Infectious Disease

HIV in the CNS: The MacLean Lab

HIV infection can lead to a number of CNS symptoms and complications. Additionally, it is postulated that the CNS, especially the brain, can act as a reservoir for virus. **Andrew "Manus" MacLean** is a cell biologist with a background in blood-brain barrier biology who is studying how the virus interacts with and infects the cells of the CNS and the resultant pathological consequences. The MacLean lab studies HIV and other viruses in the CNS using a mixture of advanced 3D cell culture techniques and non-human primate models.

Modeling HIV Infection in Glial Cells

Glial cells are responsible for maintaining neurons and helping to protect the CNS and can be infected by HIV. The MacLean Lab uses mathematical modeling to interrogate the interactions between different cell types in the CNS. By employing both *in vivo* and *in vitro* systems, the lab examines how HIV reduces the connectivity between microglial cells and astrocytes. This work is expected to prove that this is one root cause of the neurological symptoms seen in patients living with HIV.

HIV Infection, THC, and Opioids

The Lab is also interested in the effects of different pharmacological agents in HIV infection. Using non-human primate models and SIV, the lab is investigating the possible protective effects of THC during HIV infection. It has been demonstrated that while THC treatment makes the astrocytes "look" phenotypically worse under the microscope, it actually provides neuroprotective benefits. Without antiviral treatment, 1/3rd of the macaques infected with SIV will get encephalitis, mirroring conditions like "AIDS dementia" seen in untreated human patients.

HIV Infection, THC, and Opioids (cont.)

The MacLean Lab is also investigating the role of opioids in HIV infection. It is known that endogenous opioid signaling is able to produce replication-competent virus from infected astrocytes. The same effect has been observed using exogenous opioids, in stark contrast to what usually happens when a virus infects astrocytes (i.e.: that infected cells become senescent and do not produce virus).

Instead, the virus does not integrate into the host genome. Opioid signaling somehow activates the astrocytes, making them capable to shedding replication-competent virus. Given the high degree of drug abuse observed in some subpopulations of patients living with HIV, this avenue of research has special relevance in the context of the current opioid crisis. The MacLean lab is employing advanced co-culture and animal models to tease out the molecular mechanisms behind this viral activation.

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