Abstract Preview - Step 3/4

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Topic: Workshop 1 Innate Antiviral Immunity and Viral Immune Evasion

Alternative Topic: Workshop 2 Restriction Factors of Viral Infection

Title: **Human Cytomegalovirus pp65 inhibits Interferon type I production through its interaction with the cGAS/STING axis**

Author(s): Pautasso S.[1], Biolatti M.[1], Dell'Oste V.[1], Gugliesi F.[1], Gariglio M.[2], De Andrea M.[1], Landolfo S.[1]

Institute(s): [1]*University of Turin, Turin, Italy,* [2]*Novara Medical School, Novara, Italy*

Text: The innate immune response against Human Cytomegalovirus (HCMV) plays a pivotal role during primary infection. Indeed, HCMV infection of primary fibroblasts rapidly triggers a strong induction of interferon-type I (IFN-type I), accompanied by proinflammatory cytokines release. This tightly regulated defense system serves to establish an antiviral state in infected and neighbouring cells and protect them against virus replication. Here, we show that primary human foreskin fibroblasts (HFFs) produce IFN-type I when infected with HCMV strain TB40/E, v65Rev. Interestingly, significantly higher IFN-type I levels are observed when HFFs are infected with HCMV unable to express UL83-encoded pp65 (v65Stop), suggesting that the tegument pp65 protein might downregulate IFN-type I production. To clarify the mechanisms pp65 relies on to inhibit IFN-type I production, we analysed the activation of the cGMP-AMP synthase (cGAS)/STING axis in HFFs infected with the v65Rev or v65Stop. The results obtained revealed that pp65 binds to cGAS and prevents its interaction with STING thus interfering with the cGAS/STING axis. These data identify a previously unknown role for pp65 that leads to inhibition of IFN-type I production and evasion from its antiviral activity.

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