Infectious Disease

Herpes and Cell Metabolism: The Zwezdaryk Lab

Herpes viruses are extraordinarily common in humans, with most of the population testing positive by adulthood. Three different viruses in the beta herpesvirus family can have seropositive rates of up to 70-90% in humans. Though these viruses are often thought of as being harmless in people, they can have severe negative effects in immunocompromised individuals such as during organ transplants. Additionally, herpesvirus infections that occur during pregnancy can cause the same array of fetal and infant symptoms seen in zika infection. The lab of **Kevin Zwezdaryk** is studying how infection with herpesvirus can alter host cell metabolism.

Betaherpesvirus Biology

With over 200 open reading frames, betaherpesviruses are some of the largest viruses known to infect humans. In addition to having such a high number of genes, herpesviruses have evolved multiple mechanisms to infect different cell types. Betaherpesviruses ability to manipulate host cell pathways in multiple ways presents a difficult obstacle to vaccine or drug development. The Zwezdaryk Lab is using a comprehensive -omics approach, taking multiple time points from infected samples, to investigate the metabolic changes in host cells.

Betaherpesvirus, Metabolism, and Mitochondrial Function

Betaherpesvirus infection has profound effects on host cell metabolism. Infection with the virus induces a Warburg-like effect on cells. This means that cells switch their energy source from oxidative phosphorylation (which occurs at the mitochondria) to glycolysis. The lab is using a systems biology "-omics" approach. This includes integrating metabolomics (LC-MS/MS), lipidomics (HPLC), transcriptomics (RNAseq) and proteomics to examine the role of the mitochondria to altered cellular metabolism during herpesvirus infection.

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Herpesvirus, Metabolism...(cont.)

The lab postulates that the virus is able to turn the mitochondria into a factory for biomolecular intermediates during infection. They have observed increased electron transport chain activity during infection, which leads to an accumulation of not only intermediates, but also reactive oxygen species. Normally, this level of reactive oxygen species would be extremely damaging to the cell's (and virus') DNA.

By exploring how the virus deals with this high abundance of reactive oxygen species, the lab intends to understand how this allows the cell and viral genetic material to be protected despite high electron transport activity. Illuminating the relationship between the cell metabolism and viral infection in this way will lead to the discovery of novel targets for advanced, specific, and efficacious antiviral treatments for herpesvirus infection.

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