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Identification and Intracellular Location of Poliovirus Eclipse Particles and the Influence of Chloroquine and R 78206. R. Vrijsen, P. Kronenberger, O. Ofori-Anyinam & A. Boeyé. Dept. of Microbiology & Hygiene, Vrije Universiteit Brussel, Brussels, Belgium

Early in infection, poliovirions (160 S) are modified to 135 S and 110 S eclipse particles, both particles retaining RNA. The 135 S particles appeared first, 110 S particles later. In addition, variable amounts of 80 S eclipse particles without RNA were formed. The location of these eclipse products in infected cells was examined by analysing a post-nuclear homogenate on isoosmotic rate-zonal and isopycnic Nycodenz gradients. Four subcellular fractions were obtained : 1) plasma membrane, 2) endosome-like intracellular vesicles, both containing almost exclusively 160 S intact virions, 3) a lysosomal fraction containing 135 S, 110 S and 80 S particles, and 4) a cytosolic fraction containing 135 S and 80 S particles. Compounds interfering with the early stages of infection were examined. The lysosomotropic agent chloroquine (50 μM) redirected the production of 135 S and 110 S particles to 80 S empty capsids, without affecting virus replication. The 80 S particles were found in the lysosomal and cytosolic fractions. Further experiments suggested that chloroquine accelerated the release of viral RNA from 135 S particles, resulting in the formation of 80 S particles. The new antiviral compound R 78206 (Janssen Pharmaceutics) inhibited the formation of eclipse products, probably by stabilizing the structure of the intact virions. Results showed that the virions were transported from the plasma membrane into endosomes-like vesicles, but not into lysosomes, indicating that R 78206 inhibited transfer of virions to the lysosomes, possibly as a result of capsid stabilization.

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Aryl-α-D-Galactosaminides Inhibit the Ability of Rotavirus to Bind to and to Infect Susceptible Cells. James H. Gilbert and Mary E. Schaefer. Glycomed, Inc., Alameda, CA 94501.

We have examined the effect of aryl-α-D-N-acetyl-galactosaminides on the ability of rotavirus to infect susceptible tissue culture cells. MA104 cells were incubated with aryl-α-D-N-acetylgalactosaminides until confluent monolayers formed. The infectivity of SA11 rotavirus on treated and untreated cells was measured by plaque assay. Treated cells showed markedly fewer plaques than untreated cells. The inhibition is concentration dependent, and requires the α-anomeric form of the N-acetylgalactosamine. The aryl-α-anomers of glucose and N-acetylglucosamine are inactive, as are aryl-β-anomeric sugars. The ability of rotavirus to bind to these cells was measured using ^{35}S labelled SA11. Incubation of MA104 cells with aryl-α-D-N-acetylgalactosaminides resulted in a significant decrease in the amount of bound virus, suggesting that the rotavirus receptor may be modified or present at lower levels. The general physiologic condition of cells treated with benzyl-α-D-N-acetyl-galactosaminide was studied by determining cell growth curves and the ability of treated cells to incorporate ^{3}H-threonine, ^{3}H-mannose, and ^{3}H-glucosamine. ^{3}H labeled benzyl-α-D-N-acetyl-galactosaminide was employed to determine if treated cells secrete oligosaccharides containing the benzyl-α-D-N-acetyl-galactosaminide into the medium, similar to mucin producing cells treated with aryl-α-D-N-acetyl-galactosaminides (Kuan, *et al*, Jour. Biol. Chem. **264**: 19271-19277, 1989). Our results suggest that the rotavirus receptor on MA104 cells is glycosylated by a mechanism similar to mucin.