Editorial

CMV- the enemy within, time for eradication

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Cytomegalovirus (CMV) is the most infectious agent globally, belongs to the Herpesviridae family, with an infection rate 80-90%.[1] The public health annual burden in the US alone of CMV infection and co-infection was estimated to exceed $4.4 billion. CMV infects a wide array of cells with the ability to produce lifelong latent infection, where CMV DNA is present, but viral replication or shedding is not detectable, and also to reactivate from latency through unknown mechanism(s). The latency reservoir of human CMV and the reactivation process has been difficult to identify due to many reasons including its inability to infect other species.[2]

CMV is genetically diverse large virus with more than 200 open reading frame producing effector proteins. A large portion of the human CMV genome encodes gene products that evade or interfere with host immune responses in ways that facilitate the persistence and dissemination of CMV in the host.[3]

CMV infection causes severe disease with high morbidity and mortality in immunocompromised patients, and is the predominant infectious cause of congenital birth defects. CMV infections might play a pivotal pathogenetic role in the development of several chronic diseases such as SLE and RA, etc.[4] To date there is no successful treatment for the viral eradication or even a safe vaccine.

In its complex battle for survival, CMV encodes a wealth of genes capable of evading host innate and adaptive immune responses. Immuno-competent host is usually able to clear the infected cells, with the virus is able to persist for the life of the host, without causing active disease (latency). Recent studies suggested that this latent state is due to epigenetic factors control, which causes hetero chromatinization of the CMV genomes to shut off the viral gene expression. CMV therefore is able to survive indefinitely in these cells invisible to the host immune response. Transcriptional repression may also be a result of an intrinsic immune response. CMV is therefore able to survive in the face of host immunity, leading to latency.[5]

CMV reactivation may then occur in response to an infection with other pathogen, or an inflammatory response, which would then compromise the host immune system. Indeed, reactivation or gene expression of CMV has been shown to be a result of inflammatory mediators such as IL-6 and TNF through unknown specific signaling pathways. Interestingly enough, IL-6 has been shown recently to play an enigmatic role in immunometabolism,[6] and its role in CMV replication is unknown. There are more work needed to elucidate the epigenetic mechanism(s) that control CMV latency and reactivation. Understanding those mechanisms, may lead to new therapeutics capable of eradicating latent CMV or at least prevent its reactivation.[5-7]

CMV vaccine has been on the top highest priority of the Institute of Medicine of the National Academy of Sciences for vaccine development. Large number of CMV vaccines is in clinical trials for clinical evaluation, including live, attenuated strains of CMV, in addition to CMV envelop proteins and DNA subunits. Current status of CMV vaccine clinical trials and passive immunization with anti-CMV immunoglobulin has been reviewed recently. Ongoing translational and clinical research will hopefully lead to the development of effective and safe therapeutics and vaccine in the near future.

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Conflicts of interest

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References

1. Kotton CN. CMV: Prevention, Diagnosis and Therapy. *Am J Transplant*. 2013;3:24–40.
2. Varani S, Landini MP. *Cytomegalovirus*–induced immunopathology and its clinical consequences. *Herpesviridae*. 2011;2(1):6.
3. Swanson EC, Schleiss MR. Congenital cytomegalovirus infection: new prospects for prevention and therapy. *Pediatr Clin North Am*. 2013;60(2):335–349.
4. Rozenblyum EV, Allen UD, Silverman ED, et al. *Cytomegalovirus* infection in childhood–onset systemic lupus erythematosus. *Int J Clin Rheumtol*. 2013;8(1):137–146.
5. Liu XF, Wang X, Yan S, et al. Epigenetic control of cytomegalovirus latency and reactivation. *Viruses*. 2013;5(5):1325–1345.
6. Pal M, Febbraio MA, Whitham M. From cytokine to myokine: the emerging role of interleukin–6 in metabolic regulation. *Immunology and Cell Biology*. 2014;92:331–339.
7. Schleiss MR. *Cytomegalovirus* in the neonate: immune correlates of infection and protection. *Clin Dev Immunol*. 2013.