

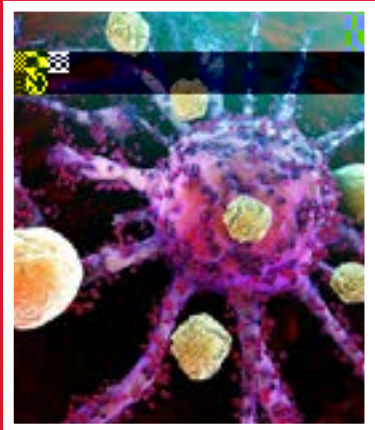
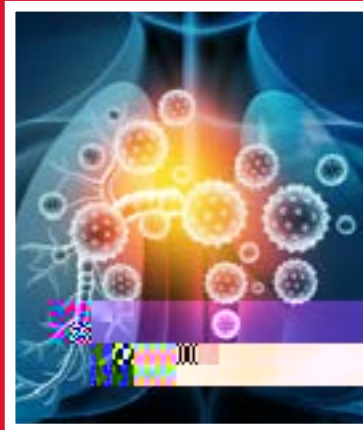
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Human endogenous retrovirus K102 (HERV-K102) is a replication competent protector foamy retrovirus located on chromosome 1 and is unique to humans. It appears to be a crucial component of the innate anti-viral response launched in macrophages in response to viruses which renders the activated macrophages foamy (FM). Various lines of evidence suggest the HERV-K102 innate defense system may be critical to host survival against emerging RNA viruses. However, the release of HERV-K102 particles from FM can be impeded by age or stress called immunosenescence (IMS). IMS is a well-established clinical risk factor for COVID-19 severity. It has been suggested IMS involves the reduced inactivation of alpha-fetoprotein (AFP) by declining levels of DHEA associated with age and/or stress. Newer evidence suggests SARS-CoV-2 may specifically target the innate anti-viral response and uniquely disrupts foam cell formation by blocking the mevalonate pathway, in addition to the deregulation of IL-6. The molecular pathways common to IMS and COVID-19 pathogenesis appear to converge on the glucocorticoid receptor (stress) regulatory network and the IL6ST (trans-signalling IL-6 receptor) inflammatory pathway both which involve active AFP and IL-6. On the other hand, CCR5 receptor blockade has shown promising clinical efficacy against COVID-19

