Volkov et al.
L-V Competition:

\[
\frac{dN_1}{dt} = rN_1 \left( \frac{K_1 - N_1 - \alpha_{12}N_2}{K_1} \right)
\]

\[
\frac{dN_2}{dt} = rN_2 \left( \frac{K_2 - N_2 - \alpha_{21}N_1}{K_2} \right)
\]

Stable Coexistence when \( K_1 > \alpha_{12}K_2 \) and \( K_2 > \alpha_{21}K_1 \)

i.e. when

competition *within* species >
competition *between* species

Other cases have unstable coexistence or one species winning
L-V Competition: 4 cases

Competitive Exclusion Principle

Complete competitors cannot stably coexist.

('complete competitors' have interspecific competition > or = intraspecific competition)

Niche Theory
L-V Competition: 5th case

If \( K_1 = K_2 \), \( \alpha_{12} = \alpha_{21} = 1 \)

Individuals of sp1 and sp2 are interchangeable
Coexistence Mechanisms

- **Neutral Theory**
  - Average per capita growth rate at low abundance ($r$)

- **Niche theory**
  - Pure fitness-equalization ($r = 0$)
  - Stabilization ($r > 0$)

- **Graphs**
  - Abundance vs. Time
  - Different scenarios illustrating coexistence mechanisms.
Hubbell’s book
Neutral communities seem to be structurally unstable

• E.g. in the L-V model, very restricted range of parameter space corresponds to neutrality

• There has to be something tuning or driving the species to be so similar

So why might neutral communities arise?

→ When there is no niche space left, there is selection for similarity with a species already present
Master equation for population size, \( n \), of species \( k \) with birth (\( b \)) and death (\( d \))

\[
\frac{dp_{n,k}(t)}{dt} = p_{n+1,k}(t)d_{n+1,k} + p_{n-1,k}(t)b_{n-1,k} - p_{n,k}(t)(b_{n,k} + d_{n,k})
\]

Gain from death in \( n+1 \) population
Gain from birth in \( n-1 \) population
Loss from birth or death in \( n \) population
Steady state solution for metacommunity

\[ P_{n,k} = P_{0,k} \prod_{i=0}^{n-1} \frac{b_{i,k}}{d_{i+1,k}} \]

Expected number of species with population size \( n \) in the metacommunity

\[ \langle \phi_n^M \rangle = S_M P_0 \frac{b_0 b_1 \ldots b_{n-1}}{d_1 d_2 \ldots d_n} = \theta \frac{x^n}{n} \]
Neutral Model:  
*Local community* connected to *Metacommunity*

**In each death and replacement event:**

- a randomly chosen individual dies, and then...

2 options: (zero-sum dynamics!)
- with probability $m$, it is replaced with the offspring of a random individual from the regional community

*or*
- with probability $(1 - m)$ it is replaced with an offspring of a random individual in the local community

$m = \text{immigration rate}$

Assumes individuals of different species are *demographically equivalent*
Algorithm for birth, death, and migration for local community connected to metacommunity

\[
b_{n,k} = (1 - m) \frac{n}{J} \frac{J - n}{J - 1} + m \frac{\mu_k}{J_M} \left(1 - \frac{n}{J}\right)
\]

\[
d_{n,k} = (1 - m) \frac{n}{J} \frac{J - n}{J - 1} + m \left(1 - \frac{\mu_k}{J_M}\right) \frac{n}{J}
\]
Steady state for local community

Using the same steady-state solution as before, we find

\[ P_{n,k} = \frac{J!}{n!(J-n)!} \frac{\Gamma(n + \lambda_k)}{\Gamma(\lambda_k)} \frac{\Gamma(\vartheta_k - n)}{\Gamma(\vartheta_k - J)} \frac{\Gamma(\lambda_k + \vartheta_k - J)}{\Gamma(\lambda_k + \vartheta_k)} \equiv F(\mu_k) \]

With the definitions

\[ \lambda_k = \frac{m}{(1-m)(J-1)} \frac{\mu_k}{J_M} \]

\[ \vartheta_k = J + \frac{m}{(1-m)(J-1)} \left( 1 - \frac{\mu_k}{J_M} \right) \]
Distribution for the local community

Using the probability density function

\[ \hat{p}(\mu) d\mu = \frac{1}{\Gamma(\varepsilon)\delta^\varepsilon} \exp\left(-\frac{\mu}{\delta}\right) \mu^{\varepsilon-1} d\mu \]
Distribution for the local community

We find

\[ \langle \phi_n \rangle = \theta \frac{J!}{n!(J-n)!} \frac{\Gamma(\gamma)}{\Gamma(J+\gamma)} \int_0^\gamma \frac{\Gamma(n+y)}{\Gamma(1+y)} \frac{\Gamma(J-n+\gamma-y)}{\Gamma(\gamma-y)} \exp(-y\theta/\gamma) \, dy \]

\[ y = \mu \gamma / \delta \theta \text{ and } \delta \theta = \theta x / (1-x) = J_M \]
Match of theory and data with comparison to log normal (black)

This analytical result provides better fit than lognormal
Problems with this test

• Simulations of stochastic niche models produce similar range of patterns

• This neutral model ignores a great deal of demographic complexity that is not necessarily part of niche mechanisms, e.g.
  – fitness-equalized life history variation
  – size structure
  – spatial structure
Different equations are useful for different types of predictions

Master equation

Fokker-Planck equation

Langevin equation
Elowitz et al.
Experimental system for detecting noise

A

Extrinsic noise

B

Intrinsic noise

Identification of noise
Measure of noise

\[ \Gamma^2 = \frac{\text{Var}(x)}{\langle x \rangle^2} = \frac{\langle x^2 \rangle - \langle x \rangle^2}{\langle x \rangle^2} \]

This is inverse of signal to noise. Also, can decompose this into intrinsic noise and extrinsic noise where the latter is anything external to that particular gene.

\[ \Gamma^2 = \Gamma_{\text{int}}^2 + \Gamma_{\text{ext}}^2 \]

Want to distinguish between these two types of noise
Direct picture of cells we can interpret in terms of level of intrinsic noise
Orthogonal axes for intrinsic and extrinsic noise
Most noise is extrinsic. Intrinsic noise matters for low copy numbers.
Most noise is extrinsic. Intrinsic noise matters for low copy numbers.
Can see similar results in many different types of cells

Table 1. Measurements of noise in selected strains.

<table>
<thead>
<tr>
<th>Modification*</th>
<th>Strain†</th>
<th>Intensity‡</th>
<th>Intrinsic noise, $\eta_{\text{int}}$ $\times 10^{-2}$</th>
<th>Extrinsic noise, $\eta_{\text{ext}}$ $\times 10^{-2}$</th>
<th>Total noise, $\eta_{\text{tot}}$ $\times 10^{-2}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constitutive (lac$^{-}$)</td>
<td>M22</td>
<td>1</td>
<td>5.5 (5.1–6)</td>
<td>5.4 (4.8–5.9)</td>
<td>7.7 (7.4–8.1)</td>
</tr>
<tr>
<td></td>
<td>JM22</td>
<td>0.88</td>
<td>5.0 (4.6–5.4)</td>
<td>6.1 (5.5–6.7)</td>
<td>7.9 (7.4–8.4)</td>
</tr>
<tr>
<td></td>
<td>MRR</td>
<td>1.21</td>
<td>5.1 (4.7–5.4)</td>
<td>5.6 (5.1–6.2)</td>
<td>7.6 (7.2–7.9)</td>
</tr>
<tr>
<td>Wild type (lac$^{+}$)</td>
<td>MG22</td>
<td>0.057</td>
<td>19 (18–21)</td>
<td>32 (29–35)</td>
<td>37 (35–40)</td>
</tr>
<tr>
<td></td>
<td>RP22</td>
<td>0.030</td>
<td>25 (22–27)</td>
<td>33 (30–35)</td>
<td>41 (39–43)</td>
</tr>
<tr>
<td>Wild type (lac$^{+}$), +IPTG</td>
<td>RP22</td>
<td>1.00</td>
<td>6.3 (5.8–6.9)</td>
<td>9.8 (9.0–11)</td>
<td>11.7 (11–12.3)</td>
</tr>
<tr>
<td>lac$^{-}$, Repressilator</td>
<td>M22</td>
<td>0.18</td>
<td>12 (11–13)</td>
<td>42 (37–45)</td>
<td>43 (39–47)</td>
</tr>
<tr>
<td></td>
<td>MRR</td>
<td>0.16</td>
<td>11 (9.8–12)</td>
<td>57 (52–62)</td>
<td>58 (53–63)</td>
</tr>
<tr>
<td>ΔrecA, lac$^{-}$</td>
<td>D22</td>
<td>0.81</td>
<td>10.5 (9.6–11.4)</td>
<td>4.6 (2.8–5.8)</td>
<td>11.4 (10.8–12.1)</td>
</tr>
<tr>
<td></td>
<td>M22ΔA</td>
<td>0.99</td>
<td>13 (12–15)</td>
<td>2.4 (0–5.3)</td>
<td>13.6 (12.8–14.5)</td>
</tr>
<tr>
<td></td>
<td>JM22ΔA</td>
<td>0.92</td>
<td>14 (11–17)</td>
<td>2.5 (0–7.3)</td>
<td>15 (12–16.4)</td>
</tr>
<tr>
<td>ΔrecA, lac$^{+}$ + IPTG</td>
<td>RP22ΔA</td>
<td>1.22</td>
<td>17 (15–20)</td>
<td>12 (8.8–14)</td>
<td>21 (20–22)</td>
</tr>
</tbody>
</table>

*Deletion refers to deletion of chromosomallacI; IPTG inducer; growth in the presence of 2×M IPTG.
†The following strain backgrounds were used: MC4100 (23) for...
Differences in cycling in time can increase noise

Repressilator induces oscillations, and if oscillations become shifted in time, this will appear to create extrinsic noise for snapshot.

RecA is involved in overcoming stalls of replication forks so can also create shifts in timing of event and increase noise

Would time averaging get rid of effects of this noise or not?
These methods are general and span levels of organization in biology and many types of processes requiring different types of predictions

1. Correlations of velocities of migrating cells

2. Absorption times for cells dividing in a crypt with selection and drift (cancer)

3. Neutral processes among individuals and species abundance distributions (SAD)

4. Noise of gene expression within a cell