NOTES

Thermal and pH Stability of Feline Calicivirus

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Molar concentration of sodium chloride partially stabilized feline calicivirus against thermal inactivation at 50 °C. One strain of feline calicivirus was relatively acid labile compared to another.

Thermal stabilization by salts and response to acids have been useful as properties characterizing certain groups of viruses (4, 7–10). Several investigators (2, 3, 5) have reported that feline caliciviruses (although the name has not been officially approved, calicivirus [kalyx, pod, or cup] has been proposed by the International Committee on Viral Nomenclature in referring to feline picornavirus) are not protected in the presence of molar MgCl₂ against heat inactivation and are intermediate in their acid sensitivity. We now report that sodium salts partially stabilize a feline calicivirus against heating, and pH stability varies from one type of feline calicivirus to another.

Two feline calicivirus strains, 17FRV (provided by S. H. Madin, California) and C14 (from J. L. Bittle, Indiana), were purified by three consecutive plaquing procedures. Virus infectivity was assayed by inoculating four tubes of primary or secondary monolayer cultures of feline kidney cells, as previously described (1, 6). The stabilization of virus by salts was tested by the technique of Wallis and Melnick (7). The effects of pH values and buffer anions on virus infectivity were studied by mixing 1-ml volumes of virus stock in 9-ml volumes of the following buffer solutions: (i) 0.1 M citric acid-sodium citrate buffer, pH 3.0; and (ii) 0.1 M phosphorous acid-sodium phosphate buffer, pH 2 and 2.5. The virus-buffer mixtures were held at room temperature, and at the desired time the sample was taken for infectivity assay.

Five series of experiments were performed in which the stabilizing effects of salts on 17FRV infectivity were studied. The combined data are displayed in Fig. 1. In all experiments the infectivity was destroyed by heating in water or in the presence of 1 M MgCl₂. These results substantiate the findings reported by previous investigators (2, 3, 5). When heated in the presence of 1 or 2 M NaCl, however, the loss of titer, although fluctuating considerably, ranged from 0.5 to 2.75 log at 30 min and from 2 to 3.25 log at 60 min. This loss was much less in a given experiment as compared to that in water, and we conclude that 1 or 2 M NaCl is partially stabilizing heat inactivation of 17FRV.

Strains 17FRV and C14 behaved differently when acidified (Fig. 2). At pH 2.5, 17FRV has appreciable stability, whereas C14 lost its infectivity by 4.25 log within 1 min. At pH 3.0, C14 was destroyed within 20 min at room temperature, in contrast to only slight loss of infectivity of 17FRV. Another experiment revealed that 0.01 fraction of 17FRV survived 33 days at pH 9, whereas C14 was inactivated within 1 week at the same pH. Thus, feline caliciviruses, not unlike picornaviruses of human origin, may differ in the resistance to inactivation by changes in pH.

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**Fig. 2.** Comparative response of C14 and 17FRV strains of feline calicivirus to acids. P, Phosphorous acid-sodium phosphite buffer; C, citric acid-sodium citrate buffer.

**LITERATURE CITED**


