Anti-Rhesus Rotavirus VP4 (Outer capsid protein VP4) (Hemagglutinin) Polyclona Antibody

Category: Polyclonal Antibody

Catalog #: AB3H193

Species Reactivity: Monkey Rhesus rotavirus

Immunogen/Specificity:
Polyclonal antibody produced in rabbits immunizing with a synthetic peptide corresponding to near N-terminal residues of Monkey Rhesus rotavirus VP4 (Outer capsid protein VP4) (Hemagglutinin)

Description: VP4 (Outer capsid protein VP4) (Hemagglutinin) functions as a spike-forming protein that mediates virion attachment to the host epithelial cell receptors and plays a major role in cell penetration, determination of host range restriction and virulence. It is subsequently lost, together with VP7, following virus entry into the host cell. Rotavirus attachment and entry into the host cell probably involves multiple sequential contacts between the outer capsid proteins VP4 and VP7, and the cell receptors. In sialic acid-dependent and/or integrin-dependent strains, VP4 seems to essentially target sialic acid and/or the integrin heterodimer ITGA2/ITGB1. VP4 is a homotrimer. VP4 adopts a dimeric appearance above the capsid surface, while forming a trimeric base anchored inside the capsid layer. Only hints of the third molecule are observed above the capsid surface. It probably performs a series of molecular rearrangements during viral entry. Prior to trypsin cleavage, it is flexible. The priming trypsin cleavage triggers its rearrangement into rigid spikes with approximate two-fold symmetry of their protruding parts. After an unknown second triggering event, cleaved VP4 may undergo another rearrangement, in which two VP5* subunits fold back on themselves and join a third subunit to form a tightly associated trimer, shaped like a folded umbrella. VP4 interacts with host ITGA2 (via ITAG2 I-domain); this interaction occurs when ITGA2 is part of the integrin heterodimer ITGA2/ITGB1. VP4 interacts with host integrin heterodimer ITGA4/ITGB1 and ITGA4/ITGB7. Proteolytic cleavage by trypsin results in activation of VP4 functions and greatly increases infectivity. The penetration into the host cell is dependent on trypsin treatment of VP4. It produces two peptides, VP5* and VP8* that remain associated with the virion.

Reference:
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Gilbert,J.M. and Greenberg,H.B., J. Virol. 72 (6), 5323-5327 (1998)
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