SIMULATION MODELS OF THE POPULATION DYNAMICS OF A NUCLEAR POLYHEDROSIS VIRUS AND ITS HOST, THE DOUGLAS-FIR TUSSOCK MOTH, ORGYIA PSEUDOTSUGATA.

by

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Simulation models of the population dynamics of a nuclear polyhedrosis virus and its host, the Douglas-fir tussock moth, Orgyia pseudotsugata

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ABSTRACT

Outbreaks of the Douglas-fir tussock moth, Orgyia pseudotsugata (McDunnough), have recurred periodically, at 7 to 10 year intervals, since the first recorded observation in 1916 in Chase, B.C. The decline of outbreaks in California, Arizona and British Columbia has been attributed to a nuclear polyhedrosis virus (NPV). The association between the Douglas-fir tussock moth and its viral disease is chosen to test the hypothesis that microparasites are responsible for the periodic population fluctuations of the insect. The test is done using Anderson and May's model and variants thereof. The parameter values for the model are derived from published data and from a laboratory experiment. The basic model is expanded to include density-dependent mortality, vertical transmission, incubation period and the effect of random fluctuations on the growth rate. Sensitivity analysis conducted for each model disclosed that none of the versions generated the observed behavior of the Douglas-fir tussock moth in the field. The periodicity of the outbreaks in field populations cannot be explained solely by the dynamics of the viral disease because the virus is too short-lived and the growth rate of the insect population too high. Therefore, other processes are likely to influence the period of the cycles and the density of Douglas-fir tussock moth populations.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

I. Introduction

Insect populations often exhibit spectacular increases and declines in density over very short time. Attempts to explain the sudden release of a population from endemic to epidemic level and its subsequent demise have led to hypotheses based upon fluctuations in weather patterns (Greenbank, 1956; Watt, 1968; Ives, 1973), insect-plant relationships (Baltensweiler, 1964; White, 1974, 1976, 1978; Dempster & Pollard, 1981), and the interaction between the insect and its natural enemies (May, 1976; Hassell,1978).

The climatic release hypothesis postulates that favorable weather conditions will promote population growth to outbreak levels as the insects increase beyond the control of natural enemies (Greenbank, 1956; Ives, 1973). According to Watt (1968), the important factor that determines the degree to which the weather influences the insect is the evolved sensitivity of the species to variations in weather patterns. If a given species is under the control of a density dependent agent , it will be more sensitive to extreme changes in weather than a species normally under density independent control. But even in those instances where the weather pattern can be correlated with the variations in the population trends, the mechanisms causing population change are still unknown. The observation that outbreaks of the spruce budworm, Choristoneura fumifera (Clem.), the forest tent

caterpillar, Malacosoma disstria, and the black-headed budworm, Acleris variana (Fern.),to mention only a few, are preceded by 2-5 years of dry summers, does not explain the mechanisms underlying such a rapid increase let alone the periodic nature of their fluctuations (Wellington, 1954; Silver, 1960; Greenbank, 1963; Ives, 1973).

One avenue of investigation is to estimate the quality of the food source and its effect on the insect. White (1974, 1976,1978) postulates that weather-induced stress of the host plant increases its nutritional quality thus enhancing survival and growth of the insect populations feeding on the plant. The nutritional value of the food source can either explain the initiation of an outbreak when nitrogen is made available or its decline when the food quality is inadequate; mature tree leaves are a relatively poor source of food for lepidopterous larvae and the result is slowly growing larvae (Scribner and Feeney, 1979). Longer development time of the insect may in turn render the larvae more susceptible to predators, parasitoids and diseases through increased exposure to enemies or lessened resistance to pathogens (Price et al., 1980). For example, a significant decline in oak leaf quality following defoliation did reduce gypsy moth larval growth and changes in population density could be driven in part by the response of the host plant to the feeding pressure of the insect (Schultz and Baldwin, 1982). However, Myers (1981) and Mason (1981b) failed to provide evidence that the Western tent caterpillar,

Malacosoma californicum pluviale (Dyar), and the Douglas-fir tussock moth, Orgyia pseudotsugata (McDunnough), are affected by host quality.

Models that only include the interaction between a predator and its prey or a parasitoid and its host, generate stable limit cycles given certain parameter values (De Bach, 1941; Beddington et al., 1975; May, 1976; Hassell, 1978). In nature, however, the situation is more complex as other factors intervene. Analysis of field populations indicates that low insect density is often maintained by either predators or parasitoids while intraspecific competition for food prevents the population from increasing (Readshaw, 1965; McNamee, 1979; McNamee et al., 1981). The upper density limit of the insect can also be determined by a pathogen. Diseases that are transmitted through contact with susceptible individuals need a threshold density of hosts in order to maintain an infection (Anderson and May 1981), and depending on the value of this threshold the disease could reach epizootic proportion before starvation takes its toll. Anderson and May (1981) propose that the cyclic pattern of low and high insect densities can be explained by the dynamics of the insect-disease interaction alone. At low host density the incidence of the disease is virtually nil and as the insect population increases the pathogens, which were present in some latent form in the environment or the host, multiply and bring about the decline of the host population. Anderson and May (1980, 1981) analysed data pertaining to the larch budmoth,

Zeirephera grisenea(Hubner), in Switzerland for evidence that a viral disease accounts for the oscillating population patterns. This insect erupts every 9 to 10 years and given the parameter values Anderson and May estimated for this insect, the model generates stable cycles of the desired period.

In Western North America the Douglas-fir tussock moth, hereafter referred to as the tussock moth, is periodically a conspicuous defoliator of Douglas-fir, Pseudotsuga menziessi var.glauca (Beissn.) Franco, as well as true firs (Abies spp.) In Oregon and California, six serious infestations have recurred at about ten year intervals since 1936 (Wickman et al., 1973) and in the interior of British Columbia, the driest stands of Douglas-fir have also been regularly subject to epidemics since 1916 (Sudgen, 1957). Survey records in Arizona also indicate an oscillating pattern of outbreaks with a periodicity of 9 to 10 years (Mason, 1977). A nuclear polyhedrosis virus (NPV) usually appears during the declining phase of an outbreak and is hypothesized to be a factor in the population dynamics of the tussock moth (Morris, 1963; Mason and Thompson, 1971). The association between the tussock moth and its viral disease raises the possibility of testing the hypothesis that this disease is responsible for the periodic population fluctuations of the tussock moth.

II. Biology of the Douglas-fir tussock moth

Life-cycle

The Douglas-fir tussock moth has one generation per year. The eggs are laid in the fall and hatch the following spring in late May or early June depending on the temperature. By the time the larvae emerge new plant shoots have flushed and the first and second instars disperse to the new foliage by spinning silk threads.The larve are then blown away by the wind. The dispersal period may vary between 10 to 20 days but the larvae usually remain within a relatively small distance from the source tree, probably no further than 200 m (Mitchell, 1979). The larvae feed on foliage for about 2.5 to 3 months and the number of instars. varies between five and six, the male having one instar less than the female. Pupation begins about mid-August and the adults emerge two weeks later, with the males preceeding the females. Adult tussock moths do not feed. Mating occurs shortly after emergence and the wingless adult female lays her eggs on the cocoon from which she emerged. The eggs are laid in a single egg mass and may contain from 150 to 200 eggs (Beckwith, 1978).

Host types

The distribution of the tussock moth over the range of host types is illustrated in Fig. 1. In California and Southern Oregon the tussock moth is mainly a defoliator of white fir, *Abies concolor* (Gord. & Glend) Linl., while in the Southwestern United States it feeds on both white fir and Douglas-fir. In the Northwestern states grand fir, *Abies grandis* (Dougl.) Lindl., and Douglas-fir are defoliated but not always at the same intensity. In Boise, Idaho, grand fir was the preferred host but Douglas-fir was severely attacked in Weiser, Idaho. The principal host in the interior of British Columbia is Douglas-fir (Sudgen, 1957).

Epidemic populations

Past outbreaks

The Douglas-fir tussock moth was first noted in 1916 near Chase B.C. Local infestations have recurred repeatedly since that first report and the interval between infestations up to 1957 in the interior of British Columbia is represented in Fig.2a. The papers from which those figures are drawn do not specify if the insect reinfested the same stands but a synchronous pattern of outbreaks among regions is apparent. In 1939, 1949 and 1955 the population collapse was attributed to a virus disease although no quantitative information on the

Fig. 1. Distribution of Douglas-fir tussock moth as determined by collecting and pheromone trapping. (From Livingston and Daterman, 1977).

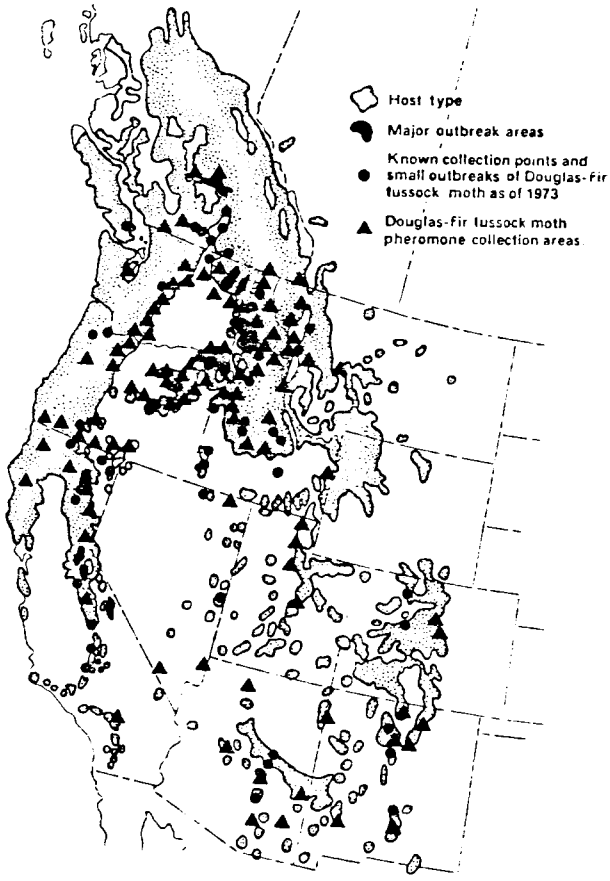
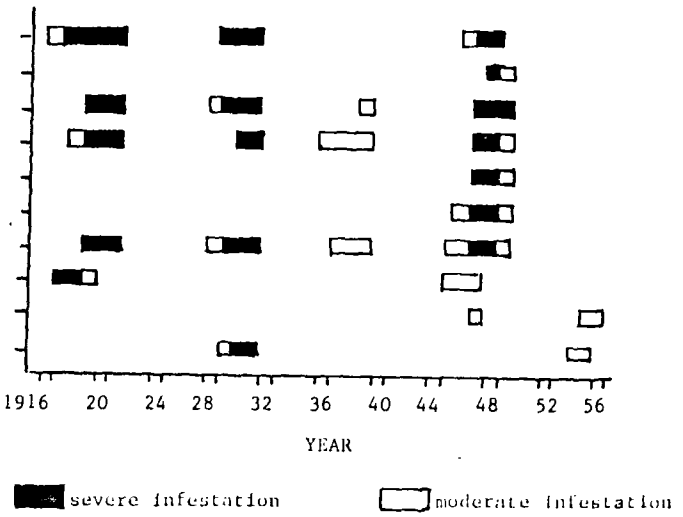
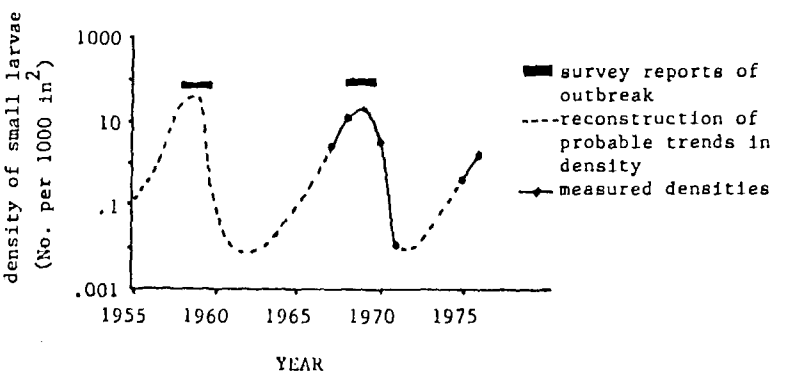

Host type
Major outbreak areas
Known collection points and small outbreaks of Douglas-Fir tussock moth as of 1973
Douglas-Fir tussock moth pheromone collection areas

Fig. 2. Records of occurence of Douglas-fir tussock moth infestations (a) in the interior of British Columbia and (b) in Aztec Peak,Arizona . (Redrawn from Sudgen, 1957 and Mason, 1977).

a)


CHASE
CACHE CREEK
KAMLOOPS
ARMSTRONG
ASHCROFT
MONTE LAKE
VERNON
HEDLEY
OLALLA
CASCADE
1916 20 24 28 32 36 40 44 48 52 56
YEAR
severe infestation
moderate infestation

b)


density of small larvae (No. per 1000 in²)
1000
10
.1
.001
1955 1960 1965 1970 1975
YEAR
survey reports of outbreak
----reconstruction of probable trends in density
measured densities

prevalence of infection was reported (Sudgen, 1957).

In the United States the first recorded occurence of the tussock moth dates back to 1927 at Janbridge, Nevada. The following year the insect was noticed at Boise and Weiser, Idaho (Balch, 1932). California and Oregon have a history of severe outbreaks that recurred at about 10 year intervals since 1936 (Wickman et al., 1973). Among the affected regions were Mammoth Lakes, California 1935-37; Stanislaws National Forest, California 1954-6; Modoc National Forest, California 1963-65; Troy, Oregon 1945-7; Burns, Oregon in 1963-65; and Clearwater National Forest, Northern Idaho 1943-45, 1962-64 and 1972-74. The most damaging outbreak on record has been reported in the Blue Mountains of Northeastern Oregon in 1972-76 when the tussock moth reached densities of 765 early instar larvae/m^2 of foliage (Mason, 1976). Aztec Peak, Arizona has been subject to at least two outbreaks (Fig. 2b) (Mason, 1974).

A tussock moth outbreak lasts from 3 to 4 years and is characterised by 3 distinct phases: the release, peak and decline phases, (Wickman et al. 1973; Mason, 1974). Outbreaks occur following several years of inconspicuous buildup of the population in a stand with usually one or two seasons of rapid increase during which noticeable damage to the trees occurs. Tree defoliation becomes apparent when densities exceed 33 early instar larvae/m^2 of foliage (Mason,1977). Defoliation is usually concentrated in discrete geographic patches with little spread to adjacent areas. In severe outbreaks the upper quarter to half

of the crown of the tree is often defoliated and a reduction of radial growth may occur if more than 50% of the tree is defoliated (Wickman, 1978). For example, the growth of white fir in California was reduced up to 74% under heavy feeding pressure. Another consequence of defoliation is top-kill. If more than 60% of the crown is defoliated, tree mortality may ensue but often mortality is due to secondary attacks by bark beetles (Wickman, 1978).

Host quality

In general, outbreaks are more prevalent on ridgetops and upper slopes, on low productivity sites and in mature and overmature stands (Stoszek et al., 1981). The interpretation put forth by Stoszek et al. (1981) is that the trees in such places where the soils are less fertile, shallower and drier, are more likely to be subject to stresses caused by water and nutrient deficiencies. Such stress factors increase the proportion of soluble nitrogen in the foliage (White, 1974, 1978) and as a result may favor tussock moth population increase via enhanced survival of the larvae.

Mason (1981b) tested the hypothesis that tussock moth outbreaks develop in response to changes in host foliage quality. He compared the quality of the foliage in typical outbreak sites with that in sites with no outbreak history and concluded that foliage quality is not responsible for the sudden

increase in insect density prior to an epidemic (Mason, 1981b). In a study done on locally abundant populations in the Eldorado National Forest, California, in 1971 , tussock moth larvae were put on caged branches and examined for adverse effects of foliage quality. The same procedure was followed for sites where no outbreaks had been reported. The production of frass, fecundity, larval survival and the proportion of late instars that pupated were not significantly different between the outbreak and non-outbreak sites.

Further research is needed to elucidate the role of host quality on the release of tussock moth populations. However, quality of the host may at least contribute to the decline of the outbreak. Current-year foliage is necessary for the survival of the first two instars .When new shoots are unavailable the insects will either starve or disperse in order to increase their chance of finding preferred foliage (Mason and Baxter, 1970). When fed old growth foliage under laboratory conditions, stresed larvae take more time to develop than non-stressed larvae fed new foliage (Beckwith, 1976). Another consequence of food shortage is a reduction in the production of eggs. Fecundity data for populations in Northeastern Oregon at the end of the first and second season of apparent tree defoliation indicate a 30% reduction in the mean number of eggs/mass (Mason et al., 1977). The mean fecundity dropped significantly from 151 eggs/mass in 1972 to 105 eggs/mass in 1973.

Virus epizootiology

Numerical changes in tussock moth populations appear to be related to a complex of natural enemies including a virus disease which is often cited as a major mortality agent in the declining phase of an outbreak (Sudgen, 1957; Morris, 1963; Wickman et al., 1973; Mason, 1974).

Two nuclear polyhedrosis viruses (NPV) are known to infect Orgyia pseudotsugata . One type of polyhedral inclusion body (PIB) contains a single virus rod and is designated as SV while the other has bundles of virus rods and is designated as BV. The SV has been found throughout the range of the host from British Columbia, Washington, Oregon, Idaho, Montana, California and Arizona. The BV virus is more limited and has been collected only in British Columbia, Washington, Idaho, Montana and Northern Oregon. Sometimes both viruses are found in the same insect population but only rarely will one individual be infected with both viruses (Hughes, 1976).

An epizootic generally develops when the eggs are contaminated with virus present in the female or the forest. As the larvae hatch they eat part of their egg shell and become infected with the virus. The virus multiplies inside the body cavity and when the larva dies, it ruptures and liberates inclusion bodies that contaminate the foliage upon which the healthy larvae will feed. These in turn will propagate the virus as they die. It appears that the initial incidence of infection

in the first instar as well as population density will influence the speed at which the epizootic will spread (Wickman et al. 1973).

Endemic populations

Tussock moths are notoriously difficult to detect between outbreaks. At densities less than 0.15 larvae/m^2 the moths are rare, being found on less than 2% of the branches. Low densities are considered to be between 0.15 and 3 larvae/m^2 with less than 25% of the branches infested. At suboutbreak level the densities are between 3 to 30 larvae/m^2 (Mason, 1977). Densities above this latter figure are termed outbreak densities.

Associated with those low densities is a complex of invertebrate predators and parasitoids that may play a regulatory role in low populations of tussock moth. In central California they appear to be preventing the populations from reaching high densities (Dalhsten et al., 1977). Among the parasitoids identified , the most common were tachinids which accounted for 73% of all parasitism. The effect of predators, however, is more difficult to determine since many of them remove the entire prey. Invertebrate predators such as certain coccinellids, pentatomids and spiders are suspected of being natural ennemies of the caterpillars but their influence has not been quantified (Mason, 1976; Dahlsten et al., 1977). Generally, the incidence of virus between outbreaks is very low or appears

to be absent. Even if the virus is undetectable in the population it may still be present in the forest floor environment and reintroduced to the insect population by airborn dust particles (Thompson & Scott, 1979).

III. The approach

From the previous discussion, it is clear that many variables change during the course of a tussock moth outbreak. The foliage condition, disease prevalence and probably predator and parasitoid densities are varying and yet there are relatively few good data on the dynamic responses of most of these components. My objective is to test the hypothesis that the explicit representation of the dynamics of the disease and host insect alone are sufficient to generate periodic outbreak patterns of the tussock moth which resemble the patterns observed in the field. In other words, I am focusing on the disease hypothesis as opposed to the food or predator hypothesis of population regulation. The unspecified effects of food or predators are implicitly included in a model where density dependent factors increase mortality at high host insect densities.

The hypothesis of the importance of disease in the tussock moth population cycles is tested in several ways. Various modifications of Anderson and May's (1981) free-living stages model (Model G) are used with parameter estimates for the tussock moth situation derived from the literature and a laboratory experiment. These versions of the model tested are different from Anderson and May's in that the free-living stages are included with each process examined and all the processes

are combined in one model in a way appropriate for the tussock moth. These processes include vertical transmission of the disease, density-dependent mortality and an incubation period of infection (see section V, "The models".) The behavior of each version of the model is examined to determine if the tussock moth population cycles seen in nature are properly reflected by the model, using parameter estimates derived for the tussock moth. Sensitivity analyses are also performed to cover a range of parameter estimates.

IV. The models

Basic model

Anderson and May's (1981) free-living stages model (Model G), which is the basic model for my purpose, is condensed into 4 differential equations describing the dynamics of the total host insect population (N), the infected hosts (Y), the susceptible hosts (X), and the long-lived infective stages of the virus (W). Generally epidemiological models describe human populations and focus primarily on the transmission of the disease from the infected to the susceptible hosts without keeping track of changes in the abundance of the host population and of the pathogen. Anderson and May (1981) break new ground by explicitly modelling the dynamics of the host as well as the microparasite populations.

The assumptions of Anderson and May's (1981) model G is that the host rate of population growth is determined by an intrinsic rate of increase (r) in the absence of the disease , minus a disease-induced death rate (α) of infected hosts where (α) is the per capita death rate. These parameters are instantaneous rates (see section on "Estimation of parameters" for their values).

$$dN/dt = rN - \alpha Y \quad (1)$$

The intrinsic growth rate r is equal to the rate at which new susceptibles are introduced (a), minus the rate at which they die (b), due to factors other than the viral disease.

The rate at which hosts acquire infection is assumed to be proportional to the number of susceptible hosts (X) and the number of infective stages of the virus (W). The infected individuals (Y) are lost at a rate $\alpha+b$.

$$dY/dt=\beta WX-(\alpha+b)Y \quad (2)$$

(β) is the transmission coefficient between infective viruses and susceptible hosts.

The infective stages are produced at a rate (λ) determined by the number of viral particles (Λ) produced during the lifetime of the infection $1/\alpha+b$. The losses from the virus population are accounted for by the rate of mortality of the viral particles (u) and by the absorption into the insect βN.

$$dW/dt=\lambda Y-(u+\beta N)W \quad (3)$$

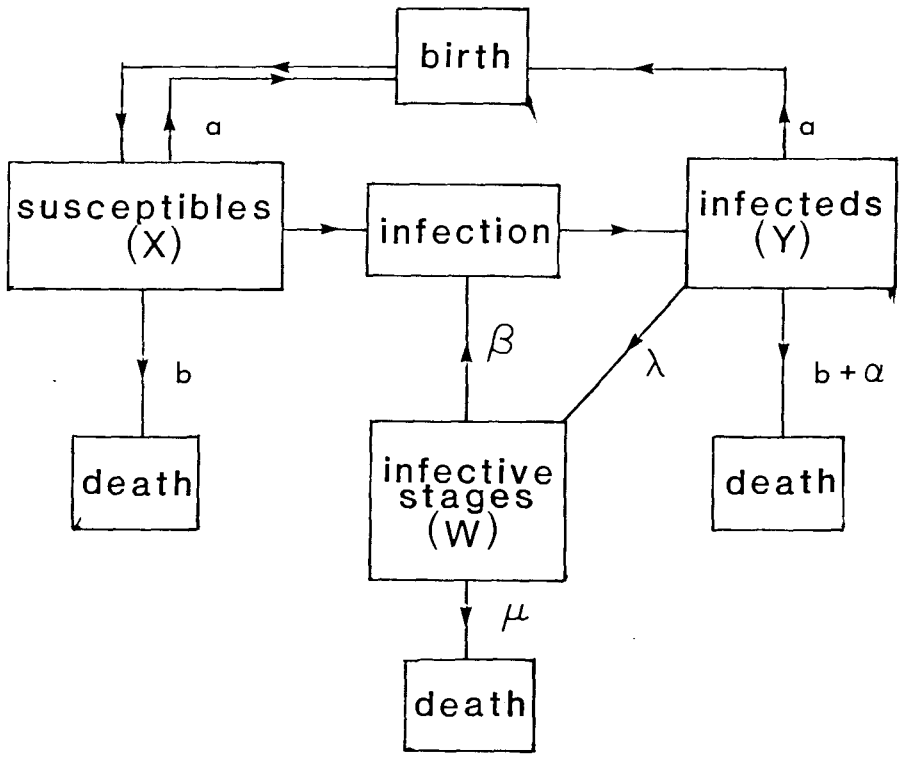
Since, by definition, the total host insect population (N) equals the sum of infected (Y) and uninfected (X) individuals, the dynamics for the susceptible hosts (X) are given by the identity equation:

$$X=N-Y \quad (4)$$

The processes incorporated in the basic model are schematically illustrated in Fig.3

Fig. 3. Schematic representation of the processes incorporated in the basic model. (Adapted from Anderson and May, 1981).

BASIC MODEL


birth
a
a
susceptibles (X)
infection
infecteds (Y)
b
β
λ
b + α
death
infective stages (W)
death
μ
death

Density-dependent model

In the basic model the only constraint limiting the growth of the tussock moth is the virus. In reality other processes such as food depletion or the action of predators and parasitoids will eventually limit population increase. I modified the basic model by adding density dependent mortality which encompasses all these processes. The equations are similar to the basic model except that the insect natural death rate (b) is replaced by the function

$$b' = b + cN \quad (5)$$

where (b) is the smallest value of the natural death rate at low host density , or the minimum natural death rate, and (c) represents the severity of density-dependent constraints on the natural mortality. The parameter (b') can be treated as a constant when (c) is equal to 0 or as a linear function of density when (c) is greater than zero (Fig. 4). The disease-induced mortality rate (α) is always density dependent and is derived from the identity function

$$\alpha = d - b' \quad (6)$$

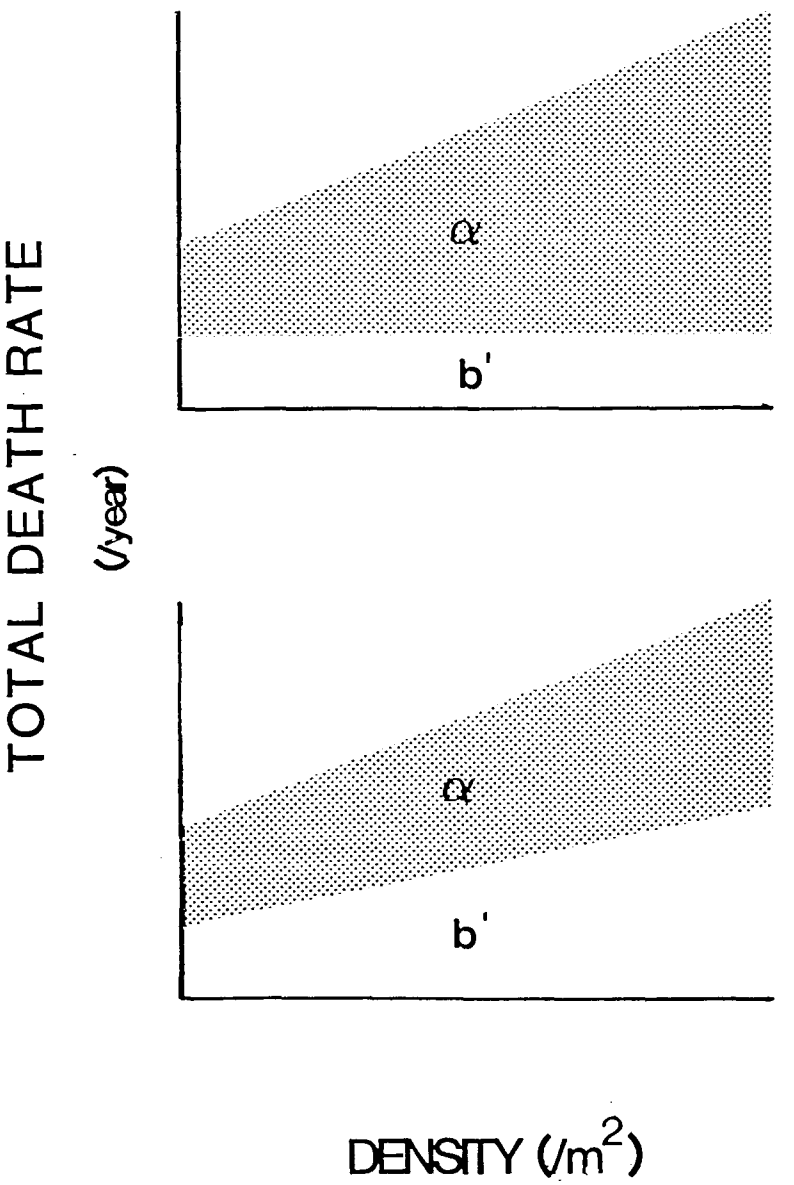
where (d) is the total host mortality.

Incubation period model

The tussock moth virus does not kill its host readily once a larva has contracted the infection and an incubation period of about 1 to 2 weeks is generally required before the insect dies.

Fig. 4. The relation between the total mortality and the density of the host (N) is illustrated under two assumptions. a) The host natural mortality (b) is constant while the disease-induced host mortality (α) is density-dependent. b) Both (b) and (α) are density-dependent.

DENSITY DEPENDENT MODEL



It is often hypothesized that a delay in the response of a mortality agent to changes in host density may produce cyclic oscillations (Berryman, 1978b; May, 1973). Whether this general statement can be extended to the tussock moth population is explored in the incubation period version of the model. The incubation period of infection is modelled by adding a new class of infected but not yet infectious individuals (M) which acquire the infection at the rate βWX, and are lost through natural death and transfer from the infected class to the infectious compartment at a rate (ν) (Fig.5).

$$dM/dt = \beta WX - (b+\nu)M \quad (7)$$

Consequently the gains in infecteds is measured by νM and the losses include natural and disease-induced mortality $(b+\alpha)Y$

$$dY/dt = \nu M - (b+\alpha)Y \quad (8)$$

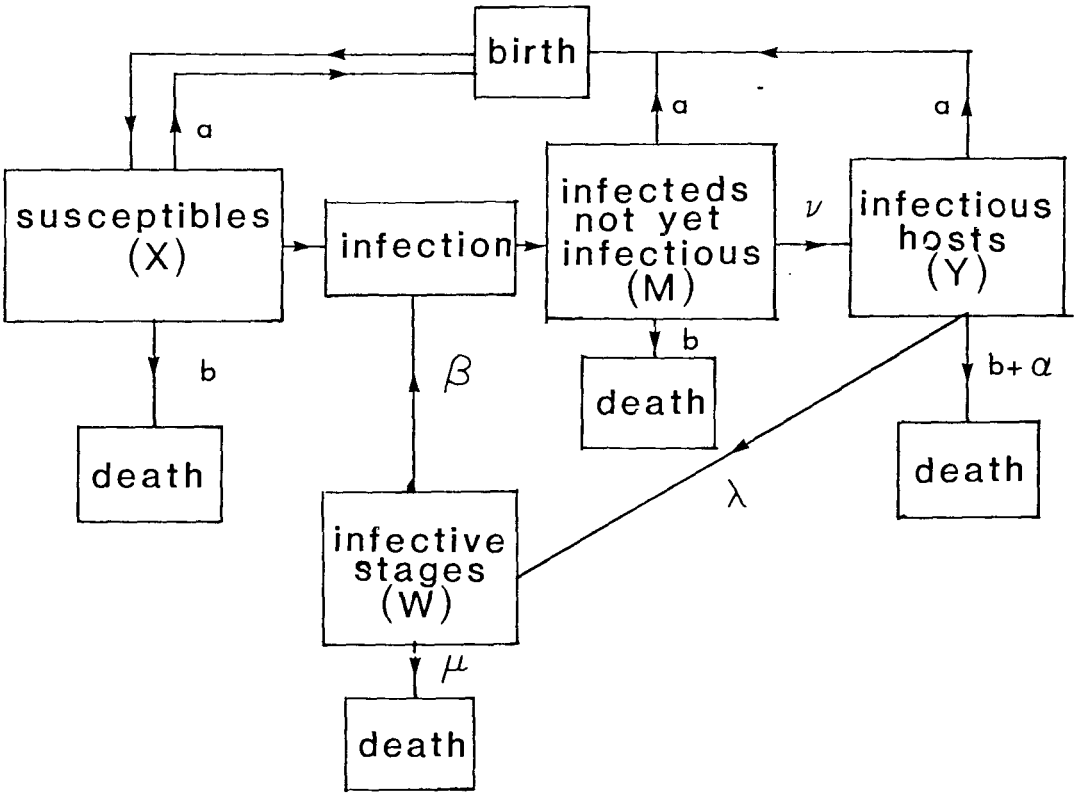
Equations (1) and (3) remain unchanged.

Vertical transmission model

In addition to contamination among conspecifics, or horizontal transmission, a female infected in the later stages of her life can transmit the virus directly to her offspring. Some pathogens are present on the surface of some eggs and the larvae become infected when they emerge and eat the egg shell. Also referred to as transovum transmission, this mechanism is one of the two means of vertical transmission, the other being the transmission of the virus directly to the embryo in the egg

Fig. 5. Schematic representation of the processes incorporated in the incubation period model. (Adapted from Anderson and May, 1981).

INCUBATION PERIOD MODEL



shell called transovarial transmission (Finn, 1975). Vertical transmission is often found in insect species which have a small probability of contracting the infection from their conspecifics (Thomson, 1958; Burges, 1973). Since the tussock moth larvae emerging from previously surface sterilized eggs will not contract the disease, so transmission is transovum. In the vertical transmission version a proportion p of the births goes directly to the infected class while the rest forms the pool of susceptibles (Fig. 6).

$$dY/dt = \beta WX - (\alpha + b)Y + apY \quad (9)$$

Equations (1) and (3) are unchanged.

Combined model

The combined model includes all the processes mentioned above in the basic, density-dependent, latency and vertical transmission models. This combined model represents the tussock moth situation as closely as is discernible from the existing data. The larvae become infectious after a given incubation period. (Fig. 7)

$$dY/dt = \nu M - (\alpha + b')Y \quad (10)$$

A proportion of the births go directly to the infecteds but not yet infectious class.

$$dM/dt = \beta WX - (b' + \nu)M + apM \quad (11)$$

The parameters (b)′ and (α) are density-dependent. The larval and the viral populations grow at the same rate as in the

Fig. 6. Schematic representation of the processes incorporated in the vertical transmission model. (Adapted from Anderson and May, 1981).

VERTICAL TRANSMISSION MODEL

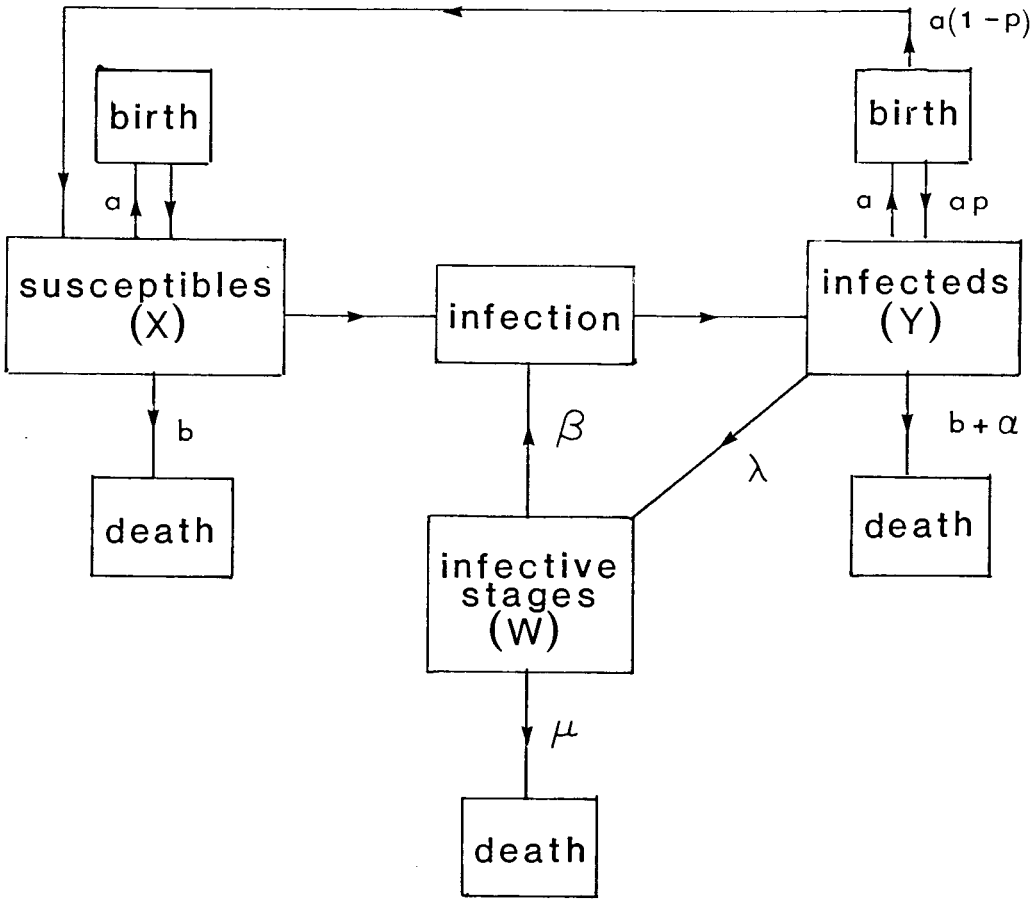
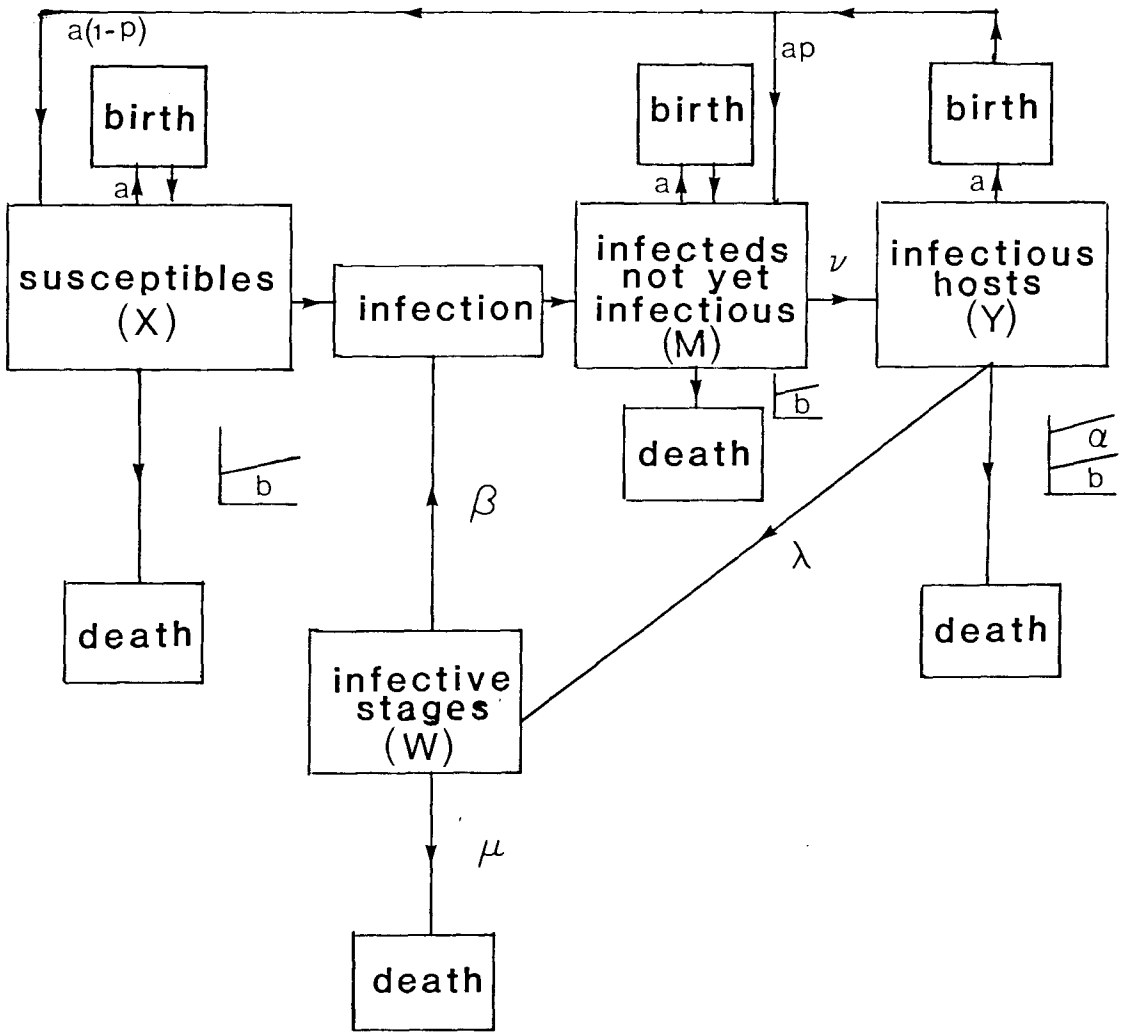


Fig. 7. Schematic representation of the processes incorporated in the combined model.

COMBINED MODEL



previous versions (equations (1) and (3)).

Stochastic model

In the previous versions of the model only one outcome is possible given certain parameter values, i.e. the models are deterministic. However, in nature those parameter values are not fixed from year to year and variation around a mean value is expected. All the processes included in the model are subject to fluctuations but I focus on the growth rate (r) because it incorporates natality and mortality and is a major influence on the dynamical behavior of the equations. Random fluctuations are incorporated in the basic and combined models in order to test whether variability in recruitment of offspring can change the periodicity or lack of periodicity observed in the deterministic versions of these models. The growth rate (r′) in the equation

$$dN/dt = r'N - Y \quad (12)$$

is either equal to

$$r' = re^{v} \quad (13)$$

or

$$r' = r + v \quad (14)$$

where (v) is a normally distributed variate with mean zero and variance .

A succinct summary of each version of the model is given in Table 1.

Table 1. Models reviewed in the sensitivity analysis.

Model	Variables and processes included	Equations
A. Basic model	Total host population (N) Infected hosts (Y) Susceptible hosts (X) Free-living infective stages (W)	$dN/dt=rN-\alpha Y$ $dY/dt=\beta WX-(\alpha+b)Y$ $X=N-Y$ $dW/dt=\lambda Y-(u+\beta N)W$
B. Density dependent model	Density dependent death rate of the host population (d)	As in model A except $b'=b+cN$ $d=\alpha+b'$
C. Incubation model	New class of infected but not infectious hosts (M)	As in model A except $dM/dt=\beta WX-(\nu+b)M$ $dY/dt=\nu M-(\alpha+b)Y$
D. Vertical transmission model	Proportion of offsprings of infected hosts go directly to the infected class	As in model A except $dY/dt=\beta WX-(\alpha+b)+apY$
E. Combined model	Inclusion of all the above processes	As in model A except $dM/dt=\beta WX-(b'+\nu)M+apM$ $dY/dt=\nu M-(\alpha+b')Y$ $dN/dt=(a-b')N-\alpha Y$
F. Stochastic growth rate model	Random fluctuations on the growth rate	As in model A and E except $r'=re^{v}$ or $r'=r+v$

β:transmission coefficient, α:disease-induced death rate, b:natural death rate, λ:virus production rate, r:host growth rate, a:host birth rate, ν :incubation coefficient, u:virus death rate, v:normally distributed random variate.

V. Data sources

The data used in the estimation of the model parameters are from the western part of the United States and Canada. Included are populations that fluctuate at low density in the absence of the virus in Mare's Egg Spring, Ore. and Eldorado National Forest, Calif. (Mason and Torgersen, 1977; Mason et al., 1983), and populations that reached high densities, up to 118 early instar larvae/m^2 and whose rapid decline was attributed to a nuclear polyhedrosis virus in Aztec Peak, Ariz. and Modoc National Forest, Calif. (Mason and Thompson, 1971; R.R. Mason, pers. comm.[1]).

Unfortunately, there is no population of tussock moth which has been studied extensively enough to provide the data necessary to estimate all of the parameters of even one version of the model. Therefore, I pieced together parameter estimates from different populations, and ensured that only comparable populations were included in the analysis.

Mare's Egg Spring, Oregon population

The 8 study plots in this area are mixed conifer stands composed of white fir, pondorosa pine, Pinus ponderosa,

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1 Range and Wildlife Habitat Lab., La Grande, Oregon

Douglas-fir and incense cedar, Libocedrus decurrens (Torr.). The area has never experienced an outbreak and the study was conducted from 1975 to 1977. Usually the branches are sampled from the mid-bole of the tree but for these low density populations the branches were taken from the lower crown and the results were comparable with the standard sampling method. Concomitantly with the sampling, larvae were stocked on branches in order to identify more precisely the mortality agents (Mason & Torgersen, 1977).

Eldorado National Forest, Calif. population

The tussock moth was sampled in a mixed conifer forest dominated by white fir, ponderosa pine and incense cedar. The samples were taken from the mid-bole of the tree. There were 4 plots at Iron Mountain and 4 other plots at Plummer Ridge. Small outbreaks of tussock moth were recorded in 1953, 1962 and 1970 in Iron Mountain but damage was not as extensive as the infestations in the other western states. Plummer Ridge has only one record of moderate populations, in 1970, but densities did not reach levels of noticeable defoliation. In 1978-79, the years from which the data are obtained, the tussock moth was believed to be in the release phase but they decreased instead of erupting. The virus was not detected and parasitoids and predators probably kept the population under control (Mason et al. 1983).

Modoc National Forest, Calif. population

The study area is composed of white fir and pondorosa pine and the mid-crown of the trees was sampled. The 5 study plots had maximum densities over 80 early instar larvae/m^2 and the virus disease was responsible for 41.3% of the total mortality (Mason & Thompson, 1971).

Aztec Peak, Ariz. population

The forest is a mixed conifer type comprised of white fir, Douglas fir and ponderosa pine. The data used in the model were obtained from 10 plots surveyed during the release phase of an outbreak and these are used to estimate the intrinsic growth rate. The outbreak was light with densities of 50 early instar larvae/m^2 and the virus was present in the collapse phase. Only early instar larvae were sampled for 8 years (Mason, pers. comm.).

Other field data sources

Situations exist where the populations do not fit into either of these two categories. This is the case in northeastern Oregon where in 1973 the tussock moth increased to extremely

high levels ($765/m^2$) without being hampered by the virus disease which appeared very late in the outbreak and accounted, in conjunction with parasites, for 11% of the total mortality (Mason, 1976). After a couple of years of heavy feeding pressure by the insect, the plots were defoliated to various degrees ranging from severe to light. Because of the starvation suffered by the larvae this population will not be used to investigate virus-related mortality at the larval stage.

Populations sprayed with the virus as a mean of biological control are also informative and in one example in Oregon the experimental spray was conducted in the Wallowa-Whitman National Forest in 1973 (Slelzer et al., 1975). Douglas-fir and grand fir were sampled to monitor the effect of the spray. Screening agents to block ultra-violet radiation were also added to the spray formulation which resulted in a 10% increase in mortality over the formula of virus only. Virus caused mortality was first observed in the treated plots about 14 days following the spray after which the natural process of contamination took over. A similar study was conducted in Kamloops, B.C. in 1975 (Stelzer et al., 1977).

VI. Estimation of Parameters

Because the equations of the various models used here are in continuous form, parameter estimates must be in instantaneous rates, not finite rates. To convert from finite rates, which are the type of data normally gathered in field studies, to instantaneous rates the natural logarithm of the finite rate is used because the general model describing changes in numbers is

$$N_{t+1} = N_t e^{rt} \qquad (15)$$

If t is set to 1 time unit, and here t is one year, the natural logarithm of the finite annual survival rate (N_{t+1}/N_t) is the instantaneous annual growth rate r.

Growth rate ,r,

The parameter r represents the maximum growth rate of the insect population and an adequate approximation is the increase in density between generations, when the insect is not food limited. In a continuous time model the instantaneous growth rate is calculated by taking the natural log of the trend index (N_{t+1}/N_t) at maximum increase. For example, during the release phase of an outbreak in Aztec Peak, Arizona, the ratio of the mean densities of 10 plots for the year 1967 to 1968, is 5.3, (Mason, R.R. pers. comm.) which when converted to an instantaneous rate is 1.7/year (Table 2).

Table 2. Table of baseline values for the parameters of the basic model.

PARAMETER	BASELINE VALUE (/YEAR)	MINIMUM AND MAXIMUM VALUES OF PARAMETER ESTIMATES
Growth rate, r	1.7	1.4-2.1
Birth rate, a	4.7	4.3-4.9
Insect natural death rate, b	3.0	3.0-5.5
Disease-induced death rate, α	8.9	6.9-11.5
Number of inclusion bodies produced/larva, Λ	2×10^8	1×10^7-4×10^8
Virus natural death rate, u	5.0	4.0-6.0
Transmission coefficient, β	1×10^{-9}	—

Birth rate ,a,

The data are from the endemic populations in order to exclude the effect of starvation on fecundity. The number of eggs per individual varied between 71 and 136 with a mean of 109 (Mason and Torgersen, 1977; Mason et al., 1983). The sex ratio was close to unity. Therefore, the average instantaneous birth rate (a) is 4.7 with a minimum at 4.3 and a maximum at 4.9 but in the sensitivity analysis the birth rate is constant at 4.7.

Natural mortality rate ,b,

In the Anderson and May model (1981), the natural mortality rate (b) includes all the mortality agents operating in populations free of the virus. Information on such agents is available from the low density populations that are sampled soon after hatching from the larval stage to the pupal stage. The data on the moth stage are an estimation of the density of adults that emerged from the pupae.

If equation (15) is modified to represent weekly changes in survival of a cohort of insects, then t=1 week and the exponent becomes the natural mortality rate b. The equation is then transformed by taking the natural logarithms

$$\ln(N_t) = \ln(N_0) - bt \quad (16)$$

where b, the instantaneous per capita death rate, is the slope of the regression line. A linear regression was performed on the

number of larvae surviving over time, and the close fit to the linear relation gives support to the assumption that the instantaneous death rate is constant throughout the insect life span (Fig. 8). The instantaneous death rate calculated for the 5 sets of field data range from 2.6 to 5.0 for the 12 week period (Table 3).

These values only account for the 3 month period while the insects are active. The rest of the year is spent in the egg stage where mortality also occurs. The instantaneous death rate, which is the natural logarithm of the proportion of the density of first instar larvae that emerged in the spring over the density of eggs the previous fall, is very low (0.5 insect/9 months or 0.17 insect/3months). Since instantaneous mortality rates are additive, the larval mortality for 3 months is added to the egg mortality for 3 periods of 3 months and the values of b for the complete life-cycle range from 3.1 to 5.5 insect/year with a mean of 4.6.

The value of 3.1 was estimated from a study done with stocked larvae, but the cohort had not been followed the following year and it is not known if this value reflects a stable or increasing population. The other values of (b) come from populations that declined the following year and the natural death rate is probably overestimated. For the purpose of the model the natural death rate should be estimated from an increasing population, when the mortality from different sources is at a minimum, since it enters in the definition of the

Fig. 8. Rate of disappearance of larvae and pupae. (Data from Mason and Torgersen, 1977).

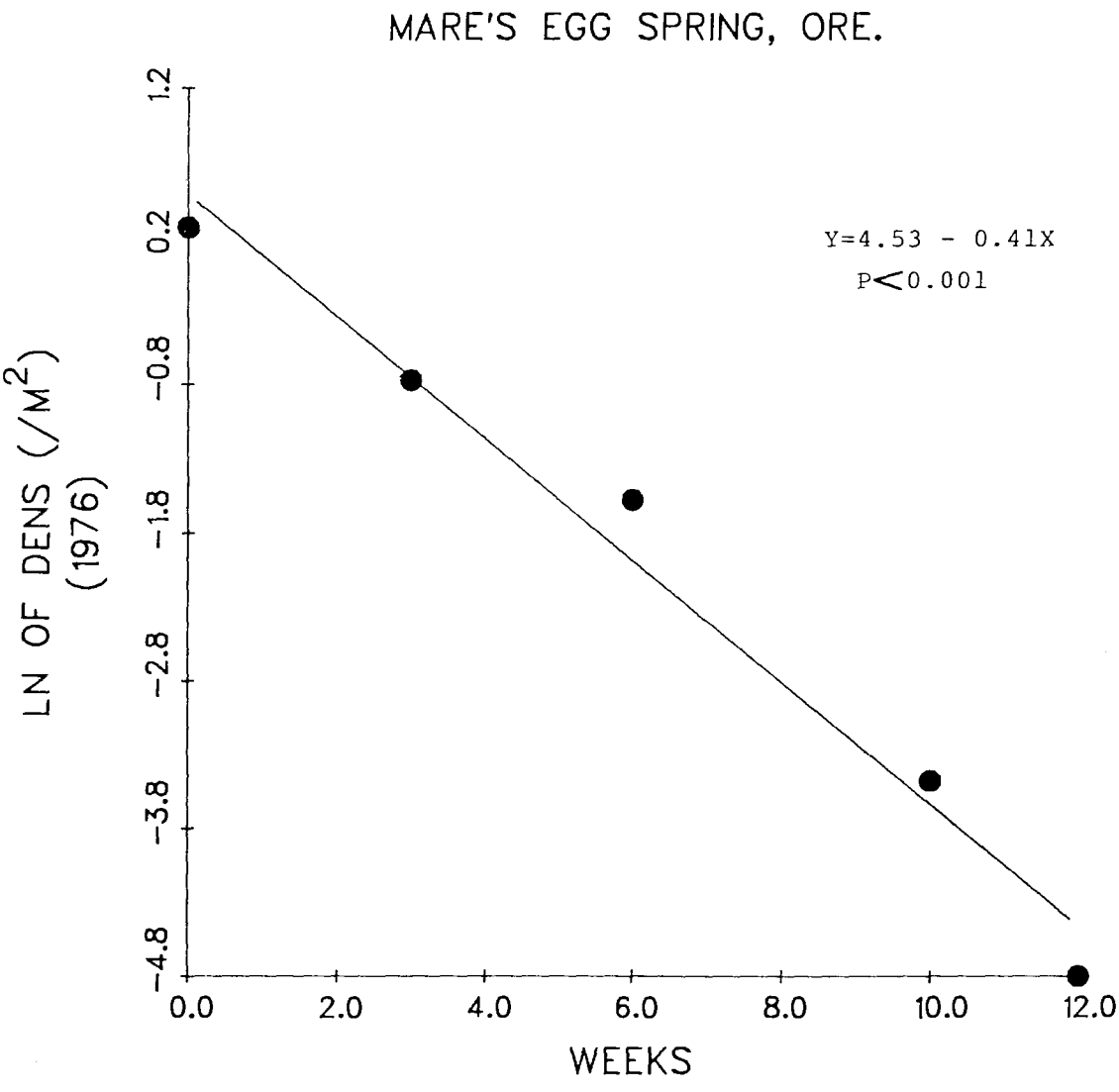

MARE'S EGG SPRING, ORE.
Y=4.53 - 0.41X
P<0.001
LN OF DENS (/M2)
(1976)
1.2
0.2
-0.8
-1.8
-2.8
-3.8
-4.8
0.0
2.0
4.0
6.0
8.0
10.0
12.0
WEEKS

Table 3. Regression statistics for field plots used to estimate the natural death rate (b). (Y=a+bx where Y=ln of dens. ($/m^2$) and x=weeks)

Plot	Larval death rate,b (/wk)	p	r^2	n
Mare's Egg Spring, Oregon				
1975	0.26	0.009	0.919	8
1976	0.41	0.001	0.976	8
Stocked cohort	0.22	0.001	0.980	8
Sierra Nevada, California 1978				
Iron Mountain	0.35	0.006	0.943	4
Plummer ridge	0.42	0.007	0.933	4

intrinsic rate of increase. The value of the intrinsic growth rate is 1.7 and the birth rate is 4.7. When those values are substituted in the equation r=a-b, the resulting death rate is 3.0. Considering the limited amount of data on natural mortality, statistical measures of deviation from the mean of 4.6 derived above are meaningless and the range of values included in the sensitivity analysis represents the best approximation of the mortality rates observed in the field.

Disease-induced mortality rate ,α,

The same regression procedure as the one used for the estimation of (b) is followed for the disease-induced death rate (α) but this time using a population in the decline phase of an outbreak in plots where the virus is an important mortality agent. The data come from 5 plots in outbreak in the Modoc National Forest in California (Mason & Thompson,1971).

The death rate calculated from the regression slope represents the mortality during the three month period when the insect is active. The mean total death rate is 9.2/3 months with a minimum at 6.2 and a maximum at 11.8. Mortality in the egg stage has to be added to those figures in order to estimate the total annual mortality. Unfortunately no information is available on the egg survival through the winter for these plots but it can be calculated from the life tables of an outbreak population in Northeastern Oregon. This population was

previously discarded because the virus was very late to appear in the larvae but the data show that for those plots where first instars were present the year following the collapse, between 13 and 32% of the larvae (n=6 plots) died from the virus disease soon after emergence (R.R. Mason, pers. comm.). The disease must have been transmitted from the adult to the eggs by absorption on the surface of the shell. The resulting instantaneous death rate (ln of proportion surviving) of 2.7/9 months is higher than in a virus-free situation and this mean value is then added to the mortality during the 3 month period. The yearly total death rate in the presence of the virus is now 11.9/year with a minimum at 8.9 and a maximum at 14.5. Since (α) is the added or excess mortality caused by the virus, the natural death rate must be removed from the total mortality to estimate the disease-induced death rate. Given a value of 4.6 for (b), (α) is 7.3/year and the maximum and minimum estimates are 9.9 and 3.4, respectively. However, given a (b) of 3.0 the value of (α) ranges between 6.9 and 11.5 with a mean at 8.9. The values of α tested in the sensitivity analysis are between 5.3 and 11.3.

The values of the disease-induced death rate (α), are very similar to the rates calculated using plots that had been sprayed with the virus in British Columbia and Oregon (Stelzer et al. 1975; 1977). In those cases the mean total death rate is 8.6 with a minimum at 7.9 and a maximum at 10.7. Since the main mortality agent is the virus and nearly all the larvae collected for rearing died of the virus, the total death rate in the

sprayed plots can be considered to represent a maximum disease-induced mortality rate, albeit in artificially enhanced conditions. Since generally no egg masses are found following a spray operation the egg survival is considered to be nil.

Rate of production of viral infective stages ,λ,

The infective stages are not released at a regular rate once an insect becomes infected. Dissemination only occurs when the infected host dies, ruptures and releases the viral particles onto the foliage. The rate of production (λ) can be estimated as follows:

$$\lambda = \Lambda(\alpha+b) \quad (17)$$

where (Λ) is the number of inclusion bodies (PIB) produced during the expected lifespan of the infection, (1/ α+b). The number of PIBs produced varies with the age and the size of the larva at the time of death. Thompson and Scott (1979) measured the average production of inclusion bodies per early instar larva to be $1x10^{7}$ PIB, as opposed to $4x10^{8}$ PIB for each late instar larva. Over a period of 50 days the average number of PIB/larva was $1.8x10^{8}$. This value is not far from the average yield/larva of $6.7x10^{8}$ PIB obtained in the pilot plant where the virus is mass produced (Martignoni, 1978). In the computer program, the value of (λ) is changed everytime (α) and (b) are modified but since (Λ) which is set at $2x10^{8}$ PIB, is so much larger than (α) and b, the overall value for (λ) in the above

equation is not greatly affected.

Rate of mortality of the infective particles ,u,

The viral particles are contained in a protein crystal that offers a certain degree of protection against environmental conditions. If not exposed directly to ultraviolet radiation from the sun, the polyhedra can survive for long periods of time. Jaques (1975)demonstrated the persistence of the cabbage looper nuclear polyhedrosis virus in the soil for as long as six years. Thompson and Scott (1979) also found active polyhedra in the forest soil and in the litter, 11 years after an NPV epizootic in the tussock moth. In another case where the last tussock moth outbreak occured in 1936-38, some soil samples, taken in 1979, revealed the presence of active virus at a very low concentration (< 45 PIB/cm^3) (Thompson et al.,1981). This persistence of viral particles in the environment permits the virus to survive periods of low host insect density. Presumably the virus is continually reintroduced to the foliage through dust transport (Thompson & Scott, 1979). Once it is present on the canopy it can spread through normal contagion if the density of the moth is high enough.

Although a small quantity of viral particles can persist for an extended length of time in the duff, the majority are deactivated on the foliage within the first year. Using Thompson and Scott's (1979) data, Anderson and May (1981) estimate the

expected virus lifespan on the foliage to be between 2 to 3 months. Data regarding this parameter are very limited but I have tentatively set a baseline value for (u) at 5.0, which corresponds to a 2.5 month lifespan (u=1/expected lifespan) and a maximum and minimum value at 4.0 and 6.0 which represent a three and two month lifespan respectively. These values reflect a low survival of the inclusion bodies. The possibility that viruses are more persistent in the environment will be explored in the sensitivity analysis; (u) values will be varied from 0.5 to 7.5.

Transmission coefficient ,β,

According to Anderson and May (1980) the transmission coefficient (β) is impossible to determine. However, they claim that since it is a scalar that only affects the magnitude of the insect density (N) and not the prevalence of infection (Y/N), they set its value arbitrarily to $1x10^{-9}$. Simulations done here confirmed that different values of (β) do not affect the existence or periodicity of the cycles as long as (r) is close to or higher thaN 1.0. However while doing the sensitivity analysis on the parameters (b) and (u), with the basic model, I observed exceptions to this generalization but these exceptions turn out to be unimportant (see section IX "Sensitivity analysis").

Density-dependent mortality

Because (r) is treated as a constant in Anderson and May's model(1981), they do not account for the possibility of decreased recruitment at high density. The evidence that such a phenomenon occurs is scant but data from Aztec Peak show a pattern of decreased recruitment at higher densities (Fig. 9). This method of analysis is not a formal test of density-dependence because of two biaises introduced by having the independent variable N_t included in the dependent variable N_{t+1}/N_t and a measurement error on the independent variable (Sokal and Rohlf, 1969). However, for the purposes of the model it is more informative to look at changes in the mortality rates. As shown in Fig. 10, there is a trend of increased mortality at higher density for the plots used in the estimation of (b) and (α).

The linear function describing the changes in total mortality is

$$d=4.7+0.07N \quad (18)$$

The modified death rate is determined by

$$b'=b+cN \quad (19)$$

where (b), which represents the minimum insect natural mortality, takes on values between 2.6 and 4.0 in the simulations and (c), which represents the severity of the density-dependent constraints, is varied from 0.0 to 0.06. Those values insure that the natural mortality does not exceed the

Fig. 9. Recruitment of Douglas-fir tussock moth in Aztec Peak for the years 1967-1970 and 1975-1978. (Data from Mason, R.R. pers. comm.).

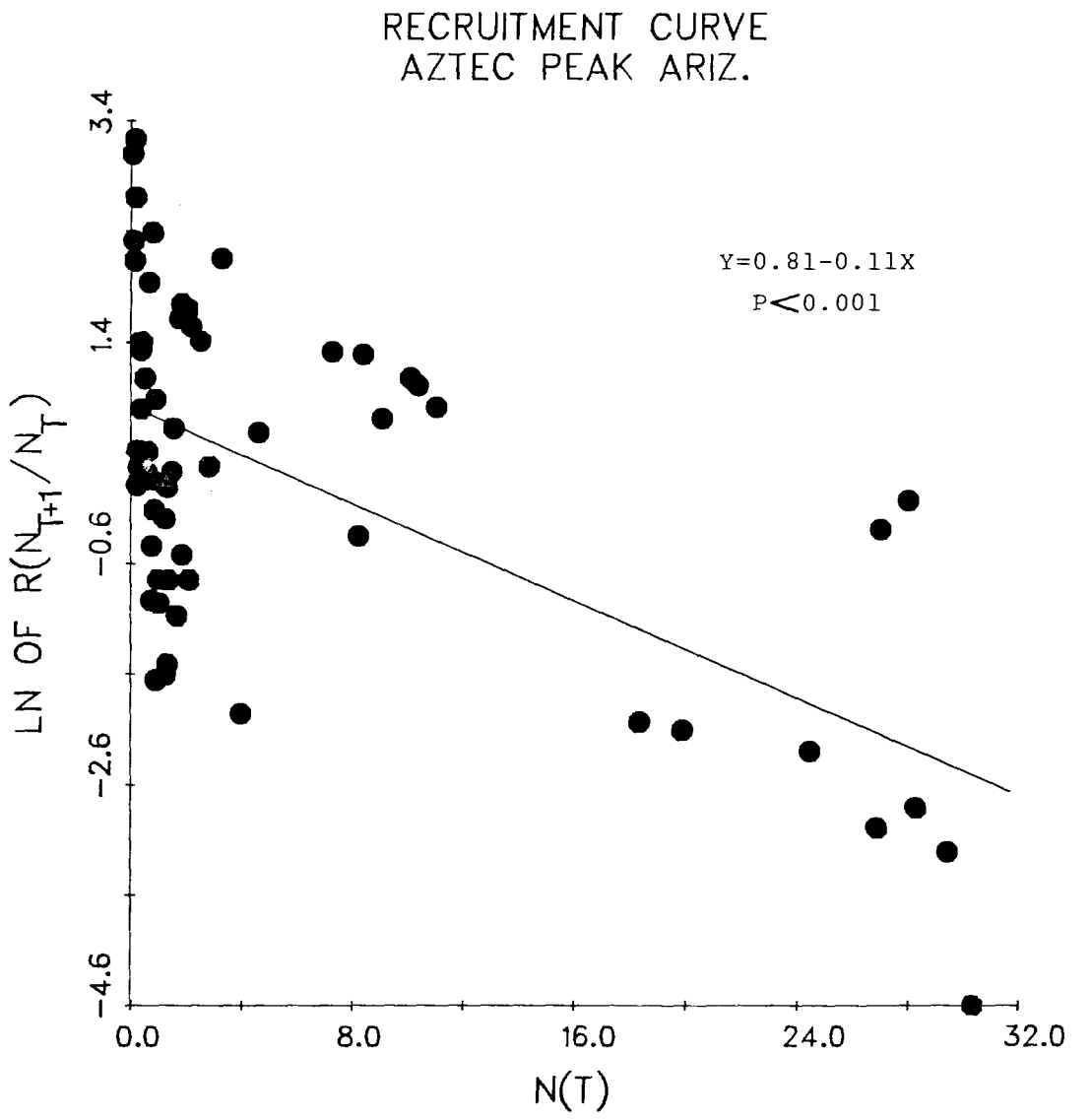
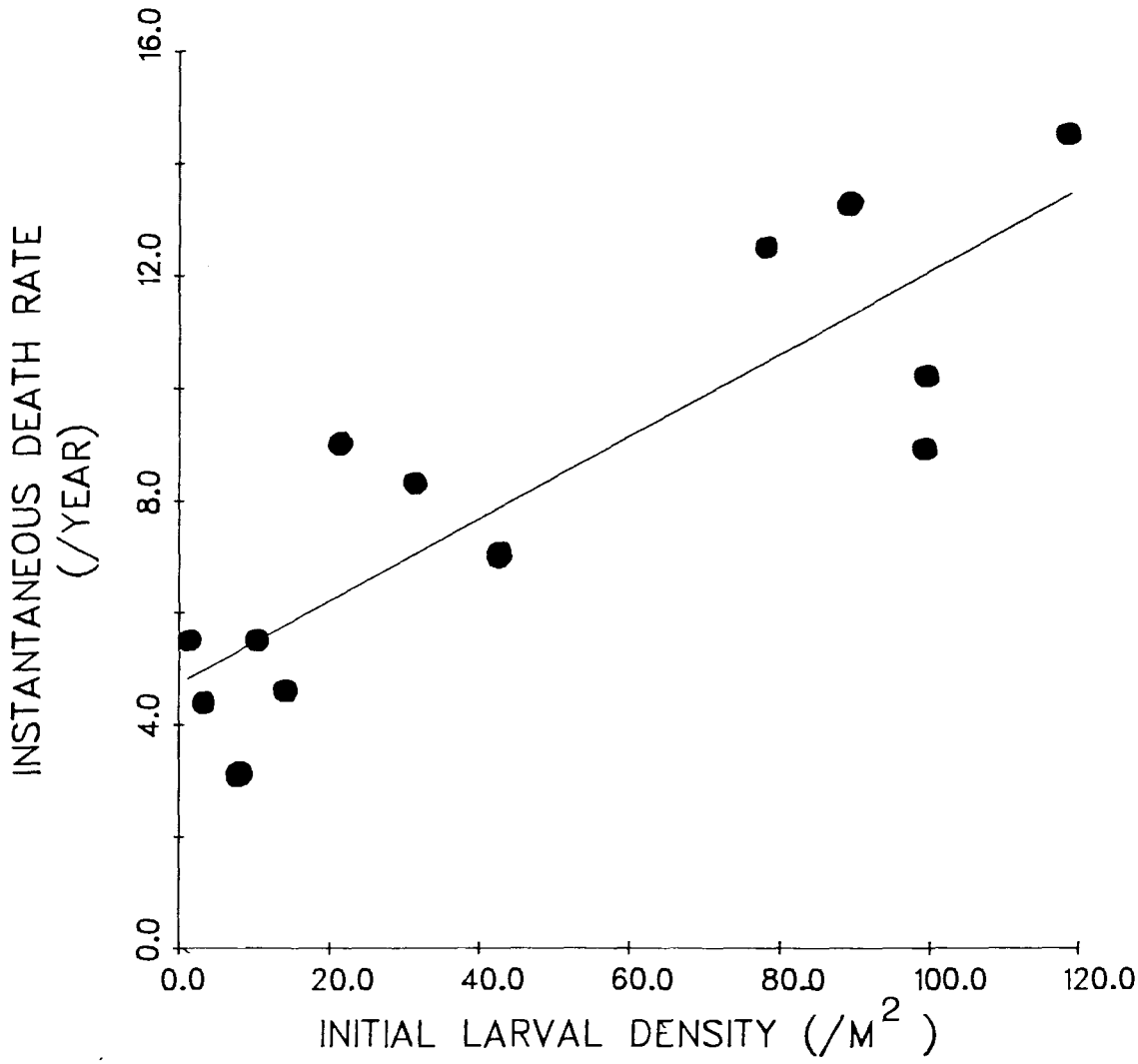

RECRUITMENT CURVE
AZTEC PEAK ARIZ.
Y=0.81-0.11X
P<0.001
LN OF R(N_{T+1}/N_T)
3.4
1.4
-0.6
-2.6
-4.6
0.0
8.0
16.0
24.0
32.0
N(T)

Fig. 10. Density-dependent total mortality rate for Douglas-fir tussock moth populations. (Data from Mason and Torgersen, 1977; Mason R.R. pers. comm.; Mason and Thompson, 1971).



Y = 4.736 + 0.07342 * X
SE(B)=0.013 P=0.0001
SS = 39.107 R = 0.868

total mortality.

Incubation period ($1/\nu$)

During the summer of 1981 I tried to simulate a virus epizootic on stocked Douglas-firs. The experiment did not provide the expected information on the rate of spread of the disease but some of the data can be used to estimate the length of the incubation period. Tussock moth larvae were obtained from egg masses collected in Hedley, British Columbia, in April 1981 where a minor infestation was under way. In June the egg masses were surface sterilized in 0.1% sodium hypochlorite in order to prevent undesirable contamination. When the larvae reached second instar, they were placed on contaminated Douglas-fir foliage that had been immersed in a suspension of virus (4×10^7 PIB/ml). The original inoculum, provided by I.S. Otvos[2], for this fresh suspension of virus was from a 1975 artificially induced epizootic in Kamloops. After 36 h of feeding on the foliage, 30 larvae were put on small Douglas-fir trees (1m high) covered with a fine mesh. The experimental plot was situated on Burnaby mountain. Larvae were enumerated every day for the following 2 weeks until all the larvae died.

The larvae started to die 7 days after being inoculated (Fig. 11). The incubation period from the day of contraction of the infection was estimated by dividing the cumulative sum of

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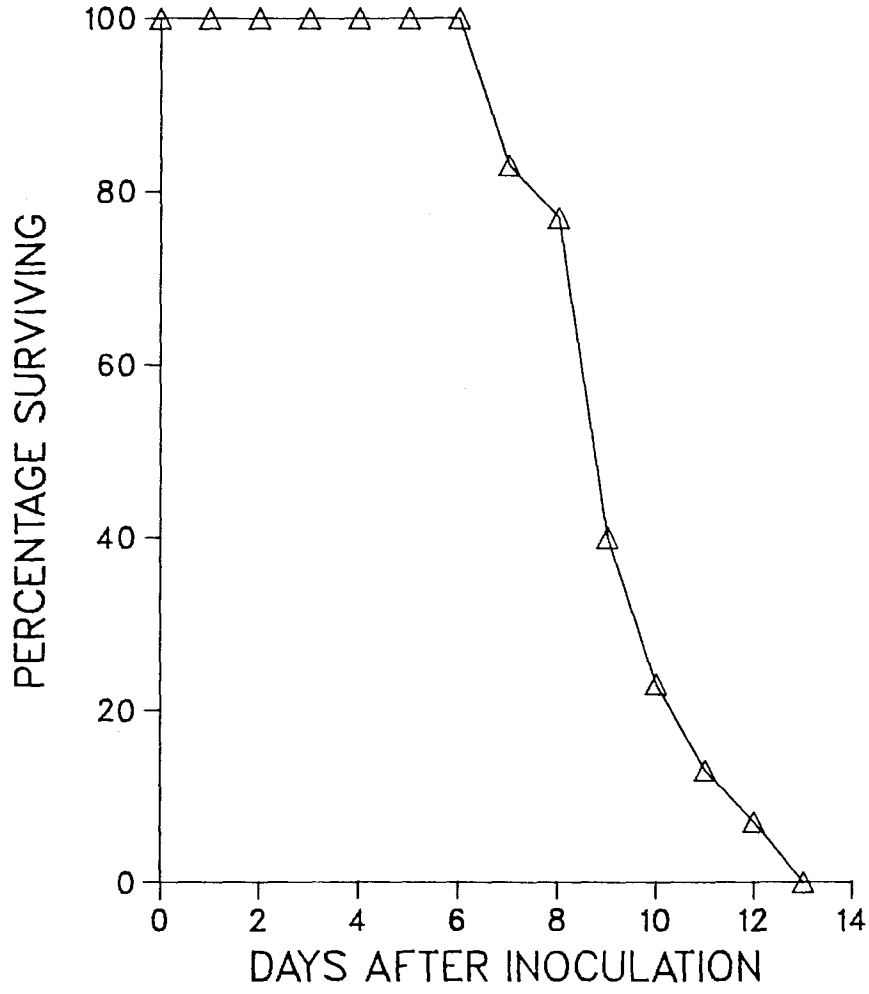
2 Pacicifc Forest Research Service, Victoria B.C.

Fig. 11. Survivorship curve for Douglas-fir tussock moth larvae experimentally infected with NPV (4×10^7 PIB/ml)


100
80
60
40
20
0
PERCENTAGE SURVIVING
0
2
4
6
8
10
12
14
DAYS AFTER INOCULATION

the average number of organism surviving from day (t) to day (t+1) by the number of individuals present at the beginning (Krebs, 1978). The incubation period is then between 8-9 days and is ajusted for the 3 month period of insect activity. Given a minimum of 7 days and a maximum of 12 days the value of (ν) ,calculated by setting ($1/\nu$) equal to the incubation period, ranges from 7 to 12 with a mean at 10/year.

Vertical transmission

The percentage of first instars dead as a result of the viral infection is a reasonable estimate of the amount of vertical transmission occuring, since at that time mortality due to contagion has not yet occured given the length of the incubation period. In the last year of the outbreak in Modoc National Forest, 10% of early instars were infected and as previously mentioned between 13 and 32% of the recently emerged larvae died from the virus in Northeastern Oregon. However lower values are more common. In the fall of 1973 in Northern Idaho, egg masses were collected in order to assess the potential for defoliation by the tussock moth the next spring (Tunnock et al., 1974). The eggs were put to hatch under controlled conditions and the percentage of larvae contracting the disease was recorded. The results from 91 collecting points range from 0% to one plot with 30% disease, but since the data are log normally distributed the geometric mean (antilog($1/n\Sigma\log p$)) of 2.0% is

used (Fig. 12).

Random variation on growth rate ,v,

The distribution of the trend indices for the 10 plots from which the growth rate (r) is estimated is shown in Fig.13a. Because of the low number of replicates it is impossible to distinguish among the fit of several different theoretical distributions of the data. However if trend indices, for the same plots, covering 8 years are included, the resulting distribution is clearly log normal (Fig. 13b) and a multiplicative log-normally distributed noise term is chosen because the survival of the larvae depends on a series of successive and relatively independent survival rates from one stage to the other (Mason, 1981a; Peterman, 1981). For comparison an additive normally distributed error term is also used.

In the multiplicative log-normal model

$$r' = re^{v} \quad (20)$$

as well as the additive normal model

$$r' = r + v \quad (21)$$

v is a normally distributed random variate with mean 0 and standard deviation of 0.3.

The value of 0.3 for the standard deviation of the ratio of the means ($r = \bar{N}_{t+1} / \bar{N}_t$) is calculated from the following formula

Fig. 12. Prevalence of the virus in newly emerged Douglas-fir tussock moth larvae in Northern Idaho (Data from Tunnock et al. 1974) .

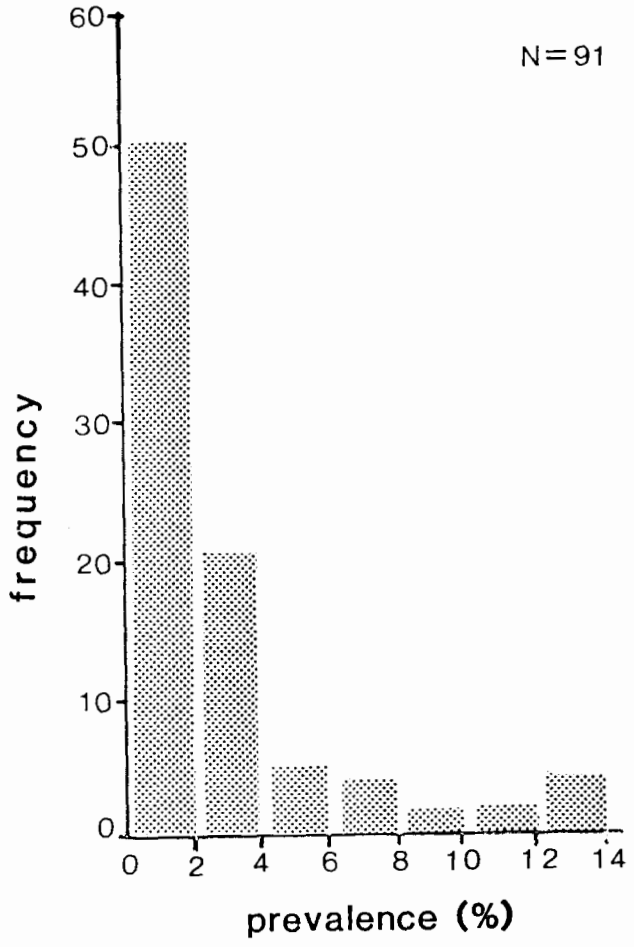
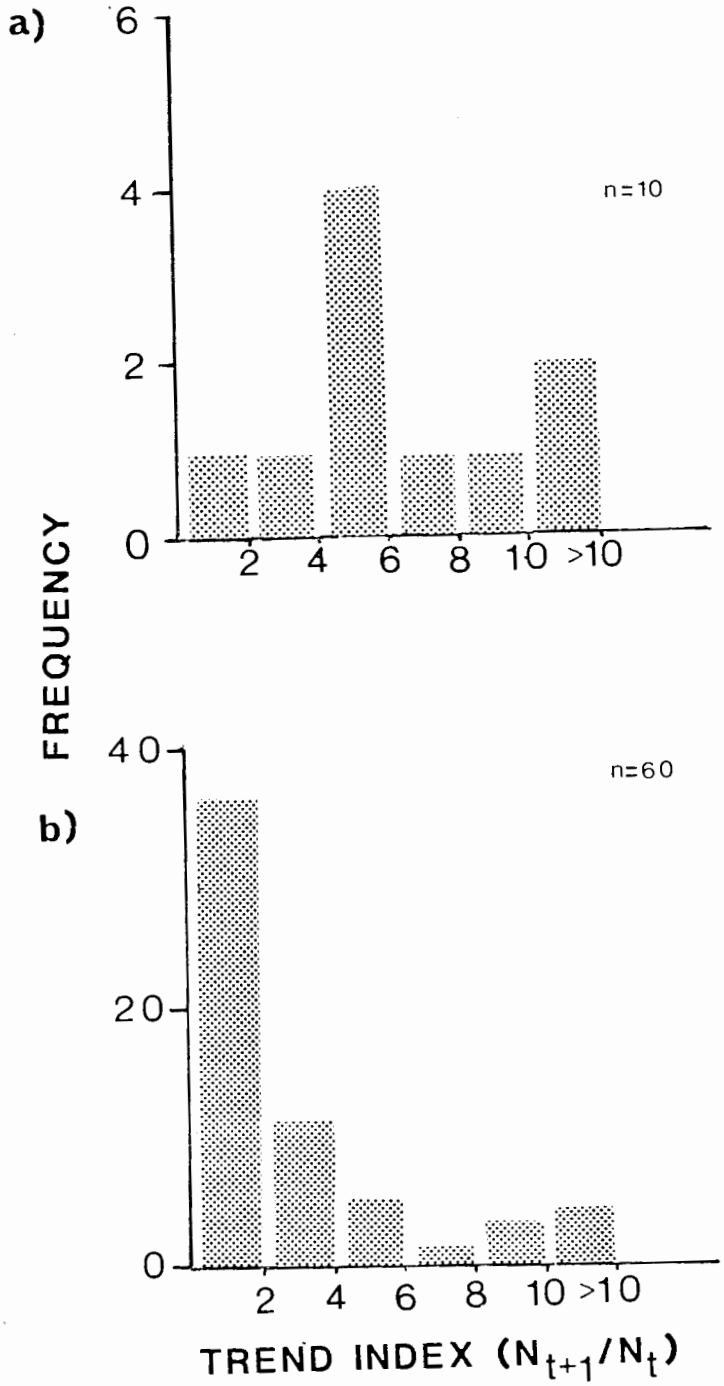

N=91
60
50
40
30
20
10
0
frequency
0
2
4
6
8
10
12
14
prevalence (%)

Fig. 13. Trend indices for populations of Douglas-fir tussock moth in Aztec Peak for the years (a) 1967-1968 and (b) 1967-1970 and 1975-1978. (Data from Mason R.R. pers. comm.).


a)
6
4
2
0
n=10
2 4 6 8 10 >10
FREQUENCY
b)
40
20
0
n=60
2 4 6 8 10 >10
TREND INDEX (N_{t+1}/N_t)

(Villegas C., pers. comm.[3])

$$\sqrt{\frac{Y^2\sqrt{\overline{X}^2} + X^2\sqrt{\overline{Y}^2} - 2XY\sqrt{\overline{XY}}}{X^2}}$$

where $\bar{N}_{t+1} = Y$ and $\bar{N}_t = X$ and since $\bar{N}_{t+1}$ and $\bar{N}_t$ are correlated ($r = .986$) the correlation coefficient is embeded in the formula.

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VII. Simulations

Simulations were performed with each version of the models by using a differential equation solver package (DVERK) (IMSL 1982). This package takes the differential equations which, given initial conditions for the state variables and parameter estimates, describe changes in state variables and calculates values for those variables at the end of 0.2 year to obtain a better resolution. Initial variable conditions used were 100 insects/m^2 for the total host population comprised of 10 infected and 90 susceptible hosts.The initial virus density is 10^6 PIB/m^2. Simulations were run for 100 years.

VIII. Criteria for evaluating model performance

Before solving the differential equations with the estimated parameter values, it is necessary to establish the criteria that describe the field situation and that will serve to evaluate the results of the simulations (Table 4). The maximum host density comprises values between 30 and 154 early instar larvae/m^2, based on studies in Arizona and California. Although greater population densities have been recorded in Oregon, they are not included in the upper limit because the course of the outbreak was different from regions where the virus is a more predominant mortality factor.

Between outbreaks the tussock moth usually persists at low levels and a value for the minimum density should be below 1 early instar larvae/m^2. Intervals between peak densities may be several years long and a group of researchers from the University of Washington came to the conclusion that outbreaks over the entire geographic range of the tussock moth occured every 8 to 9 years (Mason and Luck, 1978). Intervals as short as 7 years and as long as 10 years have been observed so a range of periods between 7-10 years is acceptable (Sudgen, 1957; Wickman et al. 1973). As for the density of viral particles, the confidence on this indicator is low since only indirect estimations are available and any result in the 10^7 and 10^9 range is acceptable. It is more informative to look at the

Table 4. Table of criteria for the behavior of Douglas-fir tussock moth populations in the field.

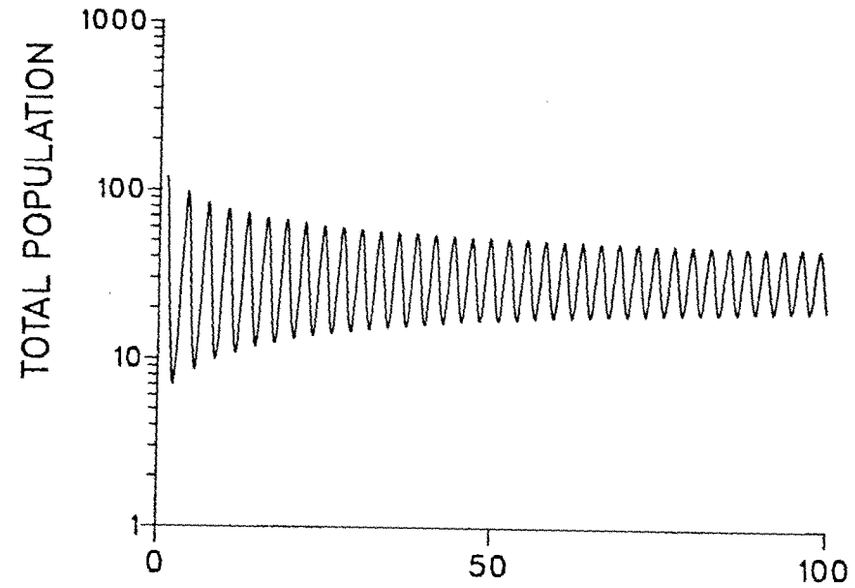
CRITERION	OBSERVED VALUES
Period	7-10 years
Prevalence of infection	25-50%
Amplitude of oscillations	30-150 hosts/m^2
Maximum host density	30-150 hosts/m^2
Minimum host density	$<$1 host/m^2
Virus density	10^7-10^9 PIBs/m^2

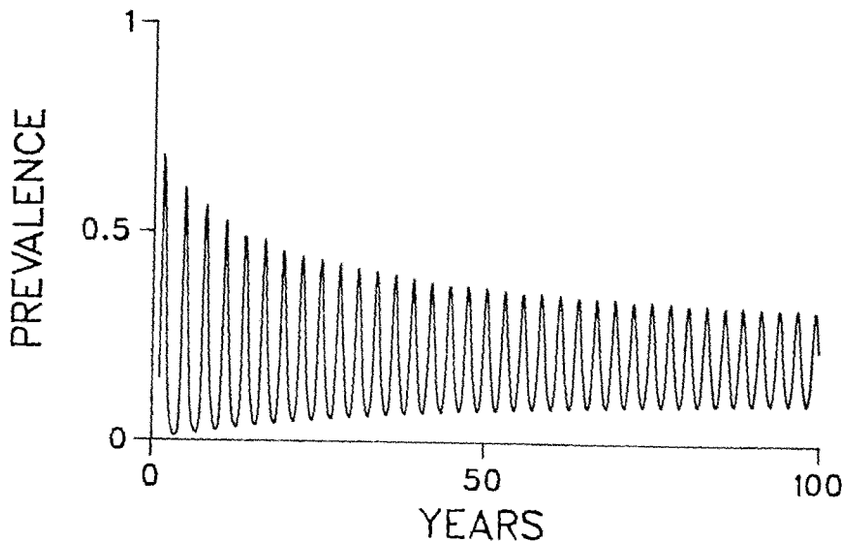
prevalence of the disease in the larval stages where 25 to 50% of the insects die of the virus. However, as in any predator-prey model, the virus lags behind the peak in host abundance which is the reason for the 2 measures of prevalence, one taken at maximum host density and one taken slightly later when virus prevalence is maximum. Since the resolution in the field data is usually not fine enough to differentiate between the two, Anderson and May's (1981) convention is followed and emphasis is on the maximum prevalence of the virus. Therefore, prevalence at maximum host density and density of virus are not important indicators upon which the decision of rejecting the applicability of the model to the tussock moth will rest. More importance is given to the period between outbreaks, the amplitude of the population cycles and the maximum prevalence of the disease.

IX. Sensitivity analysis

It would be time consuming to go through a set of simulations one by one, changing one parameter at a time and analysing the time series, such as the one presented in Fig.14. A faster method is to incorporate in the simulation program equations that calculate the above-mentioned characteristics of the time series. Among the statistical indicators chosen are the maximum and minimum densities from which the amplitude is calculated. The amplitude is 0 if the solution is an equilibrium point or greater than 0 when cycles are generated. To determine the stability of the cycles, the difference in amplitude from one cycle to the next is measured. The oscillations are stable if the difference is 0, increasing in amplitude if the difference is greater than 0, or decreasing in amplitude if the difference is less than 0. The other indicators are the prevalence of infection at maximum host density; the maximum prevalence; the maximum density of virus particles (PIB); and the period of the cycles. The performance indicators are calculated for the last 50 years of the 100 years simulations. If more than one cycle appears in these 50 years, then the value of the performance indicator is averaged over the cycles (e.g. if there are 3 cycles, with periods of 8, 10 and 12 years then the average period would be 10 years).

Fig. 14. Time series for the basic model (α=8.9, r=1.7, b=3.0, a=4.7, $\Lambda=2\times10^{8}$, $\beta=1\times10^{-9}$, u=5.0)


TOTAL POPULATION
1000
100
10
1
0
50
100


PREVALENCE
1
0.5
0
0
50
100
YEARS

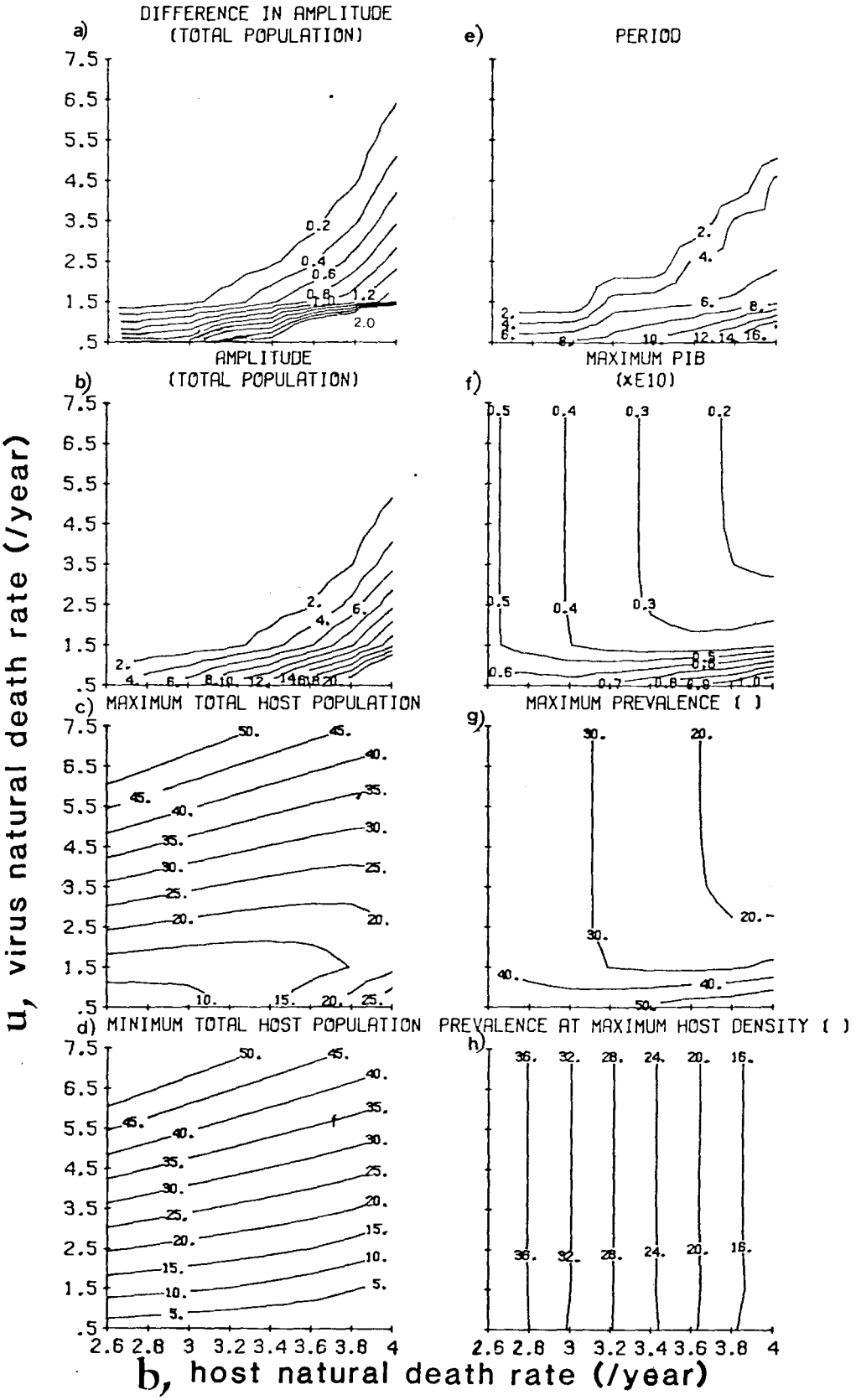
New values for the indicators are generated each time a parameter is changed and all these results are summarized in the form of nomograms also known as response surfaces or isopleth diagrams. These graphs illustrate the numerical change in a given indicator when 2 parameters are modified. Except for those 2 parameters under scrutiny, the baseline conditions remain constant throughout the simulations. This way the sensitivity of certain parameters considered important is evaluated for the effect they have on each indicator. These nomograms graphically summarize the results of different simulations. Nomograms with a finer grid resolution do not affect the results and the same scale for the virus natural death rate is used for all the nomograms except for the basic model nomograms where some intermediate values are omitted.

The next step is to determine the range of parameters that most closely reflect the field situation. This is achieved by isolating the region of each graph where the isopleths correspond to the behavior of that particular indicator in nature.

Basic model

On each nomogram in Fig.15, the virus natural mortality rate (u) increases along the Y axis and the natural death rate of the insect (b) varies along the X axis . When the value of (b) increases the corresponding insect population growth rate

Fig. 15. Nomograms of the basic model with α =5.3 (a=4.7, Λ =2x10^{8}, β=1x10^{-9})


a) DIFFERENCE IN AMPLITUDE (TOTAL POPULATION)
b) AMPLITUDE (TOTAL POPULATION)
c) MAXIMUM TOTAL HOST POPULATION
d) MINIMUM TOTAL HOST POPULATION
e) PERIOD
f) MAXIMUM PIB (xE10)
g) MAXIMUM PREVALENCE ( )
h) PREVALENCE AT MAXIMUM HOST DENSITY ( )
u, virus natural death rate (/year)
b, host natural death rate (/year)

(r) decreases ; when b=4.0 (r) has a value of 0.7. Values of the growth rate lower than 0.7 have not been included in the sensitivity analysis because at low values of (r) a slight change in the transmission coefficient affected the pattern of infection (Table 5) to the point where sometimes the model produces periods of host population fluctuation near those observed in the field. Those results are unreliable because the value of the period is not constant for different values of the transmission coefficient, and even if the desired period is obtained (e.g. 9 years), the performance of the other indicators is inadequate.

The model is run with a single set of parameter estimates and the value for each of the 8 indicators is generated, e.g. the period for the last 50 years., The model is run again for a new combination of (u) and (b) while keeping all the other parameters constant. In order to explore all the possible combinations, of (u) and (b) 64 simulations are necessary and each nomogram in Fig. 15 represents the result of those 64 runs. As shown, there exists different pairs of (u) and (b) which give rise to the same period, e.g. 8 years. Those points which represent a period of 8 years are joined and referred to as an isopleth. The same procedure is applied to all the indicators. In Figs.15 to 18, the same simulations are done for different disease-induced mortality rates.

When (α) is small the maximum and minimum total host populations are relatively insensitive to variations in (b) but

Table 5. The effect of the transmission coefficient on the pattern of infection at low growth rate with the basic model. ($a=4.7$ to 5.6, $b=4.6$, $\alpha=8.9$, $\Lambda=2\times10^{8}$).

Virus death rate (/year)	Growth rate (/year)	Transmission coefficient (/year)	Max. host density ($/m^2$)	Min. host density ($/m^2$)	Maximum prevalence	period (years)
1.0	0.1	$1x10^{-8}$	—	$0.16x10^{-6}$	—	94
		$1x10^{-9}$	20.2	0.52	0.198	44
		$1x10^{-10}$	117.0	15.0	0.085	28
	0.3	$1x10^{-8}$	13.7	$0.25x10^{-5}$	0.836	67
		$1x10^{-9}$	22.6	0.38	0.279	19
		$1x10^{-10}$	182.0	6.8	0.219	17
3.0	0.1	$1x10^{-8}$	14.2	0.02	0.747	71
		$1x10^{-9}$	20.9	10.7	0.421	14
		$1x10^{-10}$	627.0	17.6	0.395	39
5.0	0.1	$1x10^{-8}$	13.1	0.26	0.643	50
		$1x10^{-9}$	35.7	17.4	0.567	13
		$1x10^{-10}$	1315.	26.0	0.601	42
	0.3	$1x10^{-8}$	9.5	0.48	0.476	12
		$1x10^{-9}$	38.6	16.8	0.111	7
		$1x10^{-10}$	951.0	47.9	0.480	12
	0.6	$1x10^{-8}$	6.7	0.91	0.360	6
		$1x10^{-9}$	45.6	14.9	0.204	5
		$1x10^{-10}$	679.0	87.9	0.365	6
	1.0	$1x10^{-8}$	4.6	1.7	0.253	4
		$1x10^{-9}$	40.1	19.6	0.210	4
		$1x10^{-10}$	464.0	159.0	0.258	4

they change more significantly with the mortality of the virus (u) (Fig. 15 c,d). Viruses that are long-lived (small virus death rate, u) depress the maximum host densities since they are more persistent in the environment and are available longer for reinfection. Viruses that have a short life-span (high death rate, u) permits the host population to stabilize at a higher density since it has little time to contaminate another host before it is deactivated (Fig.15 c). On the other hand, the life expectancy of the virus is not a major influence on the number of viral particles produced and the maximum prevalence of infection, both of which vary mainly with (b) instead of (u) (Fig. 15 f,g). The turnover rate of the host population determines how many viral particles are eventually produced and at the same time determines the prevalence of the disease. When new susceptibles are produced at a low rate the pathogen is maintained in a small fraction of the population.

Paradoxically, if the pathogenecity of the virus is increased (large α) the maximum host density attained is higher for all the combinations of (u) and (b) (Fig. 15c, 16c, 17c, 18c). Intuitively a more efficient virus should depress its host density to a lower equilibrium. But a virulent pathogen kills its host before a sufficient amount of viruses is produced to infect the remainder of the population, thereby leaving a higher proportion of the host population free of the disease. When (α) is increased the prevalence changes with (u) and not (b) as previously observed (Figs.16g, 17g, 18g). So the capacity of the

Fig. 16. Nomograms of the basic model with α =7.3 (a=4.7, $\Lambda=2\times10^{8}$, $\beta=1\times10^{-9}$)

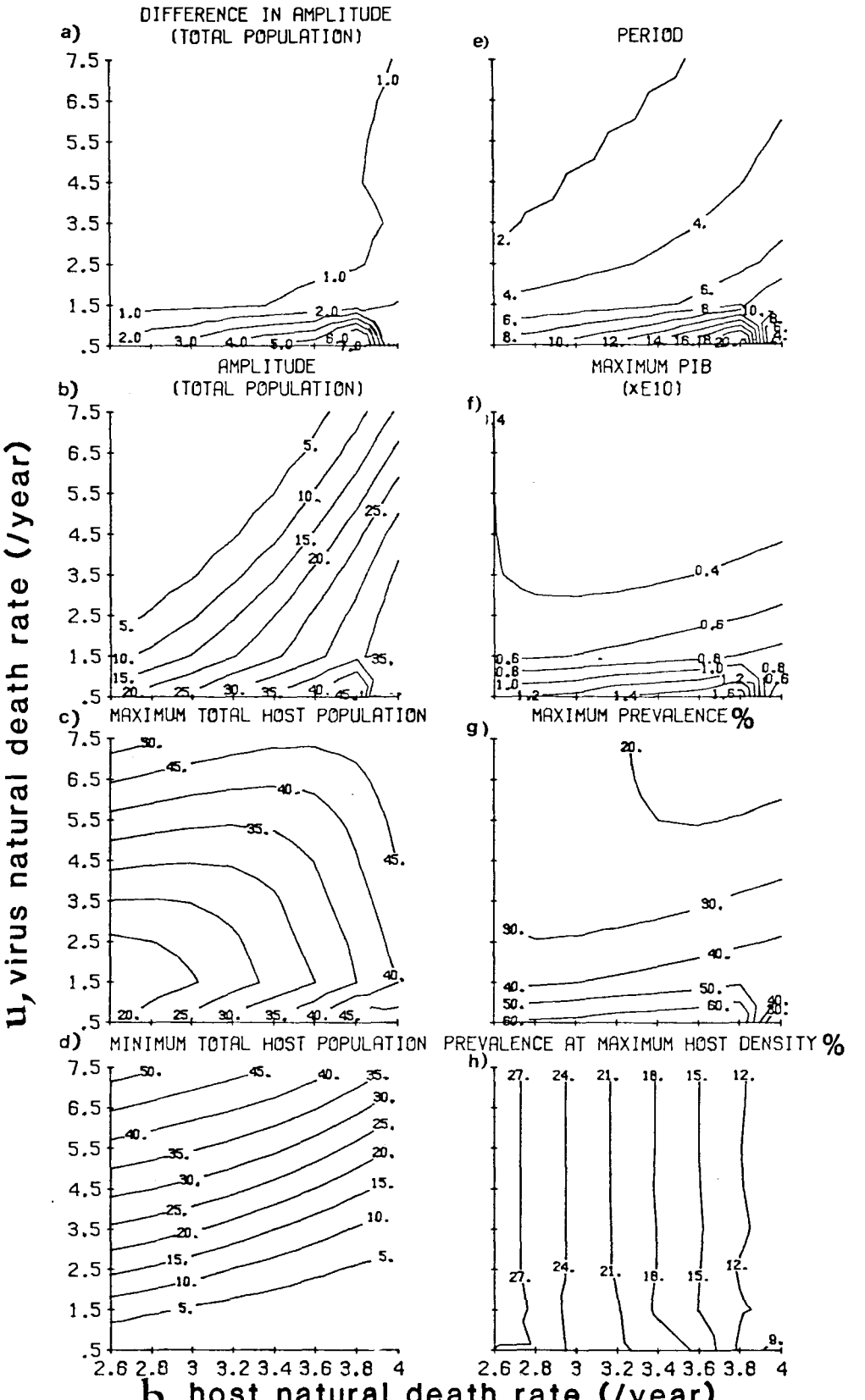

a)
DIFFERENCE IN AMPLITUDE
(TOTAL POPULATION)
b)
AMPLITUDE
(TOTAL POPULATION)
c)
MAXIMUM TOTAL HOST POPULATION
d)
MINIMUM TOTAL HOST POPULATION
e)
PERIOD
f)
MAXIMUM PIB
(xE10)
g)
MAXIMUM PREVALENCE %
h)
PREVALENCE AT MAXIMUM HOST DENSITY %
u, virus natural death rate (/year)
b, host natural death rate (/year)

Fig. 17. Nomograms of the basic model with $\alpha=9.3$ ($a=4.7$, $\Lambda=2\times10^{8}$, $\beta=1\times10^{-9}$)

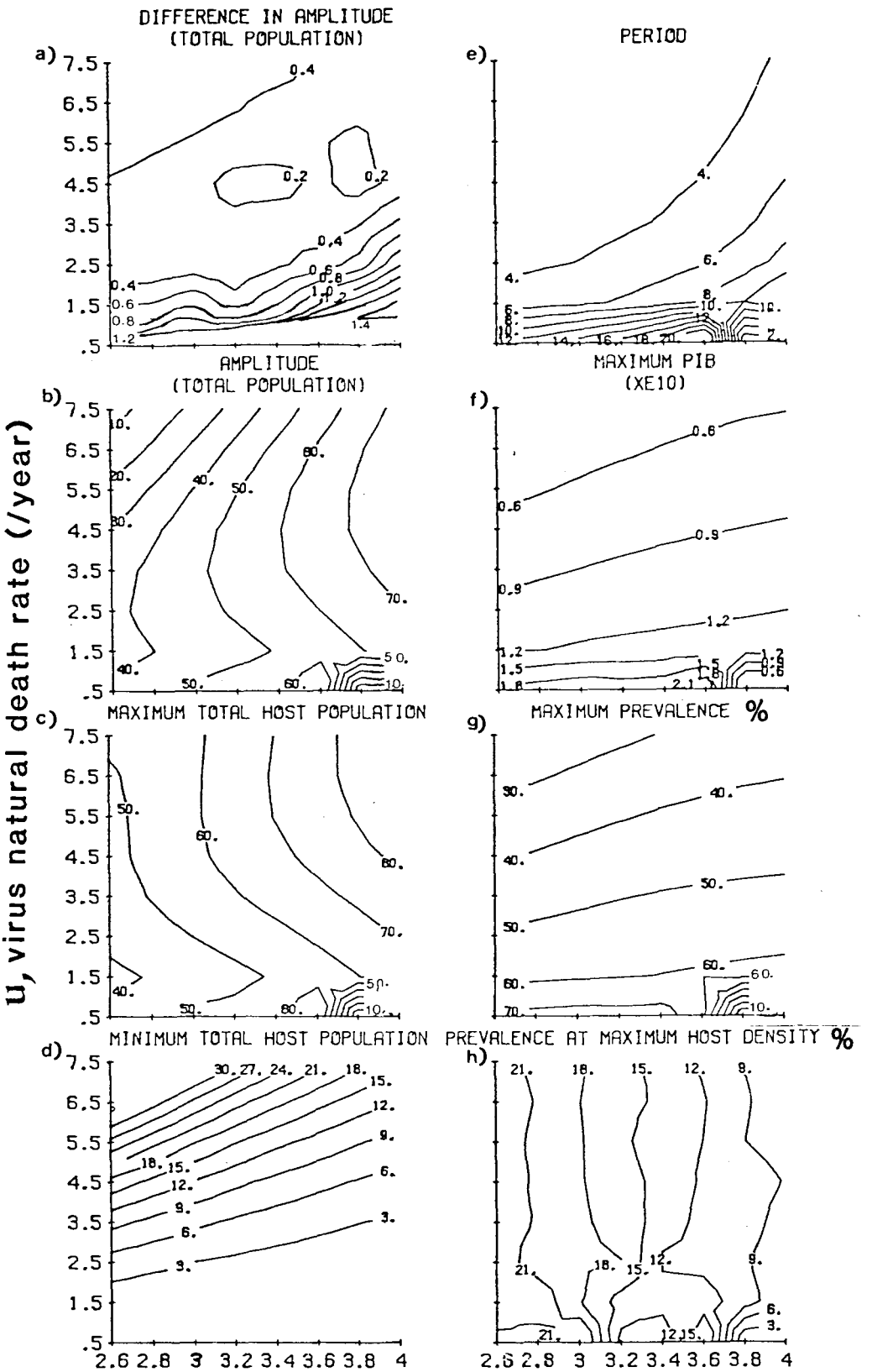
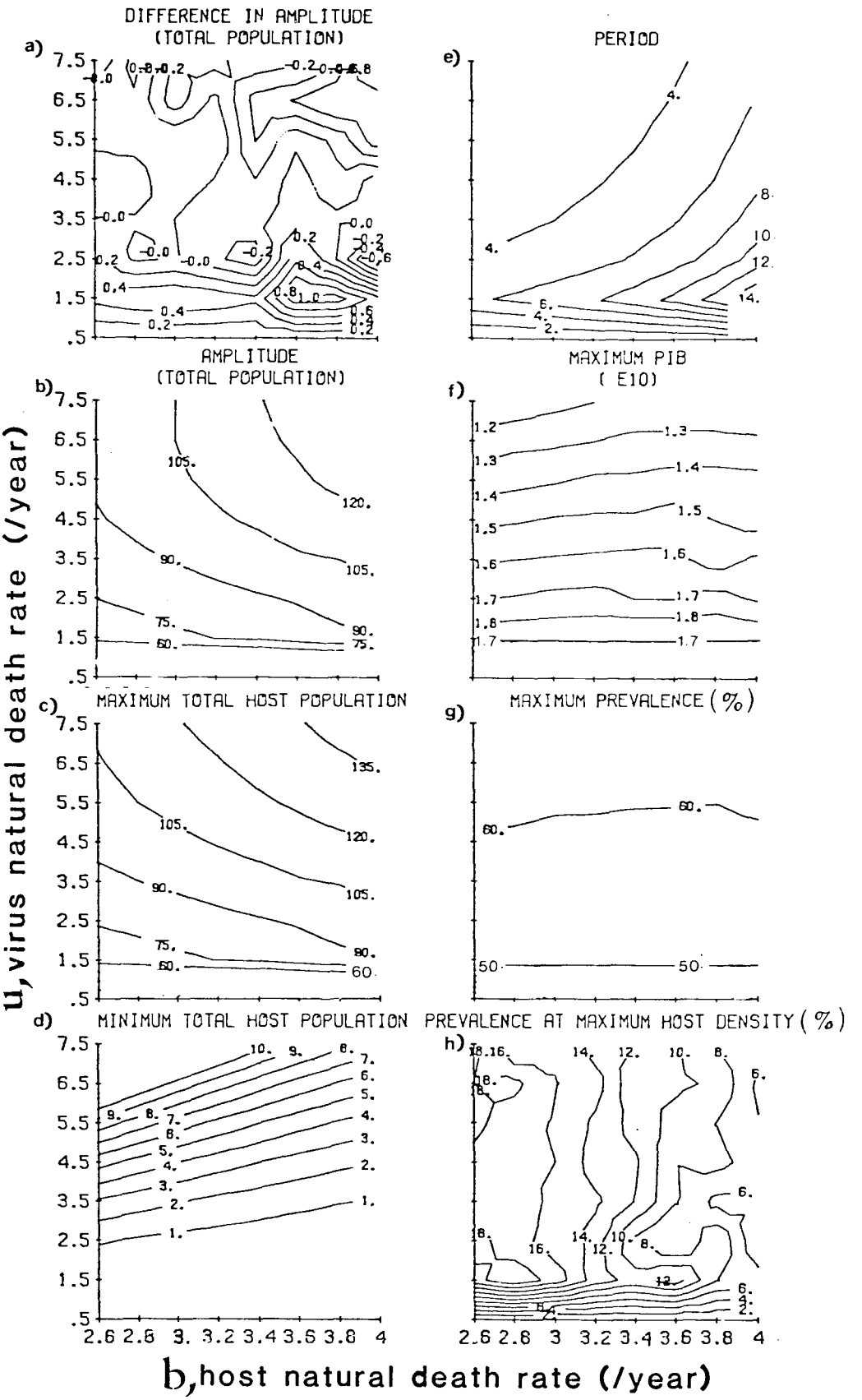

DIFFERENCE IN AMPLITUDE
(TOTAL POPULATION)
a)
AMPLITUDE
(TOTAL POPULATION)
b)
MAXIMUM TOTAL HOST POPULATION
c)
MINIMUM TOTAL HOST POPULATION
d)
PERIOD
e)
MAXIMUM PIB
(XE10)
f)
MAXIMUM PREVALENCE %
g)
PREVALENCE AT MAXIMUM HOST DENSITY %
h)
u, virus natural death rate (/year)
b, host natural death rate (/year)

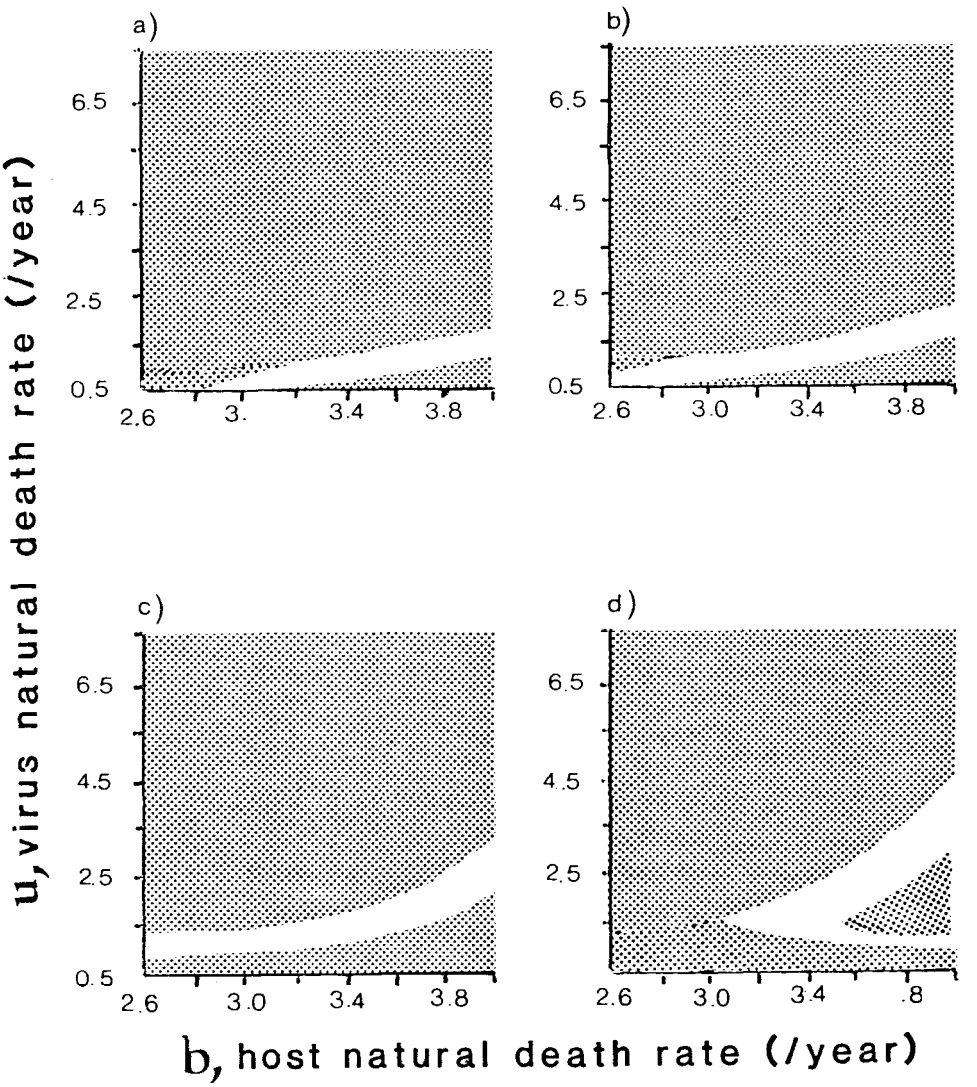
Fig. 18. Nomograms of the basic model with $\alpha = 11.3$ ($a = 4.7$, $\Lambda = 2 \times 10^{8}$, $\beta = 1 \times 10^{-9}$)


DIFFERENCE IN AMPLITUDE
(TOTAL POPULATION)
a)
PERIOD
e)
AMPLITUDE
(TOTAL POPULATION)
b)
MAXIMUM PIB
(E10)
f)
MAXIMUM TOTAL HOST POPULATION
c)
MAXIMUM PREVALENCE (%)
g)
MINIMUM TOTAL HOST POPULATION
d)
PREVALENCE AT MAXIMUM HOST DENSITY (%)
h)
u, virus natural death rate (/year)
b, host natural death rate (/year)

virus to persist outside its host becomes important as fewer pathogens are produced. A pathogen which is able to survive long periods of time outside its host depresses its maximum host density to the point that if (u) is smaller than 1.0 a highly pathogenic virus exterminates its insect host (Figs. 16-18c,g). At the opposite end of the spectrum a short lived virus does not generate periodic oscillations (Figs. 16-18e).

The unshaded area in Fig. 19a represents the combination of parameter values which give periods of host population cycles of 7 to 10 years, the periods observed in the field. It is apparent that for (α) equal to 5.3 the value of the natural virus death rate (u) necessary to generate reasonable periods is much lower than the baseline condition of 5/year. As (α) is increased the range of parameter values giving rise to a period between 7 to 10 years, is enlarged but not enough to include a reasonable combination of (u) and (b) near those observed in the field. For example, at (α) equal to 11.3, which is larger than the baseline condition of 8.9, when (u) is an acceptable 4.0, (r) is equal to 0.8 (b=3.9) which is too low, and when (r) is 1.5 (b=3.2), (u) is too low at 1.5 (Fig. 19d). Therefore, none of the simulations of the basic model fulfill, with reasonable parameter values, the major criteria already specified for the tussock moth in Table 4.

Fig. 19. Period of Douglas-fir tussock moth population cycles (years) at different values of disease induced mortality (α). (a) $\alpha = 5.3$ (b) $\alpha = 7.3$ (c) $\alpha = 9.3$ (d) $\alpha = 11.3$. ($a = 4.7$, $\Lambda = 2 \times 10^{8}$, $\beta = 1 \times 10^{-9}$).

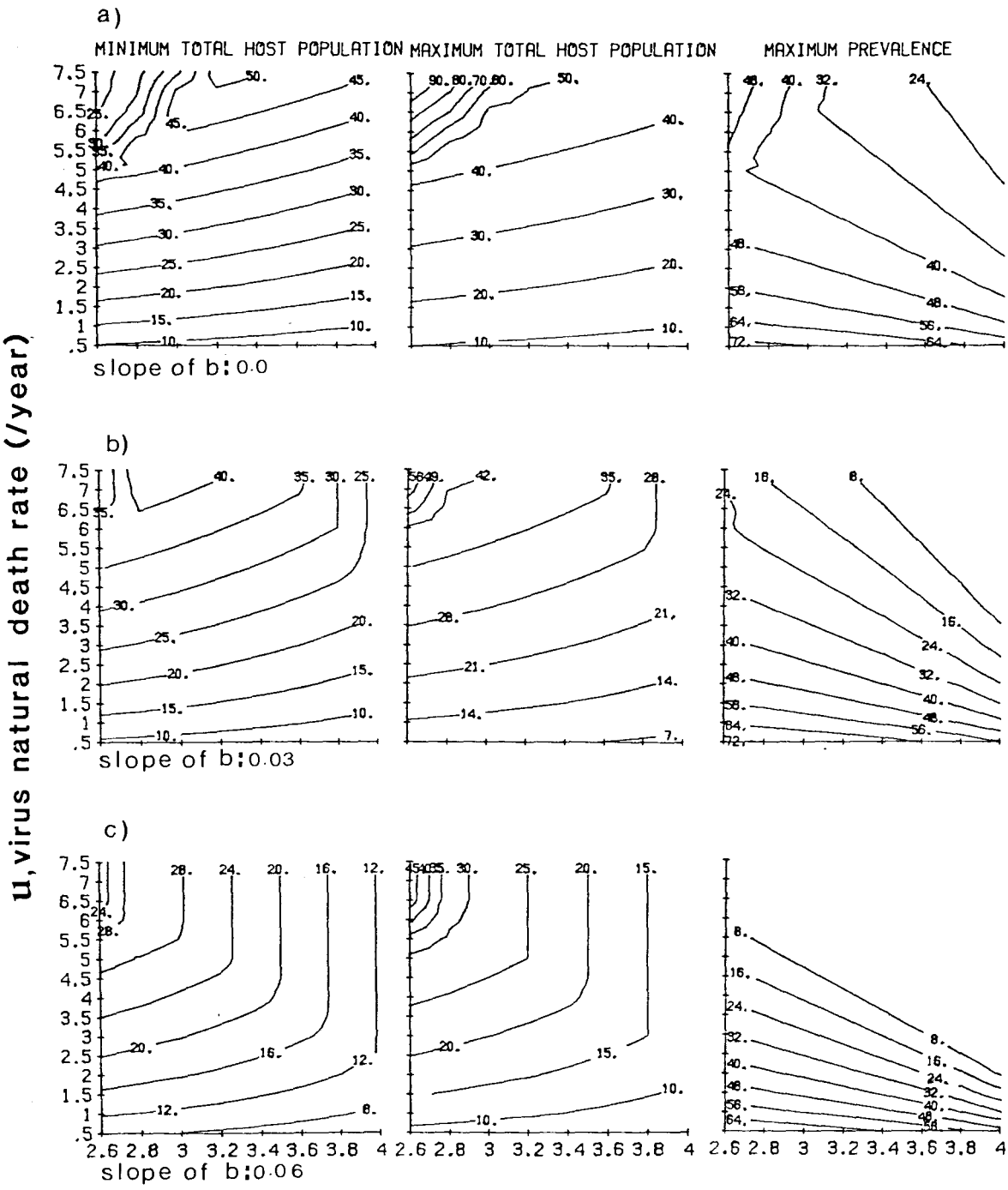

a)
b)
c)
d)
6.5
4.5
2.5
0.5
2.6
3.0
3.4
3.8
u, virus natural death rate (/year)
b, host natural death rate (/year)

Density-dependent model

In Fig.20 the X axis represents the minimum insect natural death rate (b) in equation (5), and the Y axis is the same as before. In the first set of simulations (b) is kept constant and only (α) is increased with density (Fig. 20a). In Fig. 20b and c both (b) and (α) are density dependent.

Including density-dependent mortality produces a single stable equilibrium point except for parameter combinations of short-lived virus (large u) and rapidly increasing hosts (small b), which generate stable cycles of 2 years. The dampening effect of host insect densities increases with the degree of density dependent constraints. When the reproductive rate is low (large b) and the virus short lived (large u), the hosts escape the influence of the virus as the latter goes to extinction and the prevalence drops to practically zero (Fig. 20b,c). With higher reproductive rates and for a given virus mortality rate, enough virus particles are produced by dying larvae to sustain higher incidences of disease in the host population. Thus the density-dependent model is rejected as a representation of the tussock moth in the field because it does not produce regular host population cycles using reasonable parameter values.

Fig. 20. Nomograms of density-dependent model for various degree of severity of density-dependent constraints. (a) c=0.00 (b) c=0.03 (c) c=0.06. ($a=4.7$, $\Lambda=2\times10^{8}$, $\beta=1\times10^{-9}$)


a)
MINIMUM TOTAL HOST POPULATION
MAXIMUM TOTAL HOST POPULATION
MAXIMUM PREVALENCE
slope of b: 0.0
b)
slope of b: 0.03
c)
slope of b: 0.06
u, virus natural death rate (/year)
b, minimum host natural death rate (/year)

Incubation period model

When an incubation period is added to the basic model the cycles observed with the basic model disappear. Only the maximum total host density is included in the nomograms on Fig. 21 since the minimum and maximum host density are equal and they each represent the equilibrium density. Since the virus is not produced immediately after the infection is contracted, fewer pathogens are present in the environment to infect other hosts and the longer the incubation period (small ν) the higher the equilibrium density. If the incubation period is short (large ν), the virus is more readily available for infection and the equilibrium point is lowered.

Fig.21 shows that the prevalence is relatively independent of changes in (u) and is largely influenced by variations in (b). When the insect natural mortality is high many infected hosts die before the end of the incubation period. The prevalence is therefore lower as (b) increases (Fig.21). Also, since the virus is present in the insect because of the incubation period, its capability to survive in the environment does not influence the prevalence. Increasing (α) does not permit the population to escape the influence of the pathogen as observed with the basic model because the latent period prevents the rapid loss of infected individuals (Fig.22).

Fig. 21. Nomograms for the incubation period model for different incubation coefficient. (a) $\nu=7$ (b) $\nu=10$ (c) $\nu=12$ ($a=4.7$, $\Lambda=2\times10^{8}$, $\beta=1\times10^{-9}$)

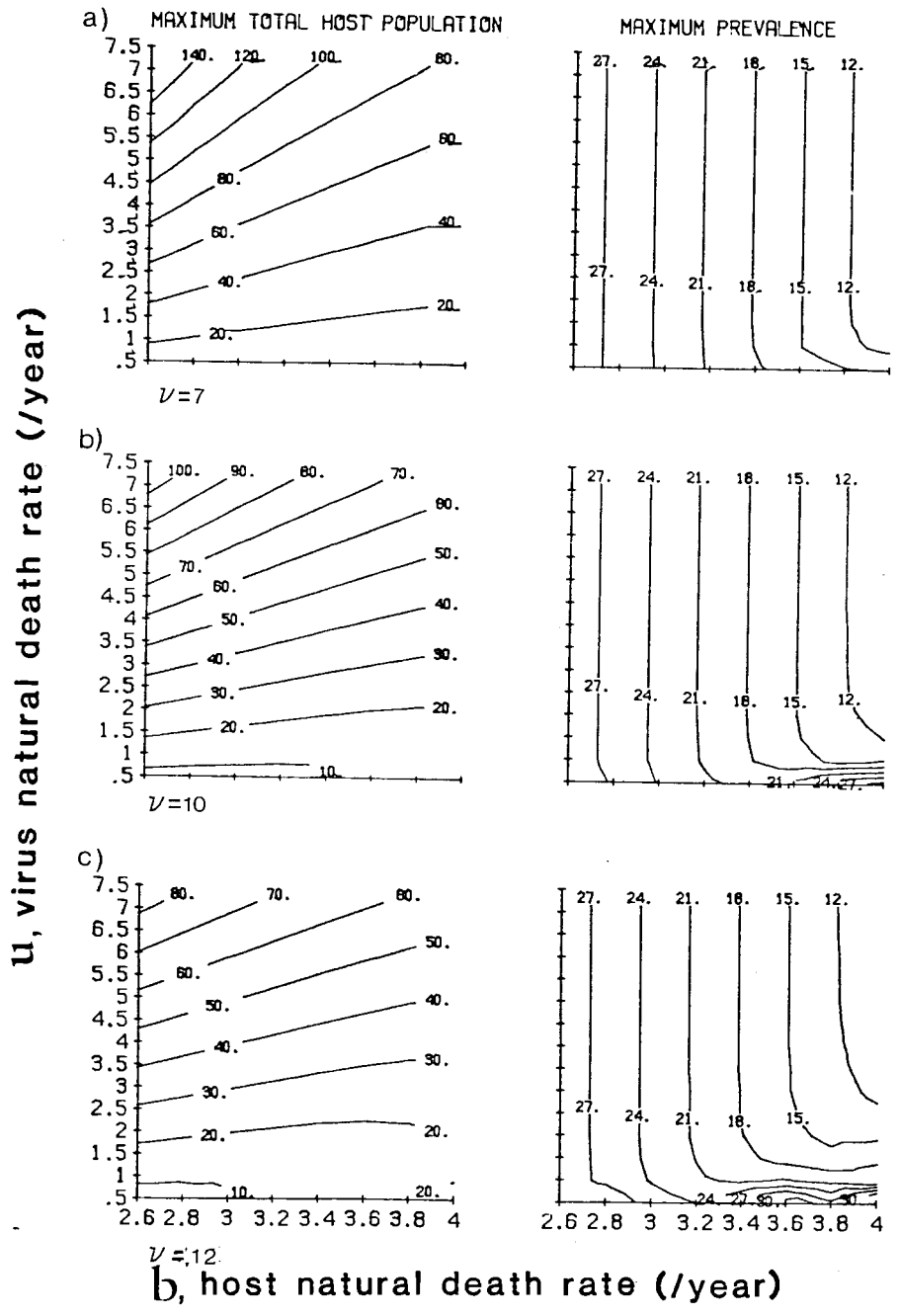
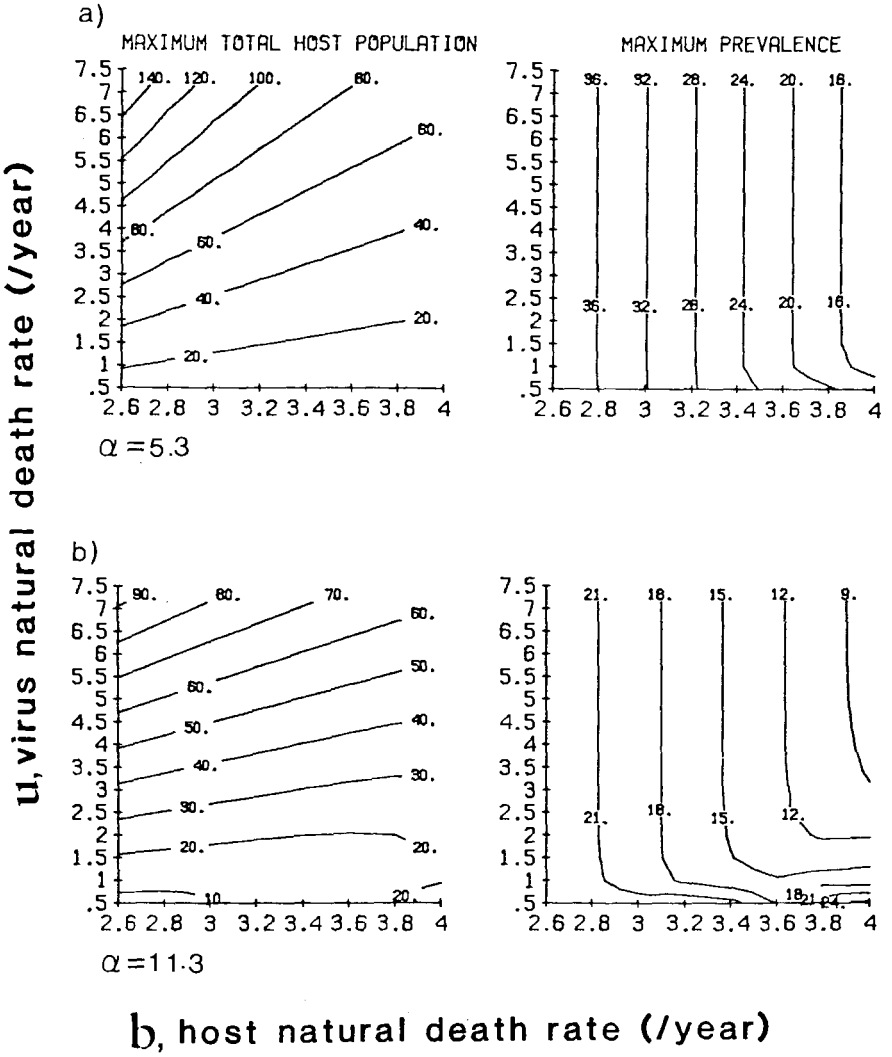

a)
MAXIMUM TOTAL HOST POPULATION
MAXIMUM PREVALENCE
b)
c)
ν=7
ν=10
ν=12
u, virus natural death rate (/year)
b, host natural death rate (/year)

Fig. 22. Nomograms for the incubation period model for two values of disease-induced death rate (α). (a) $\alpha = 5.3$ (b) $\alpha = 11.3$. ($a = 4.7$, $\Lambda = 2 \times 10^{8}$, $\beta = 1 \times 10^{-9}$)


a)
MAXIMUM TOTAL HOST POPULATION
MAXIMUM PREVALENCE
α=5.3
b)
α=11.3
u, virus natural death rate (/year)
b, host natural death rate (/year)

Vertical transmission model

Adding vertical transmission does not modify the results obtained with the basic model. Vertical transmission occurs when a female larva becomes infected late during its life cycle but is still able to reproduce. The virus contaminates the surface of the egg when the female is ovipositing so that the larvae emerging from those eggs contract the disease the next spring (Finn, 1975). Viruses from the environment can also contaminate the eggs. The simulations were done with 2% of the offspring of the infected hosts passing directly to the infected class (Fig. 23). The nomograms for other values of (α) are similar to Fig.15 to 18. Even with a proportion of 30% the results are not drastically different from the basic model (Fig. 24). Prevalence is higher since the infected class has more recruits at birth. The vertical model behaves similarly to the basic model except that the region of the desirable period is slightly enlarged but not enough to encompass a reasonable combination of u and r even when the proportion of offfspring passing directly to the infected class is increased to 30%.

Combined model

In the combined model all the processes previously mentioned and which are known to occur in the field population of the tussock moth are included and the result of the

Fig. 23. Nomograms for the vertical transmission model. ($a=4.7$, $\Lambda=2\times10^{8}$, $\beta=1\times10^{-9}$, $\alpha=7.3$, prop=0.02)

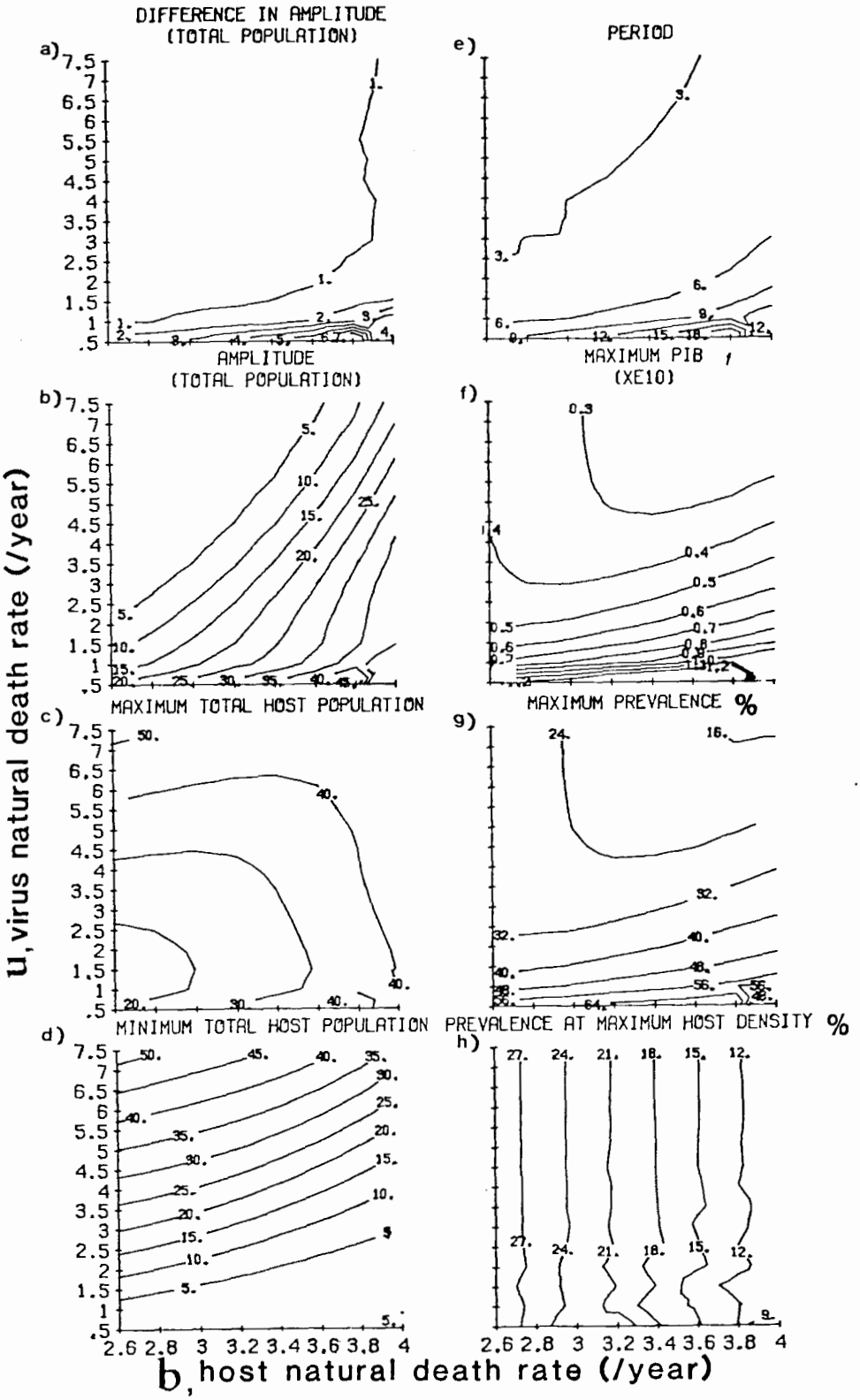
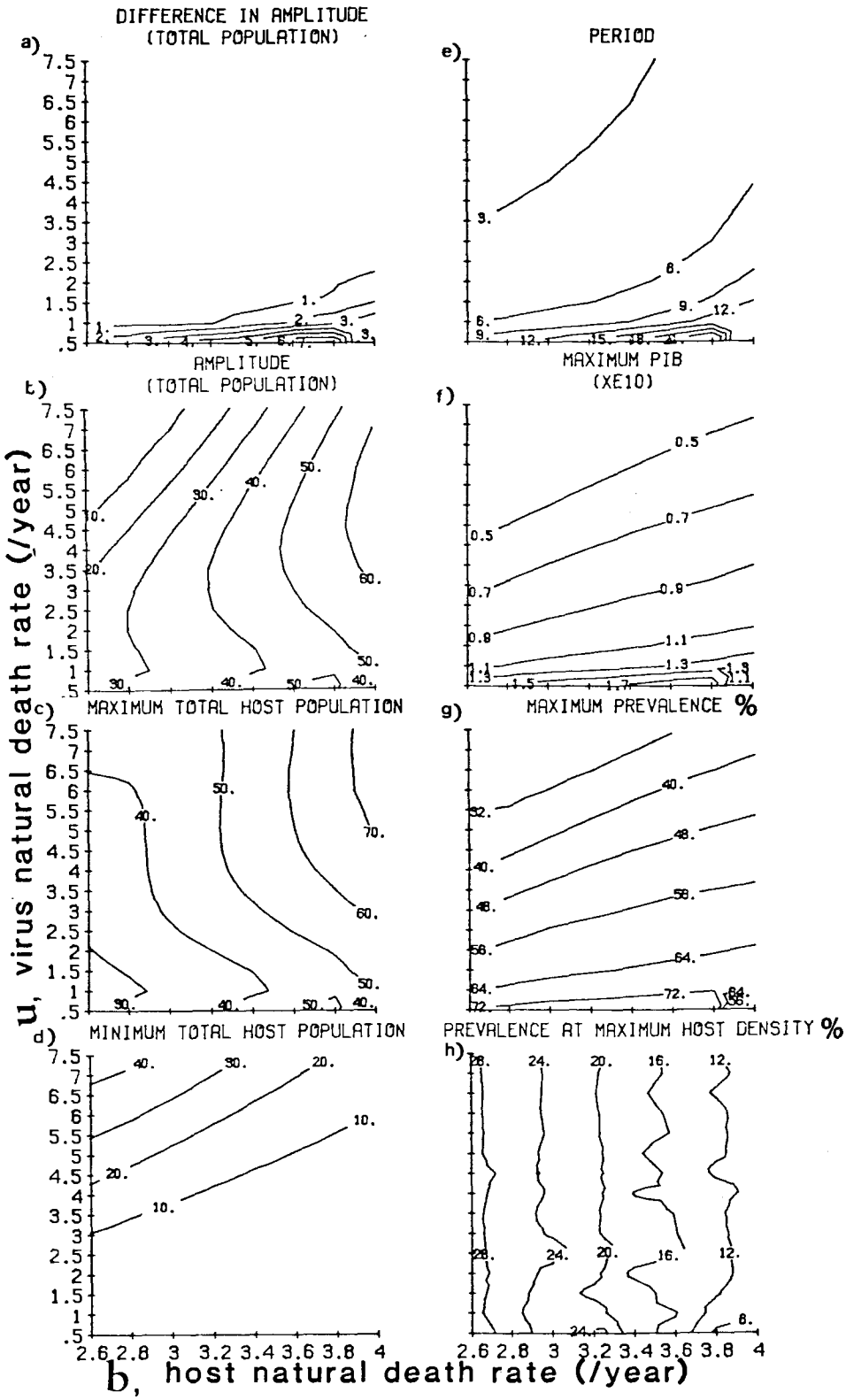

DIFFERENCE IN AMPLITUDE
(TOTAL POPULATION)
a)
AMPLITUDE
(TOTAL POPULATION)
b)
MAXIMUM TOTAL HOST POPULATION
c)
MINIMUM TOTAL HOST POPULATION
d)
PERIOD
e)
MAXIMUM PIB
(XE10)
f)
MAXIMUM PREVALENCE %
g)
PREVALENCE AT MAXIMUM HOST DENSITY %
h)
u, virus natural death rate (/year)
b, host natural death rate (/year)

Fig. 24. Nomograms for the vertical transmission model. ($a=4.7$, $\Lambda=2\times10^{8}$, $\beta=1\times10^{-9}$, $\alpha=7.3$, prop=0.30)


DIFFERENCE IN AMPLITUDE
(TOTAL POPULATION)
a)
PERIOD
e)
AMPLITUDE
(TOTAL POPULATION)
b)
MAXIMUM PIB
(XE10)
f)
MAXIMUM TOTAL HOST POPULATION
c)
MAXIMUM PREVALENCE %
g)
MINIMUM TOTAL HOST POPULATION
d)
PREVALENCE AT MAXIMUM HOST DENSITY %
h)
u, virus natural death rate (/year)
b, host natural death rate (/year)

simulations is a stable equilibrium point for various combinations of the parameter values (Fig. 25). The dampening effects of density-dependent mortality and latency are largely responsible for the loss of periodicity when compared with the basic model and hence the combined model is not an adequate representation of the dynamics of the tussock moth in the field since no stable cycles are generated.

Stochastic effects

The oscillations are not as regular when random variation is introduced (Fig. 26a,b). For this reason the previous algorithm which measured the amplitude and the period is no longer suitable. An alternative approach is time series analysis which calculates the period of host population cycles using the Box-Jenkins method of analysis (Dixom, 1981). A function C_K is calculated at lag K

$$C_k = \frac{1}{n} \sum_{t=1}^{n-k} (N_t - \bar{N})(N_{t+k} - \bar{N}) \qquad k=0,1,2...$$

where K is expressed in years and N is the total insect population. Each value of C_K is compared with the result when the lag is set to zero (C_0) and the ratio ($r_K = C_K / C_0$) is the sample autocorrelation function (SACF). The period of the insect population cycles is the value of K for which r is maximum (Fig. 26c).

Fig. 25. Nomograms for the combined model for different values of incubation coefficient (ν) and of severity of density-dependent constraints (c) ($a=4.7$, $\Lambda=2\times10^{8}$, $\beta=1\times10^{-9}$, $\alpha=7.3$)

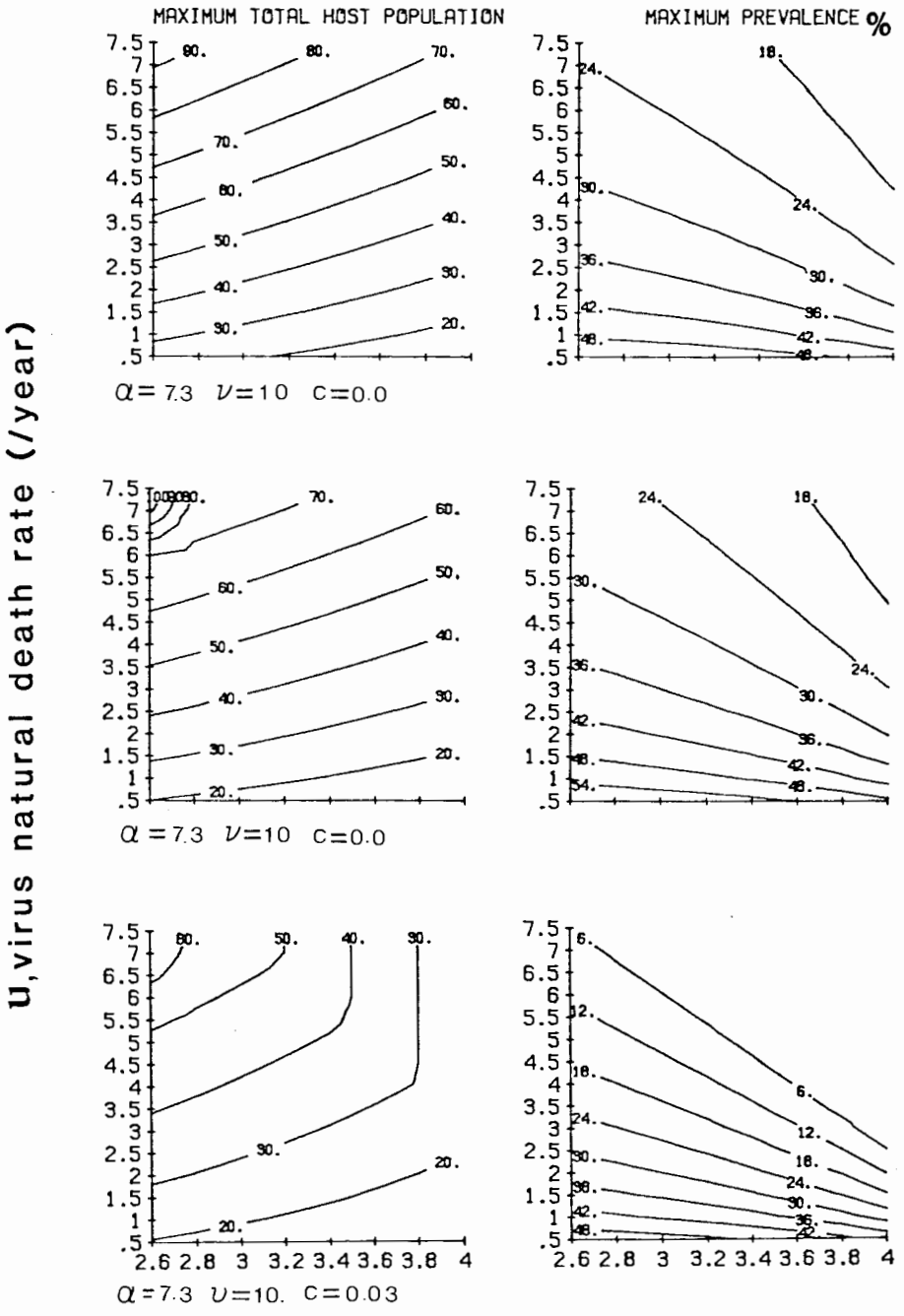
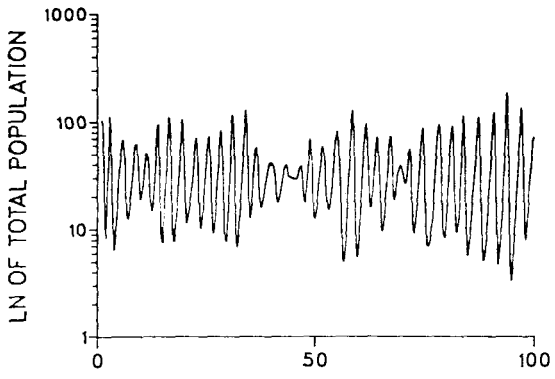
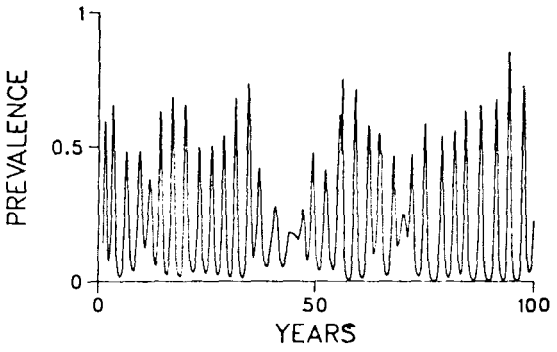
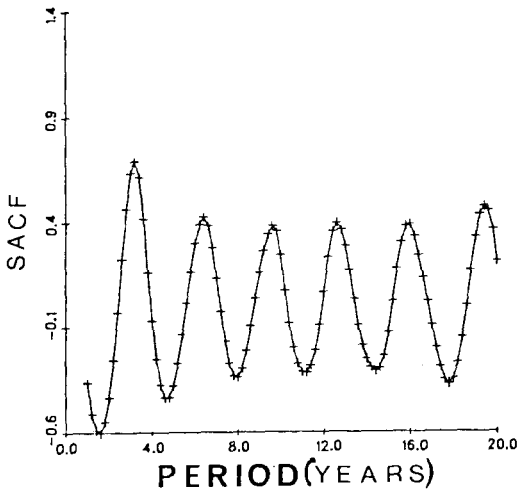

MAXIMUM TOTAL HOST POPULATION
MAXIMUM PREVALENCE %
α= 7.3 ν=10 c=0.0
α= 7.3 ν=10 c=0.0
α= 7.3 ν=10. c=0.03
u, virus natural death rate (/year)
b, minimum host natural death rate (/year)

Fig. 26. (a) and (b) Sample time series of the stochastic version of the basic model ($u=5.0$, $\Lambda=2\times10^{8}$, $\beta=1\times10^{-9}$, $\alpha=8.9$) (c) Correlogram of the above time series of the insect population.


LN OF TOTAL POPULATION
1000
100
10
1
0
50
100


PREVALENCE
1
0.5
0
0
50
100
YEARS


SACF
1.4
0.9
0.4
-0.1
-0.6
0.0
4.0
8.0
12.0
16.0
20.0
PERIOD (YEARS)

The results in Table 6 show that the distribution of the error term does not substantially modify the period and the introduction of random variation shortens the period observed with the deterministic version of the basic model having the same parameter values. The solution for the deterministic combined model was a stable point equilibrium and in this case stochasticity produced cycles but the strong dampening influences in the model confine the period to approximately 3 years as shown in the example of Fig. 26.

Table 7 summarizes the performance of the indicators for each model analysed.

Table 6. Period of simulations with the stochastic version of the basic and combined models using baseline conditions.

Virus death rate (u)	Stochastic basic model				Stochastic combined model				Deterministic model
	Distribution of r				Distribution of r				
	lognormal		normal		lognormal		normal		
	mean period (years)	range (n=30)	mean period (years)	range (n=30)	mean period (years)	range (n=30)	mean period (years)	range (n=30)	period (years)
1.0	5.0	0.0-10.	5.0	1.2-9.6	3.0	2.0-5.0	3.2	1.6-5.0	7.2
2.0	3.7	1.2-6.4	3.7	1.2-6.2	2.9	1.2-5.0	3.2	1.2-5.0	4.4
3.0	2.9	1.2-5.6	3.1	1.2-5.0	3.4	1.4-5.0	3.1	1.2-5.0	3.5
4.0	2.7	1.2-5.2	2.8	1.2-4.8	3.2	1.4-5.4	3.0	1.2-5.0	3.0
5.0	2.6	1.2-4.6	2.6	1.2-4.0	3.0	1.2-5.2	2.8	1.2-5.0	2.7
6.0	2.7	1.2-4.2	2.6	1.4-4.0	2.9	1.2-5.0	2.8	1.2-5.0	2.5
7.0	2.4	1.2-4.0	2.2	1.2-4.0	2.8	1.2-5.0	3.0	1.4-5.0	2.4
8.0	2.2	1.4-3.8	2.1	1.2-3.6	2.8	1.2-5.0	2.9	1.6-4.4	2.3

Table 7. Performance of the indicators for each model.

MODEL	HOST DENS.		OSCILLATIONS		VIRUS	MAXIMUM
	MAX.	MIN.	AMPLITUDE	PERIOD	DENS.	PREVALENCE
Basic model	Y	N	N	N	Y	Y
Density-dependent model	Y	N	N	N	Y	Y
Incubation period model	Y	N	N	N	Y	N
Vertical transmission model	Y	N	N	N	Y	Y
Combined model	Y	N	N	N	Y	Y
Stochastic model				N		

Y: CRITERION MET N: CRITERION NOT MET

X. Discussion

When addressing the topic of population fluctuations in insects, 2 questions must be considered. First, what are the agents responsible for the release of an outbreak and second, are they different from the agents causing the decline of the population? In a cyclic population the agent(s) that determine the periodicity of the cycles must also be identified. In Anderson and May's (1981) model G, one factor, a directly transmitted disease, is responsible for the periodic fluctuations in the density of its host. The sensitivity analysis done on this basic model, using parameter estimates derived from the Douglas-fir tussock moth/virus association, does not support this hypothesis on the role of the virus. Of all the versions of the model tested this version did come the closest to generating behavior similar to that observed in the field for the tussock moth. The results suggest that the virus-host interaction is not the main driving force in the tussock moth cycle. However, before discarding the hypothesis that the observed population cycles are a result of virus dynamics we must examine closely the assumptions and parameter estimates for biases which might affect the results. The parameters are discussed in the context of the basic model but the arguments raised are valid for the different versions of the model.

In the basic model the virus is the only factor constraining the growth of the host population and the insect mortality caused by the virus is encompassed in the parameter α. The linear regression method outlined in the "Estimation of Parameters" section is not significantly biased if the mortality is independent of age and if all individuals are infected at time zero. The logarithm of larval density plotted over time is a linear relationship which indicates that mortality is relatively constant for all ages of the larvae. Second instar larvae need to ingest between 10-20 inclusion bodies before becoming infected (Hughes, 1978). Older larvae, because of their larger size, need a higher dosage of virus to initiate infection which would generate age-dependent mortality if sublethal infections were common in the field. However, given the large number of viral particles liberated when an infected host dies, a susceptible larva probably ingests enough viruses to initiate an infection regardless of the size of the larva.

In populations sprayed with the virus the probability of ingesting the pathogens is even higher. In addition, those experimental populations are sprayed early in the life cycle in order to prevent defoliation. So the populations that come closest to meeting the aboved-mentioned requirements regarding the timing of mortality and infection, are sprayed populations. The value for the disease-induced death rate estimated from those cases is in the same order of magnitude as the results from the Modoc National forest in California, which were used to

estimate (α). This method is therefore not noticably biased and the range of values of (α) is representative of the field situation.

During the epizootics in California and Arizona (Mason and Thompson, 1971; Mason, 1974) the viral disease contributed to the decline of the population, but even in the absence of the virus the populations would eventually have decreased as food became scarce. For example, during the Blue Mountains outbreak in Oregon, where the virus appeared late in the life cycle, starvation limited the number of insects (Mason, 1976). None of the versions of the model discussed in this thesis simulate the dynamics of foliage growth and destruction but, it is unlikely that the period of the cycles in the field depends on the regeneration time of the food source. Wickman (1980) found that the growth of white fir reached pre-outbreak level 5 years after a tussock moth infestation while outbreaks occur after 7 to 10 years. More important could be changes in the nutritional quality of the foliage, and future research should examine its possible effects on the release and decline of outbreaks.

In the field the action of a virus on a population is not easily dissociated from other mortality factors that operate simultaneously. The estimated disease-induced death rate is underestimated if infected individuals in the population are killed by a predator or a parasitoid before the end of the incubation period of the disease. On the other hand (α) is overestimated when stresses, such as starvation and temperature,

favor viral infections (Steinhaus, 1958). Other pathogens, including bacteria, protozoa and fungi, may either have a synergistic effect or delay mortality of the host (Tanada, 1976). The interplay of these factors can lead to a very complex situation but it is unclear to what extent it affects the estimated value of the pathogenicity of the virus. If (α) is overestimated the only way to obtain the desired period would be to modify either the values of (r) or (u) which is unlikely. A more pathogenic virus would produce cycles with a longer period but not enough for the desired period to fall within the range of observed values for the tussock moth population growth rate (r) and the virus natural death rate (u).

In the basic model many simplifying assumptions are included in the definition of the growth rate (r), as defined by the birth rate (a) minus the natural death rate (b). First, the birth rate is assumed to be unaffected by infection or any other process that would tend to reduce fecundity at high host density. There is no evidence that the virus decreases the reproductive potential of the female However, in Oregon there was a significant drop in fecundity and high mortality due to starvation during the decline phase of the outbreak following extensive defoliation (Mason et al. 1977). The Blue Mountains outbreak is the most severe on record but during a typical virus epizootic moderate defoliation is more the norm and starvation related mortality is not as widespread (Mason, 1974; Mason and Thompson, 1971). There could still be a reduction in fecundity

even if starvation related mortality is low, but is not likely to be important. On the other hand the release of an outbreak could be associated with increased fecundity but the relatively small changes observed cannot be responsible for the very high rate of population increase (Mason, 1981). The analysis of low density populations indicates that larval survival is the most important influence on inter-generation trends (Mason and Torgersen, 1977; Mason and Overton, 1983; Mason et al. 1983).

The basic model incorporates larval survival in the parameter (b), the natural death rate. In the simulations, the natural mortality is not influenced by changes in the efficiency of the predators and parasitoids. It is not known which mortality agent is relaxed in the field in order to permit the population to increase rapidly but parasitism and predation are the most likely candidates. Telenomus californicus, an egg parasite, is more common in areas with no history of outbreaks or where outbreaks have not been frequent or severe than in areas that have had recent severe outbreaks (Mason and Torgersen, 1977). The degree of parasitization varied between 15-60% of the eggs being parasitized in the non-outbreak areas while it was virtually nil in the other instances. Mason and Torgersen (1977) surmise that the parasite, in the absence of alternative hosts, cannot survive periods of extremely low host densities between outbreaks.

Other parasitoids include larval parasitoids which cause a higher percentage of mortality at low host density (Mason and

Torgersen, 1977). The percentage of parasitized larvae was less than 15% during the decline of an outbreak (Mason, 1976) and over 25% in a low density population (Torgersen and Dahlsten, 1978). As for cocoon parasitism it is usually high, frequently over 50% (Torgersen and Dahlsten, 1978;Dahlsten et al. 1970; Dahlsten et al. 1977).

Another component of tussock moth mortality is predation which is more difficult to quantify because the prey is usually removed from the branch but attempts have been conducted to separate losses due to predation and dispersal. Observations on stocked cohorts of tussock moth suggest that losses of young larvae are mostly due to arthropod predation while birds were mostly responsible for predation on the mature larvae (Mason and Torgersen, 1983). Predation accounted for 47.2% of the total loss and 40.5% was attributed to dispersal. More studies on the spatial distribution of parasitoids and predators in relation to the density of the insects and the presence of alternate preys or hosts, are needed to clarify the impact of those natural enemies on tussock moth populations.

The available information indicates that T. californicus may be an important agent in constraining the tussock moth at a low density and its absence could favor population growth if predators and other parasitoids are unable to compensate for the greater number of larvae emerging. The fluctuations in the insect growth rate are probably mediated through changes in its natural mortality rather than in its fecundity and this

possibility is explored in the density dependent version of the model.

In the basic model the value of (r) is greater than 1.0 and as a result the cycles generated are shorter than the desired value since the population has the potential to increase rapidly following a crash. The period of the cycles could be lengthened if the virus natural mortality rate (u) was lower.

The virus death rate (u) is probably one of the most difficult parameters to estimate in the field. The virus is very susceptible to deactivation by sunlight so its survival is largely dependent on the amount of shade provided by the tree but once it has leached to the soil it can survive for many years. It should be taken into consideration that the viral particles present in the soil are not a total loss from the point of view of the virus population since they are still available for future reinfection for up to 40 years mostly through wind transport (Thompson et al. 1981). In addition predators and parasitoids often contribute to the spread of the disease by passing out infective feces or contaminating healthy larvae after picking up pathogens from infected hosts and ovipositing in new hosts (Reardon and Podgwaite, 1976; Entwistle, 1977; Raimo et al., 1977; Lautenschlager and Podgwaite, 1979). The net effect on the quantity of virus present in the environment should be measured in the field at the time of an epizootic. Branches could be collected at intervals and tested in bioassays for the presence of the virus.

One problem with this approach is that the virus is not distributed uniformly over the tree so branches with different initial densities of virus are compared. If the same experiment were done under controlled conditions then the physical setting of the forest, which may or may not enhance the survival of the virus, is not duplicated. More detailed information on the mortality process in the virus population would be an important contribution but the model only requires a value for the mean survival time of the virus. An expected lifespan of 2 to 3 months, the baseline value used in the simulations, is realistic but the sensitivity analysis indicates that it is not long enough to generate cycles of the desired period. The combination of a short-lived virus with a host population with a high reproductive potential produces cycles of a shorter period than the ones observed in the field. According to the basic model, cyclic behavior tends to be produced by highly pathogenic diseases with long lived infective stages reproducing in a slowly growing host population. Incorporating a density dependent host population growth rate does not increase the likelihood of generating cycles of the desired period.

The inclusion of a density-dependent growth rate, singly or in combination with other processes, dampens the oscillations to the point that the cyclic behavior is lost and the tussock moth population reaches a single equilibrium point. Because of the lack of information on the exact shape of the density dependent function only a linear relationship has been assumed but

nonlinear functions would probably result in dampened oscillations also.

The addition of processes documented in the field, i.e. virus incubation period and vertical transmission, also failed to produce cycle periods in the observed range. The results with the vertical transmission model are virtually the same as the basic model but the incubation period of infection version generates a single equilibrium point. Interactions with other trophic levels, i.e. predators and food, probably have more influence on the periodicity of the outbreaks than what is assumed in these modified versions of Anderson and May's (1981) model.

Anderson and May's model falls in the category of general models (Oster, 1981) that are "aimed at understanding general properties of ecosystems" but which are not useful for analysing a particular set of data. Anderson and May (1981) analyse data from the larch budmoth population in Switzerland, but their method of estimating the parameter values is not clearly defined. At one point they generalize that forest insect pests "exhibit relatively low rates of annual population growth...typically around unity" (Anderson and May, 1981). However the intrinsic growth rate (r) is an important component in determining the cyclic behavior of the model. As shown here a small variation in (r) affects the periodicity of the oscillations and it is curious that "their rough estimate" of 1.0/year for the larch budmoth falls in the range required to

generate the period of 10 years observed in the field. In their sweeping generalization about the growth rate of forest insects equalling 1.0, they include the spruce budworm. Referring to the Morris (1963) data they approximate (r) to be 1.0 which is quite different from the value of 1.6 put forward by Ludwig et al.(1978) using the same set of data. This discrepancy points to the subjectivity involved in the process of estimating parameter values. In addition, the limited amount of detailed processes included in the model precludes a quantitative validation of the model. Anderson and May (1980) emphasize the qualitative performance of the model, i.e., the generation of cycles is a step in the right direction. But the generation, by Anderson and May's model, of cycles of the right period for the larch budmoth may just be fortuitous and a result of the way the model was built.

It is justifiable to simplify and compress birth, mortality and virus transmission processes into as few parameters as possible. Such a simplification facilitates analysis of the equations by focusing on one element of the system considered to be important, in this case the virus. The assumptions may describe few, if any, insect populations. But even in a crude form this model is still a valuable aid in formulating hypotheses and designing experiments. But one objection to Anderson and May's model (1981) is that it is expressed in differential equations which usually apply for continuously reproducing organisms. Neither the larch budmoth nor the tussock

moth fall into that category. The pathogens fulfill that requirement during the period when the hosts are available but the viruses are not reproducing for the rest of the year. So we are faced with the problem of transforming parameters that represent discrete increments in time into instantaneous rates. When this type of model is applied to univoltine insects realism is sacrificed for ease of analysis. My attempt to translate the differential equations of the basic model into difference equations generated numerical instability because the virus is continually reproducing during one generation of the insect. One alternative would be to break down the host population into instar or size classes. But a model partitioned into size classes would have to incorporate submodels describing the growth of the foliage and the effect of climatic factors and foliage consumed on the growth and survivorship of the larvae because the production of inclusion bodies increases with the size of the larvae. The result would be a very detailed and complex model.

A certain degree of complexity is necessary if the model is to be used in evaluating management options. The tussock moth model developed by the USDA was constructed for management purposes and contains many state variables (Colbert et al. 1979). It consists of a series of submodels arranged in a hierarchical structure with resolution at the regional, forest, stand, tree and branch level. Defoliation by the tussock moth is incorporated in the branch model. An outbreak is invoked by the

user so that the length of the inter-outbreak period is not the result of interactions between the processes described by the equations but by an arbitrary decision based on past observations of cycle length. The whole exercise is a mere recapitulation of the events that occurred during the Blue Mountains outbreak in 1971-74. Most of the parameters and functions describing growth, mortality, fecundity and feeding are derived from the data collected at the time but they can be modified to some extent to accomodate different situations. This type of model may be reasonably reliable if the features of the next outbreak do not differ considerably from the previous records but correspondence between the predictions and the events is no guarantee that the assumptions are still valid. Other assumptions may lead to the same predictions. This type of model provides little insight into the mechanisms underlying the tussock moth/forest association let alone the periodicity of the outbreaks.

Using a simple logistic model Berryman (1978a) postulates that population cycles in the tussock moth are caused by time-delays in the response of density-dependent processes. Contrary to the USDA model his model is very simple but as he himself acknowledges "the identification of the biological processes giving rise to the time delays remains an unsolved problem". They could be caused by predators, parasitoids, diseases or depletion of the food source.

McNamee et al. (1981) developed an approach which is halfway between simple theoretical models and detailed empirical process models and which uses number of key processes to determine the equilibrium structure of many forest insects, including the tussock moth. The equilibrium structure is derived from a set of recruitment curves which illustrate the rate of population change for a range of defoliator densities under different forest conditions. The intersection of the recruitment curve with the line representing a constant population from generation to generation is the equilibrium point which can either be stable or unstable. Similar recruitment curves are developed for the forest biomass and the parasite/disease complex and the temporal behavior of the system is derived from the combination of these processes. According to this framework, McNamee et al. (1981) conclude that the periodicity of the tussock moth outbreaks is "determined largely by the interaction between...the defoliator and the parasitoid or disease". This conclusion is reached using a minimum of qualitative information regarding the impact of natural enemies and the extent of intraspecific competition so their conclusions are very speculative.

There is reasonable empirical evidence that pathogens may play an important role in the natural control of certain forest insects (Katagari, 1969; Stairs, 1972; Henry, 1981). The data that support this hypothesis come mostly from the introduction of viruses on non-native pests. The best known examples are of

the European spruce sawfly, Gilpinia hercynia, and the European pine sawfly, Neodripion sertifer. In New Brunswick an introduced viral disease stopped the increase in density of the spruce sawfly populations until introduced parasitoids maintained the population at a low endemic level (Bird and Elgee, 1957). In Ontario where parasitoids were absent, recurring epizootics of the virus caused the decline of the pine sawfly (Bird and Burke, 1961). In the first example the disease and the parasitoids were complementary and compensatory while in the second case repeated epizootics limited sawfly increases (Bird and Burk, 1961; Bird and Elgee, 1957; Neilson and Morris, 1963; Stairs, 1972; Burges, 1973). In another example in western Samoa, the introduction of the rhinoceros beetle caused extensive damage to coconut trees. A viral disease (ROV) was introduced from Malaya and it is apparently keeping the beetle population at a stable equilibrium density (Zelazny, 1973).

In populations of gypsy moth and tent caterpillar a viral disease is commonly found naturally when densities are high and is held responsible for the decline of the insects (Clark and Thompson, 1954; Clark, 1955,1958; Campbell, 1963; Doane, 1970). The epizootics follow a similar pattern of a low initial source of contaminants that spread among the population and reach a high incidence of infection at the late instars. Whether such cyclic patterns will persist or eventually dampen out is unknown.

Taking into account the genetic configuration of the parasite and host populations, the parasitic association will either tend towards homeostasis (Pimentel, 1968) or cyclic oscillations (Person, 1966). Diseases select for resistant individuals but because the pathogen has a short generation time it can respond quickly to the appearance of resistance in the host population and evolve greater pathogenecity. However, the evolution of greater virulence in order to overcome the increased resistance of the host may not be desirable for the parasite since too virulent a pathogen could cause the extinction of the host as well as of its own population. There should exist an upper limit to the degree of pathogenecity that will not endanger the parasite itself and theoretically a stable association may follow as less virulent pathogens are selected for (Levin and Pimentel, 1981; Anderson and May, 1982; Bremermann and Pickering, 1983). If less virulent strains of the myxomatosis virus had not appeared, the rabbits in Australia would have been eliminated, but once those strains were present they were selected for and the association has stabilized (Fenner and Myers 1978).

Another possibility is that an increase in virulence would leave the host at a selective disadvantage and as the old mode of resistance in the presence of a highly virulent disease becomes useless, selection for virulence is decreased and a cyclic oscillation may become established (Person, 1966). In cyclic populations, such as the tussock moth where the virus is

undetectable between outbreaks, the selection for resistance in the host is great during the epizootic phase but once the infection has subsided and the population is at a low density, the selection is relaxed and there is a return to susceptibility (Martignoni and Schmid, 1961; Briese and Mende, 1981). Increases in the resistance of the tussock moth to the virus following an outbreak have never been studied but it would be difficult to detect given the short course of an outbreak. On the other hand bioassays done on 3 successive generations of an inbred strain of tussock moth have failed to show significant changes in the virulence of the virus as measured by the LD50 (Martignoni and Iwai, 1978).

It would probably be possible to select for a more virulent strain of virus artificially, with the aim of using it as a biological control agent. But caution should be exercised as the outcome of spraying a highly pathogenic virus is not known. According to the basic model increasing the disease-induced mortality rate increases the maximum host density because the virus kills its host before adequate transmission can occur and before the larvae can produce a high number of viral particles. Small larvae produce fewer inclusion bodies and even though it is preferable to spray early in the life cycle in order to limit defoliation of the trees, the quantity of viral particles available for future reinfection may be decreased. Instead of being a self-sustained system as is observed in California, Arizona and British Columbia, it may become necessary to

reintroduce the virus continuously or run the risk of precipitating a severe outbreak such as the Blue Mountains outbreak in Oregon.

The viral disease is only one of the possible explanations regarding the periodicity of the tussock moth outbreaks. Anderson and May's approach does not explain the periodicity of the outbreaks but the role of the virus should be given careful consideration because of its potential as a biological control agent and because of the evolutionary consequences of the association between the virus and its host. But the role of the virus cannot be isolated from the other levels of interaction of the host with its natural enemies and its food source. It appears that the tussock moth may be kept at low densities through the action of predators and parasitoids but once the insects increase beyond a certain threshold density, a viral disease or food depletion causes the population to decline. The viral disease will cause the population to decrease before heavy defoliation which protects the trees. However long term management practices should be based on the knowledge of the mechanisms that favor the release of the insect population to high densities, so that those mechanisms can be elimated.

Although Anderson's and May's model does not provide a satisfying answer to the question whether the virus causes the periodic outbreaks of the tussock moth, it raises the possibility that the virus might play an important role. Comparisons between populations with virus and without virus

would be most informative.

BIBLIOGRAPHY

Anderson R.M. and May R.M., 1980, Infectious diseases and population cycles of forest insects. Science vol. 210:658-661.

Anderson R.M. and May R.M., 1981. The population dynamics of microparasites and their invertebrate hosts. Philos. Trans. Royal Soc. London Series B. 291(1054):451-524.

Anderson R.M. and May R.M., 1982. Coevolution of hosts and parasites. Parasitology. 85:411-426.

Balch, R.C., 1932. THe fir tussock moth (Hemerocampa pseudotsugata Mc.D.) J. Econ. Entomol. 25(6):1143-1148.

Baltensweiler W., 1964, Zeiraphera griseana Hubner in the european Alps. A contribution to the problem of cycles. Can. ent. 96:792-800.

Beckwith R.C.,1976. Influence of host foliage on the Douglas-fir tussock moth. Envir. Entomol. 5(1):73-77.

Beckwith R.C., 1978. Biology of the insect:hosts. In the Douglas-fir tussock moth: A Synthesis, p. 25, M.H. Brookes et al. ed. USDA Tech. Bull. 1585. Wash. D.C.

Beddington J.R., Free C.A., Lawton J.H., 1975. Dynamic complexity in predator-prey models framed in difference equations. Nature 255:58-60.

Berryman A.A., 1978a. Population cycles of the DFTM: the time-delay hypothesis. Can. Ent. 110:513-518.

Berryman A.A., 1978b. Towards a theory of insect epidemiology. Res. Popul. Ecol/ 19:181-196.

Bird F.T. and Elgee D.E., 1957. A virus disease and introduced parasites as factors controlling the European spruce sawfly, Dyprion hercynia, in central New Brunswick. Can. Ent. 89;371382.

Bird F.T. and Burk J.M., 1961. Artificially disseminated virus as a factor controlling the European spruce sawfly, Dyprion hercynia, in the absence of introduced parasites. Can. Ent. 93:228-237.

Bremerman H.J. and Pickering J., 1983. A game-theoretical model of parasite virulence. J. Theor. Biol. 100:411-426.

Briese D.T. and Mende H.A., 1981. Differences in susceptibility

to a granulosis virus between field populations of the potato moth, Phloremea operculella. Bull. Entom. Res. 71:11-19.

Burges W. D.,1973. Enzootic diseases of insects. N.Y. Acad. Sci. 217:32-49.

Campbell R.W., 1963. The role of disease and dessication in the population dynamics of the Gypsy moth Porthetria dispar. Can. Entom. 95:426-434.

Clark E.C., 1955. Observations on the ecology of a polyhedrosis of the Great Bassin Tent Caterpillar, Malacosoma fragilis. Ecology 36:373-376.

Clark E.C., 1958. Ecology of the polyhedrosis of tent caterpillars. Ecology 39:132-139.

Clark E.C. and Thompson C.G., 1954. The possible use of microorganisms in the control of the Great Basin Tent Caterpillar. J. Econ. Entom. 47:140-149.

Colbert J.J., Overton W.S., White C., 1979. Documentation of the Douglas-fir tussock moth outbreak population model. Pac. Northwest For. & Range Exp. Station. USDA. Gen. Tech. Rep. PNW-89.

Dahlsten D.L. Luck R.F. Schlinger E.I. Wenz J.M. and Copper W.A., 1977, Parasitoids and predators of the Douglas-fir tussock moth in low to moderate populations in Central California. Can. Ent. 109:727-746.

De Bach P. and Smith H.S., 1941. Are population oscillations inherent in the host-parasite relation? Ecology 22:263-369.

Dempster J.P. and Pollard E., 1981. Fluctuations in resource availability and insect populations. Oec. 50:412-416.

Dixom W.J. (ed), 1981. BMDP Statistical software. Univ. of Calif. Press. Berkeley. 726p.

Doane D., 1970. Primary pathogens and their role in the development of an epizootic in the gypsy moth. J. Inv. Pathol. 15:21-33.

Entwistle P.F., 1977. Epizootiology of a NPV in European Spruce Sawfly,Gilpinia hercynia. The status of birds as dispersal agents of the virus during the larval season. J. Inv. Pathol. 29:354-360.

Fenner F. and Myers K., 1978. Myxoma virus and myxomatosis in retrospect: the first quarter century of a new disease. in Viruses and environment. Kurskake E. and Maramorosch K. ed.

Acad. Press N.Y. p539-571.

Finn P.E.M., 1975. Vectors and vertical transmission: an epidemiologic perspective. Ann. N.Y. Acad. Sci. 266:173-194.

Greenbank D.O.,1956. The role of climate and dispersal in the initiation of outbreaks of the spruce budworm in New Brunswick. Can. J. Zool. 34:453-476.

Greenbank D.O., 1963. Climate and the spruce budworm. In Morris, R.F. (ed.): The dynamics of epidemic spruce budworm populations. Mem. Ent. Soc. Can. 31:174-180.

Hassel M.P.,1978. The dynamics of arthropod predator-prey systems. Princeton University Press. 237pp.

Henry J.E., 1981. Natural and applied control of insects by protozoa. Ann. Rev. Entomol. 26:49-73.

Hughes K.M., 1976. Notes on the nuclear polyhedrosis viruses of tussock moths of the genus Orgyia. Can. Ent. 108:479-484.

IMSL, 1982. IMSL Library Vol. 1 Houston, Texas.

Ives W.G.H., 1973. Heat units and outbreaks of the forest tent caterpillar,Malacosoma disstria, (Lepidoptera:Lasiocampidae) Can. Ent. 105:529-540.

Jaques R.P., 1975. Persistence, accumulation and denaturation of NPV and GV. In Baculoviruses for insect pest control: safety considerations, Summers et al. ed. Am. Soc. Micro. 90-100.

Katagari K., 1969. Review on microbial control of insect pests in forests in Japan. Entomophaga 14:203-214.

Krebs C.J., 1978. Ecology: The experimental analysis of distribution and abundance. Harper & Row 677pp.

Lautenschlager R.A. and Podgwaite J.D., 1979. Passage of NPV by avian and mammalian predators of the Gypsy moth, Lymantria dispar. Env. Entom. 8:210-214.

Levin S. and Pimentel D., 1981. Selection of intermediate rates of increase in Parasite-Host systems. Am. Nat. 117:308-315.

Livingston R.L. and Daterman, 1977. Surveying for Douglas-fir tussock moth with pheromone. Entomol. Soc. Amer. Bull.. 23(3):172174.

Ludwig D. Jones D.D. Holing C.S., 1978. Qualitative analysis of insect outbreak systems: the spruce budworm and forest. J. Anim. Ecol. 47(1):315-332.

Martignoni M.E., 1978. Production, activity and safety. In The Douglas-fir Tussock Moth: A Synthesis. p.140-147, Brookes M.H. Stark R.W. Campbell R.W. eds. USDA Tech. Bull. 1585. Wash D.C.

Martignoni M.E. and Iwai P.J., 1978. Activity standardisation of technical preparations of Douglas-fir tussock moth Baculovirus. J. Envir. Entom. 71:473-476.

Martignoni M.E. and Schmid P., 1961. Studies on the resistance to virus infections in natural populations of Lepidoptera. J. Ins. Pathol. 3:62-74.

Mason R.R., 1974. Population change in an outbreak of Douglas-fir tussock moth in Central Arizona. Can. Ent. 106:1171-1174.

Mason R.R., 1976. Life tables for a declining population of the Douglas-fir tussock moth in Northeastern Oregon. Ann. Ent. Soc. Amer. 69(5):948-958.

Mason R.R., 1977. Advances in understanding population dynamics of the Douglas-fir tussock moth. Bull. Ent. Soc. Amer. 23(3):168-171.

Mason R.R., 1978. Synchronous patterns in an outbreak of the Douglas-fir tussock moth. Env. Ent. 7(5):672-675.

Mason R.R., 1981a. Numerical analysis of the causes of population collapse of the Douglas-fir tussock moth. Ann. Ent. Soc. Amer. 74(1):51-57.

Mason R.R., 1981b. Host foliage in the susceptibility of forest sites in Central California to outbreaks of the DFTM. Can. Ent. 113:325-332.

Mason R.R., and Baxter, 1970. Food preference in a natural population of the Douglas-fir tussock moth. J. Econ. Ent. 63(4):1257-1259.

Mason R.R., and Luck R.F., 1978. Population growth and regulation. In the Douglas-fir tussock moth: A Synthesis. Brookes et al. ed. USDA Tech. Bull. 1585. Wash. D.C.

Mason R.R., and Overton W.S., 1983. Predicting size and change in nonoutbreak populations of the Douglas-fir tussock moth (Lepidoptera:Lymantriidae). Envir. Entom. 12:799-803.

Mason R.R., and Thompson C.G. 1971. Collapse of an outbreak population of the Douglas-fir tussock moth, Hemerocampa pseudotsugata, (Lepidoptera:Lymantriidae). USDA For. Serv. Res. Note PNW-139. 10p.

Mason R.R., and Torgersen T.R., 1977. Dynamics of low density populations of the Douglas-fir tussock moth with special emphasis on natural ennemies. USDA DFTM Res. & Dev. program. Final Report 62p.

Mason R.R., and Torgersen T.R., 1983. Mortality of larvae in stocked cohorts of the Douglas-fir tussock moth Orgyia pseudotsugata (Lepidoptera: Lymantriidae). Can. Entom. 115: 1119-1127.

Mason R.R., Beckwith R.C. and Gene Paul H., 1977. Fecundity reduction during collapse of a Douglas-fir tussock moth outbreak in Northeastern Oregon. Env. Ent. 6(5):623-626.

Mason R.R., Torgersen T.R., Wickman B.E., Paul H.G., 1983. Natural regulation of a Douglas-fir tussock moth (Lepidoptera:Lymantriidae) population in the Sierra Nevada. Envir. Entomol. 12:587-594.

May R.M., 1973. Stability and complexity in model ecosystems. Princeton Univ. Press. N.J.

May R.M.,1976. Models for two interacting populations. In Theoretical Ecology: principles and applications, May R.M. ed. Saunders Co. 317pp.

McNamee P.J., 1979. A process model for eastern black-headed budworm. Can. Ent. 111:55-66.

McNamee P.J. and McLeod J.M. and Holling C.S., 1981. The structure and behavior of defoliating insect/forest systems. Res. Popul. Ecol. 23:280-298.

Mitchell R.G., 1979. Dispersal of early instars of the DFTM. Ann. Entom. Soc. Amer. 72(2):291-297.

Morris O.N., 1963. The natural and artificial control the Douglas-fir tussock moth by a NPV. J. of Ins. Path. 5:401-414.

Myers J.H., 1981. Interactions between western tent caterpillars and wild rose: a test of some general plant herbivore hypotheses. J. Anim. Ecol. 50:11-25.

Nelson M.M. and Morris R.F., 1964. The regulation of European spruce sawfly numbers in the Maritime provinces of Canada from 1937 to 1963. Can. Entom. 96:773-784.

Oster G., 1981. Predicting populations. Amer. Zool. 21:831-844.

Person C., 1966. Genetic polymorphism in parasitic systems. Nature 212:266-267.

Peterman R.M., 1981. Form of random variation in salmon smolt-to-adult variations and its influence on production estimates. Can. J. Fish. Aquat. Sci. 38:1113-1119.

Pimentel D., 1968. Population regulation and genetic feedback. Science. 159:1432-1437.

Price P.W., Bouton C.E., Gross P., McPherson B.A., Thompson J.N. and Weiss A.E., 1980. Interactions among three throphic levels: influence of plants on interactions between insect herbivores and their natural ennemies. Ann. Rev. Ecol. Syst. 11-41-65.

Raimo B., Reardon R.C., and Podgwaite J.D. 1977. Vectoring Gypsy moth NPV by Apanteles melanosalus. Entomophaga. 22:207-215.

Readshaw J.L.,1965. A theory of phasmatid outbreak release. Austr. J. Zool. 13:475-490.

Reardon R.C. and Podgwaite J.D., 1976. Disease-parasitoid relationships in natural populations of Lymantria dispar in Northeastern United States. Entomophaga. 21:333-341.

Schultz J.C. and Baldwin I.T., 1982. Oak leaf quality declines in response to defoliation by Gypsy moth larvae. Science 217:149-151.

Scribner J. and Feeney P., 1979. Growth of herbivorous caterpillars in relation to feeding specialisation and to growth form of their food plants. Ecology 60:829-850.

Silver G.T., 1960. The relation of weather to population trends of the Black-headed budworm, Acleris variana(Fern.) (Lepidoptera:tortricidae). Can. Entom. 92:401-410.

Sokal R.R. and Rohlf F.J.,1969. Biometry. W.H. Freeman Co. San Francisco. 776pp.

Stairs G.R., 1972. Pathogenic microorganisms in the regulation of forest insect populations. Ann. Rev. Ent. 355-372.

Steinhaus E.A. 1958. Crowding as a possible stress factor in insect disease. Ecology 39:503-514.

Stelzer M.J. Neisses J. Thompson C.G., 1975. Aerial applications of a Nucleopolyhedrosis virus and Bacillus thuringiensis against the Douglas Fir Tussock Moth. J. Econ. Entom. 68(2):269272.

Stelzer M. Neisses J. Cunningham J.C. McPhee J.R., 1977. Field evaluation of Baculovirus stocks against Douglas-fir tussock moth in British Columbia. J. Econ. Ent. 70(2):243-246.

Stoszek K.J. Mika P.G. Moore J.A. Osborne H.L., 1981. Relationship of Douglas-fir tussock moth defoliation to stand and site characteristics in Northern Idaho. Forest Sci. 27(3):431-442.

Sudgen B.A., 1957. A brief history of outbreaks of Douglas-fir tussock moth in British Columbia. Entom. Soc. B.C. Proc. 54:37-39.

Thomson H.M., 1958. Some aspects of the epidemiology of a microsporidian, parasite of the spruce budworm. Can. J. Zool. 36:309-316.

Thompson C.G. and Scott D.W., 1979. Production and persistence of the NPV of the Douglas-fir tussock moth in the forest ecosystem. J. Inv. Pathol. 33:57-65.

Thompson G.C. Scott D.W. Wickman B.E., 1981. Long-term persistence of the NPV of the DFTM in forest soil. Environ. Entomol. 10:254-255.

Torgersen T.R. and Dahlsten D.L., 1978. Natural mortality. In: The Douglas-fir tussock moth A synthesis. Brookes M.H. Stark R.W. Campbell R.W. eds. USDA Tech. Bull. 1585 Wash. D.C.

Tunnock S. Livingston R.L. Bousfield W.E., 1974. Impact of egg viability, egg parasitism and virus on 1974 DFTM defoliation potential in Northern Idaho. USDA For. Serv. North. Reg. Insect and Dis. Rep. 74-79, 10 p. Div. State and Prev. For., Missoula, Mont.

Watt W.E., 1968. A computer approach to analysis of data on weather, population fluctuations and disease. In Biometeorology: Proc. 28th annual biology colloquium, 1967, p. 145-159. W.P. Lowry, ed. Oreg. State Univ. Press., Corvallis.

Wellington W.G.,1954. Atmospheric circulation processes and insect ecology. Can. Ent. 86:312-333.

White T.C.R., 1974. A hypothesis to explain outbreaks of looper caterpillars with special reference to populations of Selidosema suavis in a plantation of Pinus radiata in New Zealand. Oecologia 16:279-301.

White T.C.R., 1976. Weather, food and plagues of locusts. Oec. 22:119-134.

Wickman B.E., 1978. Tree injury In: The Douglas-fir tussock moth A synthesis. Brookes M.H. Stark R.W. Campbell R.W. eds. USDA Tech. Bull. 1585 Wash. D.C.

Wickman, B.E. 1980. Increased growth of white fir after a

Douglasfir tussock moth outbreak. J. For. 31-33.

Wickman B.E. Mason R.R. Thompson C.G., 1973. Major outbreaks of the Douglas-fir tussock moth in Oregon and California USDA For. Serv. Gen. Tech. Rep. PNW-5, 18p. Pac. Northwest For. and Range Exp. Stn. Portland Oreg.

Zelazny B., 1972. Studies on Rhabdionvirus oryctes I. Effect on larvae of Oryctes rhinoceros and inactivation of the virus. J. Invert. Pathol. 20:235-241.