Modelling with-in host trypanosome infections: the interplay of ecology and genetics(*)

African Trypanosomes are among the many parasites that exhibit antigenic variation as a mechanism to sustain chronic infections within their hosts.

The parasite obtains protection against specific antibody immune responses by switching of expression between distinct parasite variants. Typical trypanosome infections consist of an oscillating parasitaemia, where each peak is composed of a group of distinct variants. The characteristics of the parasitemia profile arise from a fine interplay between parasite differentiation, variant-specific immune control and the magnitudes of variant switch rates, a property related to the parasite genome. We construct a mathematical model to investigate within-host dynamics of African trypanosomes and the role and limitations of antigenic variation. We show that the host-body size ultimately enforces a critical switch rate for each antigenic variant, below which generation and consequently infection prolongation is impossible. The model is also used to infer the magnitude range of variant activation rates and our findings suggest that host-body size may be an influential factor in driving trypanosome evolution.

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