3. Viral dynamics and phylogeny -- a finite site model for viral evolution.

（ウイルスの宿主内抗原ドリフト進化ー有限抗原サイトモデル）

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We theoretically study the antigenic drift of viruses within an infected host, as observed in human immunodeficiency virus (HIV) and equine infectious anemia virus (EIAV) infections, assuming that a finite number of antigen determining sites at viral envelop gene are responsible for the specific immune response. The pattern of antigen evolution becomes more complex than that predicted from the previous one-dimensional antigen space models. If the viral growth rate is sufficiently large, the demographic stochasticity for the fate of a new antigen mutant can be neglected. The high dimensionality in the way a virus escape the immune defense in genotype space could then causes a rapid increase in the antigenic diversity and the total viral density, until finally the whole antigen genotypes are used up. The viral population is then driven to extinction in a host by the enhanced immune response to all genotypes. In contrast, if the viral growth rate is moderate or small so that only a small fraction of new antigen mutants can survive during the initial endangered period of random extinction, the viral antigenic diversity and the total density remain bounded, thereby enabling them to persist for a prolonged period by shifting the dominant antigen types. The phylogenetic pattern of antigen divergence is well characterized by the mean number of surviving antigen mutants from an antigen genotype. The substitution rate at antigen determining sites increases as the efficiency of host immune response increases. An extended model to include the effect of target cell limitation is discussed.