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Reconceptualizing within-host disease models to characterize the roles of cellular coinfection and viral complementation in the replication dynamics of influenza.

Within-host models of viral pathogens are key to better understand the virological and immunological processes regulating these infections. For influenza, models range from simple target cell limited models to models that include aspects of the immune response. At their core, however, these models are structurally similar in that they classify target cells as being simply infected or uninfected. Recent research, however, has shown that only a small subset of virions express the full set of viral genes required for productive infection and are thus fully-infectious (capable of productive infection when singly infecting cells). The vast majority of virions are only capable of expressing a limited subset of essential viral genes and are thus semi-infectious (incapable of productive infection when singly infecting cells, yet capable of replicating through complementation). Here, we propose a structurally different, yet low dimensional, modeling approach based on epidemiological macroparasite models to more accurately represent influenza virus within-host dynamics. We define the structures of these models, with cells akin to hosts and virus particles akin to macroparasites, and discuss ongoing work to identify the parameters that are measurable through experiments. (Received September 12, 2016)