

PEDv in the Literature

Porcine epidemic diarrhea virus infects and replicates in porcine alveolar macrophages

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Porcine epidemic diarrhea virus (PEDV) is a causative agent of porcine epidemic diarrhea; consequently, the small intestine was believed to be its only target organ. In this study, we found that PEDV infected not only the small intestines, but also the respiratory tract. Infection and replication of PEDV in the respiratory tract from naturally PEDV-infected piglets were examined by reverse transcription polymerase chain reaction, immunohistochemistry, and virus re-isolation. Our observations were confirmed by experimental inoculation, and we found that PEDV infection in the respiratory tract was specifically associated with alveolar macrophages in the lung. Vero cell-adapted PEDV was able to replicate in both primary alveolar macrophages and continuous porcine alveolar macrophage cells. Sequencing analysis of the spike (S) glycoprotein revealed that mutations in S might be a potential determinant of auxiliary targets for PEDV. The discovery that PEDV infects and replicates in alveolar macrophages provides new insights into its pathogenesis.

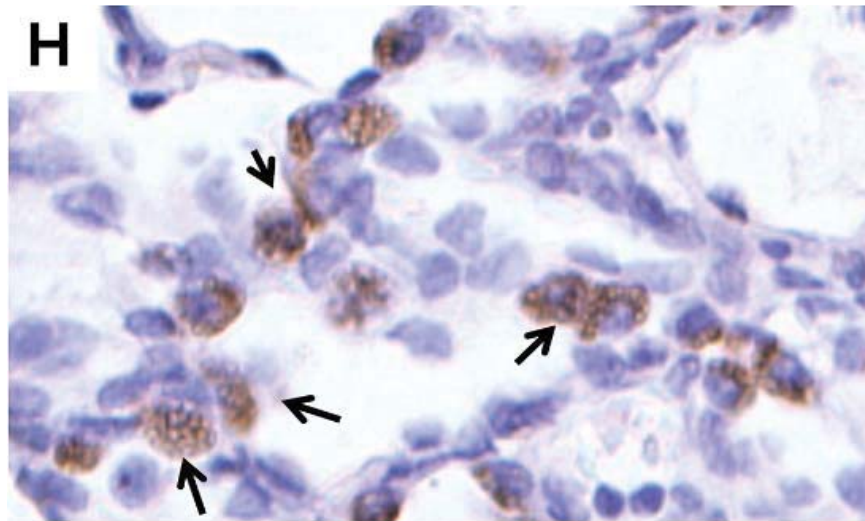


Figure 1) Lung sections from homogenate-inoculated piglets at 96 hpi. Arrows indicate PEDV-infected porcine alveolar macrophages.

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Editor's comment: This is a nice pathogenesis study with an included interest in replication of PEDv in alveolar macrophages in the lungs. Most of us have by now seen the effects of the disease in a herd and see no pathologic indication that lung tissue is infected, but the authors have found and confirmed with immunohistochemistry and re-isolation with 3D4 cells that there is at least a transient period of time in which this replication occurs during acute intestinal infection. It is unclear to us whether presence of PEDv in the lungs initially could have been a result of the oral inoculation or if migration of alveolar macrophages lead to its presentation there as the authors suggest. Moreover, we do not yet know the clinical implications (if any) of replication in the lungs. The authors present an interesting possibility that this alveolar macrophage infection could harbor and replicate just enough virus to remain infectious. This virus could at some point re-inoculate a herd throughout elimination efforts which may be the cause of prolonged infection or cyclical increase in pre-weaning mortality during recovery from the initial outbreak.