

## Mammalogy Lecture 14 – Arvicoline Population Cycles

Population Cycles: For the most part, populations tend to be fairly stable. They may fluctuate, but there's usually an identifiable correlate that we can associate with population size.

A. This is not true for many murid rodents in the subfamily Arvicolinae - voles and lemmings.

1. Examples - *Clethrionomys rufocanus* in the Kola peninsula

- *Dicrostonyx* in Alaska

- Several well-studied species of *Microtus*.

- Cycle also occur in *Lepus americanus* / *Lynx canadensis*

2. Characteristics of arvicoline cycles

1) Peak population densities may actually be 1-2 orders of magnitude higher than trough densities. The highest reported were *M. montanus* of 20,000 per hectare.

2) Growth phase is very rapid

3) Crash is very abrupt

4) Typically in arvicolines there are 3-5 years between peaks; there is a lag.

5) This periodicity seems to be fairly regular - that is non-random, at least in the temporal aspect, although the amplitude of the peak can vary.

There are some questions regarding the statistical regularity of these cycles, but not about the existence of the cycles themselves.

- In addition, there is a latitudinal component. There is a correlation between the tendency to cycle and latitude - northern -- more cyclicality.

**Especially true in Scandinavia**

B. Biologists have been struck by this phenomenon for some time, and a great deal of work has been undertaken to uncover the cause of these cycles.

1. Need to explain:

1) Rapid growth – (easy...these are quintessential r-selected mammals).

2) Why does growth stop?

3) Why is decline so steep?

4) Why is there a time lag?

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### 2. Extrinsic Hypotheses

a. Food - Herbivore/Plant interactions --- Batzli and others

At low vole densities, plants are unaffected by herbivory.  
At high densities, there is a selective reduction of plant biomass.

A high rate of herbivory induces the production of chemical defenses by plants.  
This (in addition to an overall reduction in biomass) compromises the quality of forage available to voles, which in turn, causes the crash.

The time lag is explained as the time that plant populations take to recover.

Slide: Graph on slide right represents a mathematical model based only on food and a time lag corresponding to plant recovery.

Actual populations seem to fit predictions of the mathematical models extremely well, so this hypothesis has been tested experimentally.

**When voles are raised in enclosures and given supplemental food, we still eventually see a population crash. Exhaustion of food resources alone can't explain crash.**

**These studies have been repeated again and again. Supplemental food just can't prevent a crash. Therefore, the food hypothesis can't be the only explanation.**

b. Predators -

In northern Scandinavia, there are specialist predators ---> *Mustela nivalis*  
*M. erminea*

The idea is that specialist predators oscillate with prey (as is the case with snowshoes and lynx), and predators drive the cycle.

In southern Scandinavia, the predators are more generalist and keep populations relatively low because the predator populations never crash.

This would explain the latitudinal variation.

**However, the correspondence between specialist predators and latitude doesn't hold in North America, and populations in predator-free enclosures still crash. Thus, predation alone can't explain arvicoline population cycles.**

### 3. Intrinsic Hypotheses

a. Chitty Hypothesis - Behavioral/Genetic hypothesis.

**Based on the observation that there is a higher proportion of large aggressive voles in peak populations.**

Idea is that there is a genetic basis to both size and aggressive behavior, and that selection is operating within a cycle.

It's complex, but the crux is as follows:

At low densities, selection will favor **Clark Kent** voles - amicable, put much energy into reproduction rather than aggression.

At high densities, selection will favor fighters - they'll get resources, but won't be very reproductively active → population will crash

At first, the Chitty hypothesis was criticized on the grounds that a single cycle is too short a time period for selection to operate.

However allozyme studies have demonstrated that allele frequencies in a sample from a trough can be significantly different than those in a sample from a peak. So significant genetic change can actually occur in this short a time period (w/in a cycle).

This led to a general acceptance of the Chitty hypothesis, and the old text listed this as the explanation.

**Problem - the traits involved (large size and aggressive behavior) have very low heritabilities ---> even though genetic change within a cycle can be driven by selection - not true for the crucial traits.**

#### 4. Multifactorial Hypothesis - Hestebek et al.

At low densities -Scattered social groups

Group size is regulated by dispersal - there is free dispersal.

Little aggression because there are plenty of resources.

Intrinsic factors will dominate the dynamics of the pop.

At high densities - Resources become scarce.

Food may become limiting.

Dispersers experience increased aggression because of lower relatedness.

Further dispersal will be curtailed and population may increase to the point of exhausting resources.

Extrinsic factors will dominate.

Population will crash.

Clearly, a multifactorial explanation will be required (see page 396 in your text). This actually includes many of the aspects of each of the single factor hypotheses.

Food becomes limiting, this contributes to other effects such as increased aggression.

Predators are incorporated by Pantry Effect; predation increases greatly at high vole densities because of a positive feedback, even among generalist predators. This contributes to crash.

Accounts for large aggressive animals present at high population densities.

Population cycles in arvicolines is a complex phenomenon and the explanation isn't going to be simple, and multifactorial hypotheses will be required.

However one difficulty that such a complex hypothesis presents is that it is incredibly difficult to test.