4. INSECT-VIRUS MODELS

4.1 Introduction

There are many interactions that can give cyclic dynamics in models: predator-prey interactions, weather-insect interactions, host-parasite interactions, plant-herbivore interactions and host-disease interactions. All the models referred to in the previous chapter, except the one by Wellington et al. (1975), are very general and do not necessarily consider the specific biology of any particular system. Indeed, as they have been formulated, they could be used to explain the population cycles either of insects or of small mammals (except for differences in immunity to disease). Obviously, the biologies of insects and mammals are different in many ways. It would therefore be interesting to examine one hypothesis, or one set of models, in more detail and to apply it to a system and see if the cycles are robust. Here, disease models for insects will be considered. Four existing models will be examined and adapted to to details of tent caterpillar populations.
4.2 Anderson and May

Anderson and May (1980) modelled host-disease interactions using differential equations for continuous overlapping generations of classes of infective ($I$) and susceptible ($S$) insects, and a free-living infective stage of the pathogen ($V$). The model was formulated in three equations. The first is for the susceptibles ($S$). The rate of change of the susceptible population depends on the birthrate ($a$) of all individuals, both susceptible and infected, minus the natural mortality ($d$) of the susceptibles minus the newly infected individuals.

$$\frac{dS}{dt} = a(S + I) - dS - \beta VS$$  \hspace{1cm} (4.1a)

The term $\beta VS$ is the infection rate of a susceptible individual and is proportional to the rate of encounters ($\beta$) between the susceptible and the free-living stage of the pathogen. The change in the infected population ($I$) results from the addition of the newly infected individuals minus dead infected individuals (natural mortality rate ($b$) + disease induced mortality ($\alpha$)). So,

$$\frac{dI}{dt} = \beta VS - (\alpha + d)I.$$  \hspace{1cm} (4.1b)

The third equation is for the free-living stages of the virus ($V$). These infective stages are produced from infected hosts at a rate $\lambda$ and die at a rate $\mu$. They are also lost through ingestion into susceptible and infected hosts. This gives,

$$\frac{dV}{dt} = \lambda I - (\mu + \beta(S + I))V.$$  \hspace{1cm} (4.1c)

Through analysis of this model the authors show that it is possible to produce limit cycle behaviour from the model. The crucial factor in the model is the mortality rate
of the free-living virus ($\mu$). If there were no free-living stage of the virus (so susceptible insects became infected directly from other infected insects), the model does not produce population cycles.

The authors compared the results from this model to the behavior of the larch bud-moth, a lepidopteran that has cyclic dynamics. Given parameter values based on data, the model produced ten-year cycles, the same period as the larch budmoth population shows. However, although the range in the predicted total population was similar to the actual population size (the log of the population size ranged from about 3.5 to 6.5 while the log of the actual population ranged from approximately 2 to 7), the predicted and actual prevalence of infection was quite different (between 30% and 50% in the actual population compared with 80% in the model).

Vezina and Peterman (1985) also used the Anderson and May model (equations 4.1) to model the Douglas-fir tussock moth (*Orgyia pseudotsugata*), another lepidopteran species that has 7-10 year population cycles. They simulated this model and several variations of the model that included more of the specific biology of the moth. The first modification was to make the mortality rates density-dependent by changing the minimum natural death rate $d$ to $d'$ where $d' = d + c(S + I)$ ($c$ is some measure of the severity of density-dependent natural mortality (Vezina and Peterman, 1985)). The disease-induced mortality $\alpha$ becomes $\alpha = m - d'$ where $m$ is total host mortality. The second modification was to add an incubation period between the time an insect became diseased and when it infected others. A new class of organisms that are infected but not yet infectious ($E$) was made. Organisms move into that class when they become
infected and move out at a rate v. The new system of equations is:

\[
\frac{dS}{dt} = a(S + I) - dS - \beta V S \\
\frac{dE}{dt} = \beta V S - (d + v)E \\
\frac{dI}{dt} = vE - (\alpha + d)I \\
\frac{dV}{dt} = \lambda I - (\mu + \beta(S + I))V.
\] (4.2a, 4.2b, 4.2c, 4.2d)

The third model Vezina and Peterman (1985) examined included vertical transmission.

This means that not all the new larvae are susceptible: a proportion \( k \) of them are born infected (and a proportion \( 1 - k \) of them are born susceptible). Thus, equations 4.1 become

\[
\frac{dS}{dt} = aS + a(1 - k)I - dS - \beta V S \\
\frac{dI}{dt} = \beta V I - (\alpha + d)I + akI \\
\frac{dV}{dt} = \lambda I - (\mu + \beta(S + I))V.
\] (4.3a, 4.3b, 4.3c)

Finally, they looked at a model in which all these components were combined.

\[
\frac{dS}{dt} = aS + a(1 - k)I - dS - \beta V S \\
\frac{dE}{dt} = \beta V S - (d' + v)E + akE \\
\frac{dI}{dt} = vE - (\alpha + d')I \\
\frac{dV}{dt} = \lambda I - (\mu + \beta(S + I))V.
\] (4.4a, 4.4b, 4.4c, 4.4d)

Venzina and Peterman (1985) obtained realistic parameter values for the models from field studies reported in the literature and adapted the values to fit into a
continuous-time model (so that all values were calculated per year and there would be discrete generations). They found that it was impossible to obtain cyclic dynamics with appropriate periodicity when realistic parameters were used. They concluded that it was not virus alone that generated population cycles so the models were not an adequate representation of the tussock moth system. They hypothesized that a more successful model would include the interactions between the tussock moth and its enemies and food.

One problem with the models of Anderson and May (1980) and Venzina and Peterson (1985) is that they assume that the transmission of the virus is proportional to the initial densities of susceptible and infected individuals or to the densities of susceptibles and free-living virus. This assumes that each instar is as infectious and resistant as all other instars. Dwyer (1991) performed some experiments in the field with the Douglas-fir tussock moth to determine the transmission coefficient ($\beta$) at different instars. He found that $\beta$ was significantly higher for older instar larvae, indicating that transmission over the entire year is not strictly proportional to the initial numbers of first instar larvae. It is possible that if the models were adapted to account for a changing $\beta$, cycles would occur using realistic parameter values.
4.3 Brown

Brown (1987) created a general simulation model for insect-virus populations. The model was quite detailed and included a five day latent period (between becoming infected and dying and infecting others), an environmental pool of free-living stages of the virus, and a random temperature component that varied insect growth rates. Also, the amount of virus infected larvae produce when they die increased with the age of the larvae. In the model, the probability of becoming infected at any time depends on what Brown called the pathogen burden ('pb') and on the density of the hosts ('de'). The pathogen burden is the number of infectious units that are available to infect hosts per host, or:

\[ pb = \frac{pa}{th} \]  

(4.5)

where 'pa' is the number of available pathogens and 'th' is the total number of hosts. Infection is considered to be a random process so a Poisson distribution is used, and the equation for the probability of infection is

\[ p = 1 - e^{-de*pb} \]  

(4.6)

Notice that if the density ('de') is large, \( p \) will be close to one. Biologically, this simulates greater stress and increased susceptibility at high densities (Brown, 1987). However, \( p \) is independent of the age of the larvae, and thus does not account for any increasing resistance to the disease.

The model simulates overlapping generations (so that on any given day there are insects of all age classes) and adult insects lay several eggs on each of several days. Both susceptible and exposed adults lay eggs, and they have the same fecundity. Even
though this simulation model shows oscillations, the cycles are unstable, with increasing amplitudes (Figure 4).

I adapted this model to the tent caterpillars system by making the following changes.

1. Exposed individuals have a lower fecundity than non-infected individuals. The original model assumed that exposed and susceptible adults would have the same fecundity, represented by the variable ‘ov’. I added a new variable ‘ove’, to represent the fecundity of the exposed individuals, with ‘ove’ < ‘ov’.

2. The disease is carried by a proportion (q) of the eggs laid by the exposed adults (i.e., the larvae will be in the exposed class when they emerge). The rest of the eggs will be susceptible. Thus the equation governing the reproduction,

\[ S_1 = ov \times (S_7 + E_a) \]  \hspace{1cm} (4.7)

becomes

\[ S_{egg} = ov \times S_7 + ove \times E_a \times (1 - q) \] \hspace{1cm} (4.8a)

\[ E_{egg} = ove \times E_a \times q. \] \hspace{1cm} (4.8b)

where:

- \( S_1 \) is the number of susceptible eggs in the original model.
- \( S_7, E_a \) are the susceptible and exposed adults.
- \( S_{egg}, E_{egg} \) are the susceptible and exposed eggs in the modified model.

3. Reproduction only occurs towards the end of the season, with the adults laying all of their eggs in a single batch and then dying. The original model had the adults
Figure 4. Output from Brown's original model, showing unstable oscillations.
laying a few eggs each day of their adult life. Thus, any time an organism becomes
an adult, she would lay a batch of eggs on that day and then die.

4. Only the eggs and the free-living virus survive between seasons. I decided that the
maximum total life expectancy (from egg to adult) for a tent caterpillar was 100
days. This means that at the end of the season, (at the end of day 100), all insects
in all classes except $S_{egy}$ and $E_{egy}$, are killed.

This modified model no longer produced cycles. The simulated population stabilized or
grew exponentially, depending on the birth rates that were used and the reduction in
fecundity attributed to the exposed insects. If there only a small reduction in fecundity
due to infection, the population would be able to grow exponentially due to high fe-
cundity. When there was a larger sublethal effect of virus on fecundity, the population
stabilized.
4.4 Régnière

4.4.1 Original model

Régnière (1984) produced a model which most closely represents forest Lepidoptera. He used difference equations which included vertical transmission of the virus \((k)\), no recovery from infection, and lower fecundity of diseased organisms due to sublethal effects of the virus (Figure 5). Difference equations are more appropriate than differential equations since the insects only reproduce once each year.

In the model, the number of susceptible individuals at the end of the generation can be calculated by examining three categories of organisms:

1. The number of surviving susceptible individuals that did not contact the disease and gave birth to susceptible offspring \(((1 - p_t)cS_t)\). Since the function \(p_t\) describes the proportion of insects that become infected (see equation 4.10), the term \(1 - p_t\) gives the proportion of insects that do not become infected.

2. Those susceptible organisms that contacted the disease but did not pass it on to their offspring \(((1 - k)bp_tS_t)\). This implies that either these organisms were carriers for the virus but they did not transmit it to their offspring, or that these were organisms that were more resistant to the disease.

3. The surviving infected individuals that did not pass the infection to their young \(((1 - k)aI_t)\).

Adding items 1, 2, and 3 leads to the equation:

\[ S_{t+1} = (1 - k)(aI_t + bp_tS_t) + c(1 - p_t)S_t. \]  

\(4.9a\)
Figure 5. A schematic diagram of Régnière's model (1984) (based on the diagram in his paper). In the model, \( a = f_r \sigma_i, b = f_e \sigma_c, \) and \( c = f_s \sigma_s \). The top of the diagram represents generation \( t \) while the bottom represents generation \( t + 1 \).
The parameters $a$, $b$ and $c$ are the net fecundities of infected, exposed and susceptible individuals respectively, and incorporate insect survival, fecundity, and the proportion of eggs that hatch. The net fecundities are related to each other by $a < b < c$, assuming that infected organisms are adversely affected by the disease more than exposed or susceptible insects. Régnière assumed that $a > 0$, so that infected individuals could reproduce. For the population to survive, $c$ must be greater than 1. Vertical transmission is included in the model in the parameter $k$, the proportion of offspring born to exposed or infected mothers, that are infected ($k \leq 1$).

Similarly, the number of new infected individuals is calculated by the number of susceptibles that contacted the disease and gave birth to infected offspring ($kb_{p_t}S_t$), plus the number of infected individuals that also passed the infection to their offspring ($kaI_t$).

$$I_{t+1} = k(aI_t + b_{p_t}S_t)$$ (4.9b)

The proportion of insects becoming infected, $p_t$, is based on assumptions of random movements of individuals and increases with the number of infecteds present.

$$p_t = (1 - e^{-hI_t})^\beta$$ (4.10)

where $h$ is the rate at which the healthy larvac contact disease propagules (Régnière, 1984). The parameter $\beta$ determines the aggregation of disease propagules near the diseased insects, where $\beta \geq 1$. (Low $\beta$ implies that the disease is highly concentrated near insects while high $\beta$ indicates that the disease is more widely dispersed (Régnière, 1984).) The notation is slightly changed from the original paper: Régnière used $x$ and $y$ where I have used $I$ and $S$ for clarity.
This model produces cycles with the eight to twelve year period similar to those observed in natural populations. However, there are aspects of the biology of forest Lepidoptera that are not yet incorporated. I modified the model to add some of these aspects. In general, as the model became more detailed, it lost any tendency to cycle.

4.4.2 Modifications

Organisms that become infected at hatching probably die before reproducing (since infected larvae usually live for about ten days (Entwistle, 1983a)). Therefore, I modified the original model to include this fact:

\[ I_{t+1} = k h p_s S_t \quad (4.11a) \]

\[ S_{t+1} = (1 - k) h p_s S_t + c (1 - p_t) S_t \quad (4.11b) \]

As shown in Figure 6, it was still possible to get ten to twelve year cycles in the population, but with a narrow range of parameter values. Specifically, stable cycles (of any length) primarily occur only when \( \beta \) is close to one (the minimum value of \( \beta \) as indicated by Régnière (1984)), meaning that the disease is highly concentrated near insects and the insects have a greater probability of contacting the disease. Biologically this would be the same as increasing the amount of virus infected larvae produced when they died, or reducing the amount of virus needed for infection of a larva. When \( \beta \) is larger than one, (implying that the disease is more scattered), there is a much narrower range of parameters that will cause cycles. In many instances, the infected organisms quickly die out and only the susceptible ones are left. Because the disease is more widely dispersed, the chance of becoming infected is very low. Consequently, fewer infections occur and the virus disappears.
Figure 6 demonstrates the effect of changing three of the parameters. In all cases, c, the net fecundity of susceptible organisms, is held constant, and is equal to the upper value of b, the net fecundity of exposed individuals. If c were to change, the graphs would also change, although the general pattern of the regions with cycles would remain the same. Figure 6 also shows that it is not necessary to have any sublethal effect of the virus (reduced survival or fecundity, characterised by \( b < c \)), for oscillations to exist.

In Figure 6, a distinction is made between cycles and persistent oscillations (for mathematical, not biological reasons). Cycles are those in which each oscillation is identical with all others. Persistent oscillations are those in which the amplitude of each cycle varies in a consistent, repeatable manner (for example, every third oscillation is identical), and the periodicity is relatively consistent. These persistent oscillations are stable to small perturbations (i.e., if the population is perturbed, it will quickly return to the same periodicity and amplitude as before the perturbation). Either of these types of cycles could be relevant to natural populations, especially since in natural populations each oscillation is not exactly identical to all the others.

To get some idea of the effect of various parameters on the period and amplitude of the cycles the model was simulated using one set of parameter values. Then one of the parameter values was increased and the model was simulated again. Table 1 shows a summary of the effects of increasing each of the parameters on the cycle period and amplitude, and any constraints on the values that the parameters can take. One important constraint is the value of c, the net fecundity of susceptibles, which must be greater than one for the population to survive. If c is less than one, the population is
Figure 6. An example of the effects on the existence and period of cycles in the modified Régnière model (equations 4.11), as parameters $k$ (vertical transmission), $b$ (net fecundity of exposed individuals), and $\beta$ (degree of clumping of disease) are varied. The numbers show the period of the cycles that are between eight and twelve years. In figure A) $\beta = 1$, in B) $\beta = 2$, and in C) $\beta = 3$. In all cases, $c = 3$. 

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not reproducing enough to replace itself, even if there was no virus present.

Figure 7 gives an example of the oscillations that are seen before and after $b$, the net fecundity of disease-exposed insects, is increased. When $b = 1$, exposed organisms give birth to exactly enough organisms to replace themselves since few of them survive but those that do survive give birth to a large number of offspring (see Table 3). As can be seen, the cycles become lower and shorter. This occurs because there are many more infected organisms being born, raising the infection rate for the susceptible organisms. Consequently, the total population will not become as dense as when this fecundity rate is lower. The cycles (as graphed on a log scale) look very different than natural population cycles, both in range and in pattern.

Next, a free-living virus pool, $V$, was added to the modified model (equations 4.11). The virus has a survival rate, $\sigma_v$, and $w$ propagules are removed per susceptible as the susceptibles become infected. Infected individuals contribute to this pool when they die and release $\gamma$ virus propagules. Thus, the equation for the virus is:

$$V_{t+1} = \sigma_v V_t + \gamma I_t - w p_t S_t.$$  \hspace{1cm} (4.12)

Since organisms are infected either from the free-living virus or from direct exposure to the dead infected individuals, the probability of contacting the infection should be changed to reflect this.

$$p_t = (1 - e^{-h I_t - h_v V_t})^3$$  \hspace{1cm} (4.13)

where $h_i$ and $h_v$ replace the $h$ of equation 4.10, and concern the rate at which a susceptible contacts the virus.
<table>
<thead>
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<th>Meaning</th>
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<td>$-$</td>
<td>$&lt; c$</td>
</tr>
<tr>
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Table 1. Effect of increasing different parameters in the modified Régnière models (equations 4.11 and 4.12). A ‘$+$’ indicates that the period length or amplitude height increased as the parameter increased, a ‘$-$’ indicates that it decreased and an ‘$=$’ that it did not change. The range indicates any constraints on the parameter, if any. All parameters are $\geq 0$. 

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Figure 7. Effect of varying net fecundity of virus-exposed individuals on equations 4.11, the modified Régnière model. Increasing $b$ gives lower amplitude, shorter period cycles.