Rabies virus protein P5 regulates virus replication via beclin 1-mediated autophagy

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Video Byte

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Abstract

Rabies causes severe neurological symptoms and has a high mortality rate, killing over 59,000 people each year. Rabies virus encodes five phosphoproteins (P) that are involved in viral genomic replication, transport, oxidative stress, interferon antagonism, and autophagy induction. Unfortunately, the specific functions of each P protein are poorly understood. A recent study zeroed in on the viral protein P5. Using cell lines, researchers found that P5 attaches to the N-terminal residues of the protein beclin1 (BECN1). P5 binding to the ring-like structure of BECN1 induced incomplete autophagy due to incomplete fusion with lysosomes. This process promoted rabies virus replication by increasing the levels of viral proteins. Silencing the gene for beclin1 did not reproduce this pattern of P5-induced viral replication. This study identifies a novel role for P5, whereby P5 binding to the BECN1 ring promotes viral replication by inducing incomplete autophagy. This pathway may be an ideal target for antiviral drugs against rabies virus.