Comment on “Stability via Asynchrony in Drosophila Metapopulations with Low Migration Rates”

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Dey and Joshi (Reports, 21 April 2006, p. 434) studied replicate laboratory populations of Drosophila and reported that low migration led to asynchrony among subpopulations. We argue that this unexpected outcome may be due to variation in the initial size of the subpopulations and uncontrolled stochasticity in the experiments.

Natural populations often fluctuate over time in close step over vast ranges (1, 2). Abundant field data show population synchrony and its decay with distance (2, 3), but few empirical studies have tested theoretical predictions about the synchronizing agents (2, 3). Dey and Joshi (4) tested the effects of migration rate on the dynamics and stability of laboratory populations of the fruit fly Drosophila melanogaster. As in theory (2, 3, 5, 6), high migration synchronized subpopulation dynamics but destabilized the ensemble of experimental populations (4). In contrast, low migration unexpectedly induced asynchrony among subpopulations (4).

Dey and Joshi (4) compared their empirical data against simulation results from a dispersal-coupled Ricker map. Ricker dynamics serve as a surrogate for dispersal-coupled Ricker map. Ricker dynamics synchronize the two populations while maintaining complex population dynamics has also been shown (2, 3, 10).

In laboratory experiments, much care is taken to keep the replicates of a given design similar to each other as possible. In the case of replicated populations, however, individuals may differ in age or physical state. Other sources of variation (diseases and infection, laboratory hazards, and the like) may also introduce unwanted stochasticity. We demonstrate our point with a simple simulation exercise using a pair of Ricker maps with carrying capacity normalized to unity:

\[ X(t+1) = (1-m)X(t) + mY(t) \]

\[ Y(t+1) = (1-m)Y(t) + mX(t) \]

To make a spatial structure, the maps are coupled by a dispersal rate \( m \) and modulated by stochasticity \( \mu(t) \) and \( \eta(t) \):

\[ X(t+1) = (1-m)X(t) + mY(t) + \mu(t) \]

\[ Y(t+1) = (1-m)Y(t) + mX(t) + \eta(t) \]

Noise terms \( \mu(t) \) and \( \eta(t) \) are from uniformly distributed random numbers in the range \([-0.1, 0.1] \). Each agent is synchronized by a dispersal rate \( m \) and modulated by stochasticity \( \mu(t) \) and \( \eta(t) \). The noise terms \( \mu(t) \) and \( \eta(t) \) are from uniformly distributed random numbers in the range \([-0.1, 0.1] \). Each agent is synchronized by a dispersal rate \( m \) and modulated by stochasticity \( \mu(t) \) and \( \eta(t) \).

Carefully planned experiments will eventually untangle theory and data that do not go hand in hand by prompting for better theory. That said, the results in (4) support theoretical predictions of the dynamics of dispersal-coupled populations if we accept that stochasticity may have influenced the outcome of the experiments.

References


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Fig. 1. Basins of attraction, in terms of synchronous fluctuations between two populations $X$ and $Y$ initiated with differing values around the carrying capacity $K = 1$. Synchrony is measured with cross-correlation with lag zero ($\phi$). Red indicates initial values that lead to synchronous oscillations, $r_0 = 1$, and blue indicates initial values that lead to asynchrony, $r_0 = -1$. The populations obey a coupled Ricker map with a dispersal rate of $m = 0.1$. The four rows cover the range of population growth rates used in (4), whereas the three columns indicate three values of correlation in the stochastic components (20% noise around the long-term population averages) influencing $X$ and $Y$. The system was set running for 200 time steps for each value of $X(1)$ and $Y(1)$, and the statistics are calculated for the final 50 time steps.
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