2. Sub-lethal virus affecting fecundity levels.

If the virus does not affect fecundity, then \( f_s = f_c \). However, cycles never appear under this circumstance, even if there is a large sublethal effect on survival in the larval stage. It is the lowered fecundity that is the primary cause of the population decline in the model. In fact, the larger the difference between \( f_s \) and \( f_c \), the more likely that cycles will occur (Figure 12). It is not necessary to also have the sublethal effects on survival during the larval stage in order to produce cycles.


If there is not enough virus present in the environment (because the survival rate is too low or because it does not survive over the winter), it allows the population to escape and grow to some stable level. Conversely, if the survival rate of the virus is too high, the population could start to show erratic cycles or become extinct. The parameter \( k \) controlling vertical transmission has a similar effect in that as it increases, more virus will move into the next generation and keep the population lower.

4. Infection rate.

The fact that the analysis and the results disagree implies that the fact that the probability of infection varies with time and density is crucial to the production of cycles. Another critical factor included in the infection rate is the degree of clumping of the virus near the larvae, something that is difficult to measure.
7. CONCLUSIONS

Models were used to test the hypothesis that the eight to ten year population cycles that are found in tent caterpillars could be produced by a virus. Four insect-virus models from the literature were examined. Each of these models initially showed that population cycles were possible. All of these models lost their tendency to cycle if realistic parameter values were used. The same results were found by Vezina and Peterman (1985) who adapted to Anderson and May (1980) model for biologically reasonable details of the douglas-fir tussock moth.

Since none of the existing models produced reasonable cycles, I created a new virus-insect model with particular attention to the tent caterpillar system. This model included many characteristics about the biology of tent caterpillars and the interactions between the larvae and the virus. One of these characteristics not included in any previous model examined was that the caterpillars’ susceptibility to the virus changes as the larvae become older. Thus, the probability of becoming infected varies during the larval growing season, depending on how much virus is available and the age of the larvae. Heterogeneity was incorporated in my model by varying the amounts of virus available to caterpillars.

The model was examined both through simulations and through analysis of a simplified version of the model in which the probability of becoming infected was held constant. This analysis showed that no cycles were possible in the simplified version of the model. However, simulations of the model showed that cycles could occur. This implies that is important that the probability of becoming infected ($p$) varies with the
amount of virus and number of larvae present.

Cycles occurred when using relatively realistic parameter values and the period of the cycles produced by the model corresponded well with those of actual cycles. However, the simulated oscillations showed that the average clutch size started to decline before the population size decreased and survival rate decreased at the same time as the population. In natural populations, average clutch size does not go down until the first year of the population decline while the survival rate decreases before the population decline. Thus there is a difference of one to two generations between the simulated and actual cycles of clutch size and survival rates. There are three explanations for this:

1. There are aspects of the biology of the virus or the virus-insect interaction that have not yet been discovered but that are crucial to the character of the cycles.

2. There is some factor or factors, not including virus, that is or are governing or influencing the cycles.

3. Virus works with some other component to produce the patterns seen in the cycles.

Each of these arguments will be examined in turn. First, there may be some crucial, as yet unknown biology about the virus or the manner in which the virus interacts with the insects. This conclusion affects how the model is actually constructed, so under this conclusion, it is still possible that virus is the single cause of population cycles and that the only reason that proper oscillations have not yet been produced is that something is missing or inaccurate.

This unknown biological factor could involve either interactions that are occurring or the measurement of key parameters. In the first case, it is possible that the virus is
transported between tents in some manner (by parasites or wind, for example) or that the virus has some other detrimental effect on exposed larvae besides reducing adult fecundity. Also, little is known about the free-living stage of the virus. As mentioned previously, there is some debate as to how long the virus can live in the environment, and whether the free-living form is available to first instar larvae. Finally, some of the mechanisms in the model, such as the probability of becoming infected, may change as the number of organisms increases. For example, as the population size grows, the larvae will be more likely to encounter larvae or virus from another tent and so increase their likelihood of becoming infected. The probability of becoming infected would also change if the susceptibility of the larvae changed during the cycle. Research in these areas may produce different or additional results than are portrayed in the current model.

The second area included in the first conclusion is the measurement of various parameter values that seem to govern the interactions. Many aspects of the biology of the virus are difficult to observe or to measure. For example, often it is not possible to tell if caterpillars have been infected by the virus until after they have died. Therefore, it is difficult to measure transmission and infection rates. As a consequence, the effect of virus on insects that have been exposed to the disease but have not died from it, is not well understood. Since there is always variability among individuals in the field, if different larval mortality rates or adult fecundities are measured, it is difficult to determine why these differences arose, or how much of the difference may be attributed to virus (e.g., are two values different due to differences in environment or due to a sublethal effect of virus?). The survival rate of the virus and the degree of clumping of
the free-living virus near larvae are both critical parameters in the production of cycles, but are almost impossible to determine. These are all aspects of the biology that can be studied further and it is possible that when searching for clarity in the determination of parameter values, additional information about interactions between the virus and the larvae will be discovered.

The second possible explanation states that there may be some other factor or factors, aside from virus, that is or are acting to create the cycles. Many of these other possible components, such as plant quality, weather, genetics, or parasites, have been modelled before by other researchers and are discussed in Section 4.2. None of the hypotheses seem entirely plausible for the tent caterpillar case unless one of the factors behaves similarly to the virus in its effect on the larvae (such as reducing fecundity). For example, if a mother experienced bad growing conditions, possibly from weather or food, she may have reduced fecundity or her offspring may have reduced survival. It is more probable that it is one or more elements acting together to produce cycles. For example (although this has not been modelled), it may be that weather and parasites combine to produce oscillations. Few models that have been produced examine the effect of multiple factors, excluding virus, on insect populations.

The third explanation is essentially a subcase of the second since it suggests that it may be virus acting with at least one other system component to create the cycles. These various combinations could include possibilities such as weather and virus, parasites and virus, plant quality and virus or even weather, plant quality and virus. It also may be that at high densities the larvae become much more susceptible to a variety of mortality
factors, including disease and that such mortality factors would act to decrease survival rates as are seen in the natural populations.

Several models have examined the possibility of a multiple component interaction producing cycles (Berryman, pers. comm.; Hochberg et al., 1990; Wellington, et al., 1975). Berryman maintains that the cycles are caused by the action of the parasites and that the virus just increases the magnitude of the cycles. Hochberg et al. (1990), show that cycles are possible when both a parasite and a pathogen are present in the system. As discussed in Section 3.3.2, Wellington et al. (1975) produced a model that combined effects of parasites, virus and weather. That model could be further tested using more recently collected data. These models provide initial support for the hypothesis that more than one factor creates the cycle. Further modifications of the models are necessary and some support from a natural system is still needed to see if the models are valid for the tent caterpillar system.

The results from my model also support this third explanation. Using only virus, it was possible to produce cycles with the right period. The model cycles differed from natural cycles in that the population decline was caused by a virus build-up which led to more exposed individuals and lower average fecundities, leading to a population decline. In the natural system, the average fecundities do not decrease until after the population declines and it is lowered survival rates that seem to influence the population decline. Therefore, if my model is a reasonable representation, some other factor (not included in the model) may be working with the virus to reduce population levels before the virus can reduce the average fecundity. In particular, some component in the natural
system must be acting to reduce survival without affecting fecundity.

The new model, like many others, is a purely deterministic model, and assumes that all parameter values such as survival rates and basic fecundities remain constant from year to year. In nature, this is obviously not the case. There are always stochastic events occurring which may increase or decrease any of these values, or change some of the relationships. A valid deterministic model should not be sensitive to small changes in parameter values. If the stochastically induced changes are small, many of the results that have been discussed remain similar but instead of all the results being smooth, there is variation around the basic lines. However if these changes are large, the deterministic model no longer produces similar results and is not an adequate representation of the real world. Finally, the use of deterministic models makes it easy to explore various hypotheses and to see the effect of various changes or assumptions. It would be possible to add to and to adapt this model to further examine some of the above explanations.

There are always more modifications that can be made to any model and more possibilities that can be explored. One obvious change that could be made to my model is to alter the clumping parameter $\beta$. It is likely that in a natural population, $\beta$ does not remain constant. At low populations, virus will be confined to the areas near the tents and $\beta$ would be low. However, at high populations, tents and caterpillars are abundant and there are more interactions between caterpillars of different tents. The virus will then be less likely to be as clumped near particular tents, and the parameter $\beta$ may be higher.

Another modification to the model would be to add some other factor that affects
the tent caterpillars, such as parasites, to see if the addition of this factor would cause
the population to cycle in the same patterns as are seen in nature. Ideally, this factor
would act to reduce survival levels, but not fecundity, near the peak of the cycle. It is
likely that simply adding further detail to the virus-insect interactions would not have
this effect. Finally, since this model is developed specifically for tent caterpillars, it
would be interesting to adapt it for other Lepidoptera to see if any of the results could
be generalized.

The results from the model also suggest key parameters that ideally should be
measured in the field. Two of these are related to the free-living virus. If the incidence
of free-living virus (i.e., where it is found) could be measured, the clumping parameter
$\beta$ could be estimated (see Krebs, 1989 pp81–89). Another key parameter was the
survival rate of the free-living virus, both during the larval growing season and during
the winter. This parameter is also key in Hochberg et al.'s (1990) model for insect-
pathogen interactions. Other parameters that it would be useful to measure would be
the vertical transmission rate (what proportion of a mother’s offspring are infected),
and the fecundity of females exposed as larvae.
7. LITERATURE CITED


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Appendix 1: ANALYSIS OF MODELS

A1.1 Introduction
Many differential models and difference models can be analyzed easily by following the same basic steps in both types of systems:
1. Find the null clines and steady states of the equations.
2. Linearize about each steady state and find the Jacobian and eigenvalues of the Jacobian for each.
3. From the eigenvalues, determine the conditions under which each steady state is stable.

In this section, I analyze two of the models discussed in Chapter 2, the Lotka-Volterra equations and the Nicholson-Bailey model. All analyses are based on Edelstein-Keshet (1986).

A1.2 Lotka-Volterra
The Lotka-Volterra model (equations 3.6) can be analyzed using the steps mentioned above.
1. Find and graph the null clines and steady states of the equations:
   - Null clines: These are found by setting the left-hand side of each of the equations to zero. So equation 3.6a becomes:
     \[ 0 = rN - bPN \]  \hspace{1cm} (A1.1)
     which implies:
     \[ N = 0 \text{ or } P = r/b. \] \hspace{1cm} (A1.2)
   Equation 3.6b gives:
     \[ 0 = cPN - dP \]  \hspace{1cm} (A1.3)
     which implies
     \[ P = 0 \text{ or } N = d/c \] \hspace{1cm} (A1.4)
   - Steady states: These are found wherever a null cline from each of equations A1.2 and A1.4 cross (but not where two null clines from the same equation cross). For the Lotka-Volterra equations, there are two such equilibria:
     trivial: \( (N, P) = (0, 0) \)
     non-trivial: \( (N, P) = (d/c, r/b) \)

2. Find the Jacobian and eigenvalues for each of the steady states:
   - at \( (0, 0) \): Jacobian:
     \[
     \begin{bmatrix}
     r & 0 \\
     0 & -d
     \end{bmatrix}
     \] \hspace{1cm} (A1.5)
     The eigenvalues are: \( r \) and \(-d\). Since one eigenvalue is greater than zero and one is less than zero, \( (0, 0) \) corresponds to a saddle point.
   - at \( (d/c, r/b) \): Jacobian:
     \[
     \begin{bmatrix}
     0 & -bd/c \\
     cr/b & 0
     \end{bmatrix}
     \] \hspace{1cm} (A1.6)
Here the eigenvalues are: \( \pm i \sqrt{r\delta} \). Notice that the real part of the eigenvalues is 0, so the equilibrium is neutrally stable with oscillations about it.

### A1.3 Nicholson-Bailey

Next, the Nicholson-Bailey model (equations 3.11) for insect-parasitoid systems will be analyzed following the steps above.

1. **Null Clines and Steady States**
   
The null clines of difference equations can be found, by definition, when the population is not changing or \( N_{t+1} = N_t \). Thus, solving for the isoclines in equation 3.11a leads to the conditions:

\[
\bar{N} = 0 \quad \text{or} \quad \bar{P} = \frac{\ln r}{a}
\]  

(A1.7)

while equation 3.11b gives the isocline:

\[
N = \frac{P}{c(1 - e^{-aP})}
\]  

(A1.8)

Thus, there are two steady states:

\[
(\bar{N}, \bar{P}) = (0, 0)
\]  

(A1.9)

and

\[
(\bar{N}, \bar{P}) = \left( \frac{r \ln r}{ac(r - 1)}, \frac{\ln r}{a} \right).
\]  

(A1.10)

The first, equation A1.9, is the trivial equilibrium while equation A1.10 is the non-trivial equilibrium.

2. **The Jacobian**
   
The general matrix is

\[
\begin{bmatrix}
  e^{-aP} & -ae^{-aP} \\
  c(1 - e^{-aP}) & e\bar{N}e^{-aP}
\end{bmatrix}.
\]  

(A1.11)

When evaluated at the trivial equilibrium (A1.9), matrix A1.11 becomes:

\[
\begin{bmatrix}
  r & 0 \\
  0 & 0
\end{bmatrix}.
\]  

(A1.12)

Clearly the eigenvalues of this matrix are \( r \) and 0. In difference equations, a steady state is stable if all the eigenvalues of the Jacobian evaluated at that steady state are between -1 and 1. Thus, in this case, the trivial equilibrium is stable if \( r < 1 \). This means that the population will die out if \( r < 1 \). The second case is more complicated. When the Jacobian is evaluated at the non-trivial equilibrium (A1.10), matrix A1.11 becomes:

\[
\begin{bmatrix}
  1 & -\frac{r\ln r}{c(r-1)} \\
  \frac{\ln r}{r(r-1)} & \ln r
\end{bmatrix}.
\]  

(A1.13)
So, let

\[ \beta = 1 + \frac{\ln r}{(r - 1)} \]  \hfill (A1.14)

and,

\[ \gamma = \frac{r \ln r}{(r - 1)}. \]  \hfill (A1.15)

Now the stability condition given by Edelstein-Keshet (1986) for difference equations, \(2 < 1 + \gamma < |\beta|\), can be checked. It is easy to show (Edelstein-Keshet, p82, 1986) that the first half, \(2 < 1 + \gamma\), is true only if \(r < 1\) while the second part, \(1 + \gamma < |\beta|\), holds only if \(r > 1\). Therefore, the non-trivial equilibrium is never stable (and if the trivial equilibrium is also unstable, the population will show increasing, unstable, oscillations).
Appendix 2: ANALYSIS OF THE MODEL

A2.1 Introduction
In this appendix, I provide the details for the analysis of the new model (equations 5.1). In the first part, I derive an approximate reproductive rate for the virus. In the second part, since the techniques for analyzing systems of equations are already mentioned in Appendix 1, the equations are simplified and are put into a form for which these techniques can be used, and the stability of the system determined. The third section describes some of the techniques that I used to attempt to numerically determine the stability of the system and the possible existence of cycles.

A2.2 Virus reproductive rate
The first thing that can be calculated is the reproductive rate for the virus. After substituting equation 5.1c into equation 5.1d, the basic equation for the virus becomes:

\[ V_{y,t+1} = \sigma_v V_{y,t} + (\gamma t q - w_t) p_t (\sigma_s S_{y,t} + \sigma_e E_{y,t}) \]  

(A2.1)

where, as before,

\[ p_t = 1 - (1 + \frac{V_{y,t}}{\beta w_t H_{y,t}})^{-\beta}. \]  

(5.3)

Equation A2.1 is non-linear, due to the form of \( p_t \). This makes any analysis difficult. Therefore, to simplify the analysis, \( p_t \) can be linearized about \( V = 0 \) to get:

\[ p_t \approx \frac{V_t}{w_t H_{y,t}}. \]  

(A2.2)

Substituting A2.2 into A2.1,

\[ V_{t+1} \approx \sigma_v V_{y,t} + \frac{1}{w_t H_{y,t}} (\gamma t q - w_t) (\sigma_s S_{y,t} + \sigma_e E_{y,t}) \]  

(A2.3)

and rearranging the terms gives:

\[ V_{y,t+1} \approx R_t V_{y,t} \]  

(A2.4)

\[ R_t = \sigma_v + \frac{(\gamma t q - w_t) (\sigma_s S_{y,t} + \sigma_e E_{y,t})}{w_t H_{y,t}} \]  

(A2.5)

where,

\[ H_{y,t} = S_{y,t} + E_{y,t} + I_{y,t} \]  

(A2.6)

Since \( p_t \) was linearized about \( V = 0 \), A2.4 is valid only when the amount of virus is small.

In theory, it would be possible to find other expressions for the reproductive rate of the virus that would be accurate at different amounts of virus. This would be done by linearizing \( p \) about some other steady state (where none of the populations are changing). In practice, this is not possible analytically without making other major simplifications to the model.

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A2.3 Equilibrium Analysis

To determine analytically the behaviour of the system near equilibrium the model must first be simplified. Since it was the term \( p_t \), that was creating the difficulties in analysis, I simplified the model by assuming that \( p \) remained constant. The analysis can then be easily performed on the adult stage of the model, by combining all tents. Therefore, merging equations 5.8–5.10, the total numbers of organisms are:

\[
S_{y+1,0} = \frac{d_y}{2} \left( f_x S_{y,0} + (1 - \kappa) f_e E_{y,0} \right)
\]

(A2.7a)

\[
I_{y+1,0} = \frac{d_y}{2} \kappa f_e E_{y,0}
\]

(A2.7b)

\[
T_{y+1} = \frac{d_y}{2} (S_{y,0} + E_{y,0})
\]

(A2.7c)

and, as before, \( d \) is given by

\[
d_y = 1 - \frac{T_y}{\alpha + T_y}.
\]

(5.7)

It follows from the assumptions in the model that the number of adults in year \( y \) is dependent on the number of young starting the same year, and, due to the simplification, that the dependence is linear. Thus,

\[
S_{y,0} = x S_{y,0}
\]

(A2.8a)

\[
E_{y,0} = z S_{y,0}
\]

(A2.8b)

where \( x \) and \( z \) are independent of the state variables. (The exact formulas for \( x \) and \( z \) are not important for the stability calculations but are shown in equations A2.19 and A2.22 and discussed further at that place.) Substituting equations A2.8 into equations A2.7 and dropping the \( t \) subscript since \( t = 0 \) on both sides of the equation, gives:

\[
S_{y+1} = \frac{d_y}{2} (f_x x + (1 - \kappa) f_e z) S
\]

(A2.9a)

\[
I_{y+1} = \frac{d_y}{2} \kappa f_e z S
\]

(A2.9b)

\[
T_{y+1} = \frac{d_y}{2} (x + z) S
\]

(A2.9c)

Using the standard techniques demonstrated in Appendix 1.3 (step 1), the equilibrium values for this system (equations A2.9) can be found at:

\[
(S, I, T) = (0, 0, 0)
\]

(A2.10)

and at,

\[
S = \frac{\alpha \psi (\psi - 2)}{2(x + z)}
\]

\[
I = \frac{\kappa f_e z \alpha (\psi - 2)}{2(x + z)}
\]

(A2.11)

\[
T = \frac{\alpha \psi}{2} - \alpha
\]

where,
\[
\psi = f_s x + (1 - \kappa)f_e z. \quad (A2.12)
\]

The term \( \psi \) represents the amount of reproduction of the organisms.

The second equilibrium, given by equations A2.11, does not exist if density-dependence was not present. To see this, the density-dependence term \( d_y \) in equations A2.9 would be replaced with the constant 1. If the equilibrium of A2.9 was calculated as before, the only possible steady state would be the trivial one given in equation A2.10.

The Jacobian for the system (equations A2.9) is

\[
J = \begin{bmatrix}
\frac{\alpha \psi}{2(\alpha + T)} & 0 & -\frac{\alpha \psi}{2(\alpha + T)^2} \\
\frac{\alpha \kappa f_s z}{2(\alpha + T)} & 0 & -\frac{\alpha \kappa f_s z}{2(\alpha + T)^2} \\
\frac{\alpha (x + z)}{2(\alpha + T)} & 0 & -\frac{\alpha (x + z)}{2(\alpha + T)^2}
\end{bmatrix} \quad (A2.13)
\]

Substituting the trivial equilibrium (equation A2.10) into matrix A2.13, and calculating the eigenvalues of the resulting matrix, it can be seen that the eigenvalues are 0, 0, and \( \psi/2 \). Similarly, the eigenvalues of the Jacobian evaluated at the non-trivial equilibrium (A2.12) are 0, 0 and 2/\( \psi \). In Appendix 1.3, it was noted that if \( \lambda_i \) is an eigenvalue, the condition for stability of an equilibrium is that \( |\lambda_i| < 1 \) for all \( i \). Clearly, 0 < 1, so the stability of each eigenvalue depends on the value of \( \psi \). There are two possibilities:

\[
\psi \leq 2 \Rightarrow A2.10 \quad \text{stable} \quad (A2.14)
\]

\[
\psi > 2 \Rightarrow A2.11 \quad \text{stable} \quad (A2.15)
\]

Cycles are not possible in either case. As \( \psi \) approaches 2 from above, the non-trivial equilibrium point approaches the trivial one, until, when \( \psi = 2 \), the non-trivial equilibrium equals the trivial one. For cycles to be possible, either both equilibria must be saddle-nodes, in which case a saddle node connector cycle may be present, or at least two of the eigenvalues of a steady state must be \( |\lambda_i| = 1 \), in which case a Hopf bifurcation leading to limit cycles may be present (Guckenheimer and Holmes, 1983).

The virus \( V \) can also be included in the analysis. Following the same simplification process that was done above to change equations A2.8 into A2.10, the amount of virus from year to year is

\[
V_{y+1} = \sigma_s^0 V_y + \omega S_y + \frac{d_y}{2} \gamma \kappa f_e z S_y \quad (A2.16)
\]

where \( \omega \) is some combination of parameters only, and is independent of all the variables. (As with \( x \) and \( z \), the exact form of \( \omega \) is not important to the stability calculations.) When the virus (in the form of equation A2.16) is included in the analysis, the only change in the results is that the resulting Jacobian has another eigenvalue that equals \( \sigma_s^0 \). Since this is always less than one, the stability conditions given in equations A2.14 and A2.15 do not change.

The parameter \( p \) was assumed to be constant for the purposes of doing this analysis. However, the value of \( p \) affects the results. To see what happens as \( p \) is varied, the forms
for $x$ and $z$ must be calculated. They are found by solving the difference equations for the within-year dynamics.

$$S_{y,t+1} = \sigma_s(1 - p)S_{y,t}$$  \hspace{1cm} (5.1a)

becomes

$$S_{y,t} = (\sigma_s(1 - p))^t S_{y,0}.$$  \hspace{1cm} (A2.17)

Equation 5.1a could not be solved explicitly if $p$ were dependent on any of the other model variables. Since there were six time-steps for this part of the model, let $t = 6$. Also, the survival from the pupal stage must be included. So, equation A2.17 becomes

$$S_{y,0} = (\sigma_s(1 - p))^6 \sigma_p S_{y,0}$$  \hspace{1cm} (A2.18)

This is now in the form of equation A2.9a so clearly

$$x = (\sigma_s(1 - p))^6 \sigma_p.$$  \hspace{1cm} (A2.19)

To calculate $z$, the same procedure is followed, using equation A2.17, and remembering that no organisms are born exposed ($E_{y,0} = 0$). Equation 5.1b can be rearranged to give

$$E_{y,t+1} = p(1 - q)\sigma_s S_{y,t} + (1 - pq)\sigma_e E_{y,t}.$$  \hspace{1cm} (A2.20)

This then becomes

$$E_{y,t} = \left( \frac{\sigma^6_s (1 - p)^6 - (1 - pq)^6 \sigma^6_e}{\sigma_s(1 - p) - (1 - pq)\sigma_e} \right) p(1 - q)\sigma_s S_{y,0}.$$  \hspace{1cm} (A2.21)

After allowing for pupal mortality, and noticing that equation A2.21 is in the same form as equation A2.9b, it is clear that

$$z = \left( \frac{\sigma^6_s (1 - p)^6 - (1 - pq)^6 \sigma^6_e}{\sigma_s(1 - p) - (1 - pq)\sigma_e} \right) p(1 - q)\sigma_s \sigma_p.$$  \hspace{1cm} (A2.22)

Upon examination of $x$ and $z$ given in equations A2.19 and A2.22 and shown in Figure 18, it can be seen that $x > z$ for small $p$ (since all parameters involved in equations A2.19 and A2.22 are $\leq 1$). As $p$ approaches one (its upper bound), $x$ goes to 0 and $z$ becomes small. Since $\psi$ depends on $x$ and $z$, it will also be small, less than 2 (unless $f_e$, the birth-rate of exposed mothers is very high). As $p$ becomes small, $\psi$ will grow. In general, there is a critical value, $p_c$ which corresponds to the case $\psi = 2$. If $p \geq p_c$ then the population will die out while if $p < p_c$ the non-trivial equilibrium is stable (see Figure 17). There is no explicit formula for $p_c$. Thus, the value of $p$ that is chosen affects the level at which the population stabilizes, and even whether or not the population will survive. Obviously, the other parameters involved in $\psi$ will also help determine what value $\psi$ has. Similar analysis could be done for the other parameter values, but since the results from these other analysis would also be dependent on $p$ having a constant value, and would have less meaning in the full model, the analysis will not be presented.
Figure 18 Parameters $x$ and $z$ versus the infection probability, $p$. Parameters are: $\sigma_s = .93$, $\sigma_c = .37$ and $q = .5$ (the base values given in Table 5).
A2.4 Stability: numerical analysis

Since the general analysis performed in section A2.3 is necessarily inaccurate due to the simplification that was made, I also attempted some numerical analysis of the full model. As with the previous analysis, if a steady state can be found, the eigenvalues of the Jacobian of the matrix will describe the behaviour of the populations near the steady state. One way for cycles to occur is to have a Hopf bifurcation. The first criterion for finding such a bifurcation is that for some set of parameters there must be at least one pair of complex conjugate eigenvalues with $|\lambda_i| = 1$ (Guckenheimer and Holmes, 1983). The bifurcation will arise at this point.

In order to find the eigenvalues, it was first necessary to find a steady state and to calculate the Jacobian at this point. Initially, I found a steady state by simulating the model until the population stabilized (the population size no longer varied). I then took the partial derivatives of the model at this steady state and created a Jacobian matrix. Next, I used an eigenvalue finding program (Press et. al., 1988, pp 387-393) to find the eigenvalues of the Jacobian.

In practice, there was a major problem with computer generated error. The method that I used to find the eigenvalues stated that the error would be on the order of the euclidean norm of the Jacobian. This was quite large (sometimes up to 80%). In order to find a Hopf bifurcation, it is important to know when the eigenvalues change from less than one to greater than one. Having large error makes it impossible to find this point.

The was evident from the simulations that I did. For example, if the steady state is stable, the magnitudes of the eigenvalues should all be less than one. The method I initially used to find the steady state (by running the model on a computer until the population size no longer changed) guaranteed that the steady state was a stable one. Simulations perturbing the population away from the steady state confirmed this since the population always returned within three generations. However, the eigenvalues that were found were not all less than one. If the true value of an eigenvalue was near one, even a small amount of error could be enough to make it larger than one, or to create a complex value when the true value was real. This error made this technique useless and no further numerical analysis was performed using this or any other method.