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A Critical Phenylalanine Residue in the RSV Fusion Protein Cytoplasmic Tail is Necessary for Viral Assembly into Filaments and Budding of Internal Virion Proteins into Virus like Particles

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Abstract

Respiratory syncytial virus (RSV) is a single-stranded RNA virus in the Paramyxoviridae family that assembles into filamentous structures at the apical surface of polarized epithelial cells. These filaments contain viral genomic RNA and structural proteins, including the fusion (F) protein, matrix (M) protein, nucleoprotein (N), and phosphoprotein (P), while excluding F-actin. It is known that the F protein cytoplasmic tail (FCT) is necessary for filament formation, but the mechanism by which the FCT mediates assembly into filaments is not clear. We hypothesized that the FCT is necessary for interactions with other viral proteins in order to form filaments. In order to test this idea, we expressed the F protein with cytoplasmic tail (CT) truncations or specific point mutations and determined the abilities of these variant F proteins to form filaments independent of viral infection when coexpressed with M, N, and P. Deletion of the terminal three FCT residues (amino acids Phe-Ser-Asn) or mutation of the Phe residue resulted in a loss of filament formation but did not affect F-protein expression or trafficking to the cell surface. Filament formation could be restored by addition of residues Phe-Ser-Asn to an FCT deletion mutant and was unaffected by mutations to Ser or Asn residues. Second, deletion of residues Phe-Ser-Asn or mutation of the Phe residue resulted in a loss of M, N, and P incorporation into virus-like particles. These data suggest that a C-terminal Phe residue in the FCT mediates assembly through incorporation of internal virion proteins into virus filaments at the cell surface.

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