Defense: 2 theories

- Apparent Theory (Feeney, Cates, Rhoades)
- Apparent plants: quantitative defenses -- make up to 60% of leaf tissue, e.g. tannins, toughness, high C:N) (are these defenses?)
- Unapparent plants: qualitative defenses -- alkaloids, cyanogenic compounds, cardiac glycosides)—ca. 2% leaf tissue, specialists can detoxify, or even sequester for own use...(Monarch or California Buckeye caterpillars)
Resource availability theory
(Coley, Chapin and Bryant 1985)

- Allocation to defense depends on realized growth rate of plant, \( dC/dt \, (g \, t^{-1}) \). Defenses will be low in resource rich environments, high in poor environments

- \( dC/dt = G \, C_0 \, (1-kD^a) - (H - mD^b) \)
  - \( C_0 \, (g) \) plant biomass at time zero
  - \( G \, (g \, g^{-1} \, d^{-1}) \) maximum inherent growth rate
  - \( D \, (g \, g^{-1}) \) investment in defense
  - \( H \, (g \, d^{-1}) \) potential herbivore pressure if no defense
  - \( (1-kD^a) \) defense cost curve, % reduction in growth due to investment in defense
  - \( (H - mD^b) \) defense effectiveness, reduction in herbivory due to defense
Fig. 1. Effect of defense investment on realized growth. Each curve represents a plant species with a different maximum inherent growth rate. Levels of defense that maximize realized growth are indicated by an arrow. Realized growth \( (dC/dt) \) is calculated as \( dC/dt = G\bar{C}((1 - kD^\alpha) - (H - mD^\beta) \) where \( G \) (g g\(^{-1}\) d\(^{-1}\)) is the maximum inherent growth rate permitted by the environment (without herbivores), \( \bar{C} \) (g) is the plant biomass at time zero, \( D \) (g g\(^{-1}\)) is the defense investment, \( k \) (g d\(^{-1}\)) and \( \alpha \) are constants that relate an investment in defense to a reduction in growth. The entire term \( (1 - kD^\alpha) \) is the percentage of reduction in growth due to investment in defenses. The term \( H \) (g d\(^{-1}\)) is the potential herbivore pressure in the habitat (assuming no defense). Potential herbivory is reduced by a function of defense investment, \( (mD^\beta) \), where \( m \) (g d\(^{-1}\)) and \( \beta \) are constants that determine the shape of the defense effectiveness curve. The entire negative term \( (H - mD^\beta) \) is the reduction in realized growth (g d\(^{-1}\)) due to herbivory. Since it is subtracted from growth, this assumes herbivores consume fixed amounts of leaf tissue and not fixed percentages of plant productivity. The model’s results depend on the extent to which this assumption is true. To further conform to biological reality, the herbivory term \( (H - mD^\beta) \) cannot be less than zero, regardless of the value of \( D \).
**Functional Responses** - the relationship between an individual’s consumption rate and local food density.

*Type 1 functional response* – the number of prey eaten per predator increases linearly with the number of prey present.

The per capita rate at which prey are dying is independent of prey density.

\[ a = \text{linear attack rate} \]
Type 2 functional response – At high prey densities, each prey individual has a lower chance of being eaten than at low prey densities.

The per-capita prey death rate decreases with increasing prey density.

Inverse density-dependent prey death rate

Destabilizing
Holling’s Disc Equation – Type 2 Functional Response

\[ P_e = \text{# prey eaten by a predator during a period of search time, } T_s \]
\[ N = \text{# of prey available} \]
\[ a = \text{searching efficiency, or attack rate of predator} \]
\[ P_e = a \cdot T_s \cdot N \]

[Box: \( \text{indivs indivs}^{-1} \text{ time}^{-1} \text{ time indivs} \)]

But, the time available for searching will be less than the total time, because of time spent handling prey

\[ T = \text{total time} \]
\[ T_h = \text{handling time for each prey item} \]
\[ T_h P_e = \text{Total time spent handling prey} \]
\[ T_s = T - T_h P_e = \text{time available for searching} \]
\[ P_e = a(T - T_h P_e)N \]

Holling’s Disc Equation

\[ P_e = \frac{aNT}{1 + aT_h N} \]
Type 3 functional response – At low densities the functional response increases in an accelerating way with increasing prey density.

For a range of low densities, the per-capita prey death rate increases with increasing prey density.

Density-dependent prey death rate

Stabilizing: negative feedback
Mechanisms leading to a Type 3 functional response:

Search images – predator learns to detect their prey faster as the density is increased.

Threshold effects – predators don’t initiate feeding unless they get a certain amount of stimulus from the environment.

Switching – if there is an environment with two different prey types, the predator generally concentrates on the most abundant prey type.

Prey refuges – if the prey have a certain number of safe sites, the predator can’t eat any prey when the density is below the number of sites. If you consider the entire range of prey densities, it looks like a type 3 functional response.
Functional Response might also depend on **Predator Density**

Mutual interference: a decrease in the predator searching efficiency with increased predator density.

![Graph showing mutual interference between searching efficiency and predator density](image)

**Figure 9.10** Mutual interference: the relationship between searching efficiency and the density of searching parasitoids or predators (log scales). (a) *Encarsia formosa* parasitizing the whitefly *Trialeurodes vaporariorum*. (After Burnett, 1958.) (b) *Phytoseiulus persimilis* feeding on nymphs of the mite *Tetranychus urticae*. (After Fernando, 1977; Hassell, 1978.)

Predatory mite attacking spider mite
Numerical Response: Change in the density of predators in response to changes in density of prey

- Reproduction: Increased prey availability leads to increased predator reproduction
- Aggregation: Immigration into areas with high prey density
Examples of predator-prey population dynamics

(a) Snowshoe Hare and Lynx populations over time.

(b) Graph showing population density of Host, Parasitoid, and Generations.

Bird and insect images indicating:
- Beetle attacked by parasitoid
- Mouse prey
- Owl predators
Population Dynamics of Predator-Prey Interactions

\[ N = \# \text{ of Prey} \]

\[ P = \# \text{ of Predators} \]

Prey population growth rate:
Assume that in the absence of the predator the prey grows exponentially:

\[ \frac{dN}{dt} = rN \]
Prey death due to predators:
Assume: Prey are killed by the predator at a rate that is proportional to the rate of encounter between prey and predators.

\[ a = \text{predator attack rate} \]

\[ \frac{dN}{dt} = rN - aNP \]

- Rate of increase without predators
- Rate of loss due to predators

**Type 1, Type 2, or Type 3 functional response?**

“Lotka-Volterra dynamics”
Per-capita growth rate of prey population is a decreasing function of predator density.

\[
\frac{1}{N} \frac{dN}{dt} = r - aP
\]

If the predator density is greater than \( \frac{r}{a} \) then the prey population with decline.

At this density of predators, the prey population doesn’t change.
Predator population decrease in the absence of prey:

Assume that in the absence of their prey, individual predators lose weight, and starve to death. No new predators are produced.

In the absence of prey, predator numbers decrease exponentially:

$$\frac{dP}{dt} = -qP$$

$q = \text{predator per-capita death rate}$
Predator population increase due to consuming prey:

Assume that the predator population increases at a rate proportional to the rate at which food is consumed, and the predator’s efficiency at turning the food into new predators:

\[ f = \text{predator conversion efficiency} \]

\[ \frac{dP}{dt} = f a N P - q P \]

- **Birth rate from consuming prey**
- **Death rate in absence of prey**
Per-capita growth rate of predator population is an increasing function of prey density.

\[ \frac{1}{P} \frac{dP}{dt} = faN - q \]

If the prey density is greater than \( q/(fa) \) then the predator population will increase.

At this density of prey, the predator population doesn’t change.
Lotka-Volterra Predator-Prey Model:

Prey: \[ \frac{dN}{dt} = rN - aNP \]

Predator: \[ \frac{dP}{dt} = fNP - qP \]

Zero-growth isoclines:
\[ \frac{dN}{dt} = 0 \]
\[ \frac{dP}{dt} = 0 \]

Phase-Plane Diagram

\[ P^* = \frac{r}{a} \]
Zero-growth isocline for predators:

\[
\frac{dP}{dt} = 0
\]

\[
\frac{dP}{dt} = faNP - qP = 0
\]

\[
faN - q = 0
\]

\[
N^* = \frac{q}{fa}
\]
Efficient “Lotka-Volterra” predation --> neutral stability, cycles...
Inefficiencies:

Prey are self limiting at high densities (K)

Predators interfere with each other at high predator densities

Or predators become limited by a non-food resource (e.g. dens) at high density....
Prey isocline with a hump -- Allee effect, can withstand more predators at higher prey densities.
One explanation (hypothesis) for outbreaks: a strongly self-limiting predator, and a prey population with a refuge and a “hump” (Allee effect at low densities) --> 3 equilibria, with the one at intermediate densities being unstable --> “Density Vague” dynamics (Strong).