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Chronic alcohol consumption exacerbates mCMV infection via impairing NK cell function in mice

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Abstract

Alcohol use disorder is a serious public health problem that – as of 2010 – is estimated to affect over 200 million people worldwide or greater than 4.0% of the world's population. Chronic Alcohol Consumption (CAC) is one of several established factors that contributes to a patient's susceptibility and treatment of infectious diseases by disrupting the immune system. Natural killer (NK) cells are one part of the innate immune system that form the body's first line of defense against infectious disease, particularly viral infections. However, only mature NK cells that have followed their developmental pathway are capable of effectively detecting and eliminating virally infected cells. CAC disrupts this development leading to reduced numbers of mature NK cells and reduced cytotoxicity. Murine cytomegalovirus (MCMV) is a member of the β -herpesvirinae family of viruses that integrates into the host DNA for long term latency. CMV infection typically does not present significant symptoms in immunocompetent individuals but clinical complications can result in immunocompromised patients such as alcoholics. Our preliminary research to date has shown that there are significant differences to the NK cell response in terms of IFN- γ production and splenic viral loads between water and alcohol drinking mice. Future projects will delineate the progression of the NK cell response in the short and long term to determine the full extent of CAC's effect on the NK response to MCMV.