Sulfoglycosphingolipid-mediated regulation of influenza A virus replication

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Influenza A viruses (IAVs) infect many animal species including birds, pigs, horses, and human. The interaction between the viruses and host cells is mediated by a viral spike glycoprotein, a hemagglutinin that recognizes glycoproteins and glycolipids containing terminal sialic acid. IAV infection is initiated by attachment of virus to terminal sialic acid. Previously, we have shown that sulfoglycosphingolipid can bind to IAV particles. Sulfoglycosphingolipid is abundantly expressed in various mammalian organs, including the intestine and trachea, in which IAVs replicate. However, the function of sulfoglycosphingolipid in IAV infection remains unknown. We found by using genetically produced sulfoglycosphingolipid-knockdown or enriched cells that sulfoglycosphingolipid regulates IAV replication. The development of antiviral drugs will probably play a critical role in the next influenza pandemic. NA inhibitors are currently used for protection against and treatment of influenza. However, the emergence of drug-resistant viruses has become a serious problem. Our findings provide new insights into IAV replication and suggest new therapeutic strategies.