Background

Cromer et. al. (2010) recently presented their findings titled 'How fast could HIV change gene frequencies in the human population.' They postulated that infectious disease my act as a strong force in selection with their host population. A potentially strong genetic selection candidate is Human Immunodeficiency Virus (HIV), owing to its ability to infect large numbers of humans. HIV has varying susceptibility due to different human leucocyte antigen (HLA) types that make HIV disease progression less tractable. In the human population the genetic variability can alter the rate of HIV infection as well resistance to the virus. This variability between the host and virus are continually providing an environment for applied selective pressures.

In the human population an association exists between individuals carrying different HLA alleles. Specifically, individuals that are homozygous for HLA class I loci, *HLA-B*35* and *HLA-B*53* have a tendency to develop acquired immunodeficiency syndrome (AIDS) faster than those individuals who are heterozygous (Carrington et al. 1999; O'Brien *et al.* 2001). Conversely, it has been demonstrated that individuals possessing *HLA-B*57* and *HLA-B*27* have a much slower progression towards developing AIDS (Carrington & O'Brien 2003). Genetic variability also seems to play a correlation in the time it takes to develop AIDS. In individuals who possessed both alleles *HLA-B*35* and *HLA-B*53* progressed to AIDS in 6 years, as opposed to 11 years for white subjects that lacked these alleles. Black subjects tended to progress marginally slower at 8 and 11 years with and without alleles, respectively (Gao *et al*. 2001).

As was the case for susceptible in individuals with specific alleles, resistance to HIV must also be present in a given population. One such allele has provided this evidence, the *CCR5*-Δ*32* deletion allele has been showing to provide resistance to HIV (Stephens *et al*. 1998). It is currently unknown what HIV's selective pressure is on the human population. One such factor for this pressure is the changes in the populations' behavior in respect to the virus's transmission. In certain sub-populations in different African countries there has been a noticeable decrease in the number of people infected (Stoneburner and Low-Beer 2004; Cheluget *et al.* 2006). This reduction has been brought about by an increase in the number of people having sex only with one partner, and practicing safer sex through condom usage. However, in certain areas of South Africa the prevalence of HIV infections still remains quite high especially in Eastern Cape Province where infection rates are as high as 30% (WHO 2008*b*).

This high rate of continual exposure to HIV may give rise to selection for protective alleles reduce the transmission of alleles the increase disease transmission and AIDS. Given the idea the HIV may exert strong selective pressure (Cromer et al. 2010) set out to model and test if the virus has already caused selection against HLA types that increase disease or death from HIV and AIDS.

Our presentation will present the initial models used in this study and outline some of their general findings. We will also present a brief overview on the pathology and general background on the virus. Lastly, we will provide some future directions for this research and present simulated data from this paper.

References

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