Appendix 1

Model specification and parameters

Following Row et al. (2014), we investigated the relationship between population cyclicity and genetic variation using a generalized population matrix consisting of five stages, with distinct juvenile and adult males and females (thus leading to five classes). The three-stage model is standard for exploring matrix population dynamics (Caswell 1989) and is a logical compromise between simplicity and generality in population demography (Hitchcock and Gratto-Trevor 1997, Engel et al. 2001, Spencer et al. 2002, Murray et al. 2006, Row et al. 2014). We generated population cyclicity using a time-delayed density dependence which can arise from a variety of factors or species interactions (e.g. parasite, predator, plant toxin, competition, maternal effect, etc.), but can be applied to single species models (Royama 1992, Ives et al. 2010) with direct and delayed density dependence.

Specifically, our populations were simulated using a general stage-based Lefkovitch matrix population model (Lefkovitch 1965, Strand 2002):

\[
\begin{bmatrix}
  n_k \\
  n_{jf} \\
  n_{jm} \\
  n_{am} \\
  n_{af}
\end{bmatrix}_{t+1} =
\begin{bmatrix}
  0 & JF(R) & 0 & F(R) & 0 \\
  K(S)/2 & 0 & 0 & 0 & 0 \\
  K(S)/2 & 0 & 0 & 0 & 0 \\
  0 & J(S) & 0 & A(S) & 0 \\
  0 & 0 & J(S) & 0 & A(S)
\end{bmatrix}
\begin{bmatrix}
  n_k \\
  n_{jf} \\
  n_{jm} \\
  n_{am} \\
  n_{af}
\end{bmatrix}_t
\]

(A1)

and individuals moved through each developmental stage by random selection based on transition and survival rates. Reproductive potential was determined by selecting the number of offspring for each juvenile or adult female from a Poisson distribution, and then randomly choosing an adult male as the father. Kittens could not reproduce and half the surviving cohort was assigned to juvenile male and females. The reproductive potential (FJ[R] and F[R]), kitten survival (K[S]), juvenile survival potential (J[S]) and adult survival potential (A[S]) were randomly chosen from a
prior uniform sampling distribution (Table A1). The strength of direct density dependence was determined through $\phi$ in the logistic equation:

$$\beta_1 = e^{\phi(N_{t-1})}$$ (A2)

and time-delayed logistic density dependence was modeled using a successive density dependence,

$$\beta_{2c} = e^{\phi(S_{t-1}^{l-T_D}N)}$$ (A3)

with the strength of regulation being controlled through $\phi$. Direct (Eq. A2) and delayed density dependence (Eq. A3) were multiplied by the survival potential and reproductive rate in Eq. A1 after each time interval (hereafter, ‘year’). Thus, as population size ($N$ – total population size of adults and juveniles) increases, the density-dependent effect increases, lowering the corresponding population parameters in the subsequent year. As with the demographic parameters, both the strength of the direct ($\phi$) and time-delayed ($\phi$) density dependence parameters were chosen from a prior distribution for each simulation (Table A1). Dispersal was limited to neighbouring populations following a stepping stone model. Population simulations were conducted using the Rmetasim (Strand 2002) package in R (<www.r-project.org>).

References


Table A1. Parameters and sampling distributions used in population time series simulations.

<table>
<thead>
<tr>
<th>Parameter (s)</th>
<th>Description</th>
<th>Min</th>
<th>Max</th>
</tr>
</thead>
<tbody>
<tr>
<td>K(S)</td>
<td>kitten survival</td>
<td>0.66</td>
<td>0.99</td>
</tr>
<tr>
<td>J(S)</td>
<td>juvenile male and female survival</td>
<td>0.80</td>
<td>0.99</td>
</tr>
<tr>
<td></td>
<td>potential</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A(S)</td>
<td>adult male and female survival</td>
<td>0.80</td>
<td>0.99</td>
</tr>
<tr>
<td></td>
<td>potential</td>
<td></td>
<td></td>
</tr>
<tr>
<td>JF(R)</td>
<td>juvenile female reproductive rate</td>
<td>4.46</td>
<td>6.14</td>
</tr>
<tr>
<td>F(R)</td>
<td>adult female reproductive rate</td>
<td>4.24</td>
<td>6.36</td>
</tr>
<tr>
<td>Φ</td>
<td>strength of direct dependence</td>
<td>10⁻⁷</td>
<td>0.002</td>
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<tr>
<td>ψ</td>
<td>strength of time-delayed density</td>
<td>0.0000001</td>
<td>0.002</td>
</tr>
<tr>
<td></td>
<td>dependence</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TD</td>
<td>time delay increment (e.g. years)</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>STRT.ind</td>
<td>initial population size</td>
<td>20</td>
<td>400</td>
</tr>
</tbody>
</table>
Figure A1. Change in a) gene diversity and b) genetic differentiation through time for low (solid – orange), medium (dotted – green) and high (dashed – blue) amplitude simulations.

Figure A2. Linear stepping model used in population simulations model. Distances between populations ranged from 1 (neighbours) to 9 (populations 1 and 10). Population sizes for demographic modeling were derived to be roughly equivalent to a cycling population within a 500 km² area.
Figure A3. Diagram depicting replicate structure for analysis of simulations examining, (a) the effects of cyclic amplitude (low, medium, high) on genetic diversity and differentiation, (b) the interaction of synchrony and phase-dependent dispersal with cyclicity for high amplitude cycles, (c) the potential genetic rescue from non-cyclic populations by comparing simulations with mixed (high and low cycling populations) and non-mixed stepping stone models, and (d) the magnitude of change in genetic variation throughout a cycle through a comparison of peaks (P) and troughs (T) for high amplitude cycles under three dispersal scenarios.
Figure A4. Differences in demographic characteristics of cycling populations within and between amplitude treatments.