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BRIEF COMMUNICATIONS

Natural Infection of Porcine Ileal Dome M Cells with Rotavirus and Enteric Adenovirus

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Porcine ileal dome M cells play a role in the pathogenesis of infection by enteric pathogens.[2–4,9] Group A rotaviruses and transmissible gastroenteritis virus have been seen in swine M cells; however, the ability of these cells to support rotaviral and coronaviral replication in the porcine and bovine species has not been documented.[6] Similarly, porcine enteric adenoviruses have been seen in enterocytes and possibly intraepithelial lymphocytes or histiocytes,[3,5,8] but not in M cells. Reported herein is a naturally occurring infection of swine M cells with both group A rotavirus and porcine enteric adenovirus.

A 5-week-old secondary specific pathogen-free piglet, weaned 2 days previously, was fasted and held in isolation 17 hours before the beginning of an experiment concerned with M cell function. No diarrhea or other clinical signs were observed while the animal was kept in isolation. While under surgical anesthesia, samples of terminal ileum were obtained for light, immunofluorescence, and transmission electron microscopy (TEM).

Paraffin sections and 1-μm plastic sections of dome epithelium were examined by light microscopy. Paraffin sections of dome epithelium contained occasional basophilic intranuclear inclusion bodies typical of adenovirus infection. Inclusion bodies were almost limited to dome epithelium. In 1-μm plastic sections, multifocal vacuolation and necrosis of absorptive enterocytes with diminution and loss of integrity of the basal lamina were seen. M cells, identified by a break in the continuity of the microvillous border and the presence of underlying lymphocytes, frequently were expanded toward the lumen and often enclosed large clear vacuoles. These lesions were limited to the upper two-thirds of domes, whereas crypt epithelium of domes and absorptive enterocytes on domes near the crypts were normal. Necrotic cell debris was in the intestinal lumen in close proximity to M cell surfaces. Villi adjacent to domes often contained degenerate and necrotic cells; they occasionally had denuded tips. Mild to moderate multifocal villous atrophy was seen. Group A rotaviral antigens were present in multiple foci on domes and absorptive villi (they were detected by direct fluorescent antibody tests on frozen sections of intestinal mucosa). Particles with the morphology of rotaviruses were seen by TEM in ultracentrifuged, negatively stained preparations of intestinal contents.

Ultrastructurally, absorptive enterocytes and M cells were diffusely degenerate. Necrotic cells were in the intestinal lumen. Degenerate M cells had swollen mitochondria, cytoplasmic rarefaction, vacuolation of rough endoplasmic reticulum, and nuclear swelling. Cytoplasm frequently contained viroplasm adjacent to vacuoles that were distended with rotaviruses and cell membrane debris (Figs. 1, 2). Rotaviruses, which averaged approximately 61 nm in diameter, were on luminal surfaces between surface projections of M cells, and in lymphocytes and mononuclear leukocytes enclosed in central hollow regions. Occasional M cells lacking central hollow regions had large, swollen, superficially displaced nuclei. Nuclei of these cells contained adenoviruses and granular material interpreted as adenoviral proteins (Fig. 3).

Rotavirus has been previously seen in M cells.[4,5] In our report, viral replication is indicated by the presence of cytoplasmic viroplasm and large numbers of virions in rough endoplasmic reticulum.[7] In previous experimental studies of rotaviral and adenoviral infection, advanced cellular damage or lack of a central hollow region may have obscured distinguishing features of M cells.[5,8] Species differences, age, and strain of virus may also be involved. The frequency of M cells in porcine dome epithelium as seen by scanning electron microscopy[9] suggests that in one plane, many of these cells will not be sectioned through the central hollow region. Lack of a central hollow in some cells may also indicate immaturity.[1]

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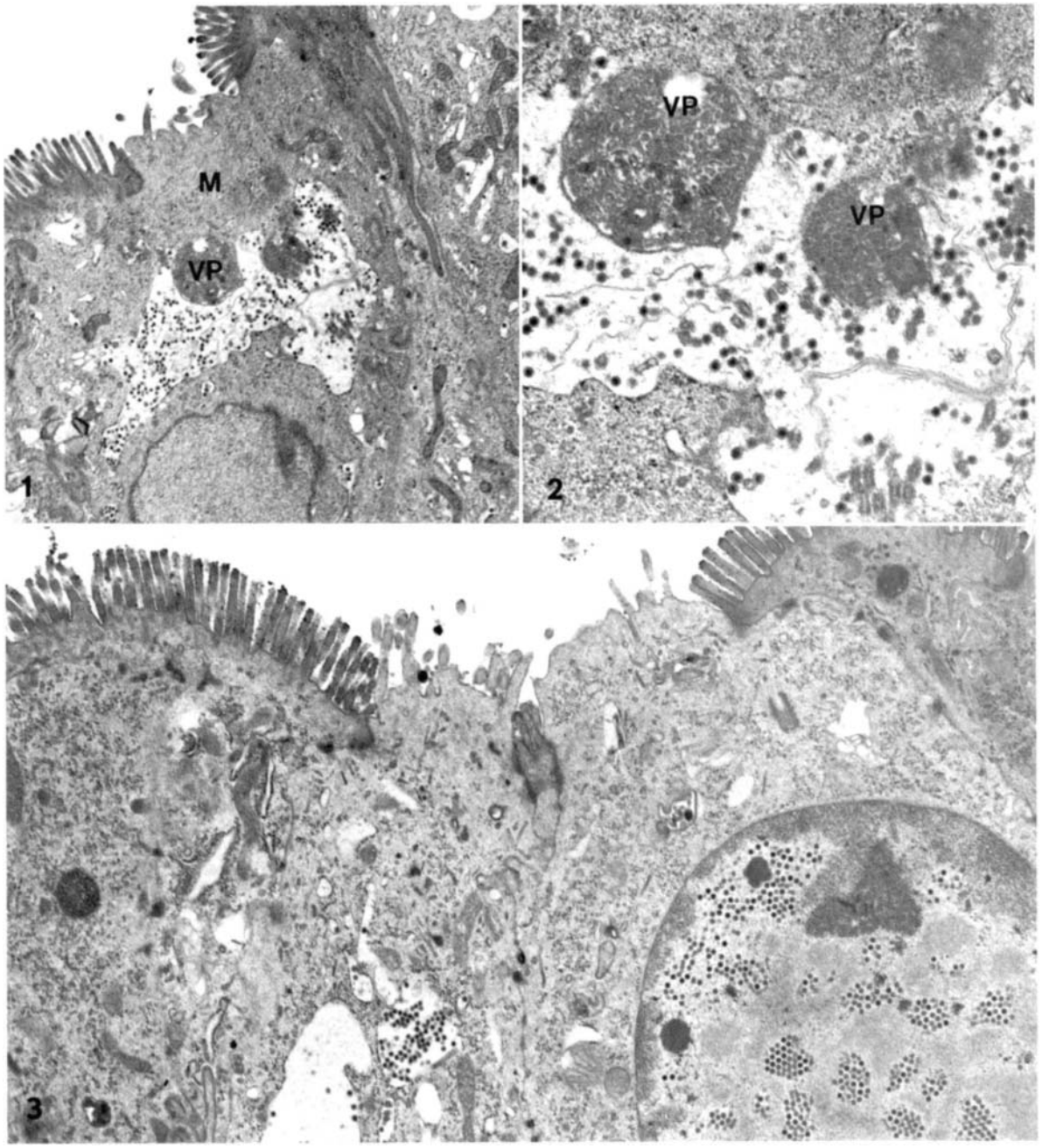

Fig. 1. M cell (M) with large cytoplasmic vacuole containing viroplasm (VP).
Fig. 2. Higher magnification of Fig. 1. Rotaviral particles and viroplasm (VP) in cisterna of rough endoplasmic reticulum.
Fig. 3. M cells containing rotavirus (left) and adenovirus (right).

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