

Lectures 28, Tu., Nov. 28

Reading homework: Chapter 7 of reference 1

1. Periodic solutions of some epidemiology models. Epidemiology is a branch of medical science that deals with the occurrence, distribution, and control of diseases in a population. A few diseases occur periodically. For example, the recurrent outbreaks of measles and other childhood diseases; the periodic outbreak of flu that occurs in the flu seasons; the periodic (with 10 year period) syphilis outbreaks in US cities; some Chinese catfish farms in Hawaii have suffered significant economic losses due to the periodic outbreak of bacterial diseases that increase fish mortality (<http://govdocs.aquaculture.org/cgi/reprint/2003/525/5250050.pdf>, 1995). This motivated many mathematical epidemiologists to seek periodic solutions in some epidemiology models. These efforts are almost inevitably futile as the causes of such periodic outbreaks are often not included in the mathematical models. For example, flu outbreak is largely caused by season change and the spread of newly evolved virus strains, while the 10 year syphilis cycle in US maybe due to the loss of immunity among those at risk of infection (<http://health.dailynewscentral.com/content/view/340/63>, 2005). In the following, we will briefly describe two such efforts.

The simplest of such model can be derived from a modification of the simplest SI model with vital dynamics by assuming that the infected ones stop producing offsprings. The model takes the form of,

$$S' = (b - \mu)S - \beta SI, \quad I' = \beta SI - (\mu + \alpha)I \quad (1.1)$$

where b is the birth rate, μ is the baseline death rate, α is the infection induced additional death rate and β is the per capita infection transmission rate. For obvious reasons, we shall assume below the birth rate is strictly larger than the baseline death rate. Clearly, (1.1) is the classical Lotka-Volterra predator-prey model in slight disguise. As a result, all solutions with positive initial values in S and I are periodic. Notice that without the disease, the population grows exponentially with exponent of $b - \mu$. Hence we may argue that the disease acts as a population control mechanism. In reality, many other mechanisms such as limited food and other resources are at work at same or different times.

A more interesting epidemiology model that capable of generating periodic solutions can be obtained by considering the often implemented isolation or quarantine practice. Quarantine can be either enforced or self-imposed. It can be forced upon on infected individuals by the sickness and/or by community orders. It can be self-imposed by susceptibles in an effort to avoid infection. We introduce below the work of Feng and Thieme (1995) on modeling the recurrent outbreaks of measles and other childhood diseases. They show that isolation (i.e., sick individuals stay at home and have a reduced infective impact) can create self-sustained oscillations provided that the number of per capita contacts is largely independent of the number of individuals present. This means that the bilinear mass action term for disease incidence is modified by dividing it by the number of nonisolated individuals. We divide the total population into susceptible (S), infectives (I), quarantined individuals (Q) and recovered individuals (R). Let $N = S + I + Q + R$ be the total population and $A = N - Q$ be the total of nonisolated population. Then the following simple model of Feng and

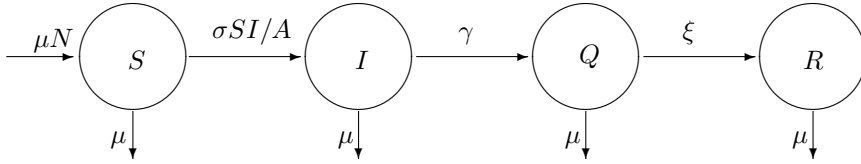


FIG. 1.1. *Flow diagram for model (1.2).*

Thieme with quarantine practice is plausible (see the model diagram Figure 1.1).

$$\frac{dS}{dt} = \mu N - \sigma S \frac{I}{A} - \mu S, \quad (1.2)$$

$$\frac{dI}{dt} = \sigma S \frac{I}{A} - (\mu + \gamma)I, \quad (1.3)$$

$$\frac{dQ}{dt} = \gamma I - (\mu + \xi)Q, \quad (1.4)$$

$$\frac{dR}{dt} = \xi Q - \mu R. \quad (1.5)$$

The model and its assumptions as well as the meaning of each parameter are self evident from the model flow diagram. Observe that the total population again stays constant ($N = S + I + Q + R = N(0)$). Using the methods and similar arguments introduced in the previous lecture, we can show that solutions of (1.2) are also positive for $t > 0$. It can be shown (see Feng and Thieme (1995)) that for some parameter values, the model produces interesting periodic solutions.

2. Periodic solutions of some predator-prey models. Although Lotka-Volterra predator-prey model succeeded in producing periodic solutions that meant to describe the oscillatory fish populations in the Adriatic Sea¹, it also allows solution with small initial conditions in both prey and predator populations to periodically reach unrealistically high and low levels. This undesirable feature is brought by the lack of prey population controls other than the predator. This prompted the addition of a self-regulation term (often called self-crowding term) in the prey equation. The resulting equation takes the form of

$$\begin{cases} x'(t) &= rx(1 - x/K) - bxy, \\ y'(t) &= y(cx - d), \\ x(0) &= x_0 > 0, y(0) = y_0 > 0. \end{cases} \quad (2.1)$$

The above model is referred in the literature as a Lotka-Volterra type predator-prey model. While solutions of this modified model no longer approach unrealistic high or low levels, the addition of this self-regulation term $-rx^2/K$ in the model also leads to the disappearance of interesting periodic solutions. Indeed, a simple application of the Dulac criterion with the Dulac function $1/(xy)$ in the positive cone shows that the above model admits no nontrivial periodic solutions.

¹Vito Volterra was an Italian mathematician who lived from 1860-1940. His son-in-law, Humberto D'Ancona was an Italian biologist who, in 1926, completed a statistical study of fish populations in the Adriatic Sea. D'Ancona asked Volterra if there was a mathematical model that could explain the increase in predator fish and decrease in prey fish which he observed during the World War I period. Within a couple of months, Volterra produced a series of models for the interaction of two or more species. Alfred J. Lotka was an American biologist and actuary who independently produced many of the same models

While there are many aspects of model (2.1) can be debated, a simple modification of the model is to replace the linear (Holling type I) **functional response** (the amount of prey mass killed by a unit of predator in a unit of time) bx in the mass action term $bx y$ by a Holling type II functional response in the form of $bx/(1 + bhx)$. This functional response can be derived from a so-called predator time-budget method. We shall briefly introduce it here. In a given unit of time, we assume that a predator spend T_s time in searching for prey and spend T_h time in handling (eating and digesting) the prey. We in fact also assume that the predator spend time on only one of these two activities. If it takes time h unit time for a predator to handle a unit mass of prey and the amount of prey caught by the predator is proportional to prey density with a proportion constant b (we shall call it the catching efficiency), then $T_h = bhxT_s$. Hence

$$1 = T_s + T_h = (1 + bhx)T_s.$$

Therefore the prey caught by a unit of predator in a unit of time is

$$bxT_s = \frac{bx}{1 + bhx}.$$

For convenience, in the following, we will denote bh by a . It is also customary to assume the predator **numerical response** (the predator mass gain in a unit of time by a unit of predator) With this modification, we obtain the following celebrated Holling type II (also known as Rosenzweig-MacArthur model) predator-prey model.

$$\begin{cases} x'(t) &= rx(1 - x/K) - \frac{bxy}{1 + ax}, \\ y'(t) &= y\left(\frac{cx}{1 + ax} - d\right), \\ x(0) &= x_0 > 0, y(0) = y_0 > 0. \end{cases} \quad (2.2)$$

It can be shown that if $\frac{cK}{1+aK} \leq d$, then model (2.2) has no positive equilibrium and the predator extinction equilibrium $(K, 0)$ is globally attractive. If $\frac{cK}{1+aK} > d$, then an positive equilibrium $E^* = (x^*, y^*)$ exists. It can be shown that E^* is globally attractive if it is locally stable (see Kuang and Freedman, 1988). Moreover, it can be shown that there is a unique limit cycle surrounding E^* in the positive cone if E^* is unstable (see also Kuang and Freedman, 1988). It can be shown and easily observed through simulation that this limit cycle grows in amplitude as one increases the carrying capacity K . Indeed, significant increasing the K value will dangerously push the predator low point of this globally attracting limit cycle very close to the prey axis (extinction of predator), prompting the so-called well-known ‘‘paradox of enrichment’’ (see Rosenzweig, 1969). However, this paradox of enrichment is hardly observed in reality. In other words, this can be viewed as an artifact of the model deficiency in Holling type II predator-prey model. In addition, we also see that the prey density at the positive equilibrium is independent of the carrying capacity K . In other words, enriching the carrying capacity of the prey will not change the prey value at the positive equilibrium. Holling type II predator-prey model thus predicts that a prey population that is severely suppressed (with $x^* \ll K$) will fluctuate violently in density. This is formulated by Luck (1990) as the ‘‘paradox of biological control’’. This again contradicts to the reality where many biological controls are successful.

A reexamination of our Holling type II functional response derivation above suggests that we shall also include the time a predator may spend in guarding its prey

sources and its catches from other predators. This amounts to include a predator interference time of T_i . An intuitive way to quantify T_i is to assume that this time is proportional to the number of predators out there in the field and it happens mainly in the searching process. In other words, we may assume that that $T_i = fyT_s$. This consideration lead to the following so-called predator-dependent functional response

$$\frac{bx}{1_bhx + fy}.$$

This functional response form is named as Beddington-DeAngelis functional response (Beddington, 1975; DeAngelis et al., 1975). With this functional response, we obtain the following Beddington-DeAngelis predator-prey model

$$\begin{cases} x'(t) &= rx(1 - x/K) - \frac{bxy}{1 + ax + fy}, \\ y'(t) &= y\left(\frac{cx}{1 + ax + fy} - d\right), \\ x(0) &= x_0 > 0, y(0) = y_0 > 0. \end{cases} \quad (2.3)$$

It can be shown that this model will no longer have a carrying capacity independent prey density at the unique positive equilibrium E^* and the amplitude of the limit cycle is much smaller when exists. This model was systematically and almost completely studied by Hwang (2004). It can be shown that E^* is globally attractive if it is locally stable and there is a unique limit cycle surrounding E^* in the positive cone if E^* is unstable (see Hwang, 2004). In the limiting case where searching time is relatively short, then the time can be divided into the handling time and the interference time among predators. That is $1 = T_h + T_i$. We can assume that the amount of prey can be caught is proportional to the interference time and the prey abundance, and is inversely proportional to the number of predators. Then we have $T_h = hb\frac{x}{y}T_i$ and the prey caught by a predator in a unit of time is $\frac{bx}{y+bhx}$. The resulting functional response $\frac{bx}{y+bhx}$ is equivalent to the function $p(x/y)$ with $p(x) = bx/(1+bhx)$. For this reason, $\frac{bx}{y+bhx}$ is referred as the ratio-dependent functional response. The resulting model is called ratio-dependent predator-prey model which take the form of

$$\begin{cases} x'(t) &= rx(1 - x/K) - \frac{bxy}{ax + fy}, \\ y'(t) &= y\left(\frac{cx}{ax + fy} - d\right), \\ x(0) &= x_0 > 0, y(0) = y_0 > 0. \end{cases} \quad (2.4)$$

This model has stirred considerable controversy in the mathematical population biology community since its debug in 1989 (Abrams and Ginzburg). This controversy arguably has contributed to the recent trend of formulating more mechanistically based population models (Loladze et al., 2000, 2004). For certain plausible parameters, the model (2.4) can generate solutions that confirm the well known Gause's classic observation of mutual extinction in the protozoans, *Paramecium* and its predator *Didinium* (Gause, 1934). The model (2.4) was systematically and globally studied by many researchers. Such work is pioneered by the work of Kuang and Beretta (1998) and to a certain degree, completed by the work of Hsu et al. (2001).

3. A geometric stability criterion for Gause type predator-prey models.

We shall introduce a simple but general geometric stability criterion for Gause type

predator-prey mode of the following form

$$\begin{cases} x'(t) &= xg(x) - yp(x), \\ y'(t) &= cyp(x) - dy, \\ x(0) &= x_0 > 0, y(0) = y_0 > 0. \end{cases} \quad (3.1)$$

Here $g(x)$ and $p(x)$ are assumed to be continuously differentiable. In addition, we assume that they satisfy the following conditions.

(H1): There is a positive constant K such that $g(x) > 0$ for $x \in [0, k]$ and $g(x) < 0$ for $x > K$.

(H2): $p(0) = 0$ and $p'(x) > 0$ for $x > 0$.

To ensure model (3.1) has a positive equilibrium $E^* = (x^*, y^*)$, we assume below that $cp(K) > d$. The Jacobian at E^* is

$$J = J(E^*) = \begin{pmatrix} g(x^*) + x^*g'(x^*) - y^*p'(x^*) & -p(x^*) \\ cy^*p'(x^*) & 0 \end{pmatrix}.$$

Obviously, $\det J > 0$. Since $y^* = x^*g(x^*)/p(x^*)$, we see that $\text{tr}J = g(x^*) + x^*g'(x^*) - y^*p'(x^*) = p(x^*) \frac{d(xg(x)/p(x))}{dx} \Big|_{x=x^*}$. Recall that the prey isocline is the curve defined by $y = xg(x)/p(x)$. Hence we have the following well-known geometric criterion due to Rosenzweig and MacArthur(1963) for the stability of E^* : Assume that the prey isocline has a single peak at $x = P$. Then E^* is stable if $x^* > P$ and E^* is unstable if $x^* < P$.

REFERENCES

- [1] P. A. Abrams and L. R. Ginzburg: *The nature of predation: prey dependent, ratio-dependent or neither?* TREE, **15**, 337-341 (2000).
- [2] J. R. Beddington: *Mutual interference between parasites or predators and its effect on searching efficiency*, J. of Anim. Ecol., **44**, 331-340 (1975).
- [3] D. L. DeAngelis, R. A. Goldstein and R. V. O'Neill: *A model for trophic interaction*, Ecology, **56**, 881-892 (1975).
- [4] Z. Feng and H. Thieme: *Recurrent outbreaks of childhood diseases revisited: The impact of isolation*, Math. Biosci., **128**, 93-130 (1995).
- [5] . F. Gause: *The struggle for existence*, Williams & Wilkins, Baltimore, Maryland, USA, (1934).
- [6] S. B. Hsu, T. W. Hwang and Y. Kuang: *Global Analysis of the Michaelis-Menten type ratio-dependence predator-prey system*, J. Math. Biol., **42**, 489-506 (2001).
- [7] T. W. Hwang: *Uniqueness of limit cycles of the predator-prey system with Beddington-DeAngelis functional response*, J. Math. Anal. Appl. **290**, 1131-122 (2004).
- [8] Y. Kuang and E. Beretta: *Global qualitative analysis of a ratio-dependent predator-prey system*, J. Math. Biol. **36**, 389-406 (1998).
- [9] Y. Kuang and H. I. Freedman: *Uniqueness of limit cycles in Gause-type models of predator-prey systems*, Math. Biosci., **88**, 67-84 (1988).
- [10] Loladze I., Y. Kuang and J. J. Elser: *Stoichiometry in producer-grazer systems: linking energy flow and element cycling*, Bull. Math. Biol., **62**, 1137-1162 (2000).
- [11] Loladze, I., Y. Kuang, J. J. Elser and W. F. Fagan: *Coexistence of two predators on one prey mediated by stoichiometry*, Theor. Popul. Biol., **65**, 1-15 (2004).
- [12] R. F. Luck: *Evaluation of natural enemies for biological control: a behavior approach*, Trends in Ecology and Evolution, **5**, 196-199 (1990).
- [13] M. L. Rosenzweig: *Paradox of enrichment: destabilization of exploitation systems in ecological time*, Science, **171**, 385-387 (1969).
- [14] M. L. Rosenzweig and R. H. MacArthur: *Graphical representation and stability conditions of predator-prey interactions*, American Naturalist, **97**, 209-223 (1963).