The significance of the Golgi apparatus in the morphogenesis of three porcine coronaviruses: Torovirus, TGEV, and CV777

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Of these three porcine coronaviruses, the Transmissible Gastric-Intestinal Virus (TGEV) and the CV777 virus (1) are enteropathogenic, causing epidemics of diarrhoea in newborn piglets. The Thermophilizing Receptorless Glycoprotein (TRG) is the protein responsible for "virophagia" and "viroplasia" in piglets. The latter virus is essentially neurotropic. All three of these viruses replicate in the cytoplasm of their host cells.

The purpose of the present study was to compare the virus-host cell interaction of these viruses.

For this purpose, a case of TGEV infection from a natural outbreak was selected. With the CV777 virus, a group of 16 casemated-derived, colostomy-derived (CD2D) piglets were inoculated experimentally at the age of two or three days. They were euthanized at different time intervals after inoculation (1). For the study of the morphogenesis of TGE, a three-day-old CD2D piglet was infected with the W211 strain in the stomach wall, laparotomy. This animal was euthanized when it started vomiting, after an incubation period of seven days. From all of these animals, tissue specimens were taken and fixed in glutaraldehyde, osmium tetroxide, and histological sections. For the study of CV777, five cell cultures of primary porcine kidney cells were fixed 24 h after they were inoculated with the same strain of virus. All specimens were processed, blocked, stained, and embedded in spurr's medium for transmission electron microscopy.

With the CV777 virus, the first morphologic evidence of infection of epithelial cells was the appearance of the virus by budding through cytoplasmatic vesicles (EP) membranes. This budding started as a crescent formation with accumulation of electron-dense material paralleled to an EP membrane. Subsequently, a bud was formed, and this bud was pressed off to form a complete virus particle inside EP cytoplasmic vesicles. Virus particles were seen inside Golgi saccs, where they caused a spindle-shaped alteration of the lamellae. The Golgi apparatus was hypertrophic, forming an onion-like structure. Subsequently, the viruses were also observed in the cytoplasm of infected cells. Similar viruses were seen to fuse with the apical cell membranes. As a result, rows of viral particles were present between the microvilli. Electron-dense, membrane-bound, cytoplasmatic structures with a fine granular matrix often were seen, especially in the early phase of infection. These structures frequently contained numerous viral particles. They were morphologically similar to secondary lysosomes.

With TGEV, assembly of virus was by budding through EP membranes frequently seen in the cell cultures. In contrast, it was rare in the infected neurons of the myenteric plexus of the stomach. Viral particles were frequently present inside the Golgi saccs, and formation of virus-filled Golgi vesicles was seen both in the "in vivo" material and in the cell cultures. Many small virus-containing Golgi vesicles were particularly striking in the Golgi area. Viral release by fusion of small vesicles with the plasma membrane was observed only in the cell cultures, and not in the neurons of the myenteric plexus. Budding of viral particles into Golgi saccs at the cell membrane was never seen. Electron-dense, membrane-bound, lysosome-like cytoplasmatic structures were seen to contain viral particles both in the cell cultures and in the neurons in vivo.

With TGEV in the enterocytes of a naturally infected piglet, budding at ER membranes was occasionally seen. Viral particles also were observed inside Golgi saccs and in Golgi vesicles. The Golgi apparatus often appeared vacuolated and hypertrophic. Viruses containing lysosome-like cytoplasmatic structures occasionally were seen.

All three viruses had a similar morphogenetic pattern. This pattern was characteristic for coronaviruses. Nevertheless, in our material, a histotoxemic effect was observed, characterized by the Golgi apparatus was observed in infected cells. Indeed, after assembly at the ER, the virus particles seem to be transported to the Golgi apparatus, where they are embedded in a small vesicle. These vesicles then seem to fuse with the plasma membrane, which is then open to fusion with the host cell.

In conclusion, piglets infected with enteropathogenic coronaviruses can excrete infectious material prior to having significant histological intestinal lesions and possibly prior to positive clinical signs of diarrhea. This study was supported by a grant of the JNWL, Brussels, Belgium.

Selected references:


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