they, too, had an inactivating power against *MHV*-3 virus; no effect was found.

From our results a certain ability to inactivate *MHV*-3 virus may be attributed to intestinal content and intestine of normal mice. Investigations on the mechanism of this inactivation and on the nature of the compound or compounds involved are now in progress.

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[1] Piazza, M., Tancredi, F., and Quagliariello, E., *Nature*, **191**, 601 (1961).
[2] Quagliariello, E., Tancredi, F., Saccone, C., and Piazza, M., *Nature*, **194**, 976 (1962).
[3] Piazza, M., and Tancredi, F., *Nature*, **197**, 903 (1963).
[4] Dick, G. W. A., Niven, J. S. F., and Gledhill, A. W., *Brit. J. Exp. Path.*, **37**, 90 (1956).
[5] Reed, L. S., and Muench, H., *Amer. J. Hyg.*, **27**, 493 (1938).

Inhibition of Localized Virus Lesions by *N*-Dimethylaminosuccinamic Acid

PREVIOUS investigations have shown that the application of 2,4-dichlorophenoxyacetic acid or its salts or certain phenoxyacetic acid analogues greatly altered the number of virus lesions or the amount of virus present in tobacco ringspot (TRSV) inoculated plants[1,2]. This confirmed the findings of several other workers[3,4].

Applications of solutions containing the growth suppressant (2-chloroethyl)-trimethylammonium chloride (CCC) reduced the amount of tobacco mosaic virus in floating leaf disks[5].

This communication describes the response of tobacco ringspot virus inoculated plants of the 'SR' cultivar[6] of *Vigna sinensis* (Torner) Savi to foliar applications of the plant growth retardant chemical *N*-dimethylaminosuccinamic acid (DMAS)[7].

Seeds of the test plants were sown in 3-in. plastic pots containing two parts soil, two parts peat moss, and one part sand. In 8–10 days the primary leaves were sufficiently expanded to permit inoculation. Ten-plant plots were dusted with 400 mesh silicon carbide and inoculated with TRSV juice expressed from inoculated tobacco plants showing good symptoms of the disease. The inoculum was diluted approximately 1 : 250 with 0·01 M neutral phosphate buffer. This inoculum containing 1 per cent silicon carbide was applied by means of a 1-in.-wide, round, T-head acrylic plastic inoculator[8]. The DMAS solutions were applied approximately 45 min after inoculation when the inoculated leaves had dried. In some cases it was mixed with the inoculum. Characteristic, large, susceptible-type, localized lesions (typically preceding systemic necrosis) appeared and were counted 3–5 days after inoculation.

The results of four experiments are illustrated in Fig. 1. Application of an aqueous solution of 5,000 p.p.m. of DMAS to leaves of *Vigna sinensis* 45 min after inoculation reduced the virus local lesions by 90·1–92·9 per cent (Table 1). The solution containing 2,500 p.p.m. of DMAS was almost as effective as the stronger solution, and that containing 1,250 p.p.m. reduced lesions 66·4–83·6 per cent. Progressively less inhibition of virus lesions was caused by solutions containing 625, 312 and 156 p.p.m. of DMAS.

Table 1. INHIBITION OF TRSV LESIONS ON BLACK COWPEA (*Vigna sinensis*) BY *N*-DIMETHYLAMINOSUCCINAMIC ACID

DMAS (p.p.m.)	Exp. 1 Lesion No.*	Exp. 1 Inhibition (%)	Exp. 2 Lesion No.	Exp. 2 Inhibition (%)	Exp. 3 Lesion No.	Exp. 3 Inhibition (%)	Exp. 4 Lesion No.	Exp. 4 Inhibition (%)
0 (control)	18·5	0	23·8	0	21·3	0	109·2	0
156	16·2	12·4	21·6	9·2	20·1	4·7	—	—
312	12·7	31·4	13·7	42·4	13·1	38·5	—	—
625	8·0	56·8	10·4	56·3	10·2	52·1	52·8	51·7
1,250	3·4	81·6	8·0	66·4	3·5	83·6	20·4	81·3
2,500	1·3	93·0	3·1	87·0	2·2	89·7	0·4	99·6
5,000	1·4	92·4	1·7	92·9	2·1	90·1	0·0	100·0

* Means of 20 leaves.

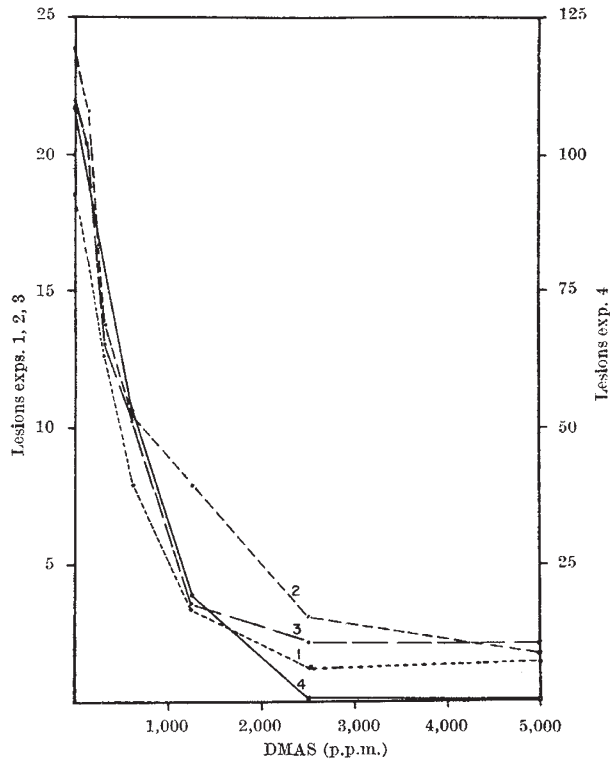

Fig. 1. Reduction of local infection of tobacco ringspot virus (TRSV), on black cowpea (*Vigna sinensis*). Foliar application of DMAS 45 min after inoculation or water spray (control, no DMAS) in exps. 1, 2, 3. Application simultaneously with inoculation (exp. 4)

Extrapolated values for zero concentration fall remarkably close to the actual values for the non-treated (water spray only) control. Lesion inhibition is consistently related to concentration of DMAS over a wide range in concentration. In some cases plants treated with solutions containing 2,500 p.p.m. or more of DMAS had irregular, small, necrotic patches. These patches probably do not represent the interference with lesion formation reported herein since they were also observed on leaves of non-inoculated plants and on non-inoculated parts of inoculated leaves. Inhibition of lesions was pronounced on many leaves with no evidence of chemical injury.

The observed response may be the result of inoculum inactivation, cell injury due to DMAS, altered host susceptibility, or viricidal action. Elucidation of the mechanism of action, however, must await the outcome of further more definitive research now in progress. Other methods and sites of application, the effects of related chemicals, and analysis of treated plants are being investigated.

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[1] deZeeuw, D. J., Karas, J. G., and Hamner, C. L., *Mich. Agric. Exp. Sta. Quar. Bull.*, **44**, 55 (1961).
[2] Karas, J. G., Ph.D. thesis, Michigan State Univ. (1962).
[3] Davis, O., *Phytopath.*, **42**, 465 (1952).
[4] Hartman, R. T., and Price, W. C., *Amer. J. Bot.*, **37**, 820 (1950).
[5] Rawlins, T. E., *Plant Disease Reporter*, **46**, 170 (1962).
[6] deZeeuw, D. J., and Crum, R. D., *Phytopath.*, **53**, 337 (1963).
[7] Riddell, J. A., Hageman, H. A., J'Anthony, C. M., and Hubbard, W. L., *Science*, **136**, 391 (1962).
[8] deZeeuw, D. J., and Timmer, L. W., *Phytopath.*, **54**, 196 (1964).