Population Dynamics of the Western Tent Caterpillar: The Roles of Fecundity, Disease and Temperature

by

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Abstract

Many populations of forest Lepidoptera exhibit regular periodic cycles in abundance. Explicit mechanisms for such dynamics however, remain a subject of debate in Ecology. I used annual field data (1977-2015) from a cyclical species of forest Lepidoptera native to southwestern B.C., the western tent caterpillar (*Malacosoma californicum pluviale*), to elucidate how fecundity, viral disease and temperature contribute to its dynamics. Using time-series analysis and relationships between lagged population density, disease prevalence and annual population growth rate, I demonstrated that cyclical dynamics can be generated. I then used AIC model selection to show that fecundity and lagged population density had the greatest contributions to annual population rate of increase, followed by disease prevalence and warmer spring temperatures during larval development. Using these factors, I constructed a population model capable of generating population cycles similar to those observed in the field. These results indicate that fecundity, density-dependent disease prevalence and temperature contribute significantly to the cyclical dynamics of these populations.

Keywords: Time-series analysis; fecundity; viral disease; temperatures; AIC model selection; density-dependent.

One of the joys of science is that, on occasion, we see a pattern that reveals the order in what initially seems chaotic. A jumble becomes part of a simple plan, and you feel you are seeing right through something to find its essence.

- Neil Shubin

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Chapter 1.

General Introduction

1.1. Population Cycles

From its infancy to modern day, a central tenet of population ecology has been the understanding of how and why populations change. In particular, population ecologists have often focused their attention toward what particular factors cause populations to change. For instance, how do specific life history traits contribute to population growth? How might finite resource depletion or intraspecific competition lead to population regulation? Can predator-prey or host-parasite linkages explain the population dynamics of particular species? Such questions (and respective factors) remain crucial to our understanding of population dynamics of species. Traditionally, population ecologists have been somewhat divided between two philosophical camps: those who sought answers to such questions through empirical (i.e. experiments, natural observations) means or those who employed more theoretical (i.e. a priori assumptions, first principles modeling) approaches. While arguments can be made for the merit of both philosophies it is important to recognize that the two are not mutually exclusive and share a common goal of identifying what factors contribute to the change of populations. Where this goal has been particular challenging historically is in the case of population cycles.

Perhaps the earliest and most famous example of population cycles comes from the early work of Charles Elton. In 1925, Elton was commissioned by the Hudson's Bay Company to study fluctuations of animal populations that were of interest to the fur trade; namely the Canadian lynx and snowshoe hare. After studying trapping records dating from 1736, Elton found large fluctuations of fur abundances over time. It was apparent to Elton that the regular cyclical fluctuations of lynx furs corresponded to the fluctuations of hare furs. The cycles of both species appeared to be closely linked, perhaps by their predator-prey

relationship. This would be the earliest documented empirical evidence of cyclical dynamics. Population cycles would later be found in a variety of species, ranging from small mammals to forest insects. In fact, the greatest incidence of cyclical population dynamics is in forest Lepidoptera.

1.2. Population Cycles of Forest Lepidoptera

Among populations that regularly cycle, forest insects, particularly Lepidoptera, are some of the most studied (see Myers, 1988; Myers & Cory, 2013). This is in part due to their frequent pest status. In the outbreak phase of a cycle, populations of forest Lepidoptera have potential for widespread defoliation that significantly impacts forest health. During a previous outbreak of spruce budworm *Choristoneura fumiferana* in the 1970's for example, more than 50 million hectares of forest were severely damaged throughout Canada (National Resources Canada, 2016). In 2001, the total area of forest (primarily trembling aspen, oak, ash, maple and white birch) defoliated by widespread outbreak of forest tent caterpillar, *Malacosoma disstria* was approximately 14.3 million hectares (National Resources Canada, 2016). Such widespread impacts to the health of forest stands throughout North America (as well as similar insect outbreaks in Europe) have largely driven research into the population dynamics of cyclic insects.

Recently, Myers & Cory (2013) summarized the requirements for cyclic populations of forest Lepidoptera, but these can apply to other species as well: (1) that fecundity is sufficiently high and/or survival sufficiently good to sustain the population increase, (2) that the initial population decline following peak density be initiated by some density-dependent limiting factor(s), and (3) that the population decline be prolonged through a delayed density-related mechanism or mechanism(s). While requirements of cyclical dynamics are understood the principle factors that generate them are still debated. Some of the most cited hypotheses for population cycles in forest Lepidoptera involve variation in food quality and quantity, parasitoids, predators, maternal effects, and pathogens (Myers & Cory, 2013).

1.3. Variation in Food Quality and Quantity

The interest in variation in food quality originates from the negative relationship between larval density of larch budmoth *Zeiraphera diniana* (LBM) and nutritional quality of their host-plant, subalpine larch (Baltensweiler, 1977). After measuring the length of subalpine larch needles over the course of several cycles, Baltensweiler noted that the highest levels of nutritional quality were associated with the increase phase of the LBM cycle, whereas lowest nutritional quality coincided with population decline. Since the time of this observation, a wealth of literature has documented the effect of plant quality on insect performance, in particular on fecundity (see review by Awmack & Leather, 2002). The concept of plant quality influencing insect performance is intuitive as a hypothesis for explaining the population cycles of forest Lepidoptera. For instance, the reduced nutritional quality of host-foliage following prolonged herbivory by high density populations could limit maternal nutrition and negatively impact egg provisioning (Awmack & Leather, 2002). If this effect is generally expressed over large areas of foliage this could be sufficient to initiate the population decline phase of a cycle. The condition of delayed density-dependence is also easily applied to this concept, provided that the response to intense herbivory is not immediate (delayed) and can be maintained over the length of the decline phase of the cycle.

While delayed herbivore-induced changes in plant quality and resulting negative consequences for insect performance have been documented in a variety of forest Lepidoptera (Kendall et al., 2005, Kaitaniemi et al., 1998, Schultz & Baldwin, 1982), the magnitude of the effect in relation to cyclical population dynamics is questionable as it does not account for a majority of the variation in insect abundance over time (Haukioja, 2005; Turchin et al., 2003).

1.4. Parasitoids

Parasitoids are common amongst most populations of forest Lepidoptera and have been shown to demonstrate delayed density-dependence, reaching peak densities during the decline phase of their host population (Berryman, 2002). As delayed density-dependence is a requirement of population cycles and parasitoids are ubiquitous across populations of

forest Lepidoptera, the role of parasitoids in contributing to cyclical population dynamics is likely. Using enclosure experiments, Klemola et al. (2014) demonstrated that egg and pupal parasitization were key determinants of growth rate of autumnal moth (AM), *Epirrita autumnata* populations and these operated in a delayed density-dependent manner. Similarly, Berryman and Münster-Swendson (1994) demonstrated that by using theoretical host-parasitoid models, 76% and 81% of the variation observed in a 20-year dataset of spruce needleminer (SN), *Epinotia tedella* and parasitoid per-capita rates of change could be explained.

Berryman is perhaps the most renowned proponent of the parasitoid hypothesis and has proposed that a majority of cyclical dynamics can be attributed to the presence of both generalist and specialist parasitoids (Berryman, 1996). Berryman's justification for this proposal is that a majority of parasitoids are capable of killing a consistently high proportion of eggs, larvae and pupae during the decline phase of many cyclical populations. Where data exists – black headed budworm (BHBW), *Acleris variana*, and Gypsy moth (GM), *Lymantria dispar* for example – Berryman claims that much of the variation in population rates of change can be correlated with parasitoid abundance. However, the population growth rate of LBM, is not well-captured (explaining only 28% of the variance) by Berryman's host-parasitoid model (1996). Interestingly though, the results of Turchin et al. (2003) contradict Berryman's, with their host-parasitoid model capturing a majority (90%) of the variance of LBM growth rate. These apparently contradictory results are difficult to interpret. In the case of the western tent caterpillar (WTC), *Malacosoma californicum pluviale*, rates of parasitization do not appear to be strongly correlated with variation in population rate of increase (R^2= 0.014, p-value=0.368), but rather seem to sustain the trough phase of the population cycles after initial decline. Perhaps in this case, parasitoids act to extend the periodicity of the population cycles, but do not specifically generate the cycles directly themselves.

Parasitoids undoubtedly play a role in contributing to the dynamics of many forest Lepidoptera; their close (and often exclusive and highly specialized) association with host populations are easily interpreted in terms of a tightly coupled predator-prey system that could easily yield stable limit cycles (Tanhuanpää et al., 2002). Caution should be made however, when making such a broad generalization as parasitoids alone generating all population cycles in forest Lepidoptera. This may not be the dominant contributing factor

to the population dynamics of all systems. More detailed records of parasitoid diversity, abundance and how these correlate with host population rate of change would greatly contribute to understanding the role of parasitoids in population cycles.

1.5. Predators

The role of predators in the population dynamics of forest Lepidoptera has received far less attention than other hypotheses (Myers & Cory, 2013). This is perhaps because many predators of forest Lepidoptera are generalists (various spiders, ground beetles, small rodents and birds) and do not rely solely on larvae and moths as a primary food source. This being said, it would seem unlikely that the relationship between such predators and their prey would be functionally strong enough to generate population cycles (see Elkinton, et al. 2004). Where predation might play a significant role however, is during periods of low prey density. For instance, in both winter moth (WM), *Operophtera brumata* and GM, it has been suggested that pupal predation could prolong the low density phase of these population cycles, extending their periodicity by one or more years (Heisswolf et al., 2009; Elkinton, 1996). The difficulty with implicating the role of predators in population cycles of forest Lepidoptera is that little, if any, long-term data exist on such an interaction, so any claims are largely speculative.

1.6. Maternal Effects

The maternal effects hypothesis suggests that density in the maternal generation will somehow affect the individual quality of resulting offspring and this in turn will determine population growth (Räsänen & Kruuk, 2007). A common example of this is the effect of density (either direct or indirect) on the quality or quantity of egg provisioning (Myers & Cory, 2013). Using pine looper data from Klomp (1966) for instance, Kendall (2005) found that pupal mass is a declining function of density and directly affects moth fecundity (egg quantity). Pupal mass also affects egg-to-adult survival of the offspring generation, suggesting the presence of maternal effects. Using these data, Kendall (2005) constructed a maternal effects model capable of generating population cycles and found that in comparison to models involving food quality and parasitoid interaction, the maternal

effects model best represented field population data. In GM, the number of eggs laid by female moths was found to be negatively impacted by density, but there did not appear to be differences in the development, survival, pupal size or fecundity of offspring from high versus low density populations (Myers et al., 1998).

As with the food quality and parasitoid hypotheses, the maternal effects hypothesis is attractive as a means to explain population cycles in forest Lepidoptera as it involves both direct and delayed density-dependent components expressed through maternal (or more broadly, parental) environment and individual quality. However, the concept of individual quality, and how such a dynamic variable should be empirically defined has drawn debate (Inchausti & Ginzburg, 2009). The features of individual quality that can be used to describe the maternal environment are numerous; and it is likely that multivariate studies are needed to appropriately identify and describe maternal effects in field populations (Plaistow et al., 2006). Additionally, while there is evidence of maternal effects in some forest Lepidoptera, the findings of many investigations appear unclear (Beckerman et al., 2002). Further studies on the impact of density and food limitation on the quality of offspring are obviously needed. In addition, these studies require a multivariate approach to determine how individual quality should be defined.

1.7. Pathogens

Many pathogens can inflict substantial mortality in insect populations and for this reason they have been implicated in the crash phase of population cycles in forest Lepidoptera. The most common pathogens associated with forest Lepidoptera are baculoviruses (Cory & Myers, 2003). These rod-shaped, double-stranded DNA viruses comprise several genera with species belonging to either genus Granulovirus (GV) or Nucleopolyhedrovirus (NPV). Currently, there are over 55 recognized species of NPVs (International Committee on Taxonomy of Viruses, 2015).

Transmission of NPVs occurs primarily through ingestion of contaminated foliage (horizontal transmission) where viral occlusion bodies (OBs) can occur (refer to Figure 1.1.). OBs consist of many virions surrounded by protein crystals of polyhedrin. Once ingested, the polyhedra are broken down by the alkalinity of the insect midgut and virions

pass through the peritrophic membrane and enter host epithelial cells lining the gut. Upon entering the nucleus of an epithelial cell, viral DNA is released from a virion and begins initial replication. A subset of virus will pass into the cytoplasm and bud from the cellular membrane, allowing the virus to then infect other cell types. The replication of viral DNA and synthesis of polyhedra occur rapidly, and within a short period of time, usually between 7-8 days of initial infection (although this is dependent on the host-virus system and temperature of the host environment), a majority of the host biomass is converted to virus. At this point, the host cadaver normally lyses, allowing OBs to contaminate the surrounding environment, including leaves, tree bark, insect shelters and soil. OBs constitute the persistent transmission stage of NPVs and can remain viable in the environment for extended periods of time, provided they are not exposed to UV light (Fuller, 2012; Jaques, 1967). These persistent OBs have been implicated in the resurgence of NPV infection within and between generations, as patches of contaminated leaves, bark or soil can serve as reservoirs for future infection (Elderd & Reilly, 2013).

As with most pathogens, the host population must exceed a critical threshold value and have sufficient exposure to infectious agents in order to initiate widespread infection. This concept is similar to the kinetics that govern the progression of a chemical reaction and is referred to as the mass-action principle. In these terms, one can think of the total proportion of NPV infected larva at the end of a generation as being the “product” of a “chemical reaction” and the “reactants” can be thought of as the initial population density and viral inoculum present upon egg emergence. The time for this “reaction” to take place, occurs over the entire course of larval development. In order for widespread infection to ensue (maximizing the yield of the chemical reaction) within such a population, the population must exceed a critical threshold and in addition must have sufficient exposure to infectious agents, in this case OBs. However, in the case of a univoltine species, the concentration of OBs present in the environment upon egg emergence is largely determined by the amount of OBs that were produced by, and remained viable from, the previous year’s population. This immediately suggests a density-dependent time lag: a requirement of cyclical dynamics.

A key difficulty in identifying the role of baculovirus infection in the population dynamics of many forest Lepidoptera is that disease prevalence, is not often directly measured in field populations, especially over the course of entire cycles. Most long-term evidence for

disease in populations is anecdotal. Also, since baculoviruses like NPVs liquefy their host, it may be very difficult to observe disease directly in the field as larva that succumb to infection may be rapidly washed away or degraded in the environment. This might lead one to believe that disease prevalence is much lower than it actually is.

The ability of pathogens such as NPVs to regulate host population dynamics was explored extensively by Anderson and May (1979, 1981) using simple epidemiological models of disease transmission. Their findings support the ability of virus to not only initiate population decline of forest Lepidoptera but to produce stable limit cycles, provided that the transmission stage remains viable for extensive periods of time (a lag effect), the virus is highly pathogenic, the production of infective stages is large and the annual population rate of increase is small (Anderson & May, 1981). Given that NPV has been observed in the decline phase of many forest Lepidoptera, particularly the GM, the tussock moth (TM), *Orygia spp.,* the forest tent caterpillar (FTC), *Malacosoma disstria,* and the WTC, combined with the fact that these systems meet the requirements of Anderson and May, it is likely that NPVs contribute significantly to the cyclical dynamics of these populations and perhaps other forest Lepidoptera as well.

1.8. Time-series analysis

Where data are available, detailed statistical analyses have made significant contributions to understanding both the expected frequency and intensity of forest lepidopteran outbreaks, as well as making plausible inferences toward potential mechanisms. One of the most useful phenomenological approaches to interpreting cyclical population dynamics is time-series analysis (Royama, 1992; Ims et al. 2007). Time series analysis attempts to interpret population cycles in terms of a series of past population densities. As previously mentioned, the concept relies on a relationship existing between some order of previous population density and future population growth. In this approach, population data are fit using a log-linear model with lagged population densities. The length of time of this lagged effect is referred to as the order of density-dependence and ultimately determines the number of independent parameters in the model. Direct density-dependence for instance refers to the effect of an immediately preceding population density (N_{t-1}) on the density of a current population (N_t). Direct density-dependent

mechanisms are perhaps more intuitive than those acting through a time delay and may be reasonably captured through a predatory functional response, or through some intrinsic mode of self-regulation (Ims et al. 2007). A delayed density-dependent mechanism however, may be a reduction in host-plant quality (initiated by defoliation in the previous year) that results in low survival of offspring (Berryman, 1996). Another example might be a sustained reduction in offspring quality initiated by high parasite loads in a previous generation (sub-lethal effects), leading to a reduction in either survival and/or fecundity (Cory & Myers, 2009). While time series analysis does not explicitly test these mechanisms, support for or against specific hypotheses can be obtained where data are available.

1.9. The western tent caterpillar

Along the south-western coast and Gulf islands of British Columbia, populations of WTC, fluctuate in density with average periodicity of 7-12 years (Cory & Myers, 2009). Western tent caterpillars are a gregarious, univoltine species native to the western coastal regions of North America from British Columbia to California. Larvae emerge from egg masses attached to branches of deciduous trees in early April and feed on surrounding foliage. Preferred plants of WTC include: red alder (*Alnus rubra*), prickly wild rose (*Rosa nutkana*), black cherry (*Prunus virginiana*), crabapple (*Malus diversifolia*) and Hawthorn (Crataegus spp.). Upon emergence, caterpillar families begin to build elaborate silken tents in the peripheral foliage of their host plant. These tents serve as shelters (internal) and basking points (surface) for family members. Larval development normally occurs over the months of April-May, progressing though a total of five instars. Throughout this period, larvae live gregariously and spend a majority of time basking in available sunlight (larvae can elevate their body temperature above ambient) and feeding on surrounding foliage in their respective family groups. Pupation normally occurs from late May to early June. Adult moths emerge in mid-to-late June, at which point they will mate. Female moths will lay a single mass of approximately 150-300 eggs that will overwinter and hatch in the subsequent year. Moths perish immediately following mating and oviposition.

Beyond exhibiting regular periodic cycling, WTC are ideal study organisms due to their gregarious behaviour and conspicuous tents that allow extensive population monitoring,

even at low density. Annual records of WTC abundance have been collected in British Columbia for 40 years (1975-2015) and the occurrence of nucleopolyhedrovirus (NPV) has been monitored for a portion of those years (Myers & Cory, personal communication). Such extensive and comprehensive time series data not only lends themselves to an extensive time series analysis, but also provides the potential to examine NPV as a potential factor causing cyclical dynamics.

1.10. Thesis aims

This thesis examines the potential role of a nucleopolyhedrovirus, as a major contributing factor in the cyclical population dynamics of the WTC. In chapter 2, I use over 40 years of time-series data collected from five WTC field sites in the Lower Mainland and Gulf Island populations of BC, to investigate the density-dependent structure of each site over time. Using partial autocorrelation, I determine the feedback or lag in the population size data and then use log-linear modeling and wavelet analysis to detect changes in the structure of density-dependence over the length of the time-series. I then use the quantitative relationships between population size, NPV infection prevalence and population rate of increase to construct a discrete host-pathogen model and determine whether it is capable of generating stable limit cycles. Chapter 3 explores how fecundity, population size, NPV infection prevalence, spring temperature and tent size contribute toward the annual population rate of increase for these sites. Using Akaike's Information Criterion (AIC), in conjunction with model averaging, I distinguish between the relative importance of factors and their quantitative effect toward changes in annual population rate of increase. I then construct two modified versions of my previous model by incorporating these top-ranked factors into the model structure. Finally, I compare population data from Mandarte Island with data from model simulations using measures of cross-correlation to determine which model best matches the dynamics of this site. As cyclical population dynamics are a phenomenon associated with other forest Lepidoptera, gaining insight into the relative importance and contributions of factors toward population growth in WTC would allow for comparison with other species to determine whether their dynamics are governed by similar factors. Models built around these factors can be used to make predictions about how populations will fluctuate over time given certain conditions. This is particularly

important for informing pest management decisions, as many outbreaking species of forest Lepidoptera inflict extensive damage to forest stands at high population levels, resulting in considerable ecological and economic loss.

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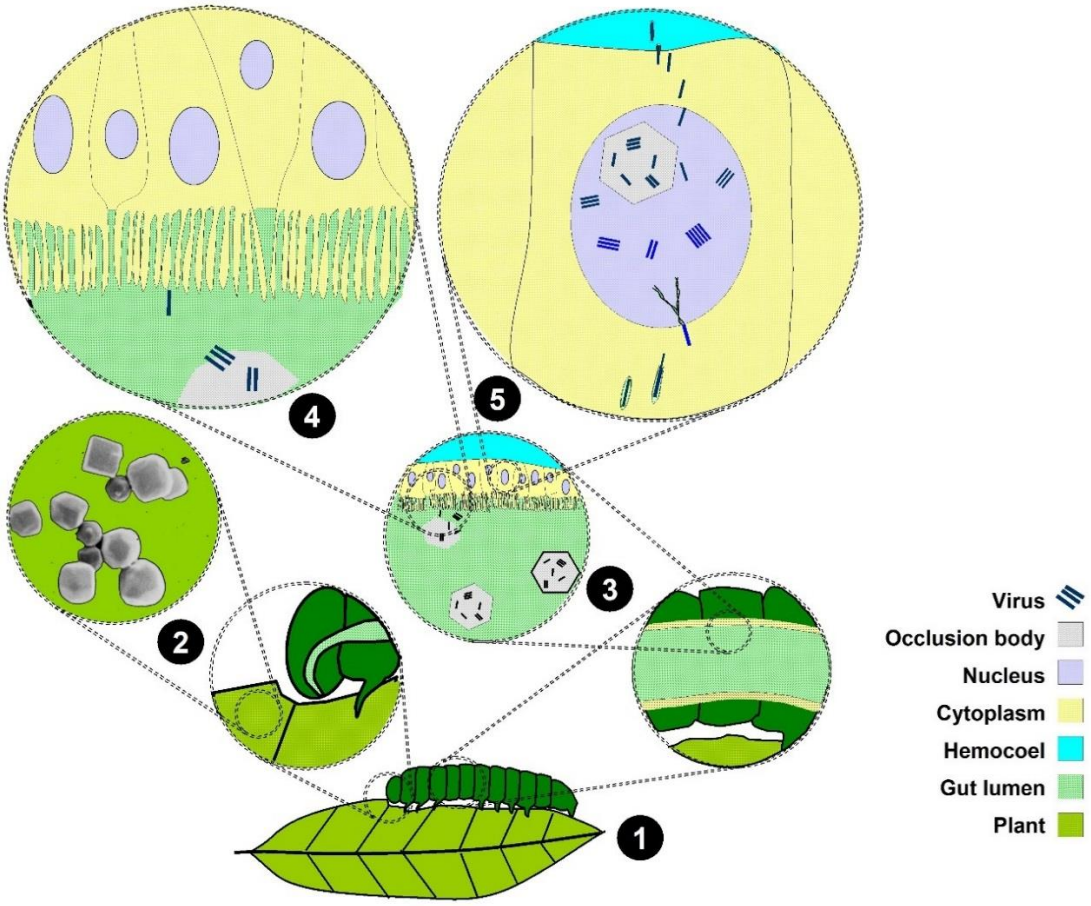

Figure 1.1. Transmission of NPVs occurs primarily through ingestion of contaminated foliage (horizontal transmission) where viral occlusion bodies (OBs) can occur (1). OBs consist of virions embedded in a proteinaceous matrix (polyhedrin) (2). Once ingested, polyhedra are broken down by the alkalinity of the insect midgut (3) and virions pass through the peritrophic membrane, entering host epithelial cells lining the gut (4). Upon entering the nucleus of an epithelial cell, viral DNA is released from a virion and begins initial replication. A subset of virus will pass into the cytoplasm and bud from the cellular membrane, allowing the virus to then infect other cell types. Replication of viral DNA and synthesis of polyhedra occurs rapidly (5) and within days of initial infection, a majority of the host biomass is converted to virus. At this point, the host cadaver normally lyses, allowing OBs to contaminate, the surrounding environment, including leaves, tree bark, insect shelters and soil. *Author Credit: Dwight Lynn (April 23, 2006).*

Chapter 2.

Time-series analysis and the potential role of viral infection in the cyclical population dynamics of the western tent caterpillar

2.1. Introduction

Animal populations fluctuate and the degree to which they do so across time and space depends on complex interactions between species and their environment (Berryman, 2002). For some species these fluctuations are cyclical and populations can fluctuate in synchrony over vast areas. Outbreak densities of cyclical forest Lepidoptera for example attract attention because they can result in widespread defoliation (Myers, 1993). A curious aspect of these populations is that they continue to decline for several generations before increasing once again.

Two approaches have been used to study the population dynamics of cyclical species: the first focuses on density-dependence and the relationship between density and the rate of population increase. The second considers mechanisms and plots the population growth rate against relevant mechanisms affecting populations (Krebs 2002). If the former fails to find a significant relationship it is common to look for a delayed density-dependent mechanism (Turchin 1990).

Delayed density-dependent mechanisms operate through interactions between a population and its food, competitors or natural enemies (Royama, 1992; Berryman, 1996, 2002; Krebs, 2013; Myers & Cory, 2013). The strength of delayed density-dependence can determine both the periodicity and stability of population cycles (Ims et al. 2007). For example, climate-induced changes are believed to have weakened the strength of delayed density-dependence in a majority of keystone herbivore populations in Europe for the past two decades, resulting in an apparent collapse of the cyclic dynamics of these populations (Butet et al. 2013).

Delayed density-dependence has been identified in numerous animal systems, ranging from small mammals (Krebs, 2001, 2013; Ims et al. 2007) to forest insects (Berryman &

Turchin, 2001; Klemola et al. 2014; Myers & Cory, 2013; Rothman, 1997). Particularly good examples are the nine-to-eleven year cycles of the snowshoe hare *Lepus americanus* in Canada (Krebs et al., 2013), the three-to-four year cycles of small rodents, including the lemmings *Lemmus trimucronatus* and *Dicrostonyx spp.* and voles *Microtus spp., Myodes spp.* and *C. rutilus* in Europe (Barraquand et al. 2014; Krebs, 2013) and the eight-to-eleven year cycles of the larch bud moth (LBM), *Zeiraphera diniana* (Baltensweiler, 1977; Baltensweiler & Fischlin, 1988; Baltensweiler et al. 2008), autumnal moth (AM), *Epirrita autumnata* and winter moth (WM), *Operophtera brumata* (Tenow et al. 2007; Klemola et al. 2014) in Europe, and western tent caterpillar (WTC), *Malacosoma californicum pluviale* (Myers & Cory, 2016) in British Columbia, Canada

While time-series analysis can identify the density-dependent structure of population dynamics, it does not identify the mechanisms (Berryman & Turchin, 2001; Krebs 2002, Turchin & Taylor, 1992; Royama, 1992). However, time-series analysis reveals the order and strength of density-dependence, allowing one to formulate probable hypotheses. Coupled with long-term empirical data on mechanisms, time-series analysis can be used to test specific hypotheses as well.

Here I analyze long-term trends for populations of WTC, a species with clear cyclic dynamics (Figure 2.1.). Of particular importance to the dynamics of this species is infection by nucleopolyhedrovirus, (*Baculoviridae*) that periodically reaches epizootic levels in high density populations of its host (Myers & Cory, 2016). First I use time series analysis PACF, log-linear models and wavelet transformations to identify the underlying density-dependent structure, stability and cyclical periodicity of five WTC field populations. Based on this information, along with data on infection by nucleopolyhedrovirus (NPV) of three island populations I suggest that infection by NPV is a potential mechanism for generating cyclical population dynamics. I then I use field data from two island populations to parameterize a simple host-parasite model to establish whether NPV infection can generate stable cycles of similar periodicity to those seen in field populations. I then test the model with data from a third population (Mandarte Island) that was not used to develop the model and compare the model results to the field data using cross-correlation analysis. This model approach is unique in the sense that it uses actual relationships between field-collected data rather than beginning with an assumed model structure and *a priori* assumptions.

2.2. Materials and Methods

2.2.1. Field populations

Field populations of WTC in the Lower Mainland and the Southern Gulf Islands of British Columbia have been monitored on an annual basis (Figure 2) since 1975 (Myers & Cory, 2013). As described in Myers (1990, 2000), the sites (and length of time series) are: (1) Cypress Provincial Park (1986-2015), located at 49.406 latitude and -123.218 longitude. This latter area was monitored at four locations ranging in elevation between ≈ 450-600 m above sea level. Caterpillars in all locations feed on red alder trees (*Alnus rubra*) dispersed throughout the south-facing slope of Black Mountain. (2) Mandarte island (1975-2015), located at 48.633 latitude and -123.283 longitude. This 7ha island in the Haro Strait supports a population that feeds on wild rose (*Rosa nutkana*). (3) Galiano island (1986-2015), located at 48.940 latitude and -123.480 longitude. This site has two monitored locations. The first is situated in a 2-ha area of Montague Provincial Park that contains a mixture of wild rose (*Rosa nutkana*) and apple trees (*Malus sp.*). The second location is a 0.5-km length of road lined with alder and black cherry (*Prunus virginiana*) trees. Data for Galiano is limited to this second site location. (4) Westham island (1977-2015), located at 49.090 latitude and -123.158 longitude. Host plants are primarily crab apple (*Malus sp.*) trees situated along 2-km of drainage ditches on the western edge of the Fraser River delta. (5) Saturna island (1996-2015), located at 48.783 latitude, -123.150 longitude. Patches of red alder (*Alnus rubra*) along roadsides is the primary food source for WTC here.

2.2.2. Population monitoring and nucleopolyhedrovirus infection prevalence

Populations are monitored annually by counting the number of tents and measuring the size of a sub-sample of tents in each site during the period of late instar development. The number of tents serves as an estimate of population size and tent size as an indirect measure of larval survival to the 4th instar (tent size is correlated with survival of 4th instar larvae). Egg masses laid by moths of the previous year are attached to branches near each tent and are collected. The number of eggs per egg mass is then counted and the mean number serves as measure of average fecundity for the population. Subsamples of

ten larvae from each tent are collected and reared in the lab to estimate the level of parasitism and NPV infection in the field. Parasitism is monitored on an individual case basis by checking for the presence or absence of parasitoid emergence from dead larva. NPV infection prevalence is measured as the percentage of subsampled families that contain infected members. Presence of NPV infection is determined visually, since the symptoms of NPV infection are usually obvious. Deaths that are not clearly NPV are examined using oil-immersion light microscopy. Both parasitization and NPV infection data are reported as percentages (NPV as a per family percentage and prasitization as a per individual percentage).

2.2.3. Determining feedback structure

To determine the underlying feedback structure of the WTC time series data, tent abundance for each site was first log-transformed and then regressed against lagged (maximum lag: t = 10) abundance data to produce a partial autocorrelation function (PACF). This process was repeated for each population. This gives the model:

$$R_t = \ln\left(\frac{N_t}{N_{t-1}}\right) = L_t - L_{t-1} = a_0 + a_1 L_{t-1} + \cdots + a_\tau L_{t-\tau} \quad (1)$$

where L represents the log transformed number of tents, τ is the maximum time-lag, a_0 through a_τ are baseline and density-dependent coefficients, and R_t is the population rate of increase at time t.

The PACF demonstrates whether the addition of a lagged term increases or decreases the coefficient of determination (note that the coefficient of determination must be less than zero in order to detect density-dependence). This information, used in conjunction with Bartlett's criterion for significance ($|PACF_d| > 2\sqrt{n}$, with n as the length of the time series) determines the order of the feedback structure (Berryman & Turchin, 2001).

2.2.4. Characterizing periodicity and stability of population cycles using log-linear modeling and continuous wavelet transformations:

Log-linear modeling:

Once the order of feedback structure was determined, population data were fit using an autoregressive approach to the model:

$$L_t = a_0 + a_1 L_{t-1} + \cdots + a_\tau L_{t-\tau} \quad (2)$$

where L represents population density, τ is the maximum time-lag, and a_0 through a_τ are baseline and density-dependent coefficients. Essentially, this process uses nonlinear least- squares method (stats package, R version 3.012) to fit equation (2) to the beginning of the time series data (the first recorded cycle). Once coefficients are calculated, the time series window "moves" ahead by a single time-step (i.e. 1 year) and recalculates the value of each coefficient. This process is repeated until the end of the recorded time series (n).

Once calculated for each site, coefficients for each time-window were plotted on the appropriate parameter plane (see Figure 2.3) to characterize the cycle periodicity for each site over time (see Flemming et al., 2002). Stability of populations was determined by calculating the cumulative absolute change of density-dependent coefficients over time. Values close to zero would indicate minimal change to cyclical dynamics, whereas increasing values would indicate either changes in cycle length or cycle amplitude (cycle dampening). Changes in periodicity over time were also visualized linearly using periodograms (see wavelet transforms below).

Continuous wavelet transform:

Using a continuous wavelet transform, log transformed time series data were decomposed into constitutive periodic components. This approach uses dilations and translations of functions referred to as "mother wavelets" to essentially determine the cross-correlation between a period in the time series data and a wavelet of a specific width or scale. The wavelet transform of a time series $x(t)$ is given by:

$$W_x(b,\tau) = \frac{1}{\sqrt{b}}\int_{-\infty}^{\infty} x(t)\varphi^*\left(\frac{t-\tau}{b}\right)dt - \int_{-\infty}^{\infty} x(t)\varphi^*_{b,\tau}(t)dt \quad (3)$$

where τ denotes the position in time, b is the wavelet scaling factor and $\varphi^*(t)$ is the complex conjugate form of the mother wavelet.

Wavelet coefficients, $W_x(b,\tau)$, are values that describe the correlation between a mother wavelet of scale b at time τ and the time series $x(t)$. These coefficients can be visualized on a wavelet spectrum periodogram to determine underlying periodic components of time series data. For a visual depiction of this process, refer to Cazelles et al. (2008).

Time-series data were transformed using a Mortlet "mother" wavelet, and wavelet spectra were visualized in a periodogram for each field site (WaveletComp package, R version 3.012). Dominant periodicity was determined by extracting the maximum value from the wavelet power plot and observing areas of high correlation indicated by areas of red (located within black significance line – significance threshold held at 95% for all sites) in the periodogram.

2.2.5. Delayed density-dependence and NPV infection as a potential mechanism for cycles

As mainland sites have a history of aerial and ground spray (aerial Btk sprays for Vancouver and Richmond in 1992 and 1993 respectively), for control of Asian GM (*Lymantria dispar*) populations (which might potentially obscure density-dependent effects), island sites were used in order to visualize the strength of delayed density-dependence on population rate of increase. Population rate of increase was plotted against lagged population size, N_{t-1}. Next, data were fit using a non-linear least squares method to the non-linear logistic model (Watt, 1990):

$$R_t = \log\left(\frac{N_{t+1}}{N_t}\right) = A\left[1 - \left(\frac{N_{t-\tau}}{K}\right)^Q\right] \quad (4)$$

where R_t is the population rate of increase at time t, A is the maximum population rate of increase, K is the equilibrium density, and Q, the coefficient of non-linearity. Parameter values were derived using the nls function in R (version 3.012).

Next, the relationship between population abundance (N_t, N_{t-1}) and NPV infection prevalence at time t was analyzed using data from Galiano and Saturna (Mandarte was excluded in order to serve as a non-biased comparison for later, when a population model was constructed from observed relationships). Both linear and logistic models were fit using least squares and non-linear least squares methods respectively (stats package, R version 3.012). The model with the highest R^2 value and lowest AIC value was selected to represent the relationship between population abundance and NPV infection prevalence. Population rate of increase was then plotted against NPV infection prevalence for Galiano and Saturna. As the relationship between population rate of increase and NPV infection was obviously linear (see Figure 2.4 b.), the relationship was analyzed using a linear model.

Models that were parameterized based on data collected from Galiano and Saturna were then used to run experimental simulations (Mandarte was excluded in order to later serve as a non-biased comparison for model accuracy). Iterations were based on the following linked functions:

$$I_t = \frac{100}{1+e^{a(Log_{10}(N_{t-\tau})+b)}} \quad (5)$$

$$R = c(I_t) + d \quad (6)$$

$$N_{t+1} = 10^R N_t \quad (7)$$

where I_t is a logistic function that represents the percent of NPV infected tents at time t; $N_{t-\tau}$ represents the population size at the appropriate time lag, τ (0 or 1, depending on AIC/R^2 criteria); a, the density-dependent transmission coefficient; b, the epizootic midpoint of the logistic curve; R, is a linear function that represents annual population rate of increase; c, the disease-dependent scaling coefficient for population rate of increase; and d, the disease-free annual population rate of increase. Stability and periodicity generated by simulations of the model were visualized through phase-plane and periodogram plots and assessed numerically using the software package Grind (Matlab

R2016a). Cross-correlation (ccf function, stats package, R version 3.012) was used to assess correspondence of simulated and population time-series data for Mandarte. As Mandarte is geographically independent of both Saturna and Galiano, this site provided an unbiased measure of model fit to population data.

2.3. Results

2.3.1. Determining feedback structure

All sites used in the analysis showed negative partial auto-correlation coefficients, with absolute values exceeding Bartlett's criterion at lag 2 (Figure 2.5), indicating second-order density-dependent structure for all populations.

2.3.2. Characterizing stability and periodicity of population cycles using log-linear modeling and continuous wavelet transformations

Absolute total change of density-dependent coefficients over time indicate Galiano as displaying the most consistent cyclical dynamics over time (ΔD.D.= 2.3), followed by Mandarte (ΔD.D.= 4.1), Westham (ΔD.D.= 6.9) and Cypress (ΔD.D.= 7.0) (Table 2.4). Due to the insufficient length of time-series data available, Saturna was excluded from the analysis, however consistency in the wavelet periodogram band (Figure 2.6) as well as a single dominant spike in the wavelet power spectrum (Figure 2.7) confirms the consistency of cyclical dynamics over time. These results are also confirmed visually through phase-plane plots that show the change of coefficients over the autoregressive fitting procedure (Figure 2.8). Changes in periodicity and consistency of cyclical dynamics are also visualized (temporally) through wavelet periodograms and phase-plane plots respectively (Figure 2.6, 2.8). With the exception of Cypress and Westham, all sites demonstrate proper multi-annual cycles over the length of the time-series with periodicity ranging from 7-11 years. Periodicity of mainland sites is markedly different from that of island sites, until the year 2000, at which point both sites appear to shift in their dynamics and converge toward the periodicity of island sites (Figure 2.6, 2.8).

2.3.3. Delayed density-dependence and NPV infection as a potential mechanism for cycles

As predicted by the second-order density-dependent structure identified in section 2.3.1., lagged population density (N_{t-1}) predicted population rate of increase for all island populations better than current population density (Figure 2.10, R^2 = 0.68 compared to R^2=0.18). NPV infection prevalence was best predicted by a lagged logistic model (Table 2.3). As all other models had ΔAIC values greater than 3, support for this model was very strong (Burnham and Anderson 2002). Population rate of increase was strongly correlated to NPV infection prevalence (Figure 2.4 b., R^2 = 0.50, p = 3.319e-06). Estimated model parameters are shown below:

$$I_t = \frac{100}{1+e^{(-2.26Log_{10}(N_{t-1})+5.205)}} \quad (8)$$

$$R = -0.023(I_t) + 0.932 \quad (9)$$

$$N_{t+1} = 10^R N_t \quad (10)$$

The composite model is represented by the following density-dependent function:

$$N_{t+1} = 10^{-0.023m(N_{t-1})+0.932} N_t \quad (11)$$

where m represents the logistic function represented by equation (8).

Simulation experiments of the model generated from equations (8)-(10) (N_0=5, n =10, 000) yielded stable multi-annual cycles for both host and NPV populations (Figure 2.11 b.). Stability was also confirmed using eigenvalue and phase plots generated using the Matlab package Grind (Appendix B). Long-term host amplitude ranged from 13 (minimum) to 769 (maximum) tents and NPV infection prevalence ranged from 6 (minimum) to 79 (maximum) percent. Average periodicity for both host and NPV populations was 6 years (Figure 2.11).

2.4. Discussion

2.4.1. Determining feedback structure:

These results reveal a strong second order density-dependent structure that largely determines the 7-11 year cycles of WTC in southwestern BC. The strength of the delay appears to be stronger for island sites than mainland sites, perhaps reflecting effects of Btk spray treatment in the mainland or distinct differences in the type or magnitude of mechanisms governing the population dynamics of island versus mainland sites. Spatial heterogeneity likely explains this difference, as both Westham and Cypress are located in areas immediately adjacent to extensive urban landscape. Cypress is also located along a steep altitudinal gradient, approximately 400-650 m above sea level. These unique habitat characteristics may be sufficient to alter small-scale migration or interactions between WTC and its natural enemies, resulting in the observed variation in density-dependence. This hypothesis is supported by Liebhold et al. (2006), who found that geographical variation in density-dependence was strong enough to alter the dynamics and overall synchrony of established gypsy moth (*Lymantria dispar*) populations along the northeastern coast of the US. Similarly, in small Fennoscandian rodent populations, Bjørnstad et al. (1995) revealed a strong latitudinal gradient in population fluctuations that was correlated with geographical changes in generalist predator abundance. Spatial heterogeneity has also been implicated in the loss of cyclical behaviour of North American snowshoe hare (*Lepus americanus*) populations (Krebs et al., 2013; Ims et al., 2007).

2.4.2. Characterizing periodicity and stability of population cycles using log-linear modeling and continuous wavelet transformations:

While cyclical periodicity for most sites ranged between 7-12 years, mainland sites were less predictable than island sites in terms of periodicity, stability and relatedness. Given sufficiently high correlation in environmental stochasticity, populations whose dynamics are determined by identical mechanisms should display a high degree of relatedness in their density-dependent structure over time (Moran, 1953). Given this, our results suggest

that the population dynamics of island sites (Galiano, Mandarte and Saturna) may be governed by similar mechanisms and that changes in these mechanisms appear (in comparison with mainland sites) quite minimal over time. This is in contrast to mainland sites (Cypress, Westham), which are also strongly related to each other, but display larger changes in their density-dependent structure over time.

Time-series analysis predicts that specialist enemies will contribute to time delays of a host population and the dimensionality of a system (Bjørnstad & Grenfeld, 2001). Given that the second-order feedback of mainland sites is significantly weaker than that of island sites may suggest that natural enemies such as NPV and/or parasitoids may not be as significant in the population dynamics as they are for island sites. This highlights both the importance and power of using time-series analysis as a preliminary investigative tool before attempting to draw comparisons between geographically disjunct populations and make mechanistic inferences. Failing to conduct such an analysis greatly increases the risk of failing to detect signatures of plausible mechanisms.

2.4.3. Delayed density-dependence and NPV infection as a potential mechanism for cycles

Several mechanisms are capable of generating second-order feedback due to the fact that they are capable of generating a time-delay (Kendall et al. 2005; Turchin et al. 1999). Maternal effects, variation in food quality, parasitoids and natural enemies are all capable of operating through a time-delay (Berryman, 1996). I have not dismissed the possibility of other factors contributing to the population dynamics of WTC, however, for the purpose of this study I have chosen to investigate whether NPV infection is capable of generating second-order cyclical dynamics. Historically, the literature on population cycles in forest Lepidoptera has emphasized the roles of food quality and quantity, along with parasitoids, while pathogens have been underemphasized (Berryman, 1996; Stiling, 1988). This is because pathogen infection is not immediately detectable (visually) in field populations and measures of disease prevalence usually require rearing larvae in the lab to determine infection status, which is not often done. With the development of molecular techniques such as PCR, it was shown that viral DNA of NPVs can persist within host populations and across generations as covert infection (Myers & Cory, 2013; Cory & Myers, 2003;

Burden et al., 2002). This realization has renewed interest in NPVs as a potential mechanism for generating population cycles.

Here, I have shown that that population rate of increase is negatively correlated with NPV infection prevalence and that NPV infection is best predicted by population abundance in the preceding generation (Table 2.3). This is intuitive if one assumes that the total amount of NPV related mortality at the end of one larval generation is proportional to the amount of viral inoculum present at larval emergence (i.e. the viral occlusion bodies that persist from one generation to the next). Even though I do not have a measure of initial NPV inoculum density at larval emergence, it is logical to assume that this would be related to the population abundance of the preceding generation (after considering factors such as larval size and/or environmental degradation). This might explain why lagged population density better predicts NPV infection prevalence than current population density (although there still is a significant correlation between current population size and NPV infection, see Table 2.3). The correlation between NPV infection and 2-year lagged population size is weak (R^2 = 0.03, p-value= 0.0479). This lagged effect of persistent virus would also explain the prolonging of the decline phase of WTC cycles over several generations. NPV occlusion bodies are known to persist in the environment and have previously been shown to remain at high concentrations on bark (Olofsson, 1988; Podgwaite et al, 1979) and remnant silken tents (although it is not known whether larva make contact with old tents) contaminated in a previous year (Kukan, 1996), if protected from UV. Contaminated egg masses are another possibility, as WTC offspring whose parents were exposed to naturally occurring virus in the field have been shown to die of infection when reared in the laboratory (Kukan, 1999). Also, viral persistence through covert infection is likely, although the frequency of overt expression from covert infection remains unknown (Cooper et al., 2003a).

Simulations run using relationships between NPV, population size and rate of increase clearly demonstrate the potential of NPV as a mechanism for generating stable population cycles. Here, instead of modifying a second-order log-linear population model to include a stochastic term (a classic time-series approach), I have constructed a mechanistic model, fit using actual population data. This is a novel approach in that it infers mechanisms from established relationships at the population level, instead of fitting model parameters using experimentally or theoretically derived (i.e. first-principles) values. The

model also supports the second order density-dependence revealed through time-series analysis. While the model itself does not accurately match the population trajectory of our test site (compare Figure 2.11. a. with Figure 2.12), it does generate cycles of similar periodicity.

Model fit to population data (Mandarte) may be improved by further investigating whether other factors influence NPV infection and population rate of increase and then incorporating these effects. For instance, reduced fecundity as a result of crowding, reduced leaf quality, selection for resistance (a possible trade-off with fecundity) or a sublethal effect of surviving virus challenge, might subsequently decrease population rate of increase and prolong the crash phase of cycles (Cory & Myers, 2009). Warmer (than historical average) temperatures may have the potential to strengthen the effect of natural enemies (Klapwijk et al. 2013). Elderd and Reilly (2013) found that NPV transmission and outbreak intensity (measured as the cumulative fraction of larva infected during an epizootic) both increased when populations of fall armyworm (*Spodoptera frugiperda*) were exposed to outdoor temperatures that were elevated using open-top greenhouse chambers. Incorporating such effects could greatly improve the accuracy and predictive power of the previously constructed model and provide better insight into what factors are important in generating cycles in forest Lepidoptera. This requires a multivariate approach that can identify key factors involved in these dynamics.

This work suggests the persistence of viral occlusion bodies (either through contaminated foliage, egg masses or covert infection) as a source of ecological “memory” responsible for producing a delayed density-dependent signature in the WTC time-series data. This idea is supported by an experiment conducted by Rothman (1997), where the proportion of NPV-killed caterpillars in a season was related to population density in the previous year. Although our model construction is based on actual data collected from years of population monitoring, more detailed experimental work is needed to test its validity. This should involve a quantitative measurement of virus persistence across entire cycle periods. For instance, there are periods of time in which there is no evidence of viral infection, and environmental persistence is unlikely to account for the long-term survival of virus. Also, models evaluating alternative hypotheses (i.e. maternal effects, host-plant interactions) should also be tested to determine whether they alone, or perhaps in

combination with other mechanisms are sufficient in describing (and predicting) the dynamics of this system.

This work is significant as it provides further support for the contribution of baculoviruses to the cyclical dynamics of forest Lepidoptera; a subject of debate among many population ecologists (Myers, 1993; Berryman, 2002; Turchin, 2003; Myers & Cory, 2013). Most data on baculovirus-host dynamics are largely theoretical, using principles derived from models first described by Anderson and May (Parker et al., 2010; Elderd et al. 2008, Dwyer, 1994; Dwyer, 1993) and lack long-term disease prevalence data. In contrast, the data I have used to construct a population model that generates stable cyclical dynamics is based on actual observed relationships between pathogen and host in the field. While theoretical models provide insight into the dynamics generated by proposed interactions between host and pathogen, they require empirical evidence (beyond population density) to validate them. Lastly, the role of baculoviruses such as NPVs in the cyclical dynamics of forest Lepidoptera needs to be re-evaluated and more long-term empirical studies on disease prevalence in populations are required.

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Tables

Table 2.1. Correlation of direct density-dependent coefficients derived from a second-order log-linear model for field populations of WTC in southwestern British Columbia. A measure of site "relatedness" is given as R^2 values. Saturna time-series data have been excluded due to the limited population data that are available for this site (1996-2015) in comparison with others (1986-2015). However, comparison of wavelet periodograms for Saturna versus other island sites (Figure 2.6) confirms the correspondence of its dynamics to other sites.

Direct Density-dependence	Mandarte	Galiano	Westham	Cypress
Mandarte		0.711	0.049	0.055
Galiano			0.0931	0.085
Westham				0.743
Cypress				

Table 2.2. Correlation of delayed density-dependent coefficients derived from a second-order log-linear model for field populations of WTC in southwestern British Columbia. A measure of site "relatedness" is given as R^2 values. Saturna time-series data have been excluded due to the limited population data that are available for this site (1996-2015) in comparison with others (1986-2015). However, comparison of wavelet periodograms for Saturna versus other island sites (Figure 2.6) confirms the correspondence of its dynamics to other sites.

Delayed Density-dependence	Mandarte	Galiano	Westham	Cypress
Mandarte		0.824	0.574	0.067
Galiano			0.540	0.097
Westham				0.396
Cypress				

Table 2.3. AIC model fitting results for models of western tent caterpillar population size (number of tents: lagged- N_{t-1} and current- N_t) regressed against NPV infection prevalence data (1986-2015). Data from Galiano, Mandarte, Saturna were used. Westham and Cypress were excluded from the analysis on the basis of their history of *Bt* spray records for Asian Gypsy Moth (*Lymantria dispar* sp.).

Independent Variable	**Model**	**R^2**	**AIC**	**ΔAIC**
N_{t-1}	Logistic	0.42	524.08	+0.00
N_t	Logistic	0.35	529.97	+5.89
N_{t-1}	Linear	0.33	530.58	+6.50
N_t	Linear	0.32	531.94	+7.86

Table 2.4. Absolute total change (cumulative) of density-dependent coefficients (direct and delayed) derived from a second-order log-linear model for time-series data taken from field populations of WTC in southwestern British Columbia over time (1986-2015). Saturna has been excluded due to the limited population data that are available for this site (1996-2015) in comparison with others (1986-2015).

Site	ΔD.D. Value
Galiano	2.3
Mandarte	4.1
Westham	6.9
Cypress	7.0

Figures

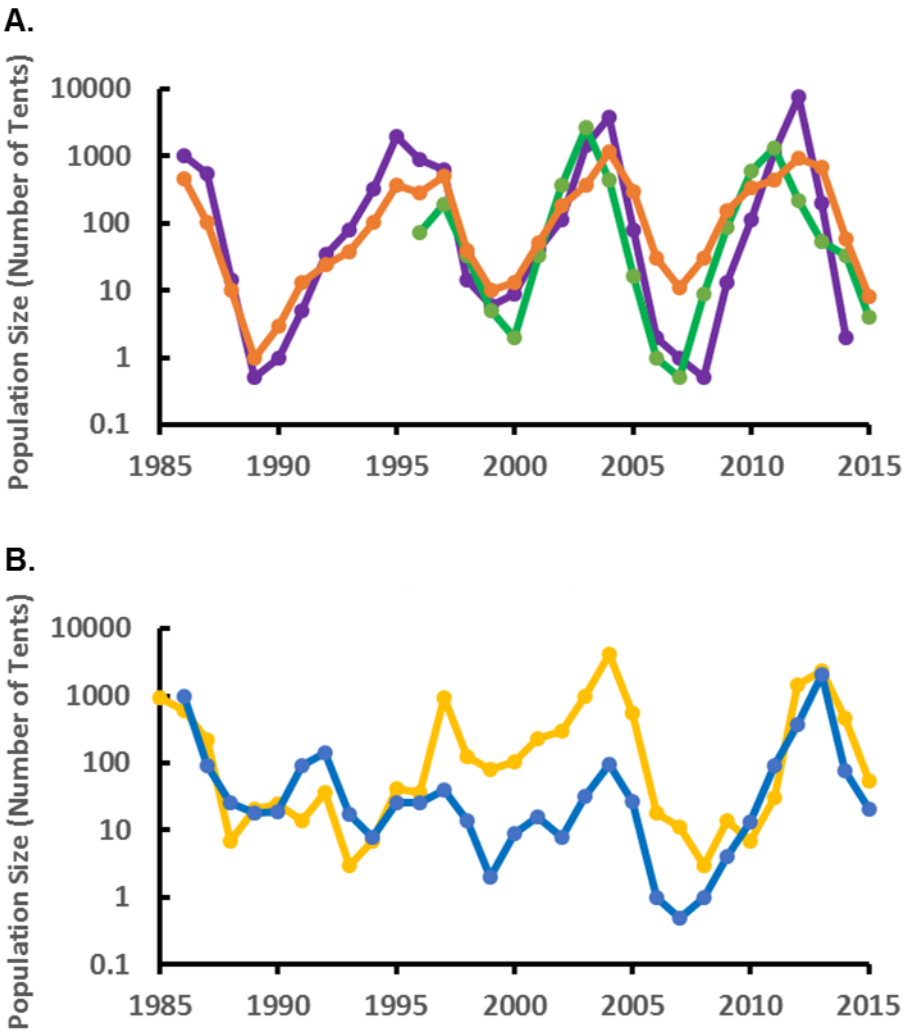

Figure 2.1. Time-series data for western tent caterpillars on (A.) Mandarte Island (orange), Galiano Island (purple) and Saturna Island (green) (B.) Westham Island (yellow) and Cypress Mountain (blue). Annual population abundance; measured as the total number of tents, is shown on the vertical axis (log scale) and time (years) is shown on the horizontal axis.

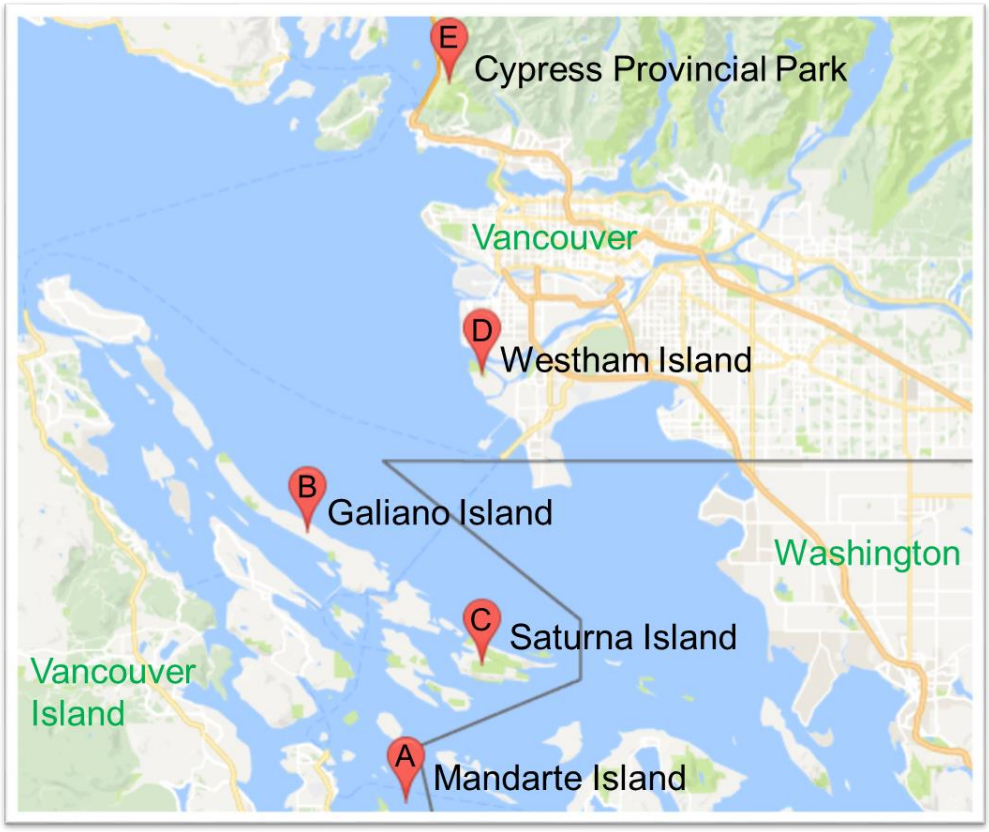

Figure 2.2. Map displaying the geographical locations of the five field populations of western tent caterpillar used in the time-series analysis. Island sites are situated within the Southern Gulf Islands of B.C. (bottom-left): (A.) Mandarte Island, (B.) Galiano Island, (C.) Saturna Island. Mainland sites are situated along the south-western coast of B.C. (upper-right): (D.) Westham Island, (E.) Cypress Mountain.

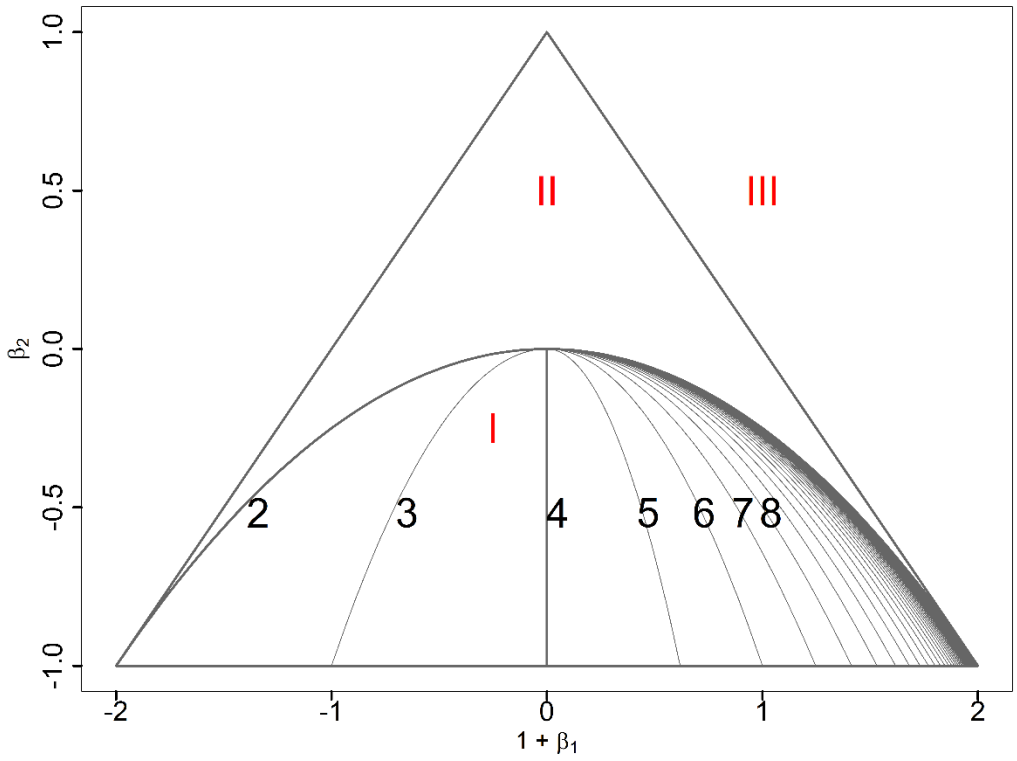

Figure 2.3. Parameter space of the second-order log-linear model. Roman numerals (red) indicate regions of unique dynamics that are possible given a combination of direct (horizontal axis) and delayed (vertical axis) density-dependent coefficients: I = Stable Cycles (isoclines indicate periodicity/cycle length), II = Cycle Convergence, III = Cycle Divergence.

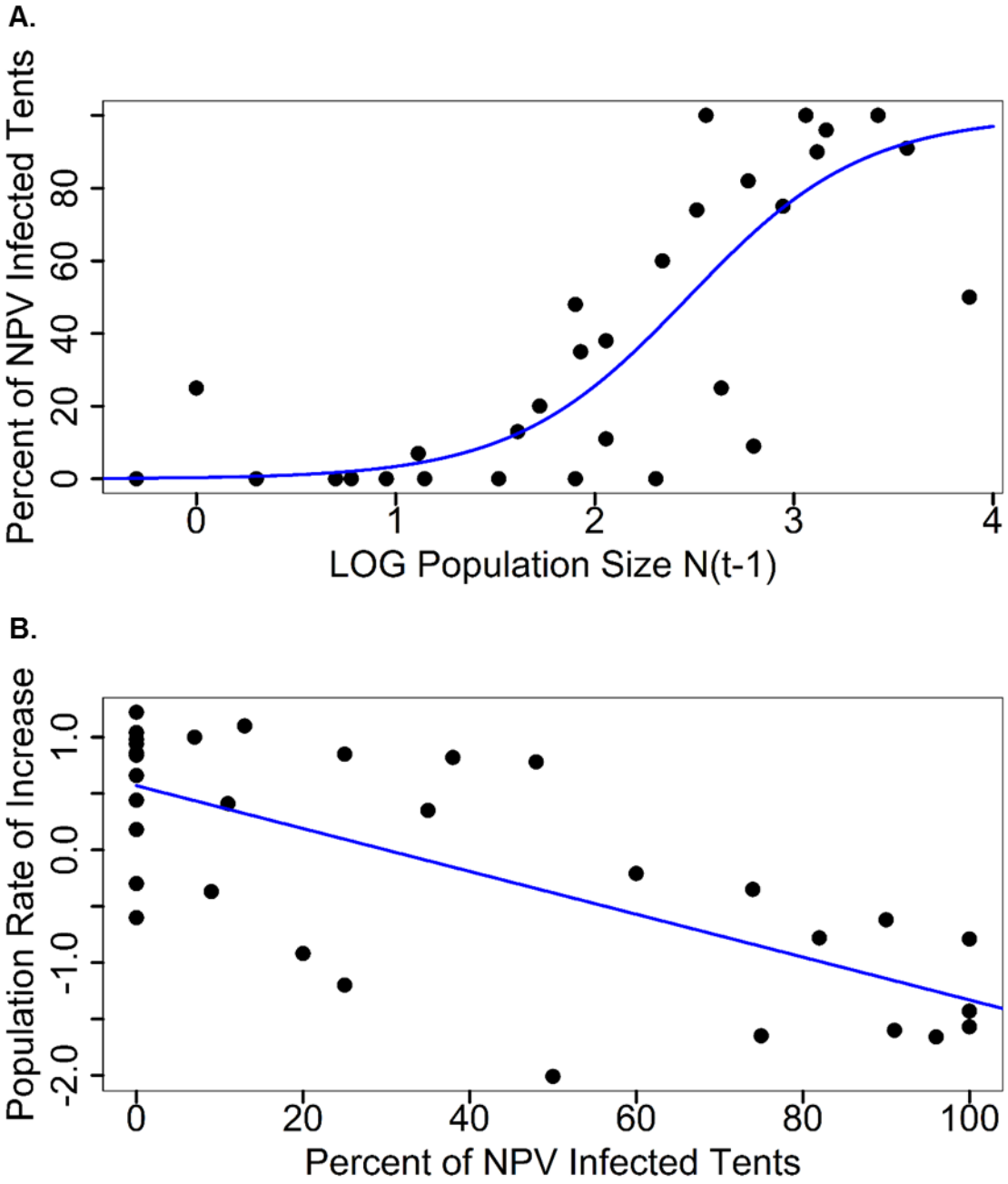

Figure 2.4 (A.) NPV infection prevalence data regressed against lagged population size data for western tent caterpillar field populations on Galiano Island and Saturna Island, R^2 = 0.75 (B.) Annual population rate of increase data ($R=\log(N(t+1)/N(t))$ regressed against NPV infection prevalence data for western tent caterpillar field populations on Galiano Island and Saturna Island, R^2 = 0.50, p = 3.319e-06.

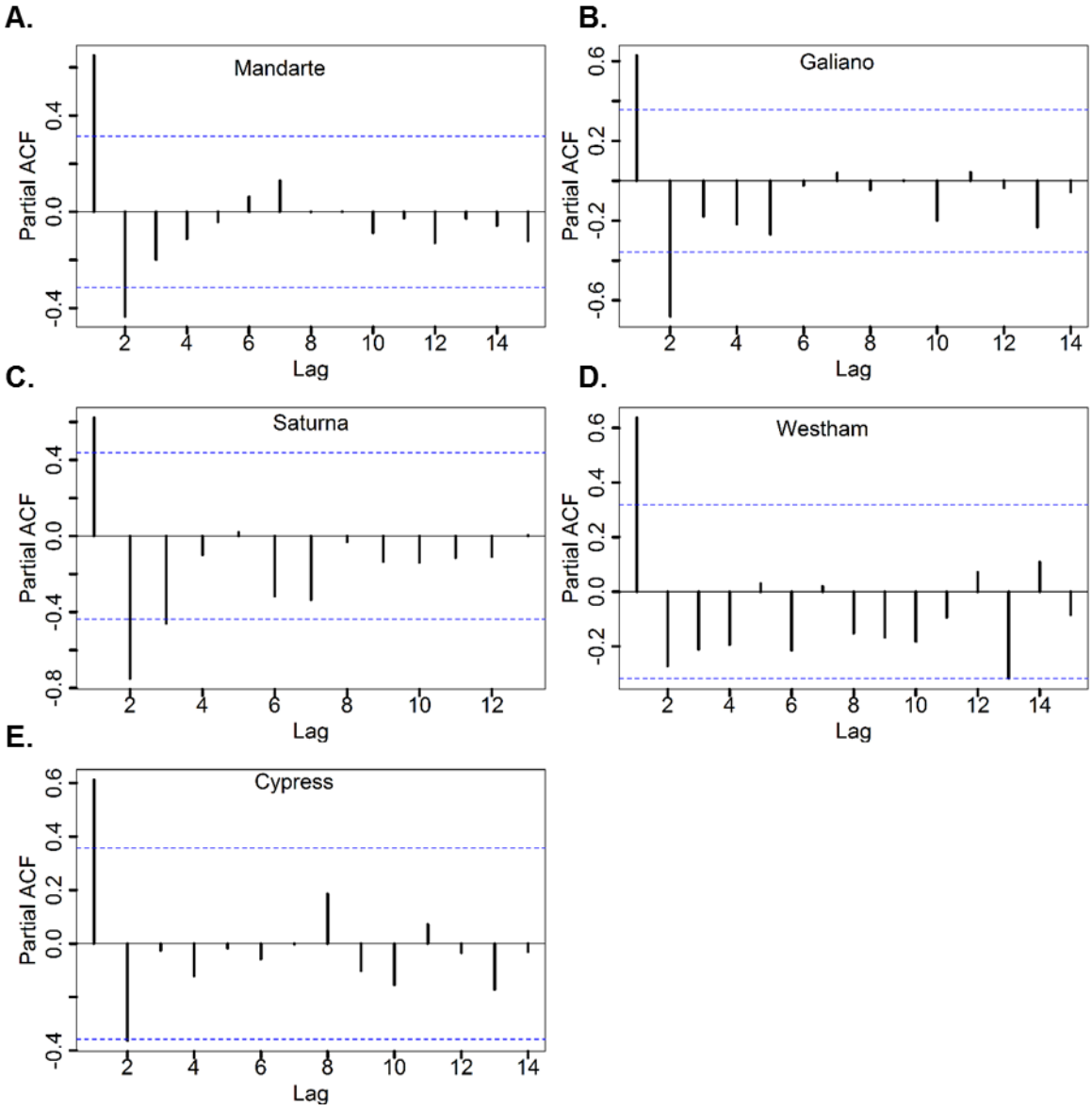

Figure 2.5. Partial autocorrelation function (PACF) plots for field populations of western tent caterpillar located in southwestern British Columbia. Sites include: (A.) Mandarte Island (B.) Galiano Island (C.) Saturna Island (D.) Westham Island (E.) Cypress Mountain. Autocorrelation is displayed on vertical axis and Lag (in years) displayed on horizontal axis. Bartlett's criterion (line of significance) is shown in blue (hashed line).

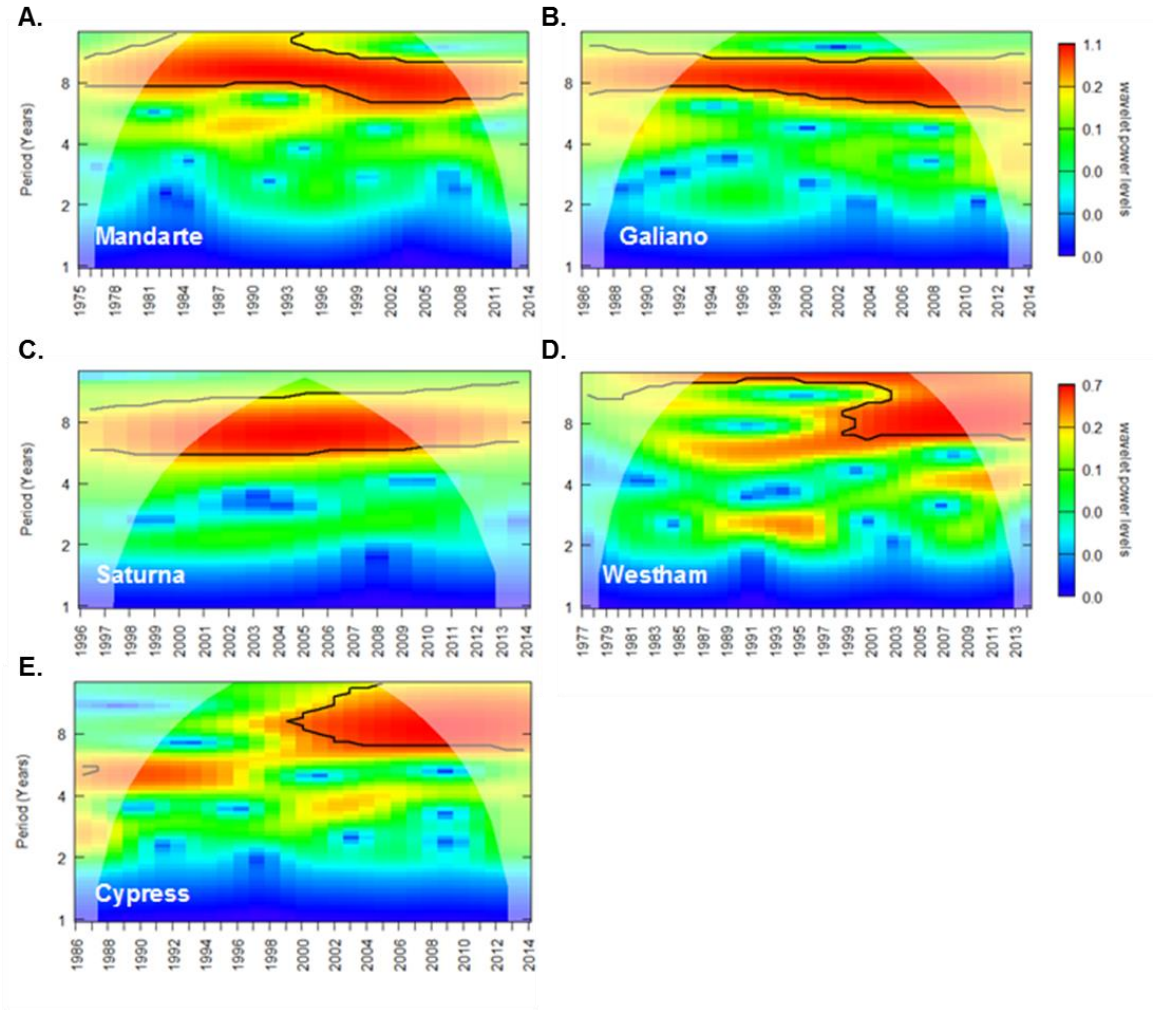

Figure 2.6. Normalized wavelet power spectra, displaying changes in cycle periodicity over time for field populations of western tent caterpillar located in southwestern British Columbia. Sites include: (A.) Mandarte Island (B.) Galiano Island (C.) Saturna Island (D.) Westham Island (E.) Cypress Mountain. Likelihood estimates of the wavelet power spectrum are shown as coloured bars (right). Areas surrounded by black contour line indicate regions of 95% confidence. White tinted regions represent the 'cone of influence' (beginning and end of the time-series data), where the accuracy of wavelet analysis declines. Inferences should thus be avoided in white tinted areas.

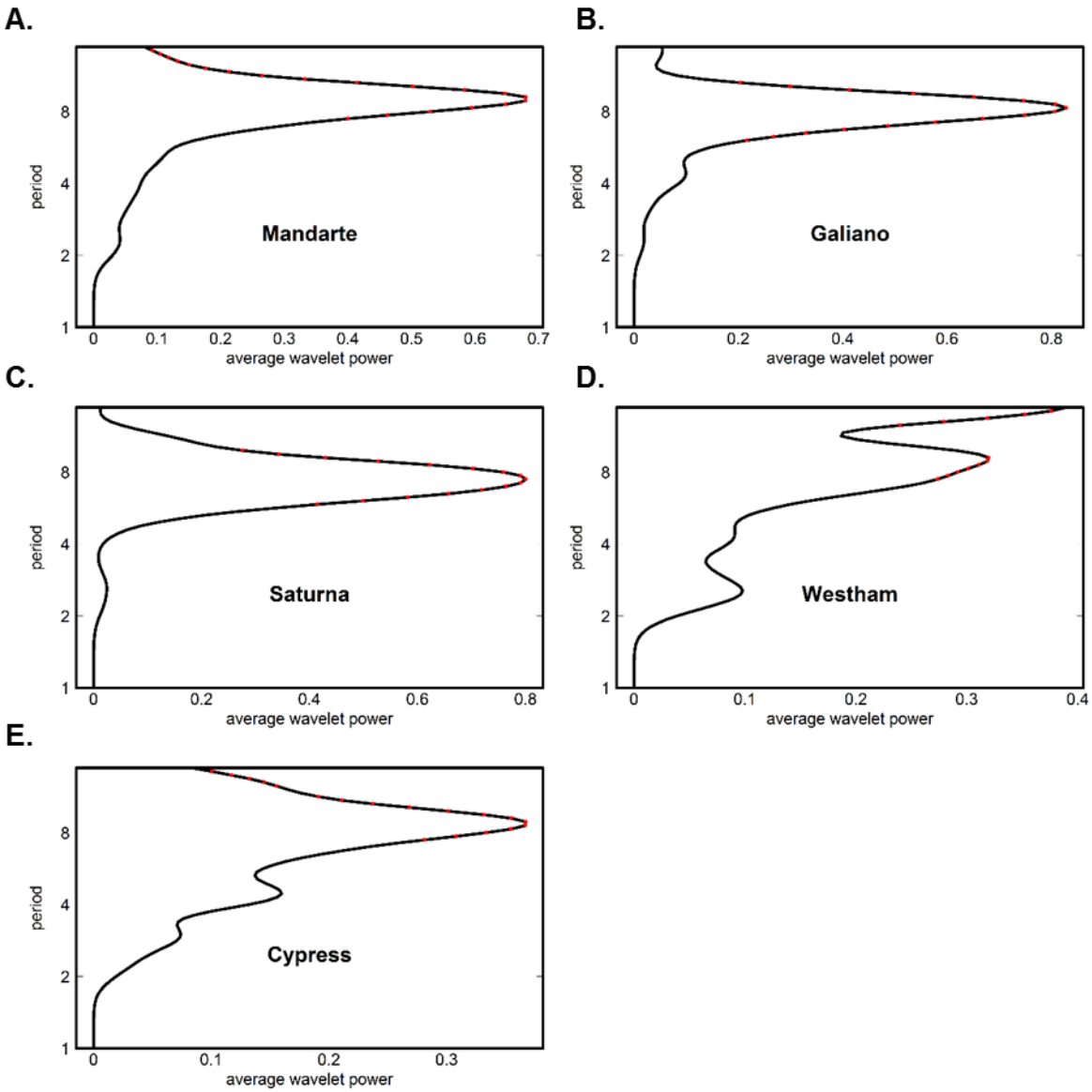

Figure 2.7. Average wavelet power spectra displaying significant periodic signatures for field populations of western tent caterpillar located in southwestern British Columbia. Sites include: (A.) Mandarte Island (B.) Galiano Island (C.) Saturna Island (D.) Westham Island (E.) Cypress Mountain. Plots with a single, dominant peak indicate populations with fairly constant (stable) dynamics. Plots with multiple peaks indicate noisy or non-stationary dynamics.

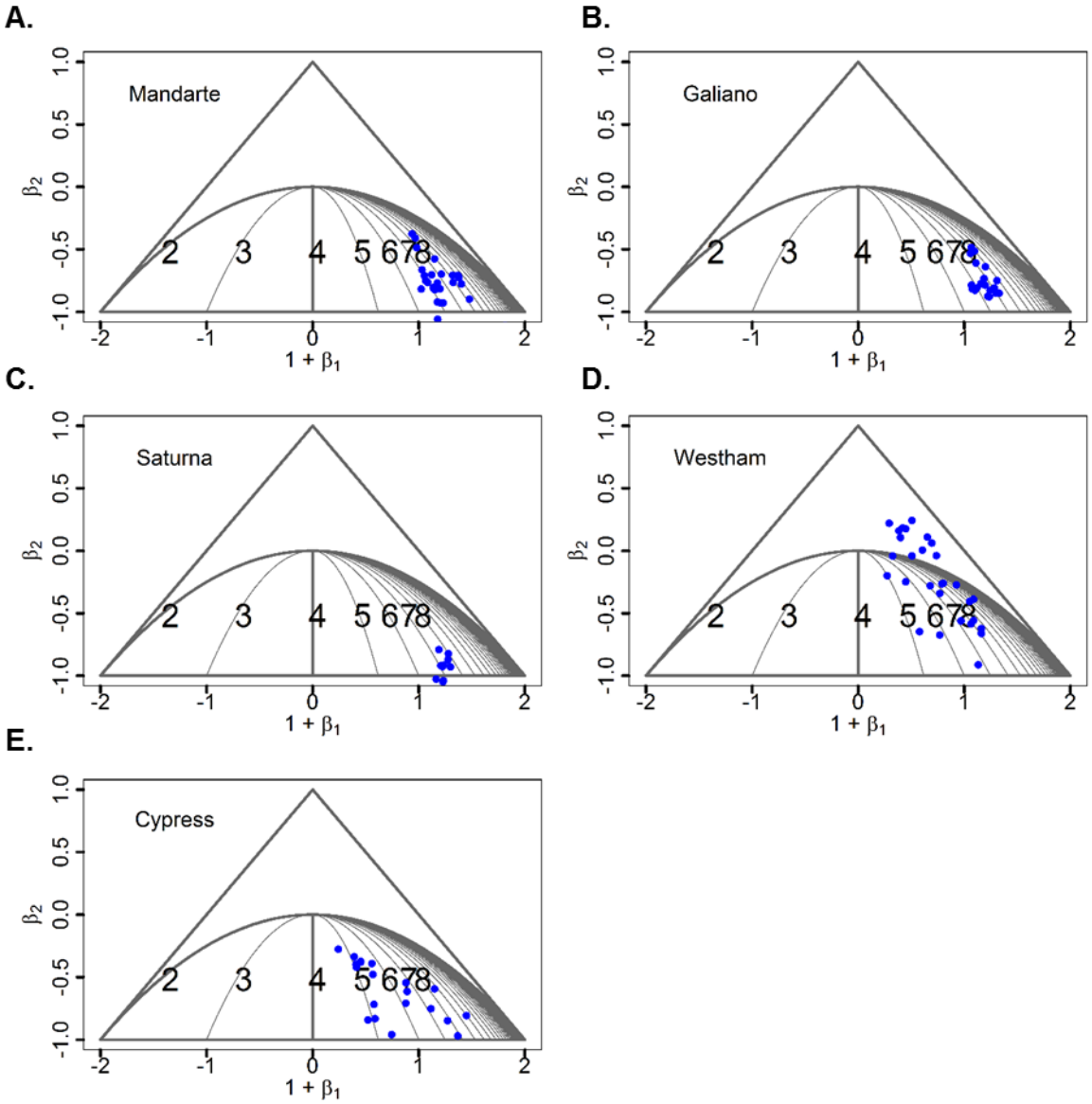

Figure 2.8. Annual measures of direct (horizontal axis) and delayed (vertical axis) density-dependence based on a second-order log-linear model fit using non-linear least squares method to time-series data for field populations of western tent caterpillar located in southwestern British Columbia. Sites included: (A.) Mandarte Island (1975-2015), (B.) Galiano Island (1986-2015), (C.) Saturna Island (1996-2015), (D.) Westham Island (1977-2015), (E.) Cypress Mountain (1975-2015).

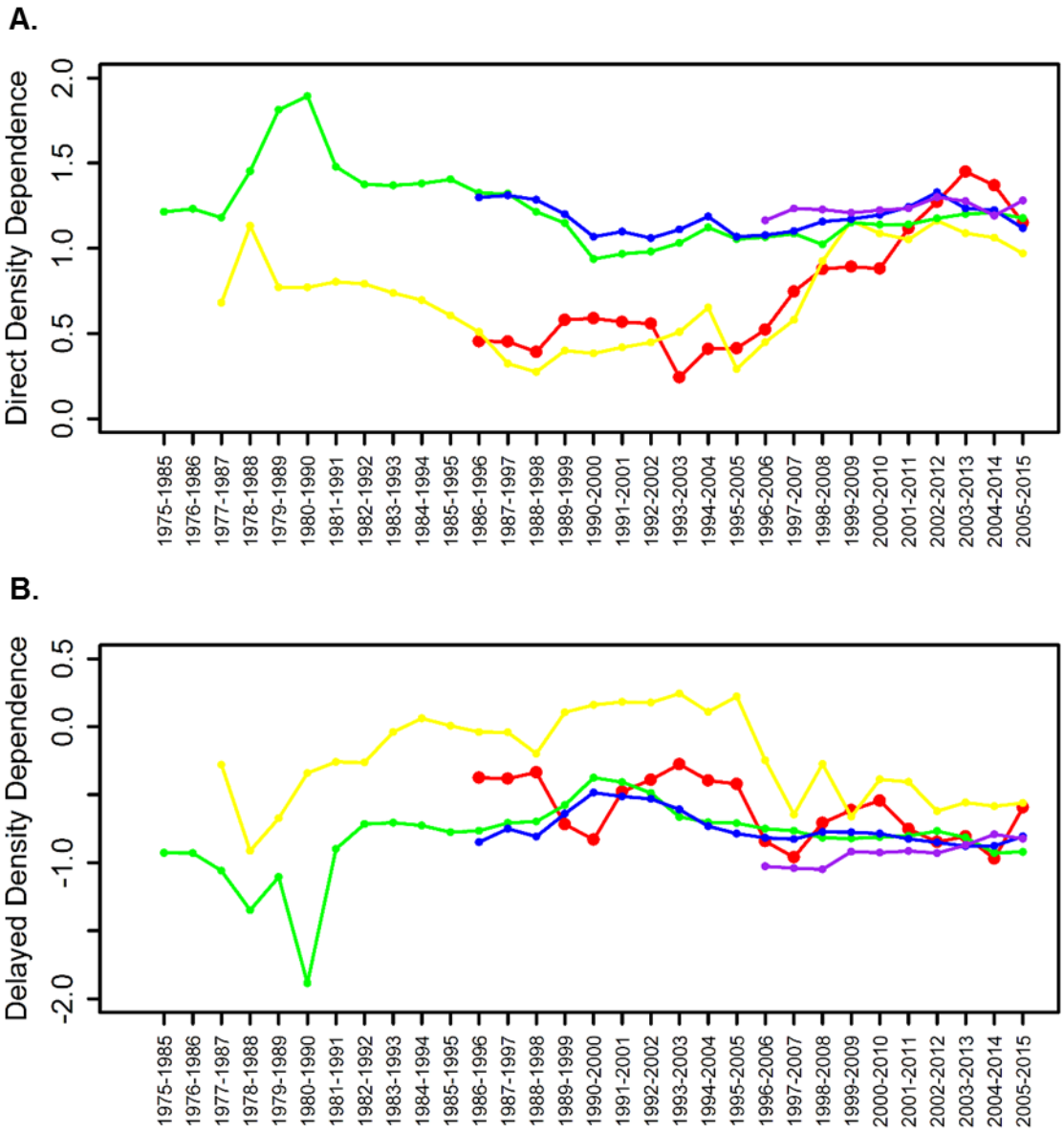

Figure 2.9. Line plots illustrating annual changes in (A.) direct and (B.) delayed density-dependent coefficients fit using a second-order log-linear model with a 10-year window for field populations of western tent caterpillar located in southwestern British Columbia. For both plots sites are indicated by colour: Mandarte Island (green), Galiano Island (blue), Saturna Island (purple), Westham Island (yellow) and Cypress Mountain (red).

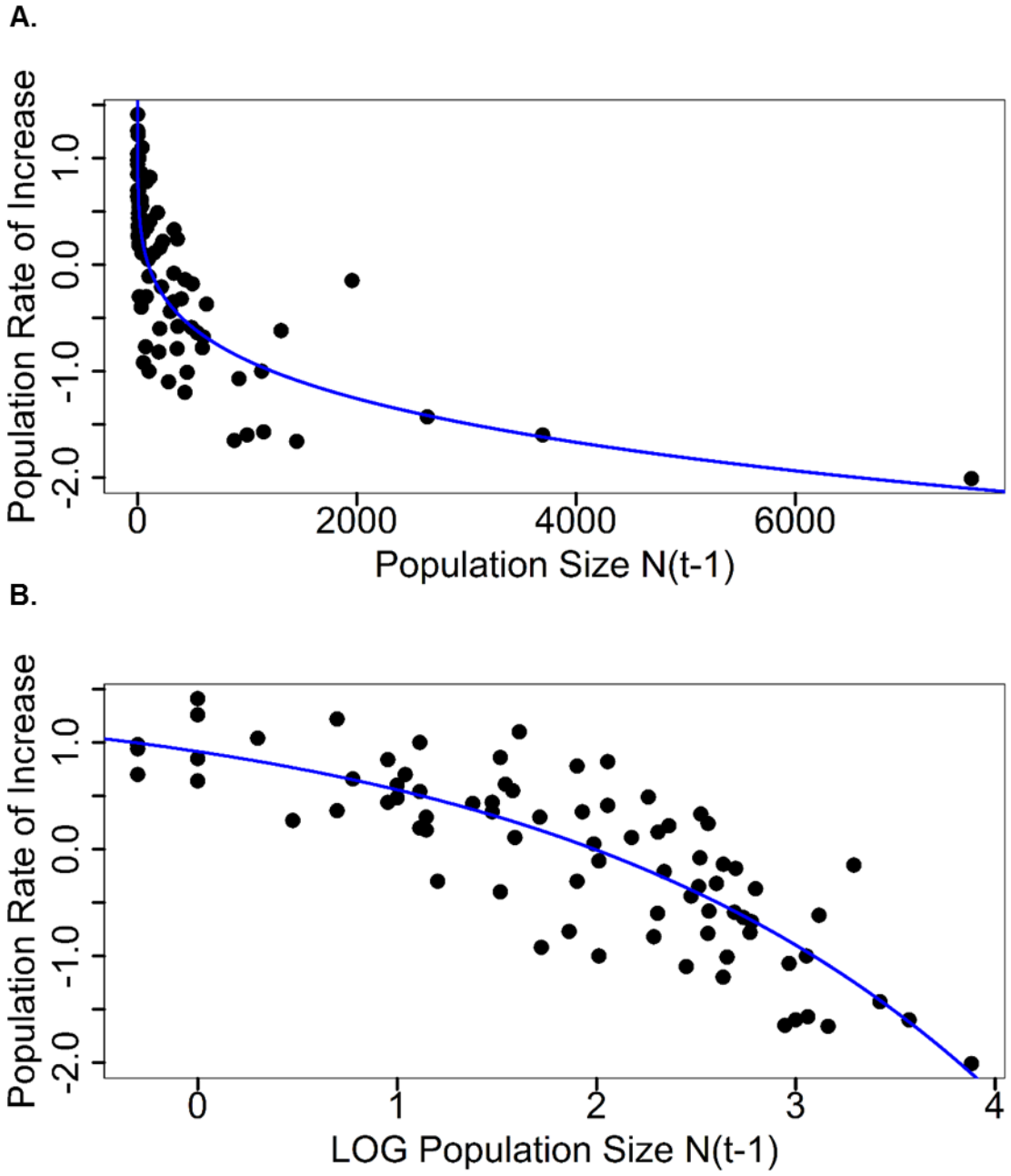

Figure 2.10. (A.) Annual population rate of increase ($R=\log(N(t+1)/N(t))$ data regressed against lagged population size data (1986-2015) for western tent caterpillar field populations located in southwestern British Columbia. Sites included: Galiano Island, Saturna Island and Mandarte Island. Data were fit using a non-linear logistic model (blue line), $R^2 = 0.68$, $A= 1.5380$, $K=97.6001$, $Q=0.1978$. (B.) Same plot as above (A.) but horizontal axis is log (base 10) transformed for clarity.

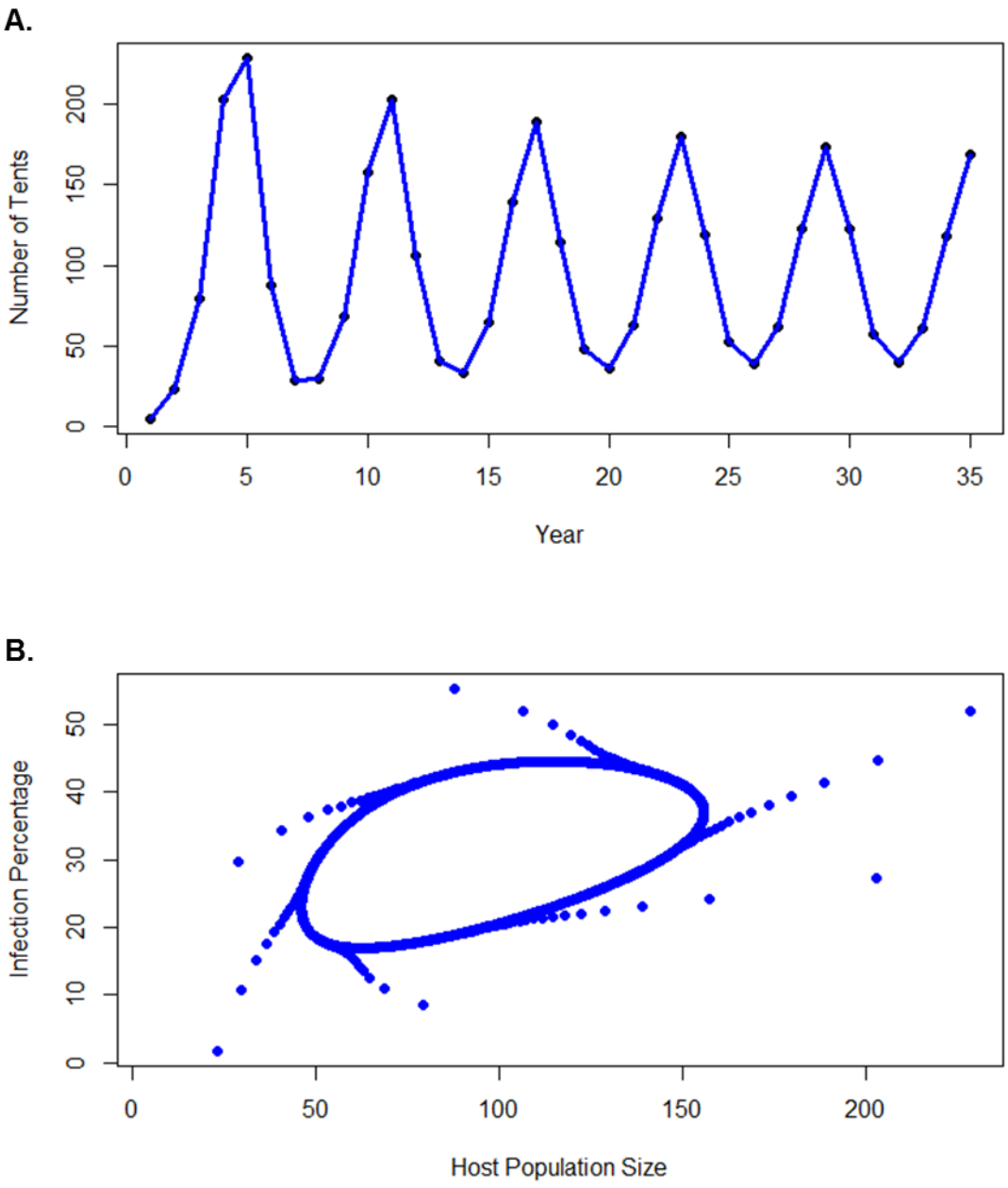

Figure 2.11. (A.) Numerical simulation of the host-parasite (western tent caterpillar-nucleopolyhedrovirus) model constructed using successive iterations of equations (5)-(7), N_0=5, t=35. (B.) Phase plane of the host-parasite model indicating dynamics as converging toward stable cyclical behavior, t=10, 0000.

Chapter 3.

Demographic and environmental variables that influence the population dynamics of the western tent caterpillar.

3.1. Introduction

The population dynamics of forest insects can be thought of as being the product of a complex interplay between intrinsic dynamics and extrinsic factors (Bjørnstad et al., 2004; Mishra, 2009). These may act directly or indirectly, having immediate and/or delayed effects on the growth, survival or reproductive success (Ratikainen et al., 2008). Of these factors, those that respond strongly to density are likely to play a significant role in the long-term dynamics of many forest insect species, provided they are not overwhelmed by exogenous processes (Brook & Bradshaw, 2006). For instance, density-dependent factors such as intra-specific competition and reduction in the quality or quantity of food have been shown to be important limiting factors in the dynamics of a number of insect species (Briggs et al., 2000; Larsson et al., 2000; Hódar et al., 2002; Percy et al., 2002). For populations of many forest Lepidoptera, density-dependent factors operating through a time-delay are thought to be responsible for periodic cyclical fluctuations in abundance (Beckerman et al., 2002; Esper et al., 2007). A difficulty in identifying the dominant factors responsible for generating such dynamics lies in the fact that many long-term datasets are not comprehensive. A majority of the annual population data for gypsy moth (*Lymantria dispar*) and larch budmoth (*Zeiraphera diniana*) populations for example, contain only measures of tree defoliation, tree ring width and/or larval density per branch (Esper et al., 2007; Allstadt et al., 2013). The lack of key information on the important intrinsic and extrinsic factors that underlie such dynamics, little can be said about the mechanisms through which density-dependence operates or about the relative importance of specific factors.

Intrinsic dynamics involve any interactions that result in feedback within the system (Koelle & Pascual, 2004). These are normally density-dependent, and can include predation,

competition, disease, parasitism and maternal effects. Delayed density-dependence can also operate through these factors and as such, all are theoretically capable of generating cyclical dynamics. In many forest Lepidoptera, density-dependent processes often operate through one or more time lags (Berryman, 1996; Berryman & Turchin, 2001; Liebhold et al., 2000; Cooke & Lorenzetti, 2006). For example, I have previously shown that the relationships between lagged population density, NPV infection prevalence and annual population rate of increase are sufficient to generate cyclical population dynamics in the WTC system (Chapter 2). However, this information does not provide any measure of the relative importance of these factors with respect to actual population dynamics. As well, there are other factors (both intrinsic and extrinsic) to consider as potential contributors to dynamics of WTC. Life-history traits, such as survival and fecundity for example, are known to be influenced by current and previous environments (Beckerman et al., 2002). Also, changes in seasonal weather patterns have been shown to impact larval performance, survival and habitat range (Hódar & Zamora, 2004; Klapwijk et al., 2013). Temperature in particular has been identified as the dominant abiotic factor affecting most herbivorous insect populations (Bale et al., 2002).

Survival probability is largely dictated by the presence of specific mortality factors, and the most dominant mortality factor in insect populations is natural enemies (Cornell & Hawkins, 1995; Cornell et al., 1998), which can include predators, parasitoids and pathogens (Hawkins et al., 1997). Among forest Lepidoptera that cycle, the most cited mortality factors are parasitoids and pathogens (Briggs & Godfray, 1995; Turchin et al., 2003). Mathematical models based on first-principles have been used extensively to demonstrate the potential of these factors in generating dynamics that are observed in natural populations and have gained tremendous popularity as a means of testing specific mechanisms, particularly where long-term data are not available (Dwyer, 1994; D'Amico et al. 1996; Elderd et al., 2008; Fenton et al. 2002). However, criticisms of such models include the possibility that the relative importance of specific factors are overemphasized and parameter values which are not biologically relevant or representative of the system.

The models of Anderson & May (1979, 1981) were the first to explore the potential role of pathogens in the population dynamics of forest insects. Since this time, models have been adapted to specific systems and modified to include more biological realism, such as

heterogeneity of host and pathogen populations, environmental persistence of pathogens, sublethal infection, travelling waves of infection and multiple mortality agents (Dwyer, 1992; Briggs & Godfray, 1996; Dwyer et al., 1997; Dwyer et al., 2000; Dwyer et al., 2004; van der Werf et al., 2011). While these model advancements have attempted to describe the mechanisms governing the dynamics of these systems, most models are parametrized using estimations from literature or short-term field experiments and quite often lack long-term field data to support specific hypotheses (Ghosh & Bhattacharyya, 2007; Elderd et al.2008; Elderd et al.2013).

While other pathogens aside from NPV certainly cause mortality in forest Lepidoptera (for instance, the fungus *Entomophaga maimaiga* associated with GM), NPVs are largely species-specific, and are most frequently associated with spectacular epizootics (Elkinton & Liebhold, 1990; Cory & Myers, 2009; Myers & Cory, 2013; Myers & Cory, 2016). The periodic population declines of GM in particular have frequently been associated with NPV infection (although long-term field data of infection levels does not exist) and the dynamics of this system have been extensively modeled (Dwyer & Elkinton, 1993; D'Amico et al., 1996; Dwyer et al., 1997; Dwyer et al., 2000; Dwyer, 2004; Parker et al., 2010). Experiments have contributed greatly to the biological realism of such models, demonstrating that transmission is not always a simple mass-action process as previously thought, and that the relationship between host and pathogen density is often non-linear (D'Amico et al., 1996). The influence of other factors, such as host behaviour and temperature, have also been modeled more recently in the GM and fall armyworm (*Spodoptera fugiperda*) systems with respect to baculovirus transmission (Elderd & Reilly, 2013; Eakin et al., 2015). The results of these modeling experiments suggest that while pathogen transmission might be a key regulating factor in the dynamics of several populations of forest Lepidoptera, the contributions of (and interactions between) other intrinsic and extrinsic factors might also be important.

Changes in fecundity over the course of population cycles are a common trend across many forest Lepidoptera (Myers, 1988; Rothman & Myers, 1996a). In the LBM for instance, up to 85% reduction in fecundity has been reported during population decline (Baltensweiler, 1977). Fluctuations in fecundity have also been reported in the AM (Klemola et al. 2008), the GM (Páez et al., 2015) and the WTC (Myers & Cory, 2013). In

the WTC, fecundity begins to decline prior to the peak in population size (Myers & Cory, 2016). During the crash phase, fecundity continues to decline and there is a lag in population recovery. While NPV may be an important factor in instigating the initial population decline, a sustained decline in fecundity might act via a time-delay to sustain the population crash (Myers & Cory, 2013). Life-history trade-offs between survival and fecundity in the context of pathogen challenge is certainly well-documented in insects (Myers et al., 2000; Dubovskiy et al., 2013; Schwenke et al., 2016). Provided that there exists a negative genetic correlation between survival and reproductive resource allocation, then at low pathogen density, selection might favour high reproductive output, whereas under high pathogen pressure, selection might favour resistance (Schwenke et al., 2016). Given the maintenance of rapid, fluctuating selective pressure and the negative correlation between immunity and fecundity, it is possible that this mechanism contributes significantly to long-term population cycles (Elderd, 2013).

Three explanations to explain the reduction in fecundity of forest Lepidoptera are: (a) food limitation; (b) costs of sublethal infection; and (c) a cost of resistance to NPV infection (Myers & Cory, 2009; Sarfraz et al., 2013). In the WTC system for instance, experimental evidence relates both population density and NPV exposure to reduced fecundity (Rothman, 1997). This is also a broader trend in many Lepidoptera that survive baculovirus challenge (Goulson & Gory, 1995; Rothman & Myers, 1996a; Myers et al., 2000; Burden et al., 2003). No evidence supports hypothesis (c), as larvae from smaller egg batches do not appear to be more resistant to NPV (Rothman & Myers, 1996b; Cory & Myers, 2009). Sublethal infection in WTC is supported by field data (Myers & Cory, 2016). In GM, female moths surviving NPV challenge have also been shown to exhibit reduced fecundity, with virus exposure causing females to be identical in pupal weight to males (Páez et al., 2015). Thus, it appears as though reductions in fecundity are either a product of food limitation and/or sublethal infection. In either case, fecundity is influenced by some density-dependent mechanism.

Changes in seasonal weather patterns can have immediate effects on survival of forest Lepidoptera (through sudden freeze events for example). Climate however, has the ability to alter dynamics over extensive periods of time through quality and availability of food, interactions with natural enemies, survival and range expansion (Bylund, 1999; Logan et

al., 2003; Kausrud et al., 2008; Murdock et al., 2013). In the Swiss Alps for example, climate warming recorded throughout the 1980s resulted in a temperature-mediated shift in the optimal elevation for LBM population growth, resulting in a change of distributional limit for the species (Johnson et al., 2010). This distribution contraction led to a dampening of the 8-10-year population cycles that have been characteristic of the population for 1,200 years (Esper et al., 2007). Climate warming and increased variability in seasonal weather patterns are also thought to contribute to the decoupling of forest Lepidoptera with their natural enemies, particularly specialist parasitoids, leading to more dramatic outbreaks (Hance, 2007). Where range shifts are occurring as a result of climate change, many outbreak species are expanding into areas where natural enemies are not present in the same order of magnitude as their previous environment (Stireman 3rd et al., 2005). If these natural enemies are not able to track their hosts range shift, this could result in altered dynamics for both populations.

The effects of climate change on range expansion and weakening or decoupling of predator-prey interactions are fairly intuitive for forest insects, however, the impact of climate on host-pathogen interactions is less understood. Previous research on the impact of temperature has mostly concerned how a warmer climate will benefit the outbreak status of pest populations (Robinet & Roques, 2010), while interactions with pathogens have received less attention. Where host-pathogen interactions in the context of insects and climate warming have been discussed, it is believed that warmer temperatures have the potential to increase transmission rates between susceptible hosts, reduce the heterogeneity of exposure risk within populations and enhance intergenerational pathogen viability (Bale et al., 2002; Harvell et al., 2002, Ghosh & Bhattacharyya, 2007; Elderd & Reilly, 2013). Generally, this suggests both an increase in the rate at which disease moves through a population and overall disease prevalence (Lafferty & Holt, 2003; Lafferty, 2009).

WTC populations in southwestern BC have been monitored on an annual basis by Myers and Cory since 1975 (Myers & Cory, 2016). The dataset resulting from their efforts is ideal for evaluating the contributions of (and interactions between) intrinsic and extrinsic factors toward the dynamics of these populations, as it contains annual measures of average fecundity, NPV disease prevalence, tent size (an estimate of late instar survival – see

Myers, 2000), population size and growth. Previously (Chapter 2), I have shown that lagged population size ($t-1$) is strongly, negatively correlated with the annual growth of these populations and NPV infection prevalence is best predicted by the previous year's population size ($t-1$) as well. NPV responds positively to increases in population size and is strongly associated with population decline (Myers & Cory, 2013). Additionally, fecundity is likely to be an important factor in prolonging population decline through costs sublethal NPV infection.

As an ectotherm, the physiology of WTC is tightly coupled to the temperature of the surrounding environment. As climate change continues this has long-term consequences on the population dynamics of this species, but immediate effects through seasonal temperatures can also be observed; this is because temperature affects growth, development and survival (Frid & Myers, 2002). Temperature may also have indirect effects, especially in the context of NPV infection. Speed of kill and viral yield per host for instance can be affected by temperature (Frid & Myers, 2002; Ghosh & Bhattacharyya, 2007). Thus, temperature, both directly and indirectly, can alter the annual growth of WTC populations.

In this study, I use demographic data from five field populations of WTC (described previously in Chapter 2), along with hourly temperature data over the course of larval development to answer the following questions: (1) do fluctuations in WTC fecundity impact population dynamics? (2) what is the relative importance of tent size, population density, NPV infection prevalence, fecundity and spring temperatures with respect to WTC population rate of increase? (3) does a model containing the most important factors (evaluated through relative contributions to population rate of increase and relative importance values) better match the field population dynamics than a host-pathogen only model? I expect that reductions in fecundity, due to either costs of resistance to NPV infection or sublethal infection, will negatively impact WTC population rate of increase. I also expect that NPV infection prevalence and population size will have the highest relative importance in relation to population rate of increase, as viral epizootics are a major mortality factor in this system, are density-dependent and are commonly associated with substantial reductions in population size.

3.2. Materials and Methods

3.2.1. Field populations and long-term monitoring

For a complete description of field sites and monitoring/sampling techniques used, please refer to Chapter 2 Materials and Methods (2.2.2.).

3.2.2. Statistical analyses

Part I: AIC model selection

Akaike's information criteria (AIC) were used to investigate the influence of annual measures of: Population size (lagged and current), NPV infection prevalence, spring temperature, fecundity and tent size on the population rate of increase in five WTC populations (described in Chapter 2). The AIC model selection process is an information theoretic (IT) approach that begins with a global model from which all possible model sets are derived. The goal of this approach is to select the optimal set of independent parameters that best explain the variation in the response variable (Grueber et al., 2011). The IT approach has numerous advantages over traditional null hypothesis testing. In contrast to null hypothesis testing, the IT approach investigates several competing hypotheses simultaneously, making inferences based on weighted support from several models (Grueber et al., 2011). Candidate models are then ranked and weighted according to AIC in order to provide support for each hypothesis. Due to the large number of candidate models usually generated by this process (this is dependent however, on the number of independent variables used), a 'top model set' (95% confidence set) was generated as a means to provide parameter and model uncertainty estimates (Burnham & Anderson, 2002; R package 'MuMIn': Barton 2013). This is advantageous over stepwise selection processes, which do not provide measures of model uncertainty.

All field populations (described in Chapter 2) were used in the analysis. Years for which one or more independent parameters were missing were excluded. Model structure consisted of a linear mixed effects model with site held as a random intercept. Independent

parameters were assessed for normality using a Shapiro-Wilk test on model residuals. Population size data were log-transformed and NPV infection values of zero were removed in order to meet the assumptions of normality. Independent parameters used in the model selection process are described below (see Model Parameters).

Significance of model parameters were assessed by whether they appeared in models that fell within the 95% confidence sets of the model averaging process (ΔAICc<2; Burnham & Anderson, 2002), had a significantly high summed Akaike weight (>0.5) across all models, and parameter estimates for which confidence intervals did not cross zero. To allow comparison between model parameter estimates, the mean was subtracted from each parameter and subsequently divided by 2 standard deviations (i.e. centering and scaling) using the scale function provided in the base package of R (R Core Team, 2015). Collinearity between parameters was checked by calculating variance inflation factors, using the vif function provided in the 'car' package (R Core Team, 2015). All vif were <3, suggesting that estimates of model parameters are sufficiently independent.

Model parameters

Spring temperature

Hourly temperature data were downloaded from the Canadian Climate Data website (http://climate.weather.gc.ca/historical_data/search_historic_data_e.html). Since weather often varies between Mainland and Gulf Island locations, temperature data were taken from two independent stations: Vancouver International Airport (used as a proxy for Cypress and Westham temperatures) and Victoria International Airport (used as a proxy for Mandarte, Galiano and Saturna temperatures). In a preliminary analysis, I used monthly average temperature data collected from Environment Canada weather stations (Vancouver International Airport and Victoria International Airport respectively) to determine which time-range of monthly temperatures had the strongest effect on WTC annual rate of population growth. The results (unpublished data) of this analysis, indicated that March-April-May temperatures (the period of larval emergence and development) had the strongest numerical effect on population rate of increase and this relationship was linear and negatively correlated. The range of temperature data was restricted over the period of egg hatch and larval development (March-April-May). I explored various representations of temperature, including daily and monthly averages and warming and

cooling degree days. In the end, I chose hourly temperature data in order to maximize the resolution of the analysis. The summed total of degree days accumulated above and below a twenty year (1995-2015) historical average were calculated for each hour and then summed over the larval period of development. This yielded two measurements for each year: the summed total of degree days spent above the historical average and the summed total degree days spent below the historical average. The twenty-year historical average for Vancouver International Airport is 6.9°C (March), 9.4°C (April), 12.8°C (May). The twenty-year historical average for Victoria International Airport is 6.8°C (March), 9.0°C (April), 12.1°C (May). This calculation allowed me to differentiate between "warmer" and "cooler" springs. An example of this procedure is detailed below:

Step 1: Calculate the summed total of degree days accumulated above historical threshold (hourly):

March, 2015	1435.5
April, 2015	813.5
May, 2015	1794.1
TOTAL	4043.1

Step 2: Calculate the summed total of degree days accumulated below historical threshold (hourly):

March, 2015	158.2
April, 2015	970.1
May, 2015	247.1
TOTAL	1375.4

Step 3: Calculate the ratio of total degree days accumulated above the historical threshold to total number of degree days (both above and below the historical threshold):

$$\frac{4043.1}{4043.1 + 1375.4} = \frac{4043.1}{5418.5} = 0.75$$

Step 4: Calculate the ratio of total degree days accumulated below the historical threshold to total number of degree days (both above and below the historical threshold):

$$\frac{1375.4}{4043.1 + 1375.4} = \frac{1375.4}{5418.5} = 0.25$$

The degree day data from Step 3 and Step 4 were used as continuous variables (ranging from 0.00-1.00) when I conducted AIC model selection, but the data were then converted to a categorical factor ("warmer spring" vs. "cooler spring") when I constructed my model in Part II (below). Warmer springs were defined as those years for which the ratio calculated in Step 3 was greater than 0.5. Cooler springs were defined as those years for which the ratio calculated in Step 4 was greater than 0.5.

The lower developmental threshold for the WTC is estimated at ~12°C (extrapolated from Figure 3.b., Frid & Myers, 2002). However, since larvae can elevate body temperature well above ambient temperature when exposed to direct sunlight (see Figure 2., Frid & Myers, 2002), the ambient temperature that reflects this lower developmental threshold is likely to be lower (possibly between 9.0-10.0°C based on Figure2., Frid & Myers, 2002). The majority of larval development occurs over the period of April-May, so this estimate of lower developmental threshold closely reflects the average ambient temperatures experienced over this period.

Fecundity

Each female moth lays one egg mass. By collecting egg masses following larval emergence and counting the number of eggs per egg mass, an average measure of fecundity can be obtained for the moths that developed and laid eggs the year before the hatched egg masses were collected.

Population size

The number of tents constructed by families of late instar larvae serves as an estimate of population size. As there is evidence for a delayed density-dependent effect (see Figure 2.10. Chapter 2), both current and previous year's population size were used as independent parameters in the model selection process.

NPV infection

Subsamples of 5-10 larvae from individual tents were reared in the lab to determine infection status. Infection is assessed on a per site basis and reflects the number of tents whose subsamples contained NPV infected larvae. This is expressed as the percentage of tents containing infection out of the total number of tents sampled. Thus, this percentage serves as an estimate for the level or prevalence of NPV infection in the field each year. As symptoms of NPV infection are obvious (liquefaction of host's tissues) larvae were assessed visually for infection status and tested microscopically only if symptoms were questionable.

Tent size

Each year, the length and width (cm) of tents is measured using a standard metric ruler. Average tent size is calculated from these measurements for each population. Tent size varies from year to year and is used (in conjunction with average fecundity) to calculate the estimate of larval survival to late instars (Myers, 2000). As tent size might correlate with survival probability, it is included in the analysis.

Aside: Parasitism

Although the WTC dataset does contain measures of larval parasitism, the collection of data on parasitoid prevalence only begins in the year 2000. For the purpose of maximizing the resolution of the AIC analysis (and not limiting sample size) I decided not to include this parameter in the model selection process. Parasitism does not appear to have a significant correlation with population rate of increase (R^2 = -0.001, p-value = 0.342) and is likely to prolong the lag phase of population recovery by preventing population growth (see Appendix A).

Part II: Model construction and testing

Using the top ranked model parameters from the AIC selection process, two discrete models were constructed based on observable relationships between parameters for all sites. As with previous model construction, data from Galiano and Saturna Islands were used to fit model parameters. This process excluded Mandarte Island, as time-series data

from this site were later used to test model fit. These models are described as the: (1) NPV-fecundity model; (2) Temperature-NPV-fecundity model.

NPV-fecundity model

This model uses the observed relationship between NPV infection and subsequent reduction in fecundity of moths surviving in the generation. Model structure is based on the following linked functions:

$$I_t = \frac{100}{1+e^{(-2.26Log_{10}(N_{t-1})+5.205)}} \quad (1)$$

$$F_t = c(I_{t-1}) + d \quad (2)$$

$$R = g(I_t) + h(F_t) + j \quad (3)$$

$$N_{t+1} = 10^R N_t \quad (4)$$

where I_t is a logistic function that represents percent of NPV families at time t, and I_{t-1} represents percent of NPV families at time t-1; F_t is a linear function that represents the average fecundity of the population at time t; c, the disease-dependent scaling coefficient for average fecundity (i.e. sub-lethal effects); d, the disease-free average fecundity; R is a linear function that represents annual population rate of increase; g, the disease-dependent scaling coefficient for population rate of increase; h, the fecundity-dependent scaling coefficient for population rate of increase and j, the disease-free annual population rate of increase. Fecundity was made a function of the level of NPV infection prevalence in the previous generation as this linear model had a significantly lower AIC score in comparison to a model with current level of infection prevalence (ΔAIC = +45.89, Table 3.6.).

Temperature-NPV-fecundity model

The temperature-NPV-fecundity model differentiates between the effect of NPV infection on the population rate of increase in warmer versus cooler years. This model is essentially the same as the NPV-fecundity model, but contains two linear functions describing population rate of increase These linear functions differ in their intercept values according to the proportion of degree days (either >0.5 or <0.5) spent above the historical average during larval development. The model is described by the following functions:

$$I_t = \frac{100}{1+e^{(-2.26Log_{10}(N_{t-1})+5.205)}} \quad (5)$$

$$F_t = c(I_{t-1}) + d \quad (6)$$

$$R_{COLD} = g(I_t) + h(F_t) + j \quad (7)$$

$$R_{WARM} = g(I_t) + h(F_t) + k \quad (8)$$

$$N_{t+1} = 10^R N_t \quad (9)$$

where I_t is a logistic function that represents percent of NPV families at time t, and I_{t-1} represents percent of NPV families at time t-1; F_t is a linear function that represents the average fecundity of the population at time t; c, the disease-dependent scaling coefficient for average fecundity (i.e. sub-lethal effects); d, the disease-free average fecundity; R is a linear function that represents annual population rate of increase; g, the disease-dependent scaling coefficient for population rate of increase; h, the fecundity-dependent scaling coefficient for population rate of increase and j and k, the disease-free annual population rate of increase in cold and warm springs respectively. Fecundity was made a function of the level of NPV infection prevalence in the previous generation as this linear model had a significantly lower AIC score in comparison to a model with current level of infection prevalence (ΔAIC = +45.89, Table 3.6.).

Cross-correlation (ccf function, stats package R version 3.012) was used to assess the correspondence of simulated data with population time-series data from Mandarte Island for which the longest time series is available. As a majority of sites contain time-series for three complete cycles, the time period of simulations was restricted over the previous 23 years (1992-2015). Cross-correlation is a measure of similarity between two time-series as a function of one lagged time series relative to another. Correlation is measured as the convolution between the two time-series and ranges from 0 (no correlation) to 1 (maximum correlation). As Mandarte is excluded from model parameterization, this site provides an unbiased measure of model fit to population data. Cross-correlation values were compared to that of the null/infection-only model (see Chapter 2) so as to select the best fitting model.

3.3. Results

Part I: AIC model selection

After examining the relative contributions of population abundance (current and lagged), NPV infection prevalence, spring temperature, fecundity and tent size on population rate of increase, the most strongly supported models contained all parameters, with the exception of current population size and tent size (Table 3.1). The model with the lowest AIC score contained lagged population size, NPV infection and fecundity (Table 3.2) and had an R^2 value of 0.67. Model dredging yielded 48 candidate models out of the total of 127 possible model combinations. A subset of five of these models was chosen based on their summed Akaike weight of 0.95 (the 95% model confidence set). Models contained in the 95% confidence set were then used in the model averaging procedure. Fecundity (1.00), lagged population density (1.00) and NPV infection (0.62) had the highest relative importance values, followed by the proportion of degree days above the historical average (0.44) and tent size (0.08). Fecundity had positive effects on population rate of increase, whereas lagged population size, NPV infection and the proportion of degree days above the historical average were all negatively correlated with population rate of increase (Figures 3.1 and 3.2, Table 3.3). Overall, lagged population size, and fecundity had the strongest effects on population rate of increase (Table 3.2) and models lacking either of these terms had significantly higher (>13) ΔAICc values.

Part II: Model construction and testing

NPV-fecundity model

Parameter estimates for the NPV-fecundity model are listed in Tables 3.7 and 3.8. Estimated model parameters are shown below:

$$I_t = \frac{100}{1+e^{(-2.26Log_{10}(N_{t-1})+5.205)}} \quad (10)$$

$$F_t = 0.348(I_{t-1}) + 213.454 \quad (11)$$

$$R = -0.015(I_t) + 0.021(F_t) - 3.773 \quad (12)$$

$$N_{t+1} = 10^R N_t \quad (13)$$

The composite model is represented by the following density-dependent function:

$$N_{t+1} = 10^{-0.015m(N_{t-1})+0.007m(N_{t-2})+0.710} N_t \quad (14)$$

where m represents the logistic function represented by equation (10).

Simulation experiments generated from iterations of equations (10)-(13) yielded stable multi-annual limit cycles with 7 year periodicity (compared with 7-to-12 year periodicity for Mandarte Island) (Figure 3.4). As this model did not contain stochastic or extrinsic variables, cycle amplitude and frequency did not vary. Long-term host population amplitude ranged from 10 to 545 tents (compared with 1 to 1131 tents for Mandarte Island). NPV infection prevalence ranged from 6 to 73 percent (compared with 0 to 73 percent for Mandarte Island). Fecundity ranged from 188 to 211 eggs (compared with 144 to 224 eggs for Mandarte Island). Cross-correlation between simulated data and Mandarte Island data surpassed Bartlett's criterion (Figure 3.6) and the maximum cross-correlation coefficient was 0.78 (occurring at lag -1). This is significantly greater than the cross-correlation coefficient of the null model (0.28), which did not surpass Bartlett's criterion.

Temperature-NPV-fecundity model

Parameter estimates for the temperature-NPV-fecundity model are listed Tables 3.7 and 3.9. Estimated model parameters are shown below:

$$I_t = \frac{100}{1+e^{(-2.26Log_{10}(N_{t-1})+5.205)}} \quad (15)$$

$$F_t = 0.348(I_{t-1}) + 213.454 \quad (16)$$

$$R_{COLD} = -0.013(I_t) + 0.019(F_t) - 3.339 \quad (17)$$

$$R_{WARM} = -0.013(I_t) + 0.019(F_t) - 3.677 \quad (18)$$

$$N_{t+1} = 10^R N_t \quad (19)$$

The composite model is represented by the following piecewise function:

$$N_{t+1} = \begin{cases} 10^{-0.013m(N_{t-1})+0.007m(N_{t-2})+0.717} N_t & \text{if } R_{COLD} \\ 10^{-0.013m(N_{t-1})+0.007m(N_{t-2})+0.379} N_t & \text{if } R_{WARM} \end{cases} \quad (20)$$

where m represents logistic function represented by equation (15).

Simulation experiments (Figure 3.5) generated from iterations of equations (14)-(18) yielded stable multi-annual cycles with 6-8 year periodicity (compared with 7 to 12 year periodicity for Mandarte). Long-term host amplitude ranged from 3 to 975 tents (compared with 1 to 1131 tents for Mandarte). NPV infection prevalence ranged from 4 to 82 percent (compared with 0 to 73 percent for Mandarte). Fecundity ranged from 185 to 212 eggs (compared with 144 to 224 eggs for Mandarte). Cross-correlation between simulated data and Mandarte data surpassed Bartlett's criterion (Figure 3.6) and the maximum cross-correlation coefficient was 0.63 (occurring at lag -1). This is also significantly greater than the cross-correlation coefficient of the null model (0.28), but less than the NPV-fecundity model (0.78).

3.4. Discussion

Part I: AIC model selection

A major problem in ecology is explaining why populations fluctuate (Berryman & Turchin, 2001). For forest insects whose populations undergo regular periodic cycling, numerous explanations have been proposed and tested extensively using first-principles based models and experiments. These methods of hypothesis testing have been useful in demonstrating how cyclical dynamics can be produced by a number of factors (Kendall et al., 1999). The relative importance of specific factors however, can be overemphasized, particularly when a model is constructed and fitted around a single mechanism (Bowers, 1993; Kendall et al., 2005). In GM populations for instance, the role of NPV is often emphasized in population models, but the roles of food limitation and the fungal pathogen *Entomophaga mamaiga* are frequently overlooked or underrepresented even though they are known to be important factors in the outbreak of this species (Elderd et al., 2008). Although life-table studies can be time-consuming and thus difficult to conduct for outbreaking species of forest insects, having measures of how specific factors contribute to vital rates within populations allows for the construction of models around relevant

factors rather than extraneous ones. A recent summary of time-series for outbreaking defoliators in Canadian forests highlights the rarity of life-table studies, with only 12 studies for seven defoliator species reported out of a known 106 species (Johns et al., 2016). Time-series data for a majority of Canadian defoliator data contains only measures of defoliation or tree ring width (see Table 2, Johns et al., 2016), also making it difficult to speculate on the importance of various factors.

Through AIC model selection I have identified the relative contributions and importance of both demographic and environmental factors towards the population rate of increase for WTC populations in southwestern BC. These variables (in order of relative importance) are: fecundity, lagged population size, NPV infection prevalence and the proportion of degree days above the historical average. The fact that the best supported model contained fecundity, lagged population size, and NPV infection prevalence provides support for the lagged population model I constructed previously (see Chapter 2), but also suggests the importance of fecundity. As there is a negative relationship between NPV infection and fecundity (Table 3.7), these results could be interpreted in terms of the disease defense hypothesis (DDH), where under peak population, viral epizootics will select for resistant individuals and there is a trade-off between resistance and fecundity. However, it is difficult to discern whether reduced fecundity is a direct result of the costs of evolved resistance to virus or whether reduced fecundity is simply a sub-lethal effect of surviving virus challenge. The disease defence hypothesis requires (i) the existence of genetic variation for virus resistance in populations; (ii) a trade-off between resistance and fecundity; and (iii) that the frequency of disease resistance will increase following an epizootic along with a reduction in fecundity (Cory & Myers, 2009). Cory and Myers (2009) found that while there is evidence for variation in resistance to NPV infection in the WTC, there was no relationship between the size of the egg mass from which larvae hatched and their susceptibility to virus. This may suggest that the relationship between NPV infection prevalence and fecundity could be the result of sub-lethal viral infection. Alternatively, since the moths for which fecundity is measured developed in the “lagged population” (N_{t-1}), they were exposed to that population size and infection level. My results show that fecundity, lagged population size and NPV infection prevalence are key factors in the population dynamics of WTC.

In addition to demographic factors, I have also shown that seasonal temperature has the potential to alter WTC population dynamics. The proportion of degree days spent above the historical average during larval development has a negative linear effect on the annual growth rate of WTC populations in southwestern BC. A plausible hypothesis is that warmer temperatures favour the performance of natural enemies of the WTC, resulting in increased mortality. This is supported by my findings, which suggest an additive effect of warmer springs and NPV infection prevalence that contributes to a decrease in annual population rate of increase (Table 3.4, Figure 3.3). The possible ways in which temperature might alter virus dynamics is by altering host time-to-death, virus yield-per-host and/or risk of infection.

Warm temperatures have been shown to decrease time-to-death in NPV infected larvae, increase the kinetic potential of virus replication, as well as increase the theoretical yield of viral inoculum (van Beek et al., 2000; Frid & Myers, 2002; Subramanian et al., 2006; Ghosh & Bhattacharyya, 2007). Frid & Myers (2002) found that both incubation time and theoretical yield of NPV in the WTC were sensitive to temperature variation in laboratory experiments. Over the range of 18-24°C, incubation time (determined by time-to-death of host) of the virus decreased by six days (14 to 8 days) and theoretical virus yield increased slightly. In a similar experiment Subramanian (2006) found that NPV production in *Spodoptera lituria* was enhanced by 6.623×10^{11} PIB (yield/100 inoculated larvae) when reared at 25°C versus room temperature. Additionally, over the range of 25-35°C, the mean incubation time of the virus decreased by three days. A similar trend between temperature and incubation time was observed in experiments infecting neonate (3-6h) cabbage loopers *Trichoplusia ni* with AcMNPV over constant ambient temperatures ranging from 14-29°C. This temperature differential resulted in an exponential decline in median incubation time from 15 (14°C) to 3 (29°C) days (van Beek et al., 2000). While these experiments were carried out on individual larvae, the collective effect of a temperature-mediated decrease in incubation time could allow for further rounds of transmission within populations (Rothman, 1997). However, temperature also alters the growth and development rates of insects and the ratio between these rates determines adult body size (Frid & Myers, 2002). Faster development rates at warmer temperatures could then result in reduced host body size and subsequently a smaller viral yield, which could reduce the probability of subsequent transmission or transgenerational viability of

virus (see for example Takahashi et al., 2015). Faster development rates could also allow larvae to escape infection via developmental resistance (Hoover et al., 2002). This effect is best observed in a time-to-death experiment with velvetbean caterpillar *Anticarsia gemmatalis* and its associated NPV (Silva & Elliot, 2016). Here, 4th instar larvae inoculated with NPV were reared at temperatures of 20, 24, 28 and 32 °C. Time-to-death decreased with warmer temperatures (from *ca*. 11 to 5 days over the range of temperatures) but total mortality also decreased from lower to higher temperatures as larvae developed faster and "escaped" infection under warmer temperatures. Results from these experiments indicate an apparent trade-off between rounds of transmission, virus production and host resistance in the context of warmer temperatures. This would imply the existence of an optimal temperature range at which transmission is maximized.

Larval host behaviour is another factor that can influence the risk of viral infection, as variation in behaviour can alter the frequency or probability of pathogen exposure (Parker et al., 2010; Eakin et al., 2014). Behaviour may in turn be influenced by warmer temperatures, particularly with regard to the rate at which individual larva feed and move (Frears et al., 1999; Parker et al., 2010; Silva & Elliot, 2016). Thus, warmer temperatures, acting through host behaviour, have the potential to alter host-pathogen dynamics. A transmission experiment using the fall armyworm (FAW) *Spodoptera frugiperda* and its NPV, for instance, demonstrated the potential for higher temperatures to increase disease transmission and outbreak intensity (Elderd & Reilly, 2013). In this experiment, temperature and virus density were manipulated inside of open top chambers containing soybean plants. Healthy larvae were introduced to the chambers, allowed to feed, and were then removed to assess the number of newly infected larvae. Although there was no appreciable change in mean transmission rate with increasing temperatures, there was a decrease in the coefficient of variation (CV) associated with transmission as larva increased their feeding rate. This decrease in CV was the result of a decrease in heterogeneity in disease risk amongst individual larvae, leading to an increase in outbreak intensity. While FAW and WTC are different in some respects (with regard to voltinism and gregarious behaviour for instance), warmer temperatures have a similar quantitative effect on WTC larval feeding rates (unpublished data). Given the correct temperature range, WTC feeding rates could be optimized to maximize exposure risk. At later instars, where the majority of feeding occurs for WTC, NPV exposure risk would be at its highest,

provided inoculum is present in the surrounding feeding environment. This would also be beneficial in the context of virus yield; as larger instar caterpillars produce more inoculum per cadaver (Shapiro et al., 1986; Ebling & Kaupp, 1998). This of course must be weighed against the potential increase in host resistance that is a characteristic quality of late instar larvae (Hochberg, 1991; Fuxa, 2004). Collectively, this suggests the existence of an optimal temperature range for which exposure risk and viral yield is maximized, while negative effects of increased host resistance are minimized. Provided that incubation time is also reduced under such a temperature range, this could result in additional transmission cycles within a season of larval development (Rothman, 1997). Secondary (or higher) transmission cycles could yield a substantial increase in the number of infected larvae and dramatically increase the quantity of inoculum produced (Frid & Myers, 2002). This would result in reduced population growth and could potentially have negative implications for the proceeding larval generation if viral persistence remains high.

Another possibility is that warmer temperatures interact with factors that have not been directly measured, such as predation or other pathogens. Generalist predators for instance, are known to feed on forest Lepidoptera, but mostly at low prey densities (Myers & Cory, 2013). If warmer temperatures favour the performance of generalist predators, then this could prolong the lag or recovery phase between population peaks. However, the support for the hypothesis that predators maintain the lag phase of population cycles in forest Lepidoptera is weak (Elkinton et al. 2004, Dwyer et al. 2004). Pathogens other than NPV are known to infect forest Lepidoptera and may also be affected by warmer temperatures (van Frankenhuyzen & Nystrom, 1987; Siegert et al., 2009). However, other than the fungal pathogen *Entomophaga maimaiga* that is associated with GM populations, pathogens other than baculoviruses are not frequently implicated in the natural occurrence of epizootics in forest Lepidoptera (Fuxa, 2004). If other pathogens (particularly those which are less readily identified than NPV) are to be implicated in the population dynamics of forest Lepidoptera, then probing of the larger pathogen community needs to be undertaken. With molecular techniques such as PCR and second-generation sequencing, this should be possible (Myers & Cory, 2013).

Part II: Model construction and testing

I have shown that models incorporating both the effects of fecundity (either through costs of resistance to virus challenge or through sub-lethal effects) and temperature are capable of generating cyclical dynamics and these models outperform a host-pathogen-only (null) model with respect to fit to time-series data for Mandarte island. These results agree with findings from AIC model selection (Part I), which strongly implicate the importance of these variables in the population rate of increase. In WTC, NPV infection has been shown to have both immediate and delayed effects on host populations, particularly at high host densities (Rothman, 1997). Immediate effects occur through virus-induced mortality, while delayed effects occur through a reduction in host reproductive potential (Myers & Kukan, 1995; Rothman, 1997). The models I have constructed demonstrate the initial dramatic population decline that is characteristic of the immediate effects of viral-induced mortality and also capture (at least in part) the prolonged "trough" phase that is sustained by reduced fecundity.

To my knowledge, this is the first time temperature has been linked deterministically to demographic parameters in a discrete-time baculovirus-host population model. It is common practice to account for the effects of exogenous or unmeasured processes by including a stochastic term in population models (Kendell et al. 2005; Berryman & Turchin, 2001). Stochastic variables amalgamate the effects of these processes into a single "noise" term that produces multiplicative lognormal perturbations to population densities. These terms allow such models to maintain cyclical behaviour, whereas without their effect dynamics tend to converge toward a steady-state equilibrium and cycles disappear (Ims et al., 2007). My model however, utilizes actual observed relationships between springtime temperature and population rate of increase; and while temperature is itself stochastic by nature, its effects in the model structure are deterministic. This measureable relationship is particularly valuable if one is wishing to consider how cyclical populations like the WTC will behave under certain climate change scenarios. Considering the fact that warmer springs seem to have a deleterious effect on population rate of increase in WTC, this may slow the rate of increase of these populations if the frequency of warmer springs increases in future years. Additionally, under high levels of NPV infection, it is likely that warmer springs could produce more dramatic crash phases of these population cycles.

By the end of the century, IPCC projections of climate change estimate an increase in global surface warming in the range of 1.8-4.0°C (Meehl et al., 2007). Furthermore, for BC, the average total number of degree days (>5°C) accumulated throughout March-May over the period of 1981-2010 was 466; by 2025 this number is estimated to be 576 (Wang et al., 2012: ClimateWNA-UBC Forestry, model CanESM2_rcp45_2025). This amounts to an increase of 110 degree days. Such warming over a relatively short period of time will likely have negative implications for WTC annual population rate of increase. Given that I have constructed a model that contains relationships between the growth of WTC populations and temperature, it should be possible to modify the current model structure and run simulations under potential future climate scenarios to estimate the magnitude of this effect on the dynamics of these populations. Future work should focus on the mechanistic underpinnings of how warmer springtime temperatures affect NPV infection dynamics in this system. In particular, focus should be directed toward the various temperature-mediated trade-offs between virus yield, incubation time (time-to-death), individual exposure risk, and developmental resistance. This would allow one to estimate the optimal temperature range required for maximal outbreak intensity, which could further refine the way temperature is represented in the current model structure.

Models of forest insect population dynamics are almost always built using *a priori* assumptions, which are then measured against time-series data to reveal whether they fit sufficiently (Dwyer et al., 2000). An issue with this method is that cyclical population dynamics can be generated by a number of mechanisms, especially where a density-dependent time-lag or delay is involved (Ginzburg & Taneyhill, 1994; Briggs & Godfray, 1995; Kendall et al., 2005). Given the appropriate parameter values (and assuming these values are biologically plausible) many mechanistic models can be fit to time-series data, but this does not validate their underlying assumptions or mechanisms. The model I have constructed is unique in the sense that it does not rely on *a priori* assumptions or parameter values that are estimated from experiments. Instead, I use actual relationships between fecundity, population abundance, disease prevalence and seasonal temperatures from long-term field data to construct a model and determine whether the relationships can produce population cycles. Another unique component to this study is that I also ensure the relative importance of specific factors toward population rate of increase before constructing my model. This provides the necessary justification for their

inclusion in the model structure. This research further supports the role of baculoviruses in contributing to the cyclical population dynamics of forest Lepidoptera but also emphasizes the impact of spring seasonal temperatures. As temperatures influence host-pathogen dynamics in forest Lepidoptera, further work is needed to predict how climate change will alter these population cycles.

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Tables

Table 3.1. Linear model parameter estimates for predictors of annual population rate of increase in western tent caterpillar field populations located in southwestern British Columbia. Sites included were: Mandarte Island, Galiano Island, Saturna Island, Westham Island and Cypress Mountain. Annual time-series data for the period of 1975-2015 were used. Parameter values have been centered and scaled to allow for comparison. Estimates represent the average of parameter values over the 95% model confidence set.

Parameter	Estimate	Std. Error	t value	Pr(>|t|)
(Intercept)	-0.05609	0.07005	0.789	0.42997
Fecundity	0.28247	0.05805	4.804	1.6e-06***
% Infection	-0.16066	0.05953	2.662	0.00777**
N_{t-1}	-0.38980	0.07053	5.471	2.0e-16***
Degree Days > Hist. Avg.	-0.14134	0.05640	2.471	0.01347*
Tent Size	0.09694	0.05143	1.858	0.06323.

Signif. codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

Table 3.2. Linear models with environmental and demographic factors as predictors of annual population rate of increase in western tent caterpillar field populations located in southwestern British Columbia. Sites included were: Mandarte Island, Galiano Island, Saturna Island, Westham Island and Cypress Mountain. Annual time-series data for the period of 1975-2015 were used. Model dredging yielded 48 candidate models and 5 models were chosen as the top model set based on delta AIC scores (models with ∆AIC<5) and cumulative weight (cumulative summed weight of 0.95).

Term Codes:

1 = (DD>Hist.Avg.) **2 =** (Fecundity) **3 =** (%Infection) **4 =** (N_{t-1}) **5 =** (Tent Size)

Model Terms	df	logLik	AICc	delta	weight
2-3-4	6	-62.22	137.45	0.00	0.34
1-2-4	6	-62.62	138.25	0.80	0.23
1-2-3-4	7	-61.54	138.45	0.99	0.21
2-4	5	-64.21	139.13	1.67	0.15
2-3-4-5	7	-62.52	140.40	2.94	0.08

Table 3.3. Linear model parameter estimates for predictors of population rate of increase in western tent caterpillar field populations located in southwestern British Columbia. Sites included were: Mandarte Island, Galiano Island, Saturna Island, Westham Island and Cypress Mountain. Annual time-series data for the period of 1975-2015 were used. Population size data were log transformed (base 10) to ensure normality criteria were met.

Formula: Population Rate of increase ~ %Infection	**Estimate**	**Std. Error**	**t value**	**Pr(>|t|)**
(Intercept)	0.202964	0.158685	1.279	0.206159
%Infection	-0.013388	0.003324	-4.027	0.000171***
Adjusted R-squared	0.2107	p-value		0.0001715
Formula: Population Rate of increase ~ Nt-1	**Estimate**	**Std. Error**	**t value**	**Pr(>|t|)**
(Intercept)	0.93878	0.09870	9.512	< 2e-16***
Nt-1	-0.54953	0.04883	-11.255	< 2e-16***
Adjusted R-squared	0.4712	p-value		< 2e-16
Formula: Population Rate of Increase ~ Fecundity	**Estimate**	**Std. Error**	**t value**	**Pr(>|t|)**
(Intercept)	-2.921769	0.513382	-5.691	7.43e-08***
Fecundity	0.014818	0.002601	5.694	7.33e-08***
Adjusted R-squared	0.1866	p-value		7.332e-08
Formula: Population Rate of Increase ~ Tent Size	**Estimate**	**Std. Error**	**t value**	**Pr(>|t|)**
(Intercept)	-0.786164	0.168658	-4.661	7.33e-06***
Tent Size	0.008359	0.001852	4.513	1.36e-05***
Adjusted R-squared	0.1223	p-value		1.357e-05
Formula: Population Rate of Increase ~ Degree Days > Historical Average	**Estimate**	**Std. Error**	**t value**	**Pr(>|t|)**
(Intercept)	1.2275	0.1943	6.317	3.00e-09***
DD>Hist. Avg.	-2.5652	0.3760	-6.822	2.18e-10***
Adjusted R-squared	0.2353	p-value		2.184e-10
Formula: Population Rate of Increase ~ Degree Days < Historical Average	**Estimate**	**Std. Error**	**t value**	**Pr(>|t|)**
(Intercept)	-1.3377	0.1967	-6.800	2.44e-10***
DD<Hist. Avg.	2.5652	0.3760	6.822	2.18e-10***
Adjusted R-squared	0.2353	p-value		2.184e-10

Table 3.4. Linear model parameter estimates for NPV infection prevalence as a predictor of annual population rate of increase in western tent caterpillar field populations located in southwestern British Columbia. Sites included were: Mandarte Island, Galiano Island, Saturna Island, Westham Island and Cypress Mountain. Annual time-series data for the period of 1975-2015 were used. Infection values of zero have been removed to ensure data are normally distributed. The proportion of degree days has been included as a categorical variable with "warm" years defined as springtime temperatures for which the proportion of degree days spent above the historical average is greater than 0.5.

	Estimate	**Std. Error**	**t value**	**Pr(>|t|)**
(Intercept)	-0.01268	0.16314	-0.078	0.938359
% Infection	-0.01313	0.00287	-4.575	2.99e-05***
as.factor(DDG>Avg.)	0.66956	0.16997	3.939	0.000245***
Adjusted R-squared	0.4357	p-value	1.298e-07	

Signif. codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

Table 3.5. Linear model parameter estimates for fecundity as a predictor of annual population rate of increase in western tent caterpillar field populations located in southwestern British Columbia. Sites included were: Mandarte Island, Galiano Island, Saturna Island, Westham Island and Cypress Mountain. Annual time-series data for the period of 1975-2015 were used. The proportion of degree days has been included as a categorical variable with "warm" years defined as springtime temperatures for which the proportion of degree days spent above the historical average is greater than 0.5.

	Estimate	**Std. Error**	**t value**	**Pr(>|t|)**
(Intercept)	-4.721993	0.834458	-5.659	6.62e-07***
Fecundity	-0.021882	0.004375	5.002	6.84e-06***
as.factor(DDG>Avg.)	0.485555	0.174732	2.779	0.00757**

Signif. codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

Table 3.6. Linear model fitting results for NPV infection prevalence data regressed against fecundity data for western tent caterpillar field populations located in southwestern British Columbia. Sites included were: Galiano Island, Mandarte Island and Saturna Island. Annual time-series data for the period of 1986-2015 were used.

Independent Variable	**Model**	**R^2**	**AIC**	**ΔAIC**
% Infection (I_{t-1})	Linear	0.34	403.93	+0.00
%Infection (I_t)	Linear	0.30	449.83	+45.90

Table 3.7. Linear model parameter estimates for (lagged) NPV ($\boldsymbol{I_{t-1}}$) infection prevalence as a predictor of fecundity for western tent caterpillar field populations located in southwestern British Columbia. Sites included were: Galiano Island, Mandarte Island and Saturna Island. Annual time-series data for the period of 1986-2015 were used.

	Estimate	**Std. Error**	**t value**	**Pr(>|t|)**
(Intercept)	213.45442	4.94163	43.195	< 2e-16***
% Infection (I_{t-1})	-0.34764	0.09789		0.00142**

Signif. codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

Table 3.8. Linear model parameter estimates for fecundity and NPV infection prevalence as predictors of annual population rate of increase for western tent caterpillar field populations located in southwestern British Columbia. Sites included were: Galiano Island and Saturna Island. Annual time-series data for the period of 1986-2015 were used.

	Estimate	**Std. Error**	**t value**	**Pr(>|t|)**
(Intercept)	-3.773207	1.099739	-3.431	0.002098**
Fecundity	0.021302	0.005042	4.225	-0.000277***
% Infection	-0.015124	0.002790	-5.422	1.26e-05***

Signif. codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

Table 3.9. Linear model parameter estimates for fecundity and NPV infection prevalence as predictors of annual population rate of increase for western tent caterpillar field populations located in southwestern British Columbia. Sites included were: Galiano Island and Saturna Island. Annual time-series data for the period of 1986-2015 were used. The proportion of degree days has been included as a categorical variable with "warm" years defined as springtime temperatures for which the proportion of degree days spent above the historical average is greater than 0.5.

	Estimate	**Std. Error**	**t value**	**Pr(>|t|)**
(Intercept)	-3.339365	1.101961	-3.030	0.005773**
Fecundity	0.019502	0.005024	3.882	0.000710***
% Infection	-0.013438	0.002908	-4.620	0.000109***
as.factor(DDG>Avg.)	-0.338288	0.212823	-1.590	0.125028

Signif. codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

Figures

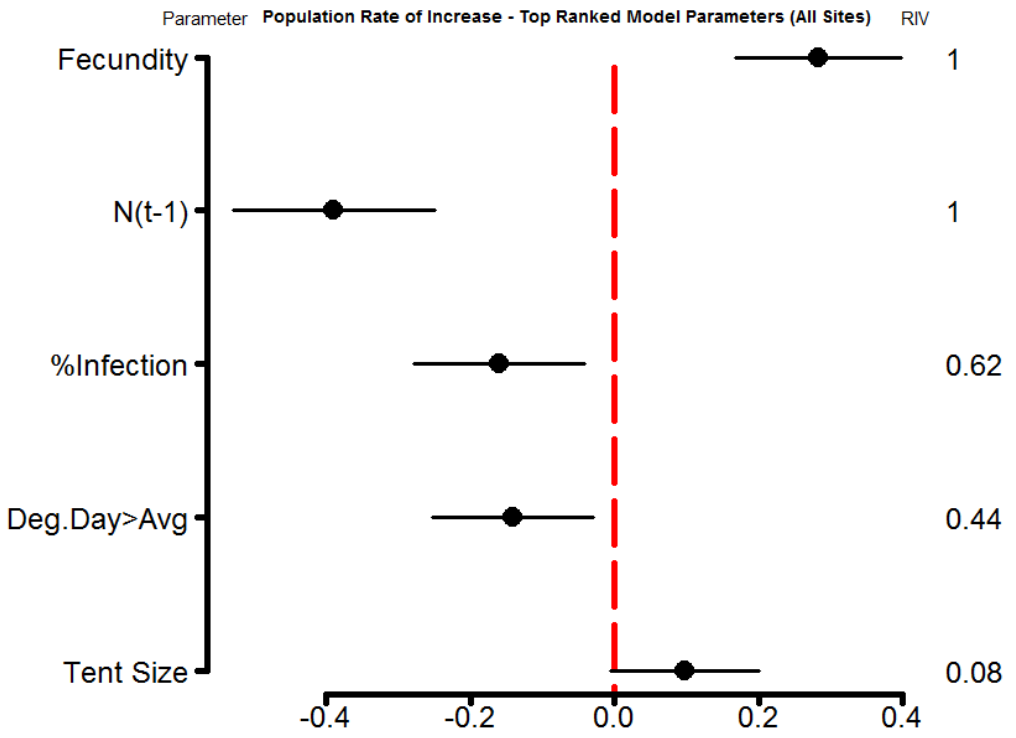


Figure 3.1 Average parameter estimates (scaled and centered) from linear mixed effects models ('site' held as a random intercept) of demographic (fecundity, lagged population size, NPV infection prevalence, tent size) and environmental (proportion of degree days above the historical average) parameters on population rate of increase for field populations of western tent caterpillar located in southwestern British Columbia. Sites included were: Mandarte Island, Galiano Island, Saturna Island, Westham Island and Cypress Mountain. Estimates (closed circles) are calculated using the top model set from the model dredging process along with 95% confidence intervals (horizontal lines). Confidence intervals that overlap with the red hashed (zero) line are unlikely to have a strong directional influence on population rate of increase. The frequency of appearance of a variable in the top model set is indicated by the relative importance value (RIV) shown on the right axis. Variables are listed in descending order according to RIV values.

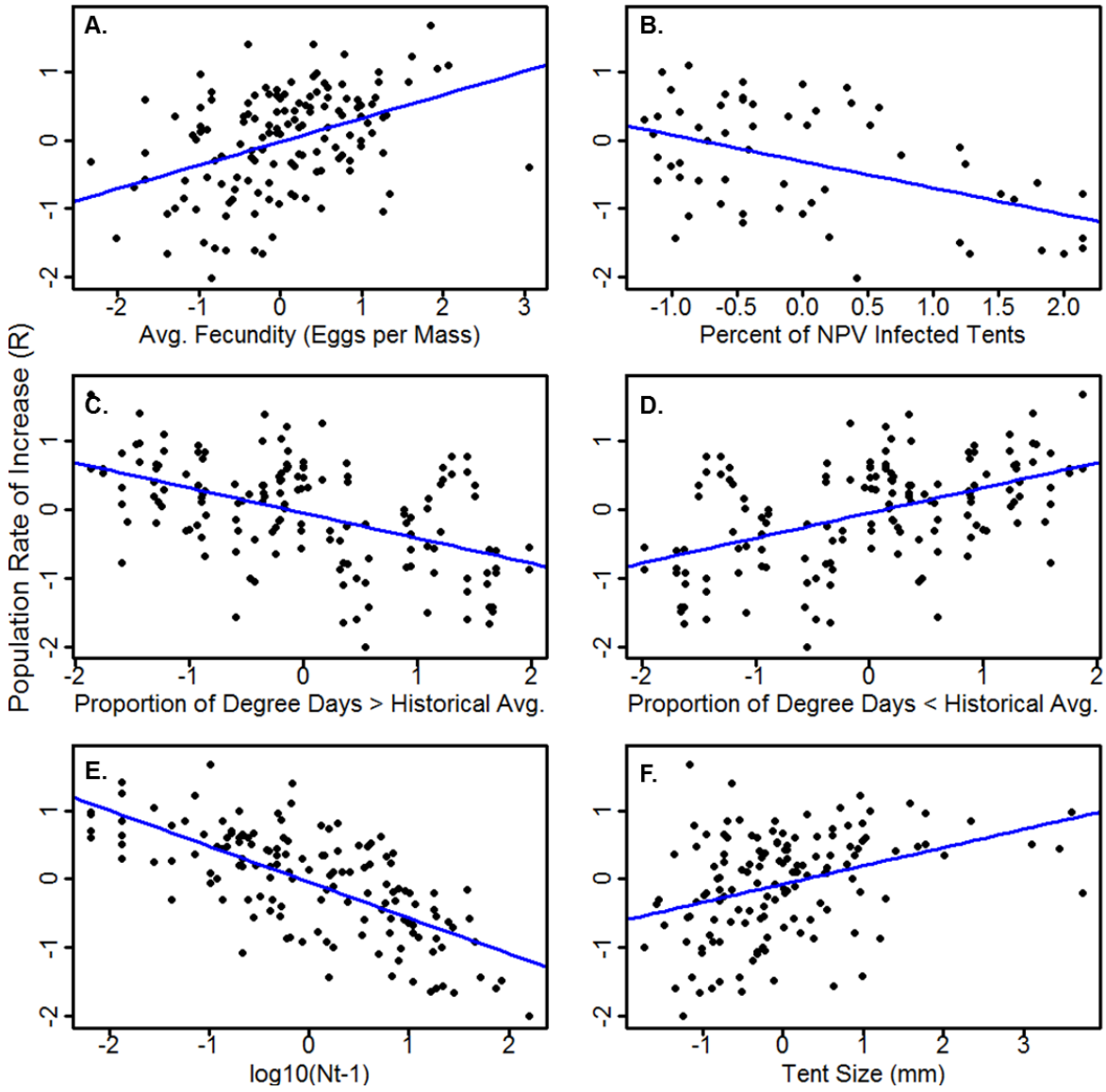


Figure 3.2. Scatterplots of demographic and environmental parameters (scaled and centered) regressed against population rate of increase for field populations of western tent caterpillar located in southwestern British Columbia. Independent parameters are: (A.) Average fecundity (B.) NPV infection prevalence (C.) Proportion of degree days > historical average (D.) Proportion of degree days < historical average (E.) Lagged population abundance and (F.) Average tent size (mm). Sites included were: Mandarte Island, Galiano Island, Saturna Island, Westham Island and Cypress Mountain. Annual time-series data for the period of 1975-2015 were used. Lines represent linear model estimates for each parameter against population rate of increase.

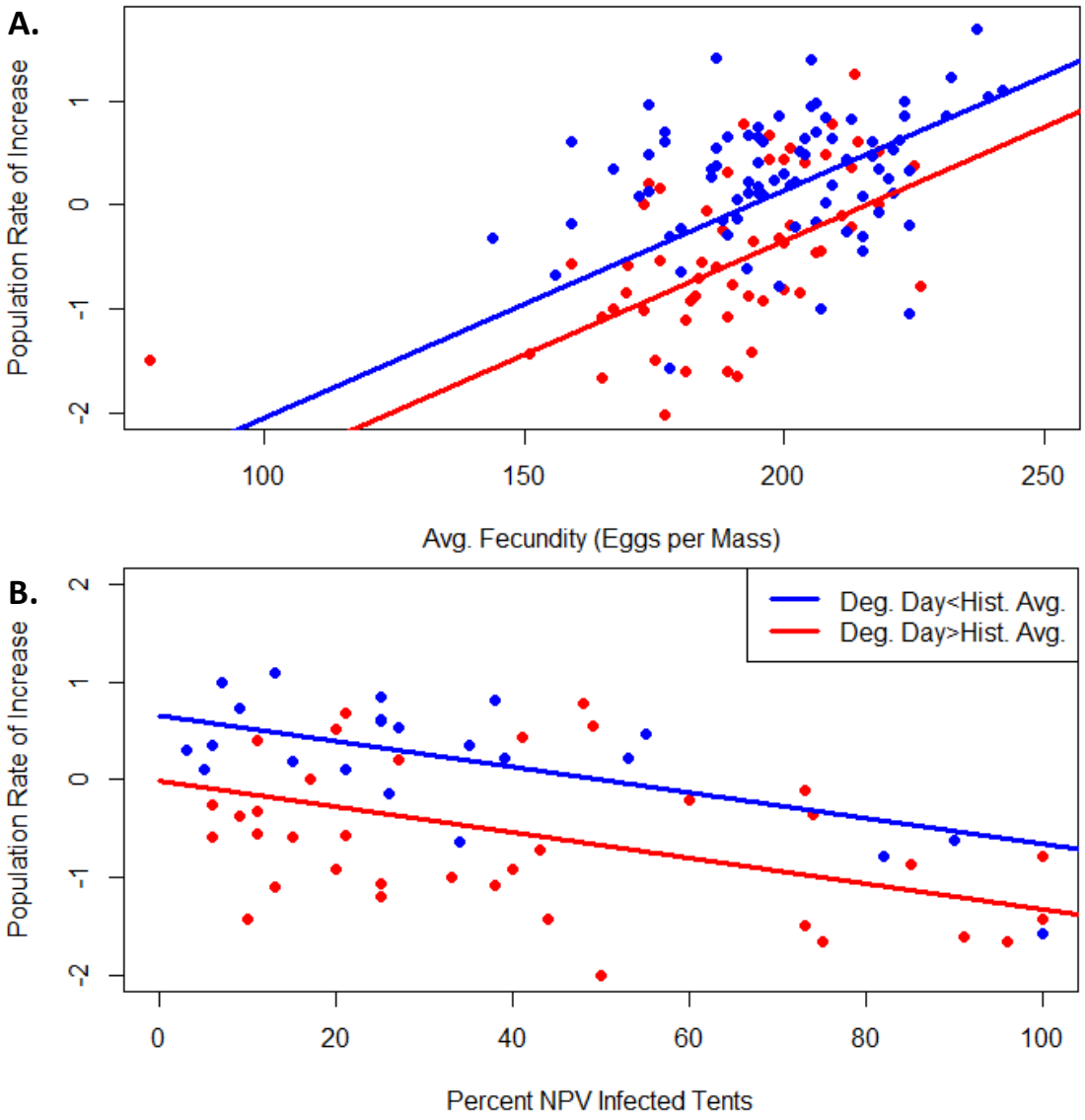


Figure 3.3. Scatterplots demonstrating fecundity (A.) and NPV infection prevalence (B.) as predictors of annual population rate of increase for western tent caterpillar field populations located in southwestern British Columbia. Infection values of zero have been removed to ensure normality of data. Lines represent model estimates with y-intercept values determined by whether the proportion of degree days was below (blue = cooler) or above (red = warmer) the historical average for a given year.

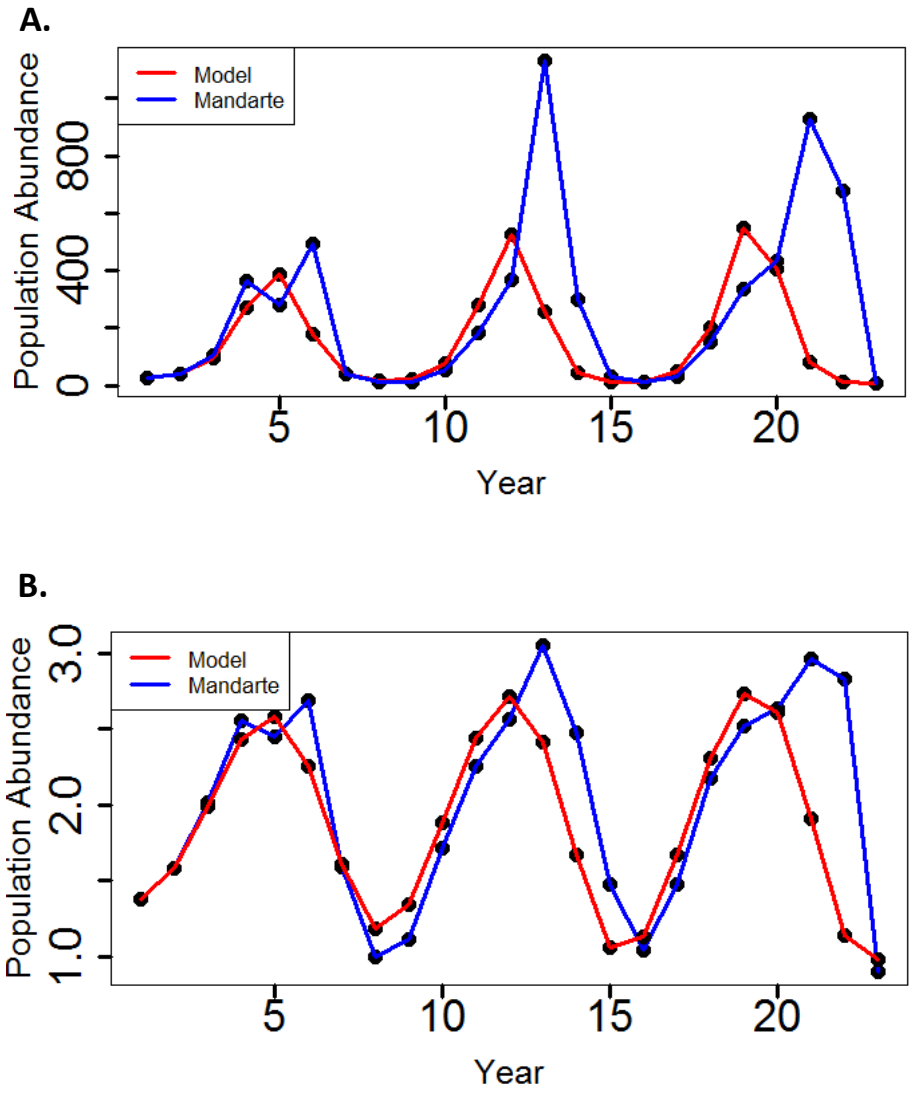


Figure 3.4. Simulation results of a western tent caterpillar-nucleopolyhedrovirus model parameterized from time-series data for Galiano Island and Saturna Island (red line). Model parameters contain both direct effects of viral infection (reduced survival) and indirect effects of virus challenge (transgenerational reduction of offspring fecundity in response to maternal virus challenge). Simulations were run over 22 generations and compared with time-series data from Mandarte island (1992-2015) (blue line). Initial starting population conditions of the model are the same as that of Mandarte for the first two time steps (1992 = 24 tents, 1993 = 38 tents). Simulation results are shown with population size (A.) untransformed and (B.) log (base 10) transformed.

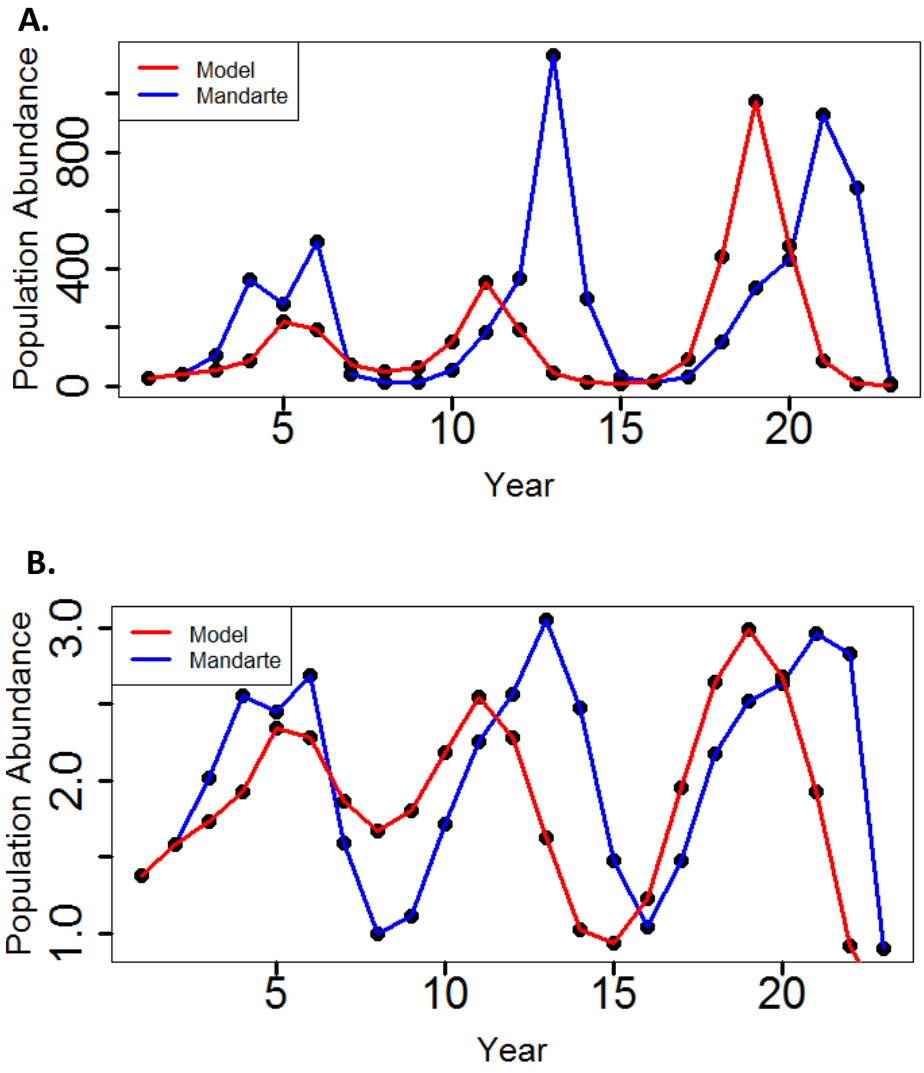
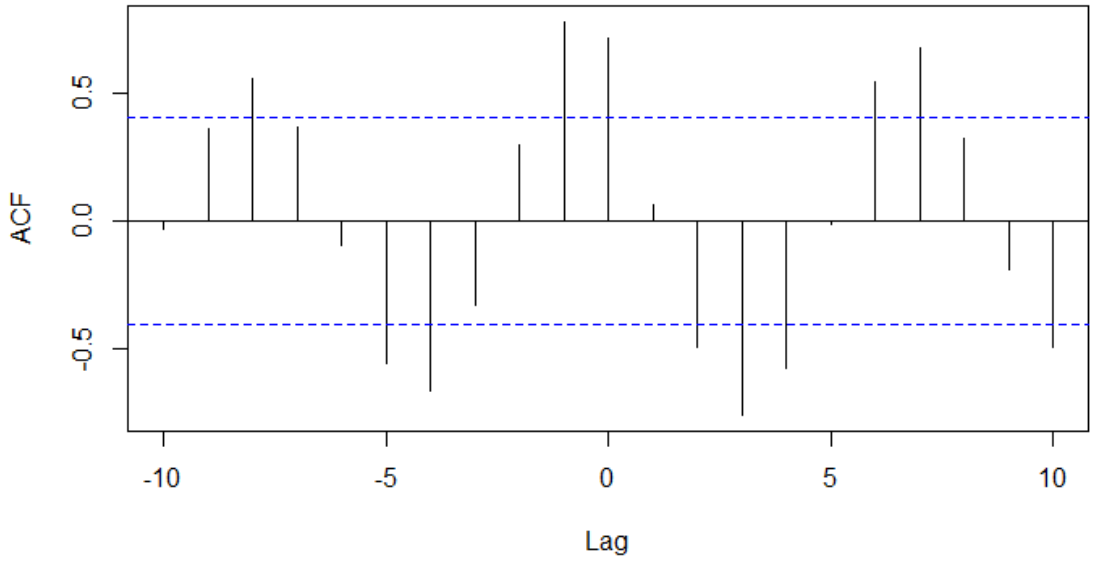


Figure 3.5. Simulation results of a western tent caterpillar-nucleopolyhedrovirus model parameterized from time-series data for Galiano Island and Saturna Island (red line). Model parameters contain the direct effects of viral infection (reduced survival), indirect effects of virus challenge (transgenerational reduction of offspring fecundity in response to maternal virus challenge) as well as the stochastic effects of annual springtime temperatures. Simulations were run over 22 generations and compared with time-series data from Mandarte island (1992-2015) (blue line). Initial starting population conditions of the model are the same as that of Mandarte for the first two time steps (1992 = 24 tents, 1993 = 38 tents). Simulation results are shown with population size (A.) untransformed and (B.) log (base 10) transformed.

A.



B.

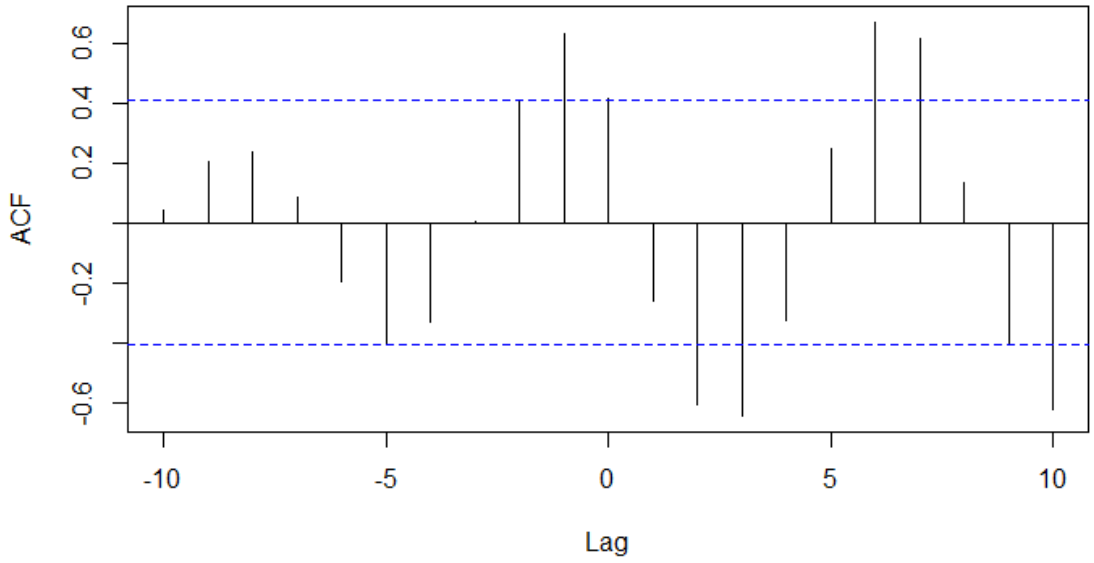


Figure 3.6 Cross-correlation plots for western tent caterpillar simulated model data regressed against time-series data for Mandarte Island. Cross-correlation is displayed on vertical axis and Lag (in years) displayed on horizontal axis. Bartlett's criterion (line of significance) is shown in blue (hashed line). NPV-fecundity model shown in (A.) and NPV-fecundity-temperature model shown in (B.).

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Chapter 4

General Conclusion

4.1. Thesis Aims and General Findings

The overall aim of this thesis was to examine the potential roles of factors (both intrinsic and extrinsic) thought to be important in the cyclical population dynamics of the WTC. Specifically, I focused on parameters that could influence vital rates of these populations. The results suggest that NPV infection prevalence is a significant factor in the dynamics of these populations and clearly responds positively to the size of its host population, reducing survival through viral death and host reproductive potential. In the context of the debate as to what factors contribute to the cyclical dynamics of forest Lepidoptera, the findings of this research further support the hypothesis of top-down regulation by pathogens, specifically NPVs.

4.2. Delayed Density-Dependence

A key component of cyclical dynamics is the existence of a delayed density-dependent negative feedback on vital rates (Klemola et al., 2008). Previous population densities negatively impact the survival and/or reproductive potential of a current generation through maternal effects, variation in plant quality, delayed response of parasitoids or pathogens (Berryman, 1996). Using time-series analysis, I demonstrated the second-order delayed density-dependent structure of field populations of WTC and distinguished between the dynamics displayed by island versus mainland sites. Island sites typically display more regular cyclical behaviour and have a higher degree of second-order density-dependence than mainland sites. If NPV is the factor through which this delay operates, then one might expect the prevalence of NPV infection to differ between island and mainland sites as this would account for the difference in strength of second-order density-dependence. Indeed, island sites typically display higher levels of infection prevalence throughout the course of their cycles (Myers, 2000), so it is possible that differences in host-NPV dynamics account

for this difference in strength of density-dependence. In fact, delayed density-dependence in the context of pathogens has not been investigated in many forest Lepidoptera (Klemola et al., 2008). Further work should then focus on populations of forest Lepidoptera whose NPV dynamics differ in magnitude to determine whether this difference correlates directly to the strength of second-order density-dependence. If a general trend is found, then this could further support the hypotheses of top-down regulation by NPV and delayed (trans-generational) effects of NPV exposure.

4.3. The Roles of NPV Infection and Fecundity

Using a relationship between lagged population size and NPV infection prevalence I found evidence for delayed density-dependence in WTC operating through reduction in host survival. This relationship was non-linear (logistic) and generated stable limit cycles. Next, by conducting AIC model selection on various factors believed to have an impact on WTC population rate of increase I was able to identify the relative importance of fecundity, lagged population size, NPV infection prevalence and warmer spring temperatures. This allowed me to further modify my model structure to determine whether incorporating the effects of these factors increased model fit to actual population data. The incorporation of fecundity into the model structure significantly improved fit. This agrees with results from model selection, which ranks fecundity highest in terms of relative importance to the population dynamics of WTC. The NPV-fecundity model provides support for a maternal effect, where the environment experienced by the maternal generation (in this case, the quality of the environment is dictated by the prevalence of NPV infection) has negative consequence for the fecundity of offspring. Under this scenario, survivors of baculovirus challenge would incur negative costs in the quality of their offspring (Myers & Cory, 2013). This could easily be mediated through a reduction in offspring provisioning following virus challenge. Field studies support this, as WTC larvae challenged with NPV exhibit reductions in egg mass size and reduced pupal weight (pupal weight is positively correlated with fecundity) of their female offspring (Rothman, 1997).

Correlations between fecundity and density-dependent factors in a previous generation are also reported in other forest Lepidoptera. In LBM for example, the extent of defoliation in years of high population density is negatively correlated with fecundity of moths in the

subsequent year (Baltensweiler, 1977). In AM, a similar (but weaker) trend exists between fecundity and defoliation in the previous year. However, in the case of AM, host trees are also shared by WM and during years of extensive defoliation, WM populations typically continue their outbreak status for an additional two years (Tenow et al., 2007). This suggests that reductions in fecundity may not be related to defoliation in the previous generation, but some other density-dependent factor(s). As measures of NPV infection prevalence are rarely collected in the field over extensive periods of time, it is impossible to implicate baculovirus as a factor in such instances. Future work should focus on relating specific density-dependent factors to changes in fecundity over the course of population cycles. Changes in fecundity are a general trend amongst forest Lepidoptera and is likely explained by some density-dependent factor(s). These factors are likely to operate with a time-delay and so the environment of the maternal generation is a likely starting point of investigation.

4.4. The Role of Temperature

In addition to demographic and life history parameters, the findings of my research suggest that temperature is an important factor in the population dynamics of WTC, particularly during the larval period of development. Temperatures above the historical mean negatively impact the annual population rate of increase. Although exact mechanisms for this are not known, there are multiple ways in which temperature could impact population rate of increase via host-pathogen dynamics. Warm temperatures could impact NPV dynamics by decreasing time to death, increasing the rate virus replication, as well as increasing the theoretical yield of viral inoculum (van Beek et al., 2000; Frid & Myers, 2002; Subramanian et al., 2006; Ghosh & Bhattacharyya, 2007). Warmer temperatures could also increase larval movement and feeding behavior, both of which could then in-turn increase the risk of exposure to persistent infectious agents (Parker et al., 2010; Eakin et al., 2014; Elderd & Reilly, 2013).

Beyond the effects of temperature on phenology and range expansion, there is a surprising lack of literature on how temperatures impact (or could potentially impact) disease dynamics in forest insects (Thomas & Blanford, 2003). With the certainty of climate change, there is a general need to know how interactions between insect pests

and their pathogens will change. In particular, future work should focus on trade-offs between pathogen fitness traits in the context of a warmer environment. For instance, warmer temperatures are known to decrease time to death of NPV infected larvae, but this is associated with decreases in viral yield, which could decrease horizontal transmission rates (Frid & Myers, 2002; Takahashi et al., 2015; Redman et al., 2016). From the perspective of the host, shorter time to death of infected hosts could increase the potential for further rounds of horizontal transmission (Frid & Myers, 2002). However, warmer temperatures also decrease development time of larvae allowing for faster rates of developmental resistance to virus (Hoover et al., 2002). How these various thermal-sensitivities will impact pathogen success in a warmer climate needs further review and theoretical models derived from these trade-offs should be relatively simple to construct and analyze for most species. Although this could be further complicated by organisms that can elevate body temperature above ambient, like WTC (Frid & Myers, 2002). How these trade-offs play-out in the field and over longer periods of time is less straightforward. In particular, pathogen virulence and host resistance are important factors in determining the progression of host-pathogen interactions, and if these factors are altered by temperature, then long-term dynamics and host-pathogen co-evolution could be affected (Thomas & Blanford, 2003).

4.5. Significance of Research

Time-series data for cyclical populations rarely contain more than measures of population abundance, making it difficult to draw conclusions about the factors responsible for such dynamics or the mechanisms through which they operate (Klemola, 2008). Confronted with data that are limited to measures or indices of host abundance, there are a limited number of options available for hypothesis testing. Time-series analysis can identify the density-dependent structure of population data, but does not provide information on the factors through which the density-dependent structure operates (Royama, 1992; Hunter & Price 2000; Turchin & Berryman 2000; Hunter, 2001). Laboratory and field experiments can test specific hypotheses, but are often limited in the number of factors they can test or the variation in strength of factors over temporal and/or geographical scales (Hunter, 2001). Models are useful theoretical tools for determining whether *a priori* assumptions

are validated by abundance time-series data, but often lack ways (beyond goodness of fit) in which to test output against ecological data (Dwyer, 2000). This work is unique in the manner in which it was conducted. Rather than relying on a single method of describing population cycles (time-series analysis, models, etc.) I synthesized information from a number of approaches to create a population model that can be tested against ecological data. The WTC dataset is unique, as it contains measures of a number of factors likely to influence population growth. However, if comprehensive datasets and/or detailed life-table analyses can be constructed for other cyclical species, I believe a similar approach to the one that I have undertaken can reveal the key factors responsible for such dynamics.

4.6. Concluding Remarks

In the introduction to my thesis, I discussed how population ecologists have traditionally been somewhat divided between two "philosophical camps": empiricists and theoreticians. While both groups have made tremendous progress toward understanding the fundamentals of population cycles, I argue that it is only through employing the methodologies from both groups that a consensus can finally be reached on what factors contribute to the dynamics of cyclical species. Furthermore, I would like to comment on first-principles approaches to modelling population dynamics. Model structure can often be assumed based on certain *a priori* assumptions (i.e. the system contains disease so a Kermack-Mckendrick model structure is used) before actually observing relationships between factors in the system under study. This is an oversight that is made too often by theoretical modelers, perhaps because of a lack of field data regarding the influence of specific factors on vital rates. Experiments can get around this dilemma, but they are often focused on the importance of a limited set of factors or cannot replicate conditions throughout the course of population fluctuation. Another difficulty is that a multitude of factors can be associated with changes in density of cyclical populations (defoliation and food quality for instance) and as a result, many of these can be argued to be relevant contributors to their dynamics.

I argue that while rare, comprehensive field data that includes measurements of factors likely to influence survival and growth (reproduction) is the best starting point for investigating the underlying mechanisms of population dynamics. Using these data and employing an information theoretic approach (or similar “key-factor” type analysis) would allow one to distinguish between factors that best describe changes in population rate of increase. From this point, theoretical models based on observed relationships between these factors could be constructed and used to ask whether these relationships are sufficient to generate the dynamics similar to actual ecological data. I believe by employing such methods, there can be greater constructive dialogue between both “philosophical camps” and improved hypothesis testing that is more holistic in its approach. With the construction of models which can readily be tested against ecological data, predictions of how the population dynamics of forest Lepidoptera are likely to behave under future scenarios can be made with greater confidence. This has obvious application toward forest pest management, where the effects of such population dynamics have significant economic and ecological impacts.

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van Beek, N., Hughes, P. R., & Wood, H. A. (2000). Effects of incubation temperature on the dose–survival time relationship of *Trichoplusia ni* larvae infected with *Autographa californica* nucleopolyhedrovirus. *Journal of invertebrate pathology*, *76*(3), 185-190.

Appendix A.

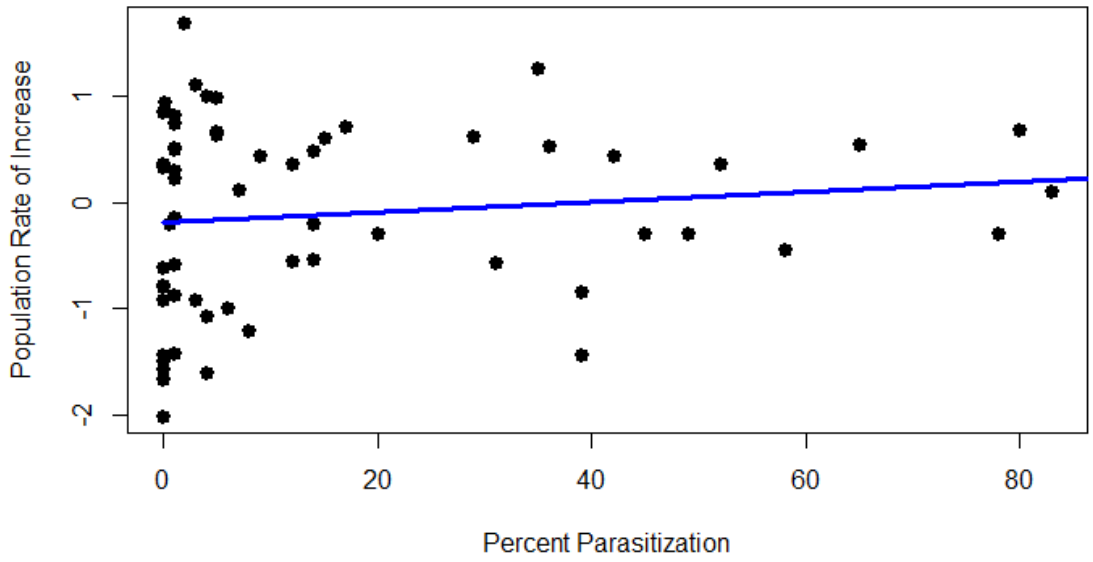

Figure A1. Plot of the relationship between percent of parasitization and annual population rate of increase for field populations of western tent caterpillars in southwestern British Columbia, R2 = -0.003, p = 0.3681.

Appendix B.

Reducing dimensionality of model for stability analysis

Begin with the formula for population rate of increase:

$$R = \log\left(\frac{N_{t+1}}{N_t}\right)$$

Using log-laws:

$$R = \log(N_{t+1}) - \log(N_t)$$

Substituting $R = aI_t + b$

$$aI_t + b = \log(N_{t+1}) - \log(N_t)$$

$$\log(N_{t+1}) = aI_t + b + \log(N_t)$$

Let $Y_{t+1} = \log(N_{t+1})$

$$Y_{t+1} = aI_t + b + Y_t$$

$$Y_{t+1} = \frac{100\text{a}}{1 + e^{(-2.26Log_{10}(N_{t-1})+5.205)}} + b + Y_t$$

Define the arbitrary parameter A_{t+1}

$$A_{t+1} = Y_t$$

So, the new model:

$$(1) \quad Y_{t+1} = \frac{100\text{a}}{1 + e^{(-2.26Log_{10}(N_{t-1})+5.205)}} + b + Y_t$$

$$(2) \quad A_{t+1} = Y_t$$

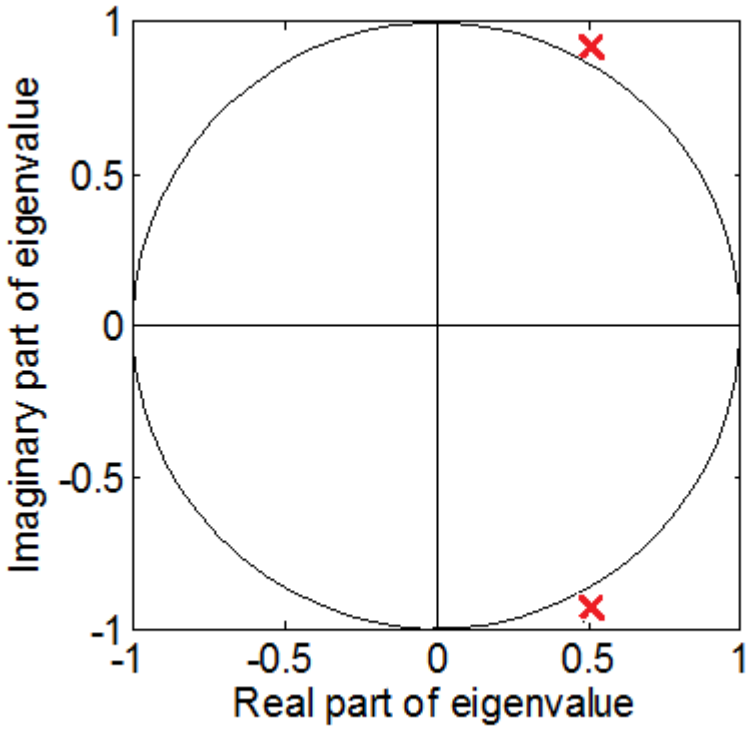

Figure B1. Eigenvalues for a western tent caterpillar-nucleopolyhedrovirus model derived from time-series data for populations residing on Saturna Island and Galiano Island. Eigenvalues were evaluated at equilibrium. Real parts of eigenvalues are positive and equal, indicating an unstable equilibrium point (proper node).

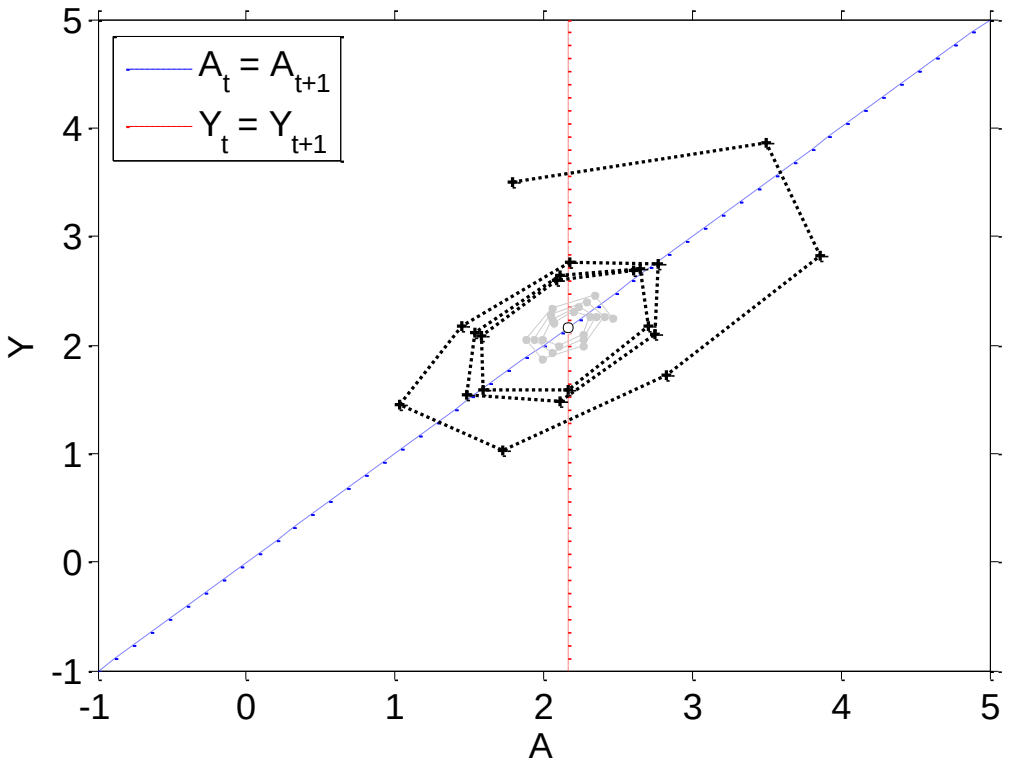

Figure B2. A phase plane demonstrating the attractor behaviour of a western tent caterpillar-nucleopolyhedrovirus model derived from time-series data for populations residing on Saturna Island and Galiano Island. When initiated far from the unstable equilibrium point (open circle at the intersection of the blue and red null clines) the system demonstrates attractor behaviour (black crosses). When initiated close to the unstable equilibrium point the system demonstrates repulsive behaviour (Grey closed circles). Between areas occupied by black and grey lines, stable limit cycles exist.