Contribution ID: **212** Type: **Oral Presentation**

A dynamical system model of host-pathogen interaction illustrates the role of the immune system in resilience to infection

Thursday, 12 July 2018 10:50 (20 minutes)

Traditionally the study of host response to an infection has focussed on the effect of the pathogen on host health and the ability of the host immune system to clear the pathogen. In this paradigm the host experiences an initial reduction in health (assessed by suitable metrics e.g. temperature rise, weight loss, subjective feelings of ill health) as the pathogen load in the body increases. When the pathogen load is sufficiently high, the immune response is activated and attempts to clear the pathogen. If the immune system is able to eliminate the pathogen, the host survives and returns to full or partial health. If not, the infection results in death of the host. This paradigm does not account for the potential deleterious effects of the immune response on the health of the host. While this concept has been well studied in autoimmune disorders, it has not been considered in the context of diseases caused by pathogens. Here we present a dynamical system model of the interplay between the host, pathogen and the host immune response. The work utilises an existing two-variable, nonlinear mathematical model of host-pathogen interaction [1] and supplements it with a variable describing the health of the host. The equation describing the health variable incorporates terms for self repair and interactions with the pathogen and immune response. The system gives rise to a rich set of behaviours that, in addition to the usual steady states of full host recovery or host death with unbounded pathogen growth, also includes steady states with incomplete pathogen clearance and partial recovery or host death with complete pathogen clearance. The trajectories in phase space recapitulate patterns that have been described empirically [2]. The model was parameterised against a published mouse malaria dataset [3] and found to adequately describe the observed trajectories. The model also suggested that the difference between host survival and death in the study was governed by the strength of the health-immune response interaction rather than pathogen mediated effects.

[1] H. Mayer *et al.*, *Chaos* 5(1), 1995
[2] D. Schneider, *PLoS Biology* 9(9) 2011
[3] B. Y. Torres *et al.*, *PLoS Biology*, 10.1371/journal.pbio.1002436, 2016

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Session Classification: Parasites & pathogens

Track Classification: Disease - infectious