments showed a loss in infectivity of the virus when treated with sodium selenate, zinc sulphate, Dithane Z78, and sulfamerizine.

In general, treatment of plants with sulfaguanidine, sodium, selenate, and proliferol seemed to be more effective in reducing symptoms when applied before inoculations, while Dithane Z78 was more effective when applied after inoculation. Malachite green was uniformly effective in reducing the number of plants expressing symptoms both before and after inoculation.

Calcium chloride had a lethal effect on many of the plants. Some drop in stand count was also noted in plants inoculated five days after treatment with sodium selenate and sulfaguanidine. Plants treated with p-aminobenzenesulfonylamide and sulfamerizine developed such an intense mottling that readings after 39 days were not taken due to possible confusion with mosaic symptoms.

Dithane Z78, zinc sulphate, proliferol, and sodium selenate appeared to increase the dry weights of treated plants, while sulfamerizine, sulfaguanidine and calcium chloride definitely depressed the dry weight.

Efforts to inactivate the virus using scopoletin as the inactivating agent proved negative. Approximately one-third of the D. barbatus plants treated with a saturated water solution of scopoletin produced virus-like symptoms. Five of these plants which appeared to have the most positive mosaic pattern, were tested for virus activity by passage into 20 D. barbatus plants. Four plants were used to test each of the five suspected plants. After an interval of 35 days, symptoms of mosaic were not detected on the 20 test plants while symptoms were still evident on the five original plants.

As facilities were not available for removing the chemicals after their application to the virus, the action of the chemical not only on the virus, but also upon the plant involved in the inoculation, must be considered. The possible masking of symptoms due to absorbtion of the chemical by the plant, and the effects of the chemical on the plant cells could possibly have influenced the manifestation of mosaic infection.

The progress of the virus in a plant is generally considered to be by the invasion of living cells and to be hindered by death of the cells. Therefore, a chemical which is toxic to the cell might hamper or stop the movement of the virus and thereby limit infection. Obvious evidence of this was noted in the use of sodium selenate, which produced a severe necrosis of the inoculated leaf. Fewer plants of D. barbatus, thus treated, appeared to express symptoms of virus infection.

From the evidence presented there seems to be little possibility of the carnation-mosaic virus being inactivated in plants by the chemicals used in these experiments, once it has become established in a plant. However, the treatment of plants with chemicals before infection by the virus occurs might meet with some success. As Dithane Z78 applied after inoculation was more effective in reducing the number of plants showing symptoms, it may be possible that its action was directly on the virus in the plant. In contrast, the action of sulfaguanidine, sodium selenate, and proliferol may be directly on the plant, thereby producing possible immunity to infection by the virus. However, due to the necrotic condition of leaf tissue treated with such a chemical as sodium selenate, the presence of a toxic compound within a cell may prevent its recovery after wounding to such an extent that the virus is unable to produce infection. As malachite green is known to produce a reduction in the increase of tobacco-mosaic virus, the effectiveness of this chemical may be merely in lengthening the time before symptoms appear. This condition would be further aggravated due to the tendency of the virus to become masked as a result of rising temperatures.

It would appear from these experiments that the chemicals with the most promise of inactivating the virus have very little effect on carnation plants and in some cases increase growth either due to stimulation or by providing nutrients to soils lacking these elements.

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