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Exploiting lipid metabolism by HSV-1 – a challenge to rethink new therapies for Alzheimer's disease

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Herpes simplex virus-1 (HSV-1) establishes a life-long latent infection and can enter the brain via retrograde axonal transport. Recurrent reactivation of HSV-1 may lead to neurodegenerative disorders, including Alzheimer"s disease (AD), although the underlying mechanisms have not been fully elucidated yet. Lipids constitute the bulk of the brain dry mass and alteration of lipid metabolism is a key component in AD.

Considering that the mechanisms for remodeling of metabolism by HSV-1 are still poorly understood, we aim at dissecting the host metabolic pathways modulated by infection in a neuronal-like cell line to identify pathways that might be targeted to prevent AD.

Specifically, we found an increase in both de novo synthesis and lipid storage following HSV-1 infection. In addition, anti-AD compounds targeting lipid metabolism (*e.g.* CMS121, C75) impaired HSV-1 infectivity.

Overall, our data unveil new aspects of HSV-1-AD interplay and uncover new potential targets to rethink new possible therapies.