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Perspective

Novel Perspectives on Parechovirus Infection in Human Brain Organoids: Beyond Neurologic Manifestations

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DESCRIPTION

One of the main causes of infections in the Central Nervous System (CNS) is porcine viruses. Although certain genotypes, such Parechovirus A3 (PeV-A3) and Echovirus 11 (E11), can cause serious neurological illness, PeV-A1, which is quite common, is not linked to central nervous system disease. Here, we use human brain organoids and clinical isolates of PeV-A genotypes to further our knowledge of these variations in PeV-A CNS illness. Our findings suggest that the variations in neurological illness caused by PeV-A1 and A3 viruses are not attributable to the infectivity of Central Nervous System (CNS) cells, as both viruses are able to infect brain organoids with a comparable cell tropism. The host cell metabolism is markedly altered by PeV-A infection, according to proteomic studies. The ensuing inflammatory reaction the potency of PeV-A3 (as well as E11 infection) was much higher than that of PeV-A1 infection. Altogether, our data support clinical observations and point to a role for inflammatory-mediated neurology in PeV-A3 (and E11) infection as opposed to viral replication.

Within the Picornaviridae family, human parechoviruses, sometimes referred to as Parechovirus A (PeV-A), are prevalent childhood diseases that can cause severe clinical symptoms, primarily in infants1. PeV-A has been reported to be in circulation in a number of nations, including the USA4, Japan3, and the Netherlands2. Enteroviruses (EVs) and PeV-As are closely related, sharing clinical traits and the potential for outbreaks. PeV-As are the second most common cause of viral CNS infections in newborns, much as EVs. PeV-A and EVs have strong similarities that are emphasized by the Polyphyletic group of "orphan" viruses within the enterovirus genus, Echoviruses 5, which includes neurotropic viruses like Echovirus 11 (E11) 6–8, was the first categorization of PeV-A.

There are 19 genotypes in the PeV-A species, the most common of which are PeV-A1 and PeV-A39. While PeV-A1 and PeV-A3 can can cause respiratory and gastrointestinal disorders, PeV-A3 is more commonly linked disorders of the Central Nervous System (CNS). Globally, there have been many reports of PeV-A3 outbreaks; the most recent occurred in the USA in 2022/12. The virus can enter the circulation and cause sepsis-like disorders after infecting the major replication sites, which are the intestinal or respiratory epithelium. It can also infect other organs, leading to CNS-related diseases such as temporary paralysis, encephalitis, meningitis, and other conditions. The majority of these incidents involve younger children than three. Months at the ages of 10 and 15. Long-term neurological consequences such as neurodevelopmental delays, impairment in auditory skills, or delay in gross motor function are observed in addition to these acute clinical manifestations. Although PeV-A1 and A3 exhibit notable variations in short- and long-term morbidity, the underlying causes of these variations are yet unknown.

This genotype-specific variation in illness may be explained by PeV-A3's propensity to infect (other) CNS cell types as opposed to PeV-A1. In a brain cell line (SH-SY-5Y), we previously observed that PeV-A3 strains had faster replication kinetics than PeV-A1 strains. Distinctions in the structure of the receptor-binding area might be another reason. An arginyl-glycyl-aspartic acid motif is present in the VP1 of PeV-A1, but not in PeV-A3. This motif allows PeV-A1 to attach to integrins that are linked to cell membranes indicating that PeV-A1 and -A3 use distinct receptors to allow entrance. A variation in cell tropism and consequent illness might result from this uneven utilization. However, it has recently been shown that the entrance receptor for both genotypes A1 and A321 is a host membrane protein called Myeloid-Associated Differentiation Marker, or MYADM.

Finally, we observed that, in primary Human Airway Epithelial (HAE) cultures22, PeV-A3 infection elicited a greater inflammatory response than PeVA1 infection. The variations in PeV-A1 and PeV-A3-induced CNS illness may potentially be explained by the changes in genotype-specific inflammatory responses.

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