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The elusive when & why of synchrony in malaria infections

Malaria parasites can proliferate in periodic, synchronized bursts within the host, dynamics underlying the regular fevers associated with the disease. Whether proliferation is synchronized or not has the potential to influence parasite transmission and the efficacy of antimalarial drug treatment. Yet the causes of synchrony remain elusive, including to what extent hosts versus parasites influence the timing of infection dynamics. Disentangling the drivers requires robust statistical methods for quantifying the degree of synchrony within an infection, a considerable challenge. I use a heuristic model (a Leslie matrix) to show that existing methods to quantify synchrony are inadequate. To develop improved methods, I simulate time series from experimental rodent malaria infections, using a system of delayed differential equations to specify different levels of synchrony. I incorporate realistic sampling error to mimic infections from replicate 'mice' and use these simulated data to validate a new method to quantify synchrony. This new approach could identify diversity in the timing of parasite life cycles within the host, a necessary step towards understanding how that timing alters disease spread and host health.