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Influenza A virus and its reliance on host cell metabolism

Inhalt

Influenza A viruses (IAVs) rely heavily on host cell metabolism for productive replication. Several studies have demonstrated that IAVs actively modulate host cellular processes. It has already been shown that suppression of glucose metabolism inhibits viral replication. Interestingly, inhibition of glycolysis disrupts IAV replication by impairing viral polymerase function resulting in reduced synthesis of viral genomic RNA. This raises the question of whether this is a glycolysis-specific effect or a general consequence of metabolic inhibition. There is limited knowledge about how IAV polymerase regulation interacts with metabolic pathways. Here, we demonstrated that inhibition of the main metabolic pathways glutaminolysis, oxidative phosphorylation (OXPHOS), fatty acid synthesis (FAS), and pentose phosphate pathway led to a reduction of viral titers. Moreover, inhibition of glutaminolysis, FAS, and OXPHOS led to an imbalance in the cellular glycolysis and respiration networks, with an extended period of viral transcription and a sharp decline in replication. Furthermore, we investigated effects of the tricarboxylic acid cycle intermediate oxaloacetate on viral replication and showed that it nearly fully reversed the glycolysis inhibition mediated reduction on viral titers. Our results suggest that the regulation of IAV replication is closely linked to the cellular metabolic state, showing the potential of modulating metabolism as a host-targeted antiviral strategy.

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Yes, I am a junior scientist.

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