

Functional Interactions Between Human, Bacterial Neuraminidases and Influenza A Viruses

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Respiratory tract infections are responsible for 3 to 5 million deaths each year worldwide. Respiratory co-infections, involving multiple pathogens simultaneously or sequentially, involve complex host-pathogen interactions whose mechanisms remain poorly understood. In particular, the role of sialic acids and microbial neuraminidases has been little explored in viral/bacterial co-infections. This study investigate the role of the human neuraminidases NEU1 and NEU3 and the bacterial neuraminidases NanA and NanI in influenza A(H1N1) and A(H3N2) virus infection. Using different infection scenarios in the A549 cells line, we identified functional interactions between influenza A viruses and NEU1/NEU3. Furthermore, experiments performed in a reconstituted human airway epithelium model revealed a dual role for bacterial neuraminidases during influenza co-infection. Indeed, administration of NanA or NanI recombinant proteins before or early after infection inhibits viral production, whereas post-infection treatment promoted A(H1N1) spread. Our findings highlight the role of human and bacterial neuraminidases throughout the influenza A virus life cycle and suggest multiple functional interactions between virus, bacteria, and host during co-infection. These observation provide new insights into respiratory co-infection mechanisms and open new avenues for the development of broad-spectrum antimicrobial strategies.

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PhD student

Junior scientist status

Yes, I am a junior scientist.

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