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ACSL4-Mediated Lipid Peroxidation Drives Ferroptosis in Vero Cells during PEDV Infection

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DESCRIPTION

Porcine Epidemic Diarrhea Virus (PEDV) is a highly contagious virus that affects pigs, causing severe gastrointestinal disease, particularly in neonatal piglets. In recent years, the mechanisms underlying PEDV infection have garnered attention, particularly how the virus interacts with host cell processes. One of the critical cellular processes implicated in the pathogenesis of viral infections, including PEDV, is ferroptosis, a form of regulated cell death associated with the accumulation of lipid peroxides. Ferroptosis is characterized by iron-dependent lipid peroxidation that leads to cellular damage and death, and it has been shown to play a role in various diseases and infections. Recent studies have suggested that PEDV infection can induce ferroptosis in Vero cells, a commonly used cell line for viral studies, via a pathway that involves Acyl-CoA synthetase long-chain family member 4 (ACSL4), a key enzyme in lipid metabolism Ferroptosis is a distinct type of cell death that differs from traditional apoptosis and necrosis. It is triggered by the accumulation of reactive oxygen species (ROS) that oxidize polyunsaturated fatty acids (PUFAs) in membrane phospholipids. These oxidized lipids lead to membrane damage, disruption of cellular integrity, and ultimately cell death. ACSL4 plays a crucial role in this process by catalyzing the esterification of long-chain fatty acids, which are important components of phospholipids. During infection, PEDV hijacks host cellular machinery, including the lipid metabolism pathways, to create an environment conducive to viral replication. The virus alters the host cell's lipid profile, and ACSL4 becomes an essential factor in the accumulation of oxidized phospholipids. These oxidized lipids are a hallmark of ferroptosis, and their presence suggests that PEDV infection manipulates the lipid metabolism machinery to facilitate ferroptotic cell death. In Vero cells, PEDV infection leads to the activation of several key signaling pathways that promote lipid peroxidation. ACSL4 is one of the key players in this process, as it regulates the incorporation of long-chain fatty acids into phospholipids, thereby influencing the overall lipid composition of the cell. Upon PEDV infection, the increased activity of ACSL4 leads to an enhanced formation of phospholipids containing PUFAs, which are more prone to oxidative damage. The oxidative stress generated by the viral infection results in the accumulation of lipid peroxides, triggering ferroptosis. The interplay between viral replication and ferroptotic pathways creates a vicious cycle that contributes to the death of infected cells, further compromising the host's immune response. The involvement of ACSL4 in the PEDV-induced ferroptosis pathway has been a significant finding. It suggests that targeting ACSL4 could offer a potential therapeutic strategy to combat PEDV infections. Inhibition of ACSL4 or its downstream signaling pathways has been shown to reduce lipid peroxidation and alleviate ferroptosis, which may limit viral replication and improve cell survival. This opens up new avenues for developing antiviral therapies that focus not only on inhibiting viral replication but also on modulating the host's lipid metabolism to prevent ferroptotic cell death. Additionally, the oxidative stress induced by PEDV infection and its role in ferroptosis also sheds light on the broader implications of viral pathogenesis. The ability of viruses to manipulate host cellular processes such as lipid metabolism and oxidative stress responses underscores the complex interaction between host and pathogen. By understanding these mechanisms in greater detail, researchers can develop more targeted and effective treatments for PEDV and other viral infections that exploit similar pathways.

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CONFLICT OF INTEREST

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