domestic animals may be reservoirs of so-called human strains of influenza virus. The view had been based on the isolation of several strains of myxovirus influenzae A from horses^{2,3}, ducks^{4,5} and pigs⁶.

Our observations direct attention to the veterinary importance of myxovirus influenzae A. This virus can cause epizootics with severe cases of illness. Thus, its presence in domestic animals may not always be considered as simple carriership.

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¹ Andrewes, C. H., in Morris Pollard, *Perspectives in Virology*, 184 (Galveston, Texas, 1959).

² Sovinova, O., Tumova, B., Pouska, F., and Němec, J., *Acta Virol.*, 2, 52 (1958).

³ Versteeg, J., Mouton, R. P., and Verlinde, J. D., *Tidjsh. Diergeneesk.*, 83, 608 (1958).

⁴ Koppel, Z., Vrtiak, J., Vasil, M., and Špiess, St., *Veterinarstvo*, 6, 267 (1956).

⁵ See Andrewes, C. H., and Worthington, G., *Bull. World Health Org.*, Geneva, 20 (2-3), 435 (1959).

⁶ Popoviet, J., Wynohradnyk, V. L., Berninski, C., Feteanu, A., and Schuler, G., *Probleme de Epizoo. Vet.*, 9, 37 (1960).

Competition between Infectious Tobacco Mosaic Virus Nucleic Acid and Intact Virus on *Nicotiana glutinosa*

PREVIOUS work by Wu and Rappaport¹ was concerned with the phenomenon of competition between intact strains of tobacco mosaic virus. In each case, a mild strain of tobacco mosaic virus (U2 or VM) was used to interfere with the establishment of infections by the common strain, U1. In this communication, competition between these same strains but involving infectious ribonucleic acid (RNA) and intact virus will be described.

Infectious RNA was prepared by the phenol method of Gierer and Schramm², and stored in a deep freeze until used. In the first series of competition experiments, a constant amount of infectious RNA from U1 was mixed with different amounts of intact VM virus. The mixed inoculum was applied to one side of *Nicotiana glutinosa* leaves. The opposite, control half-leaves were inoculated with an equal concentration of U1-RNA alone. Leaves were detached immediately after inoculation and incubated at 22° C. under constant light according to the procedure described by Wildman and Ford³. Inactivation of the RNA preparation by leaf extract was prevented by inoculating each half-leaf with a separate glass paddle. In this way, contamination of the inoculum by tissue fluids was avoided.

The two strains, U1 and VM, differ markedly in lesion size⁴. VM produces a minute lesion which is readily distinguishable from the much larger one produced by the intact strain U1 or its infectious nucleic acid. The extent of interference in the production of U1 lesions, resulting from the competition of U1-RNA and VM, was estimated by counting the large lesions characteristic of the U1 strain.

A number of experiments were performed in which the ratio of U1-RNA to VM virus, as well as the total concentration of infectious units in the mixture, were varied over a wide range. Representative results are given in Table 1.

Table 1. COMPETITION BETWEEN AN INTACT STRAIN OF TOBACCO MOSAIC VIRUS, VM, AND INFECTIOUS RNA EXTRACTED FROM THE COMMON STRAIN OF TOBACCO MOSAIC VIRUS, U1

U1-RNA concentration (μgm./ml.)	VM concentration (μgm./ml.)	Fraction U1 lesions appearing
50	25	0.93
50	250	0.85
38	25	0.91
38	250	0.84
3.8	25	1.73
3.8	250	0.84

Although some interference is discernible, the results suggest that the inhibitory effect of VM on U1-RNA lesion appearance is slight in comparison to that observed between intact viruses. These RNA preparations contain approximately 1 per cent of the infectivity by weight of the intact virus. An RNA concentration of 50 μgm./ml. is thus equivalent to 0.5 μgm./ml. of virus, with respect to the number of lesions produced. Ordinarily, a mixture containing 0.5 μgm./ml. U1 virus and 250 μgm./ml. VM would result in extensive interference, reducing the U1 lesion count to approximately 2 per cent of the controls¹. Similar results to those shown in Table 1 were obtained when the infectious RNA from another mild tobacco mosaic virus strain, U2, was substituted in these experiments for the U1-RNA. These data appear in Table 2, and again indicate that little interference is encountered by the RNA in the presence of intact tobacco mosaic virus.

Table 2. COMPETITION BETWEEN AN INTACT STRAIN OF TOBACCO MOSAIC VIRUS, VM, AND INFECTIOUS RNA EXTRACTED FROM A MILD STRAIN OF TOBACCO MOSAIC VIRUS, U2

U2-RNA concentration (μgm./ml.)	VM concentration (μgm./ml.)	Fraction U2 lesions appearing
38	25	0.89
38	250	0.74

These results indicate that the nucleic acid has a decided advantage, in terms of time, in initiating an infection over the intact virus. This advantage is most reasonably attributed to the elimination of those preliminary steps in infection associated with the removal of the protein and the release of the infectious nucleic acid. All the results so far accumulated suggest that competition in the tobacco mosaic virus system occurs at the nucleic acid-level. For this reason, the likelihood of an intact virus interfering with the establishment of an infection by the nucleic acid is slight indeed. This is in accord with evidence obtained in different ways by other investigators⁵, suggesting that the RNA of tobacco mosaic virus initiates an infection and completes its reproductive cycle sooner than does the intact virus.

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¹ Wu, J. H., and Rappaport, I., *Virology*, 14, 259 (1961). Rappaport, I., and Wu, J. H., *Virology* (in the press).

² Gierer, A., and Schramm, G., *Nature*, 177, 702 (1956).

³ Wildman, S. G., and Ford, C. V., *Phytopath.*, 50, 677 (1960).

⁴ Wu, J. H., Hildebrandt, A. C., and Riker, A. J., *Phytopath.*, 50, 587 (1960).

⁵ Siegel, A., Ginoza, W., and Wildman, S. G., *Virology*, 3, 554 (1957). Schramm, G., and Engler, E., *Nature*, 181, 916 (1958). Franckel-Conrat, H., Singer, B., and Veldee, S., *Biochim. Biophys. Acta*, 28, 636 (1958).