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Viral Mechanisms of Persistence and Pathogenesis

My laboratory investigates the mechanisms of rhesus cytomegalovirus (RhCMV) persistence and pathogenesis to develop clinically relevant strategies that can prevent and/or treat human cytomegalovirus (HCMV) disease. My lab developed the rhesus macaque model of HCMV to enable studies in nonhuman primates that translate to human clinical trials. Our primary research focus is to identify viral determinants of latency, persistence, and pathogenesis. Our goals include (1) define RhCMV mechanisms of persistence, (2) characterize determinants of pathogenesis, (3) identify protective immune responses, (4) design novel strategies that prevent RhCMV infection and disease, and (5) develop reagents for clinical trials in humans.

Our data validate rhesus macaques as a relevant animal model to study how chronic viral infections modulate host immunity and impact aging of the immune system, potentially leading to new therapies promoting healthy aging in humans.

This figure (adapted from Chang and Barry, Proc Natl Acad Sci 107:22647, 2010) illustrates the effects of how HCMV weakens our immune response when humans are infected with this virus. HCMV expresses a viral protein (cmvIL-10) that suppresses our immune response to the virus. This viral process greatly complicates the strategies for HCMV vaccines that could limit development of a protective vaccine against congenital HCMV infection.

Deere JD and Barry PA. Using the Nonhuman Primate Model of HCMV to Guide Vaccine Development. Viruses 6:1483-1501, 2014. PMC4014706
Deere JD, Chang WL, Castillo LD, Schmidt KA, Kieu HT, Renzette N, Kowalik T, Barthold SW, Shacklett BL, Barry PA, and Sparger EE. Utilizing a TLR5-adjuvanted cytomegalovirus as a lentiviral vaccine in the nonhuman primate model for AIDS. PLoS One 11:e0155629, 2016. PMC4868283

To contact Dr. Peter Barry and for more information on his research, see: http://www.cnprc.ucdavis.edu/peter-barry/