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# Influenza A virus NS1 targets the host ubiquitin ligase ZNF598 to promote innate immune evasion

## Inhalt

RIG-I-mediated sensing of viral RNA initiates type I interferon (IFN-I) expression, which is essential for innate immune defense. Because aberrant activation of this pathway can lead to autoimmune disorders, RIG-I signaling is tightly regulated. The E3 ubiquitin ligase ZNF598 indirectly suppresses IFN-I expression by conjugating ubiquitin D (FAT10) to RIG-I, thereby inhibiting its activation and attenuating the antiviral response. During its infectious cycle, Influenza A virus (IAV) exploits ubiquitin-mediated mechanisms to promote viral replication. The non-structural protein NS1 plays a key role in counteracting the host antiviral response. Preliminary work identified ZNF598 as a conserved interactor of NS1 from multiple IAV strains. Here, we characterize the interaction between ZNF598 and NS1 from three distinct IAV strains. We also investigate the impact of NS1 on ZNF598-dependent RIG-I FAT10ylation.

## Keywords

ZNF598, NS1, RIG-I, FAT10

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## Professional status of the speaker

PhD student

## Junior scientist status

No, I am not a junior scientist.

**Track Klassifizierung:** Innate immunity

**Typ des Beitrags:** Poster presentation