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Modeling dengue dynamics: unraveling the impact of homologous reinfections

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Dengue fever, caused by the dengue virus and transmitted by Aedes mosquitoes, remains a major public health concern in tropical and subtropical regions. The co-circulation of multiple serotypes increases the risk of severe disease due to antibody-dependent enhancement (ADE) during heterologous reinfections. Mathematical models have long explored the complex dynamics of dengue transmission, incorporating key factors such as temporary cross-immunity (TCI) and ADE [1, 2].

Traditionally, homologous reinfections, secondary infections with the same serotype, were not considered, as individuals were assumed to acquire lifelong immunity to a single serotype. However, recent studies suggest that homologous reinfections, though rare, can occur [3]. While typically asymptomatic due to a rapid immune response, these reinfections may contribute to viral circulation and influence epidemiological patterns.

We present a modeling framework that captures dengue immune dynamics mediated by antibodies, integrating both homologous and heterologous reinfections. Our model reproduces the viral load and antibody production dynamics observed in primary and secondary infections, aligning with empirical immunology studies [4].

This framework lays the foundation for an extended multi-strain population model incorporating primary and secondary infections, TCI, ADE, and homologous reinfections. We explore the epidemiological impact of homologous reinfections in endemic settings and assess their broader implications for dengue transmission, particularly in temperate regions like Europe, where established vectors and local transmission are primarily driven by viremic imported cases.

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