Species interactions and coevolution

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What is coevolution?

"Thus I can understand how a flower and a bee might slowly become, either simultaneously or one after the other, modified and adapted to each other in the most perfect manner, by the continued preservation of all the individuals which presented slight deviations of structure mutually favourable to each other."

— Charles Darwin, The Origin of Species

Coevolution: Reciprocal evolutionary change in interacting species (Janzen, 1980)
My lab uses mathematical models to study coevolution.

- Spatially structured species interactions
- Structure of biological communities
- Genome structure

Focus on this project today.
An example of spatially structured coevolution: toxic newts and resistant snakes

- Predator-prey interaction

Butch Brodie
Toxic newts

*Taricha granulosa*

- Newts contain Tetrodotoxin, a potent neurotoxin
- Some newts contain enough toxin to easily kill a human
- Toxin causes snakes to only “taste” the newts
Resistant snakes

*Thamnophis sirtalis*

- Some snakes have evolved modified sodium channels
- These snakes are more resistant to tetrodotoxin
- Consequently, resistant snakes can eat toxic newts
Toxic newts and resistant garter snakes

(Hanifin et al. 2008, PLoS Biology)

Observation #1:

Newt toxicity and snake resistance
Are spatially variable
Toxic newts and resistant garter snakes
(Hanifin et al. 2008, PLoS Biology)

Observation #2:
Newt toxicity and snake resistance are positively correlated
Summarizing the Data
(Hanifin et al. 2008, PLoS Biology)

- Newt toxicity varies across space
- Snake resistance varies across space
- Toxicity and resistance are positively correlated

These observations have led to the development of a coevolutionary hypothesis
A coevolutionary hypothesis

*** We can test this coevolutionary hypothesis using mathematical models ***
Developing an appropriate model

• The data consists of toxicity and resistance measured in many populations

\[ \bar{z}_1 = 1.8\text{mm} \]
\[ \bar{z}_2 = 1.9\text{mm} \]

\[ \bar{z}_1 = 1.2\text{mm} \]
\[ \bar{z}_2 = 1.6\text{mm} \]

\[ \bar{z}_1 = 1.3\text{mm} \]
\[ \bar{z}_2 = 1.6\text{mm} \]

\[ \bar{z}_1 = 1.4\text{mm} \]
\[ \bar{z}_2 = 1.7\text{mm} \]

\[ \bar{z}_1 = 1.1\text{mm} \]
\[ \bar{z}_2 = 1.3\text{mm} \]

⇒ Our model must predict mean trait values in replicate populations
Let's start by modeling one of these populations

If we assume that additive genetic variance is constant:

\[
\Delta \bar{z}_1 = G_1 \left( \frac{1}{W_1} \frac{\partial \bar{W}_1}{\partial \bar{z}_1} \right)
\]

\[
\Delta \bar{z}_2 = G_2 \left( \frac{1}{W_2} \frac{\partial \bar{W}_2}{\partial \bar{z}_2} \right)
\]

⇒ To predict (co)evolution we need to calculate mean fitness
Defining individual fitness

\[ W_T(z_i) = W_A(z_i)W_B(z_i, z_j) \]

**Abiotic environment:**

**Species interactions:**

Abiotic environment:

Species interactions:

Optimal phenotype, \( \theta \)

Probability of consumption

Snake resistance - Newt toxicity
Developing recursions for trait means

\[
\bar{W}_1 = \int \int W_A(z_1)W_B(z_1, z_2)\phi(z_1)\phi(z_2) \, dz_1 dz_2 \\
\bar{W}_2 = \int \int W_A(z_2)W_B(z_2, z_1)\phi(z_1)\phi(z_2) \, dz_1 dz_2
\]

Assume weak selection

\[
\Delta \bar{z}_i = G_i \left( \frac{1}{\bar{W}_i} \frac{\partial \bar{W}_i}{\partial \bar{z}_i} \right)
\]

Incorporate genetic drift

\[
\Delta \bar{z}_i \approx G_i \left[ 2\gamma_i (\theta_i - \bar{z}_i) + s_{Di} \right] + \zeta_i + O(\epsilon^2)
\]

Abiotic selection

Biotic selection

Drift
Model predictions for local coevolution

Weak selection on newts
Strong selection on snakes

Strong selection on newts
Weak selection on snakes

Strong selection on newts
Strong selection on snakes

Toxicity and Resistance

→ Equilibrium trait values depend on the strength of biotic selection
But we need a model of MANY populations!

Empirical Data

- $\bar{z}_1 = 1.2 \text{mm}$
- $\bar{z}_2 = 1.6 \text{mm}$
- $\bar{z}_1 = 1.3 \text{mm}$
- $\bar{z}_2 = 1.6 \text{mm}$
- $\bar{z}_1 = 1.4 \text{mm}$
- $\bar{z}_2 = 1.7 \text{mm}$

Minimal model

- Multiple populations
- Gene flow (island model)
  ➔ Requires more equations
Adding multiple populations and gene flow

\[ \Delta \tilde{z}_{i,1} \approx G_i \left[ 2\gamma_i \left( \theta_{i,1} - \tilde{z}_{i,1} \right) + 2s_{M_i} \left( \tilde{z}_{j,1} - \tilde{z}_{i,1} \right) + s_{D_i} \right] + \left( 1 - m_i \right) \tilde{z}_{i,1} + m_i \tilde{Z}_i + \zeta_i + O(\varepsilon^2) \]

\[ \Delta \tilde{z}_{i,2} \approx G_i \left[ 2\gamma_i \left( \theta_{i,2} - \tilde{z}_{i,2} \right) + 2s_{M_i} \left( \tilde{z}_{j,2} - \tilde{z}_{i,2} \right) + s_{D_i} \right] + \left( 1 - m_i \right) \tilde{z}_{i,2} + m_i \tilde{Z}_i + \zeta_i + O(\varepsilon^2) \]

\[ \vdots \]

\[ \Delta \tilde{z}_{i,n} \approx G_i \left[ 2\gamma_i \left( \theta_{i,n} - \tilde{z}_{i,n} \right) + 2s_{M_i} \left( \tilde{z}_{j,n} - \tilde{z}_{i,n} \right) + s_{D_i} \right] + \left( 1 - m_i \right) \tilde{z}_{i,n} + m_i \tilde{Z}_i + \zeta_i + O(\varepsilon^2) \]

- In principle, we could then just solve this system of 2n equations
- In practice, this is impossible

➤ This difficulty can be overcome by making a change of variables that reveals a tractable approximation
What does our final approximation predict?

At equilibrium and assuming weak selection:

The spatial variability in toxicity or resistance is:

\[ \sigma^2_{z_i} = \frac{G_i}{2N_i(m_i + 2\gamma_i G_i)} \]

The correlation between toxicity and resistance is:

\[ \hat{\rho} \approx 0 + O(\varepsilon^2) \]

What is missing from these equations?

What does this tell us?

Does this provide support for the coevolutionary hypothesis?