Meghan Holdorf, PhD [1]

Co-Mentor: Qianting Zhai, PhD

Infectious Diseases

Emeryville, California, United States
Our group is focused on discovering novel antiviral mechanisms involved in regulating persistent and chronic viral infections such as the chronic Hepatitis B virus. Persistent and chronic DNA viruses in particular have developed many mechanisms to both manipulate and co-exist within the nucleus, evading the host immune system. During acute infections, CD8+ T cells eliminate virus-infected cells through direct killing and the production of antiviral cytokines; however, this is not achieved during chronic viral infections. In patients chronically infected with Hepatitis B virus, there is evidence showing that this effect is due to the loss of virus-specific CD8+ T cell function, though the mechanism(s) responsible for the CD8+ T cell failure is still not fully understood. Virus-specific CD8+ T cells isolated from the peripheral blood of chronically HBV-infected patients fail to proliferate, produce cytokines, and show elevated expression of checkpoint inhibitory molecules, a status defined as exhaustion. This CD8+ T cell dysfunction is hypothesized to be attributed to the high levels of persisting viral antigens composed of circulating soluble HBV surface antigen (HbsAg) and HBV e antigen (HbeAg). A better understanding of the mechanisms leading to viral persistence and T cell dysfunction is critical to guiding new therapeutic strategies aimed at treating such diseases.

Selected Publications

Site-specific association with host and viral chromatin by Kaposi’s Sarcoma-associated herpesvirus LANA and its reversal during lytic reactivation. [2]
Mercier A, Arias C, Madrid AS, Holdorf MM, Ganem D.

Occupancy of chromatin organizers in the Epstein-Barr virus genome. [3]
Holdorf MM, Cooper SB, Yamamoto KR, Miranda JJ.

Click here [4] for additional publications.

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