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

Esa Ranta* and Veijo Kaitala

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 Drosophila population dynamics (7, 8), and estimates of growth rate \( r \) are in the range where the Ricker function yields chaotic dynamics (9). However, the phase of a deterministic dispersal-coupled Ricker map has been shown to be dependent on initial population size (10). In particular, when two isolated chaotic subpopulations are coupled by redistributing individuals, “low” migration [10% in (4)] simplifies the chaotic population fluctuations into periodic dynamics (10). More importantly, in-phase and out-of-phase population dynamics form fractal basin boundaries where the match of phase is sensitive to the initial size of the two subpopulations (10). That high migration [30% in (4)] synchronizes 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 as 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:

\[
X(t + 1) = \left[1 - m\right]X(t) + mY(t) \mu(t), \quad Y(t + 1) = \left[1 - m\right]Y(t) + mX(t) \eta(t).
\]

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

\[
X(t + 1) = \left[1 - m\right]X(t) + mY(t) \mu(t), \quad Y(t + 1) = \left[1 - m\right]Y(t) + mX(t) \eta(t).
\]

Noise terms \( \mu(t) \) and \( \eta(t) \) are from uniformly distributed random numbers in the range \( 1-w, 1+w (5) \), where \( w = 0.2 \% \) variation around the long-term mean of \( X \) and \( Y \). Such noise, when perfectly correlated (11), acts as a synchronizing agent. We therefore used \( \text{COR}[\mu(t), \eta(t)] \) values 1, 0.5, and 0, which correspond respectively to perfectly correlated stochasticity, partially correlated stochasticity, and stochasticity with no correlation at all. We calculated synchrony between \( X \) and \( Y \) using cross-correlation with lag zero, \( r_0 \) (3). We restricted the system to 10% migration (\( m = 0.1 \)), the condition under which subpopulation asynchrony was reported in (4).

Either the system sets on perfect synchrony in fluctuations of \( X \) and \( Y \) (\( r_0 = 1 \)) or the two populations fluctuate in opposite phase (\( r_0 = -1 \)). Under low migration, the phase of the two populations depends on the values used to initialize the Ricker map (Fig. 1). Consider now an experimenter who wants to initialize the two populations with exactly the same values (preferably close to \( K = 1 \) and \( \text{COR}[\mu(t), \eta(t)] = 1 \)). With increasing growth rate \( r \) and stochasticity, the experimenter will face problems, because even a minor deviation might tip the initial values so that the populations end up in different phases in terms of synchronous fluctuations.

Our analysis suggests that the finding in (4) that low migration induces asynchrony in subpopulations can be explained by weak dispersal coupling and stochasticity, which can have different effects on low and high dispersal regimes among populations with high growth rates. 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 (3). 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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