

Influenza A virus (IAV) NS1 mediated host gene shut off takes place in nuclear speckles

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IAV-NS1 proteins are known to inhibit host gene expression. In both transfection and infection experiments a strong attenuation of reporter gene expression is observed when NS1 proteins are fused to protein domains guiding NS1 exclusively to nuclear speckles (nsps). IAV H7N7 SC35M NS1 protein that is fused to domains, that guide it to nuclear or non-nuclear compartments other than nsps, show little or no ability to attenuate reporter gene expression. A nsp-localised NS1-effector domain is sufficient to inhibit gene expression. The protein SON is an essential component of nsps. In cells treated with siRNA targeted against SON the ability of NS1 to suppress the expression of a reporter gene is reduced relative to cells with fully functional nsps. Lastly, we demonstrate that the NS1 mediated suppression relies on transcriptional inhibition. Our data suggest that IAV-NS1 suppresses nuclear speckle promoted gene expression by inhibition of transcription.

Reportedly the f2f3 domain of CPSF30 binds to NS1. The f2f3 domain will be fused to domains that guide it to nuclear or non-nuclear compartments. In reporter gene assays we try to link subcellular localisation of the NS1 binding recombinant f2f3 domain with inhibition of host gene expression and/or APA (alternative polyadenylation of host transcripts) to investigate whether CPSF30-NS1 interaction takes place in nsps and how this will affect APA and reporter gene transcription.

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No, I am not a junior scientist.

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