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Research on interaction of HIV and the human innate immune system:

Of the 6.7 billion people worldwide, around 33 million people are infected with the human immunodeficiency virus (HIV). This is half of one percent of worldwide human population. Untreated, the virus limits the life expectancy of an infected individual to typically less than ten years. Due to the failures of many strategies that have been exerted in the hope of developing a treatment or a vaccine for the disease, the current researches have been modified from being data driven to knowledge driven. This current way of thinking calls for intervention that is focused on the interaction between the HIV and the human immune system.

Apolipoprotein B mRNA editing enzyme, catalytic polypeptide-like (APOBEC) is a protein family that interferes with the replication of the HIV virus and other retroviruses by altering the genomic sequence of the virus. The virus, to combat this, has evolved a countermeasure in the form of a protein called Viral Infectivity Factor (vif) that can bind to APOBEC and reduce its effectiveness. One outcome of APOBEC altering the genomic sequence of the HIV virus is that the immune system becomes unable to recognize the virus and therefore the virus cannot be eliminated from the immune system. Another outcome is that the severe genomic alteration of the virus causes the virus to become mutated making it incapable of producing proteins and leading to the virus,Äô death. Thus, depending on the extent of the genomic sequence of the HIV virus that was altered by APOBEC, it can result in being beneficial or detrimental to the proliferation of the virus.

Since data are available about the pattern of APOBEC alteration of HIV genomic sequence, using quantitative statistical techniques one can measure to what extent the HIV genomic sequence can be altered by APOBEC, and hence determine when the alteration can be beneficial or detrimental to the proliferation of the virus in the human organism. This research naturally has bearing on drugs that are designed to deactivate vif in an attempt to control the proliferation of the virus.