University of Alberta

Interactions between the forest tent caterpillar (*Malacosoma disstria* Hübner) and its natural enemies: the effects of forest composition and implications for outbreak spread

by

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For my family: Jim, Betty, Trish and Nancy.

Abstract

Forest tent caterpillar (*Malacosoma disstria* Hübner; FTC), a major defoliator of aspen trees, occupies both aspen and mixedwood forest stands in Alberta's boreal forest. Forest stand composition could influence the spatial pattern of FTC outbreaks if mortality from natural enemies differs between stand types. I conducted field experiments to determine whether predator- or parasitoid-caused mortality of FTC differed between aspen and mixedwood forest stands and developed a spatial population model to determine the effects of variation in generalist predation on the spread of an FTC outbreak, including the effects of potential predator-caused Allee effects. Generalist predation on FTC was higher in aspen stands than in mixedwood stands, and the spatial model suggests that these observed differences may be sufficiently large to impact FTC outbreak spread rates. Forest stand composition may contribute to the spatial pattern of FTC outbreaks through variation in the impacts of predators on FTC populations.

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Chapter 1

General Introduction

Many forest insect populations, of both native and introduced species, periodically outbreak and cause widespread defoliation (e.g. Bjørnstad et al. 2010, Tenow et al. 2007, Cooke and Lorenzetti 2006, Royama et al. 2005, Bjørnstad et al. 2002, Cooke and Roland 2000). These periodic outbreaks may be generated by a variety of mechanisms including lagged density dependence resulting from feedback from host plants (Turchin et al. 2003) and natural enemies, especially pathogens and parasitoids (Royama et al. 2005, Dwyer et al. 2004, Turchin et al. 2003, Roland and Taylor 1997, Berryman 1996). In addition to their complex temporal dynamics, outbreaks can also be spatially dynamic, frequently spreading out or travelling across forested landscapes (Cooke et al. 2009, Tenow et al. 2007, Johnson et al. 2006a, Johnson et al. 2004, Bjørnstad et al. 2002). Despite both theoretical and forest management interest in forest insect population dynamics, very little is understood about the factors that govern the rate or direction of spread of forest insect outbreaks (but see Johnson et al. 2006a, Johnson et al. 2004).

Spatial Spread of Populations and Allee Effects

Population spread rates have been a central focus of both theoretical and empirical invasive species research (Liebhold and Tobin 2008, Hastings et al. 2005, Fagan et al. 2002, Andow 1990) where population spread occurs by transient invasion waves. For invading populations that disperse through diffusion, spread rates can be estimated from the dispersal and population growth rates included in single-species

models (Andow 1990). However, spread rates are more difficult to estimate directly from models that include detailed descriptions of either population or spread dynamics, such as those that include demographic Allee effects (Wang and Kot 2001, Lewis and Karieva 1993), long-distance dispersal (Kot et al. 1996) or interacting species like natural enemies (Fagan et al. 2002, Owen and Lewis 2001). Because population spread is a direct result of both dispersal and population growth in low-density populations (Hastings et al. 2005), processes affecting growth rates of low-density populations can be critical to spread rates.

Allee effects cause declines in individual fitness or realized population growth rate with decreasing population density and are particularly important for small populations, affecting many population processes including establishment, spread and extinction (reviewed in Kramer et al. 2009, Courchamp et al. 2008, Courchamp et al. 1999, Stephens et al. 1999). Allee effects may be strong, whereby low-density populations decline, or weak, whereby low-density populations continue to grow but at a slower rate (Courchamp et al. 2008, Berec et al. 2007, Courchamp et al. 1999) and both strong and weak Allee effects can theoretically reduce the spread rates of invading populations (Taylor and Hastings 2005, Wang and Kot 2001, Kot et al. 1996, Lewis and Karieva 1993). For example, spatial variation in the invasion speed of gypsy moth (*Lymantria dispar*) has been associated with the strength of local Allee effects, with stronger Allee effects slowing invasion speed (Tobin et al. 2009, Tobin et al. 2007, Johnson et al. 2006b). Although Allee effects have typically been considered in the context of spread of invading populations (e.g. Taylor and Hastings 2005), the same processes may also affect spatial dynamics, including spread, of native forest insect outbreaks.

Natural Enemies and Outbreak Spread

Interactions with natural enemies are widely considered an essential component of the dynamics of forest insect populations (e.g. Klemola et al. 2010, Royama et al. 2005, Dwyer et al. 2004, Turchin et al. 2003, Roland and Taylor 1997, Berryman 1996, Gould et al. 1990, Hassell and May 1986, Southwood and Comins 1976) and can influence both temporal and spatial population dynamics. Interactions between forest insects and specialist enemies such as parasitoids may drive temporal population cycles (e.g. Klemola et al. 2010, Berryman 1996), but specialist enemies cause very little mortality in low-density populations (Dwyer et al. 2004, Turchin et al. 2003, Berryman 1996). In contrast, generalist enemies, including generalist predators, frequently have their greatest impact in low-density populations and can maintain forest insect populations at endemic densities (Klemola et al. 2002, Gould et al. 1990).

Spatial variation in community composition or abundance of natural enemies, due to heterogeneity in forest or landscape composition, can have consequences for the dynamics of forest insect outbreaks, resulting in geographical variation in population cycle period (e.g. Bjørnstad et al. 2010) or in outbreak duration (Roland and Taylor 1997, Roland 1993). Highly mobile natural enemies may cause spatial patterns in herbivore population abundance, restricting the spatial extent of outbreaks by dispersing beyond regions of high prey abundance and causing high mortality in peripheral low-density populations (Maron and Harrison 1997). Depending on whether there is an Allee effect in the prey population, natural enemies, especially generalists, can also slow or reverse the spread of invading prey populations by causing high mortality in low-density populations on the edge of the spreading front (Taylor and Hastings 2005, Fagan et al. 2002, Owen and Lewis 2001).

Although natural enemies can affect the spread rates of prey populations by increasing mortality in the spreading population (Fagan et

al. 2002), natural enemies may further influence spread rates by causing an Allee effect in the prey population (Courchamp et al. 2008, Gascoigne and Lipcius 2004). The occurrence of predator-induced Allee effects in the prey population depends on the nature of the aggregative and functional responses of the predator to the prey (Gascoigne and Lipcius 2004). For example, a generalist predator with a type II functional response to prey imposes higher per-capita prey mortality in low-density populations than in larger prey populations, resulting in a reduced growth rate in low-density prey populations (Kramer and Drake 2010, Gascoigne and Lipcius 2004). Because generalist predators can potentially influence local dynamics of forest insect populations by inducing Allee effects in the prey population, they may also affect the spatiotemporal dynamics of forest insect populations, including outbreak spread rates. However, the potential for generalist predators to do so through an induced demographic Allee effect in low-density populations has never been explicitly considered.

Forest Tent Caterpillar Life History and Population Dynamics

Forest tent caterpillar (*Malacosoma disstria* Hübner; FTC) is a widespread defoliator of aspen (*Populus tremuloides*) and sugar maple (*Acer saccharum*) in hardwood and mixedwood forests in North America. This native defoliator provides an excellent opportunity to investigate the effects of natural enemies on the spatial dynamics of forest insect outbreaks. Forest tent caterpillar populations cycle with an approximately 10-year periodicity across much of their range (Cooke and Roland 2007, Cooke and Lorenzetti 2006, Sippell 1962) and in the boreal forest and aspen parkland regions cycle peaks frequently result in outbreaks that last 2-3 years and cause severe defoliation of aspen trees (Cooke et al. 2009, Cooke and Roland 2007, Cooke and Lorenzetti 2006, Sippell 1962, Hodson 1941). Forest tent caterpillar outbreaks are influenced by landscape

features such as forest fragmentation, which can increase outbreak duration and therefore the temporal extent of defoliation (Cooke and Roland 2000, Roland 1993). Defoliation caused by FTC reduces aspen growth (Brandt et al. 2003, Hogg et al. 2002), and consecutive years of defoliation are associated with tree mortality (Brandt et al. 2003, Hildahl and Reeks 1960) and stand decline (Man and Rice 2010), making outbreaks of this insect a legitimate concern for forest managers of hardwood resources.

Forest tent caterpillars are univoltine, overwintering as first instar larvae within the eggs (Fitzgerald 1995, Hodson 1941). Hatch of first instar larvae in early spring coincides with aspen budbreak (Parry et al. 1998) and newly hatched larvae typically feed on aspen foliage as a colony until the end of the third larval instar, at which time they begin to disperse (Fitzgerald 1995). Fourth and fifth instar larvae cause the greater part of aspen defoliation until fifth instar larvae spin cocoons in aspen foliage and pupate in early to mid summer (Fitzgerald 1995, Hodson 1941). Adult moths are short-lived (~5 days) and females typically lay a single egg mass of between 150 and 300 eggs in the terminal branches of aspen trees (Batzer et al. 1995, Fitzgerald 1995). During the egg, larval and pupal stages, FTC are attacked by a suite of dipteran and hymenopteran parasitoids and a variety of generalist arthropod and avian predators (Witter and Kulman 1972).

Interactions between FTC and natural enemies, in particular predators and parasitoids, appear central to the dynamics of FTC populations. High parasitism rates by the sarcophagid fly *Arachnidomyia aldrichi* and the tachinid fly *Leschenaultia exul* are implicated in the collapse of outbreaks (Parry 1995, Witter and Kulman 1979, Hodson 1977, Sippell 1962). Furthermore, disruption of host-parasitoid interactions might explain the spatial variation in FTC dynamics in fragmented habitat (Roland and Taylor 1997). In contrast to specialist parasitoids, generalist

predators cause high mortality in low-density FTC populations, although they do not appear to regulate FTC populations (Glasgow 2006). Given the apparent influence of natural enemies on the local dynamics of FTC populations, spatial variation in interactions between FTC and their natural enemies may determine the spatial pattern of outbreaks, including the rate and direction of outbreak spread. Spatial variation in generalist predation in low-density FTC populations is of particular interest because of the potential for generalist predators to affect spread rates by inducing Allee effects in those low-density populations.

Forest Stand Composition

In general, the impacts of natural enemies on insect populations may be greater in more diverse habitats, especially habitats with greater vegetational species diversity, because of higher natural enemy abundance or diversity in those more diverse habitats (Andow 1991, Russell 1989, Root 1973). More diversehabitats provide a greater variety of prey species and microhabitats for generalist natural enemies, a greater diversity of resources for adult parasitoids, or a greater abundance of refuges for prey species that allows for persistence of specialist enemy populations (reviewed by Jactel et al. 2005). In the mixedwood boreal forest of northern Alberta, forest stand composition and diversity are highly variable, ranging from pure aspen stands to more diverse mixedwood stands comprised of aspen and other deciduous and coniferous tree species, and FTC populations exist throughout this range of habitats. Although natural enemy-caused mortality of FTC has been widely studied (e.g. Glasgow 2006, Roth et al. 2006, Parry et al. 1998, Parry et al. 1997, Roland and Taylor 1997, Parry 1995), there has been little consideration of the effects of forest composition on predation and parasitism of FTC, nor have the effects of forest composition on FTC population dynamics been thoroughly investigated (but see Sutton and

Tardiff 2007, Roland 1993). For other forest defoliators, such as the spruce budworm (*Choristoneura fumiferana*) and autumnal moth (*Epirrita autumnata*), predation and parasitism, especially by generalists, are higher in more diverse forest stands (Riihimäki et al. 2005, Quayle et al. 2003, Cappuccino et al. 1998). Forest stand diversity may therefore also alter the interactions between FTC and its natural enemies. In particular, differences in generalist predation of low-density populations between stand types may have implications for the spatial dynamics of FTC populations in the mixedwood boreal forest, including the spread of outbreaks.

Thesis Overview

My objective is to determine whether variation in interactions with natural enemies, specifically generalist predators, mediated by forest composition can influence the spread of a FTC outbreak. My thesis addresses two questions concerning the consequences of forest composition for the spread of FTC outbreaks: 1) What are the relative impacts of specialist and generalist natural enemies on low-density FTC populations, and do their impacts vary with forest composition? and 2) Does generalist predation alter the spread rate of a FTC outbreak, in particular through an Allee effect?

In Chapter 2, I describe the results of a field experiment in which I assessed the effect of forest stand composition on FTC mortality caused by both generalist predators and parasitoids throughout the larval and pupal stages in low-density populations. I used exclusion treatments to isolate different sources of mortality of early instar and late instar larvae and pupae in aspen and mixedwood forest stands. I examined the predator and parasitoid communities in each habitat and related differences in FTC mortality between stand types to differences in natural enemy abundance or community composition. In addition to addressing differences in

natural enemy-caused mortality between forest stand types, this experiment is also the first to describe the relative importance of generalists and specialists to FTC mortality across all larval and pupal stages in low-density populations.

In Chapter 3, I develop a discrete time model to describe the local interactions between FTC, a specialist parasitoid and a generalist predator, with parameters estimated from field data. With this non-spatial model, I examine the effects of generalist predation on local FTC population dynamics, including cycle period and the potential for generalist predation to induce an Allee effect in the FTC population. This model also provides an opportunity to examine the role of stochasticity in the generation of outbreak densities of FTC. I extend the model spatially to examine the effects of generalist predation on FTC outbreak spread rates, and to determine how predator-induced Allee effects further influence outbreak spread.

If natural enemy-caused mortality, in particular generalist predation, varies with forest stand composition, and if the strength of generalist predation influences FTC outbreak spread rates, then landscape-scale forest composition may influence the spatial pattern of FTC outbreak spread in the mixedwood boreal forest. The outcomes of my research have implications for forest management in areas of the mixedwood boreal forest affected by FTC outbreaks and contribute to the growing body of knowledge regarding native forest insect outbreaks and population spread.

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Chapter 2

Experimental Assessment of the Effects of Forest Stand Composition on Interactions Between the Forest Tent Caterpillar (*Malacosoma disstria* Hübner) and its Natural Enemies

Introduction

Interactions with natural enemies are widely considered an essential component of the dynamics of forest insect populations. Natural enemies including predators, parasitoids, and pathogens, have been proposed both as regulators of low-density endemic populations in some forest insect populations and as drivers of periodic outbreaks in others (e.g. Klemola et al. 2010, Heisswolf et al. 2009, Klemola et al. 2002, Tanhuanpää et al. 2001, Kidd and Jervis 1997, Parry et al. 1997, Berryman 1996, Gould et al. 1990, Hassell and May 1986, Southwood and Comins 1976). Generalist enemies frequently have their greatest impact in low-density populations and density-dependent predation may be responsible for maintaining forest defoliator populations at endemic densities (Klemola et al. 2002, Gould et al. 1990). In contrast, lagged density-dependent interactions with specialist enemies, where specialist enemy populations respond numerically to prey populations but with a time-lag, may drive defoliator population cycles but cause very little mortality in low-density populations (Dwyer et al. 2004, Turchin et al. 2003, Berryman 1996). Because both specialist and generalist enemies can affect forest

insect dynamics, spatial variation in natural enemy communities can have consequences for the dynamics of forest insect populations, resulting in geographical gradients in dynamics or variation in dynamics associated with habitat heterogeneity (e.g. Bjørnstad et al. 2010, Klemola 2002, Roland and Taylor 1997).

The forest tent caterpillar (*Malacosoma disstria* Hübner; FTC) is a widespread cyclic defoliator of aspen (*Populus tremuloides*) in the boreal forest. Forest tent caterpillar populations cycle with an approximately 10-year periodicity across much of their range and localized outbreaks typically last 2-3 years, although both cycle length and outbreak duration vary geographically (Cooke et al. 2009, Sippell 1962). A variety of mechanisms may contribute to the cyclic dynamics of FTC populations, including: maternal effects (Myers 1990), weather, (Roland et al. 1998, Daniel and Myers 1995, Ives 1973), pathogens (Stairs 1966) and natural enemies, especially parasitoids (Roland 2005, Roland and Taylor 1997, Parry 1995).

Forest tent caterpillars are attacked by a suite of dipteran and hymenopteran parasitoids (Parry 1995, Witter and Kulman 1972). Declines from peak density have been associated with high parasitism by the sarcophagid fly *Arachnidomyia aldrichi* and the tachinid fly *Leschenaultia exul* (Parry 1995, Witter and Kulman 1979, Hodson 1977). Furthermore, analyses of short population time series indicate the presence of lagged density-dependent processes (Roland 2005), suggesting parasitism may be partly responsible for promoting cycles in FTC populations. A variety of generalist arthropod and avian predators also attack FTC throughout their life cycle (Glasgow 2006, Parry et al. 1997, Witter and Kulman 1972) and, although predation on pupae does not appear to regulate FTC populations, generalist predators may still cause high FTC mortality at low-density (Glasgow 2006). Interactions between FTC and their natural enemies have been widely studied across a range of FTC densities (e.g.

Glasgow 2006, Roth et al. 2006, Parry et al. 1998, Rothman and Roland 1998, Parry et al. 1997, Roland and Taylor 1997, Parry 1995, Witter and Kulman 1979, Hodson 1977, Witter and Kulman 1972, Hodson 1939), however, individual studies focus on a single guild of natural enemies (i.e. predators, pathogens, or parasitoids), and on only one or a few life stages (but see Rothman and Roland 1998). It is therefore unclear how specialist and generalist natural enemies interact across FTC life history to contribute to generational mortality in low-density populations.

Landscape heterogeneity influences FTC population dynamics, with outbreaks typically lasting longer in regions where deciduous forest is highly fragmented by agriculture (Cooke and Roland 2000, Roland 1993), and disruption of host-parasitoid interactions might explain this spatial variation in FTC dynamics (Roland and Taylor 1997). Fragmentation appears to reduce the strength of lagged density-dependence evident in FTC population time series, resulting in outbreaks that develop more quickly (Roland 2005). However, controlled fine-scale experiments have failed to find an effect of forest fragmentation on parasitism rates or generalist predation on pupae (Glasgow 2006, Roth et al. 2006). Although the effects of deciduous forest fragmentation on FTC population dynamics have been studied, the mixedwood boreal forest also comprises a large part of the range of FTC and habitat effects on FTC populations in this ecosystem have never been thoroughly considered (but see Sutton and Tardiff 2007, Parry et al. 1997).

In the mixedwood boreal forest of northern Alberta, forest stand composition and diversity is highly variable, and aspen can be found in pure stands or with other deciduous and coniferous trees. The 'natural enemies' hypothesis (Andow 1991, Russell 1989, Root 1973) proposes that the impacts of natural enemies on herbivore insect populations are greater in more diverse habitats, in particular habitats with greater vegetational species diversity, because more diverse habitats support a higher

abundance or diversity of natural enemies. A variety of mechanisms may lead to higher natural enemy abundance or diversity in habitats with diverse vegetation: Diverse habitats may provide a greater variety of prey species and microhabitats for generalist natural enemies, promoting more abundant, diverse and stable enemy populations. Similarly, diverse habitats may provide refuges for prey species, allowing persistence of specialist enemies. Finally, diverse habitats may provide a greater diversity of resources for adult parasitoids, such as nectar and pollen, thereby promoting parasitoid populations that are more abundant (reviewed by Jactel et al. 2005).

Although the natural enemies hypothesis was originally motivated by observations from agricultural systems, observations of reduced forest pest damage in more diverse forest stands has advanced its consideration in forest systems (e.g. Jactel and Brockerhoff 2007, Koricheva et al. 2006, Jactel et al. 2005). Furthermore, in some forest defoliator populations such as the spruce budworm (*Choristoneura fumiferana*) and autumnal moth (*Epirrita autumnata*), mortality from generalist parasitism or predation does increase with forest diversity (Riihimäki *et al.* 2005, Quayle et al. 2003, Cappuccino *et al.* 1998), although the response by specialist parasitoids may be opposite (Herz and Heitland 2005, Sheehan 1986). Forest stand diversity may therefore alter the interactions between FTC and its natural enemies, with potential implications for the dynamics of FTC populations in the mixedwood boreal forest.

The objectives of this study were to determine: 1) the relative contribution of generalist predators and both specialist and generalist parasitoids to mortality of low-density FTC populations, and 2) if natural enemy-caused mortality of FTC is greater in more diverse forest stands and if it is related to natural enemy abundance or diversity, as predicted by the natural enemies hypothesis. I use several enemy exclusion treatments to identify the contribution of predators and parasitoids to

generational mortality of FTC and compare natural enemy-caused mortality between aspen (low diversity) and mixedwood (high diversity) forest stands. I expect generalist enemies to cause most of the natural enemy-caused mortality in the low-density FTC populations. Furthermore, I expect greater natural enemy-caused mortality of FTC in more diverse mixedwood stands as a result of greater abundance or diversity of generalist predators and parasitoids.

Methods

Study Location and Site Selection

I conducted field experiments from April to August in 2009 and 2010 in the mixedwood Boreal forest of north-central Alberta, Canada, in low-density, endemic FTC populations. Forest in this region is dominated by stands of aspen (*Populus tremuloides*) and balsam poplar (*Populus balsamifera*) with deciduous-conifer mixedwood stands including white spruce (*Picea glauca*) and balsam fir (*Abies balsamea).* The understory is varied, but commonly includes *Alnus crispa*, *Amalanchier alnifolia, Cornus stolonifera, Rosa acicularis, and Salix* spp.

I selected 20 forest stands (ten aspen stands and ten mixedwood stands) in the spring of 2009 based on forest composition (assessed visually), accessibility, and the availability of aspen saplings suitable for exclusion treatments. Selected sites extended over a transect approximately 100km long and were a minimum of 1 km apart (Figure 2-1). Stands ranged in size between 0.9 ha and 27.5 ha (median: 2.86 ha). Aspen stands had canopies comprised of >80% aspen and <10% coniferous species. Mixedwood stands were aspen dominated (>50% aspen in the canopy), but contained a minimum of 20% white spruce either co-dominant or sub-dominant in the canopy. Detailed data on both canopy and understory variables were collected subsequently at each site

and are reported in Appendix 1. Yearly male moth abundance was estimated by the average trap-catch from two pheromone traps at each site (Schmidt et al. 2003).

Experimental Exclosures

To determine the relative impacts of generalist predators and parasitoids on FTC mortality, I established four exclusion treatments on aspen saplings (approximately 3.5 m tall) at each site. All treatments were established at the onset of the experiment with the exception of the arthropod exclusion treatment, which was added in 2010 to distinguish between arthropod and avian predation. The treatments were as follows:

1. *Enemy exclusion:* All natural enemies (predators and parasitoids) were excluded by enclosing the sapling in a light-coloured, fine mesh bag and applying an approximately 20 cm-wide band of tanglefoot (The TangleFoot Company, Michigan, USA) around the tree base. Velcro or zippers on one side of the bag allowed access for sampling (Fig. 2-2a).

2. *Predator exclusion:* Both epigaeic arthropod predators and avian predators were excluded, while still allowing access by parasitoids. Epigaeic arthropods were excluded with tanglefoot. Birds were excluded using a bamboo frame draped with 1-inch mesh gill netting erected around the sapling canopy (modified from Glasgow 2006; Fig. 2-2b). I attached a 15 cm tall aluminium funnel around the base of the exclosure to prevent larvae from escaping down the tree trunk. These exclosures were not effective at preventing predation by spiders or pentatomids (Hemiptera: Pentatomidae), which were removed by hand when they were observed.

3. *Arthropod exclusion:* Epigaeic arthropods were excluded while allowing access by birds and parasitoids by applying Tanglefoot around the base of the sapling. Spiders and pentatomids were removed by hand when observed.

4. *Open:* All natural enemies were allowed access to FTC larvae and pupae.

Saplings selected for exclusion treatments were of similar size and were a minimum of 10 m from the edge of each forest stand. The enemy exclusion and predator exclusion treatments were placed near each other, but a minimum of 50 m from the arthropod exclusion and open treatments (which were placed together). This separation prevented the conspicuous exclosures from affecting predation and parasitism at the less conspicuous treatments. Because saplings were limited, they were re-used between years if they suffered no visible damage. Over the two field seasons, enemy and predator exclosures were each damaged on six occasions.

In 2010, I used HOBO data loggers (Onset®) to monitor temperature for one month (11 May to 13 June) at 30-minute intervals in the enemy exclosures, predator exclosures and on the open trees at two sites. Average temperatures in the three treatments differed by less than 1°C (Table 2-1). Average daily maximum temperatures were more variable among treatments (Table 2-1), with the enemy exclusion treatment warmer than the open treatment by 0.5 – 5 °C.

Experimental Assessment of Natural Enemy-Caused Mortality of FTC

Natural enemy-caused mortality in aspen and mixedwood forest stands was assessed using exclusion treatments for three life history stages of FTC separately: early instar larvae, late instar larvae and pupae.

Early-instar larval mortality

In late April of 2009 and 2010, I stocked each experimental tree with two egg masses attached near terminal buds. Egg masses were obtained the previous winter from high-density FTC populations in northern Alberta (2009) or near Prince George, British Columbia (2010). Upon hatching, larvae were censused biweekly for abundance and instar. Because FTC larvae typically disperse at the 4th instar (Fitzgerald 1995),

survival was monitored until the second census of 3rd instar larvae or until molt to the 4th instar, whichever occurred first. At the end of the census, larvae were re-collected from the saplings. In 2009, approximately 50 larvae were left and allowed to pupate in each enemy exclusion treatment, and were subsequently used in the assessment of pupal mortality factors (see below). The initial number of first instar FTC larvae on each tree was determined from hatched egg masses. Mortality of early instar larvae was calculated as (1-*s*), where *s* is the proportion of hatched larvae that remained at the end of the census. Early instar larvae rarely disperse from their natal colonies (Fitzgerald 1995) so all losses of larvae from the experimental saplings were interpreted as mortality, with the source inferred according to exclusion treatment.

Late-instar larval mortality

Because 4th and 5th instar FTC larvae disperse from their natal colonies (Fitzgerald 1995), it was necessary to tether these later instar larvae to the experimental trees to ensure recovery and fate determination. Tethers, consisting of cotton thread adhered transversely to the abdomens (A7 and A8 segments) of the larvae with cyano-acrylate adhesive (Krazy Glue®), were attached to larvae between 12 and 24h prior to deployment. Larvae were tethered to experimental saplings within easy access to leaves previously eaten by FTC larvae, and with approximately 20 cm of slack thread. Larvae were frequently tangled to some extent upon retrieval. Fifth instar larvae were tethered in sets of two or three per tree for 24 h on two separate days in late June 2010, for a total of five, 5th instar larvae per exclusion treatment per site. After 24 h, I recorded fate of the larvae (dead, alive or preyed on). Partial larval remains were classified as preyed on. Live larvae were collected and subsequently classified as healthy (if an adult moth eclosed), parasitized (if a parasitoid emerged from the larva or pupa) or dead from unknown sources. Unknown mortality may be a

result of viral or fungal pathogens (Stairs 1972, Stairs 1966) or, in this study, as a side effect of the tethering protocol, but I did not identify specific causes.

Most experimental trees used for the assessment of early-instar larval mortality were re-used for the late instar experiment. In five cases, it was necessary to apply the exclusion treatment to a new sapling (three predator exclusions, one enemy exclusion and one arthropod exclusion). Because defoliation is used as an oviposition cue by some tachinid parasitoids of FTC (Mondor and Roland 1997), I allowed approximately 10 4th and 5th instar FTC larvae to defoliate each new sapling one week before the addition of tethered larvae. Defoliation levels among the experimental trees were variable.

Pupal mortality

Large 5th instar cage-reared FTC larvae not previously exposed to parasitoids were placed in mesh bags on branches of each experimental tree and allowed to spin cocoons and pupate in the leaves. Within three days of pupation, mesh bags were removed so pupae were exposed to predation and parasitism. In July 2009, between three and nine (median = 4.5) pupae were deployed using mesh bags on each sapling in the predator exclusion and open treatments. Pupae in the enemy exclusion treatment developed from the approximately 50 FTC larvae that remained in the exclosures since hatch, although pupae were not recovered from all replicates. All pupae were collected over two days in early August. In the predator exclusion and open treatments, exposure times of pupae ranged from 12 to 26 days (median: 17 days; Appendix 2); the exact exposure time of individual pupae in the enemy exclusion treatment is not known. In July 2010, five pupae were deployed in all three exclusion treatments using mesh bags. All pupae were re-collected after having been exposed

for 12 days. Several replicates did end up with fewer than five pupae because not all deployed larvae survived to pupate.

Pupal fates were identified based on a suite of characteristics: 1) Healthy: a healthy moth eclosed or the puparium had clean sutures and the cocoon silk had an exit hole with scales remaining; 2) Parasitized: presence of parasitoid larva, pupa, or adult; characteristic parasitoid exit hole from puparium or characteristic staining of cocoon silk (Hodson 1939); 3) Preyed on: absence of puparium but silk cocoon remaining or silk cocoon not intact and puparium not intact; 4) Unknown mortality: all pupae that could not be otherwise classified. Pupal fates were identified blindly.

Generalist Predator and Parasitoid Surveys

Generalist arthropod predators

The abundance of potential arthropod predators of immature FTC was monitored at each site using pitfall traps (Spence and Niemelä 1994). Five traps, 10 m apart, were installed along a transect between the enemy/predator exclusion treatments and the open/arthropod exclusion treatments at each site and were checked twice per week. In 2009, pitfall traps were installed for a two-week period (30 June to 14 July) corresponding to the FTC late-larval, early-pupal period. In 2010, traps were installed for one week in May (15 May to 24 May) corresponding to the early-larval period, and one week in July (2 July to 11 July) corresponding to the late-larval/early-pupal period. This change allowed for the detection of seasonal effects (corresponding to FTC life history) on the arthropod community (Spence and Niemelä 1994). Primary arthropod predators of FTC larvae and pupae, including ants (Hymenoptera: Formicidae), spiders (Araneae) and carabid beetles (Coleoptera: Carabidae), especially the genera *Calosoma*, *Carabus* and *Pterostichus* (Larochelle 1990, Witter and Kulman 1972) were identified and

enumerated using published keys (Lindroth 1969). In each year for each site, I pooled the data from the five pitfall traps within each collection period to determine the abundance/trap/day of each predator group.

Avian predators

I conducted three avian point counts at each site in both years to estimate the abundance of potential avian predators of FTC larvae and pupae. All three counts were conducted approximately halfway between the enemy/predator exclusion treatments and the open/arthropod exclusion treatments at each site from sunrise to 0730 h between 24 May and 19 June in both years. All birds heard or seen within a 50 m radius of the point during the 5-minute count were recorded. Observations from all three counts within a year were averaged to estimate avian species' abundances at each site (Toms et al. 2006).

Parasitism

Parasitism rates of tethered larvae exposed for 24 hours were very low (see Results). Therefore, larval parasitism rates were estimated and the parasitoid community characterized with independent collections of FTC larvae in 2010. Between five and seven egg masses were placed on four or five saplings at each site to facilitate larval collections for parasitoids. I applied tanglefoot to the tree bases to prevent arthropod predation and increase the probability of recovering late 4th and early 5th instar larvae. At least 30 late 4th or early 5th instar larvae were collected from all but one site (median=73) between 17 June and 27 June.

Re-collected tethered 5th instar larvae and larvae collected to estimate parasitism rates were reared in plastic cups (Solo®) in groups of three and fed fresh aspen foliage every other day until the emergence of parasitoids, pupation or death. Re-collected pupae were stored at room temperature in plastic cups until emergence or death. All parasitoids

recovered from larvae or pupae were identified using published keys (Williams et al. 1996, Goulet and Huber 1993, Dasch 1971, Sippell 1961).

Data Analysis

I used linear mixed effects models to compare mortality of all FTC life history stages between forest stand types and among exclusion treatments, unless otherwise indicated. In all models, site nested within forest type was included as random factor. To analyze early-instar larval loss rates, I also included the identity of each experimental tree as a random factor. For analysis of tethered larvae, tethering date was additionally nested within site. All proportions were arcsin-square-root transformed. The significance of main effects and their interactions were evaluated with F-ratio tests, and likelihood ratio tests of nested models were used for model simplification. Significant main effects or interactions were further investigated using *a posteriori* orthogonal contrasts evaluated at α/n where n is the number of contrasts.

Early-instar larval mortality

I examined the effects of exclusion treatment and forest type on both the total mortality of early instar FTC larvae and the loss of FTC larvae over time using linear mixed models. Total early-instar larval mortality in 2009 and 2010 were analyzed separately because of the addition of the arthropod exclusion treatment in 2010. Total mortality in the enemy exclusion, predator exclusion and open treatments was also compared between years.

I also used a linear mixed model to compare the loss of larvae over time from the open treatments between forest stand types and between years. To determine loss rates of early instar larvae, I assumed hatch occurred on the census date immediately before the census date with the maximum count of 1st instar larvae. This adjustment assumes that all mortality of FTC larva over the duration of hatch occurred over three days

and therefore overestimates the loss rate between the initial FTC number and the maximum 1st instar census observation. This bias is consistent however among all sites and treatments. Larva counts were ln-transformed to linearize the mortality rate, assuming a negative exponential reduction in larval number with time.

Damage to saplings and premature larval dispersal reduced replication of the predator exclusion treatment in both the aspen and mixedwood forest (n=9 each) in 2009, and replication of the predator exclusion treatment in aspen stands (n=6), the open treatment in mixedwood (n=7) and aspen (n=9) stands, and the arthropod exclusion treatments in aspen stands (n=9) in 2010.

Late-instar larval mortality

The effects of exclusion treatment and forest stand type on both total mortality and the proportion of larvae preyed on were analyzed using linear mixed models, and for each I only considered those treatments in which each fate actually occurred (e.g. for the analysis of the proportion of larvae preyed on the enemies exclusion treatment was omitted).

Predation and mortality from unknown sources can cause larval death before the emergence of parasitoids so that actual parasitism rates may be obscured in the presence of these mortality factors. To adjust the apparent parasitism rate to account for the contemporaneous effects of predation and unknown mortality, I calculated marginal parasitism rates ($m_{Parasitism}$; Elkinton et al. 1992):

$$m_{Parasitism} = \frac{p_{Parasitism}}{1 - m_{Unknown} - p_{Predation}}$$

$$\text{where: } m_{Unknown} = \frac{p_{Unknown}}{1 - p_{Predation}}$$

and p_x is the observed proportion of larvae dying from cause x. This calculation assumes that parasitoids are entirely out-competed by pathogens that cause unknown mortality, and corrects for predation preventing the detection of both unknown mortality and parasitism.

Parasitism of tethered larvae was rare, so I compared apparent and marginal parasitism rates between forest types and among exclusion treatments using Kruskal-Wallis tests. If larval predation is independent of parasitism, marginal parasitism rates should be the same in larvae protected from predators and those exposed to predation.

Pupal mortality

In 2009, there was a low incidence (8.3 %) of pupal parasitism in the enemy exclusion treatment, primarily because many larvae spun cocoons on the exclusion cage rather than in the foliage; these pupae were excluded from the analysis. The duration pupae were exposed to natural enemies in 2009 did not affect the probability of parasitism, or predation (Appendix 2). In 2010, several saplings were broken during the exposure period, but there was no effect of tree-breakage on the probability of pupal predation, or parasitism (Appendix 2). I combined data from both years.

The effects of exclusion treatment and forest stand type on total pupal mortality, the proportion of pupae preyed on and the apparent and marginal parasitism rates (calculated as for tethered larvae) were analyzed using separate linear mixed models, and for each I again considered only those treatments in which each fate actually occurred. Marginal parasitism rates should be equal for pupae in both the predator exclusion and the open treatment if predation is random with respect to parasitism. I also compared apparent and marginal parasitism rates of the two main pupal parasitoid guilds, the sarcophagid fly *Arachnidomyia aldrichi* and ichneumonid wasps, between exclosure types using Kruskal-Wallis tests.

Generational mortality

I examined the effects of exclusion treatment and forest type on generational mortality using *k*-values (-$\log_{10}$(survival); Elkinton et al. 1992, Varley and Gradwell 1970). For each exclusion treatment and forest type, I calculated separate *k*-values for early instar and late instar larvae and pupae. *k*-values for late instar larvae and pupae included only mortality attributed to natural enemies whereas *k*-values for early instar larvae included all mortality. No correction was made to account for the relative duration of each life-history stage; this is especially notable for late-instar larval mortality, which was only assessed over 24 h, whereas late-instar larval stages normally last up to two weeks. Observed mortality of late instar larvae over 24 h could not be directly scaled to the cumulative duration of the 4th and 5th instars because tethering likely elevated predation and parasitism rates above natural levels. However, the sum of the *k*-values across life-history stages in each treatment and forest type reflects the relative contribution of predators and parasitoids to total mortality over the larval and pupal stages in this experiment.

Natural enemy abundance and diversity

I compared generalist arthropod predator communities between forest stand types for the three trapping periods separately (July 2009, May 2010, July 2010) using MANOVA (Everitt 2005). For each time period, I conducted two separate analyses: one examining the arthropod community using abundances of ants, beetles and spiders, and a more detailed analysis that considered the carabid beetle community. To determine if specific components of the arthropod predator community contributed to mortality of early instar larvae or predation on pupae, I compared mortality of early instar larvae (May 2010) and pupal predation rates (July 2009, July 2010) to abundances of spiders, ants, and the two most abundant beetle species (*Pterostichus adstrictus* and *P. pennsylvanicus)*

using logistic regressions that included forest type as a main effect. A quasibinomial error distribution was assumed to account for overdispersion (Crawley 2007). Likelihood ratio tests were used to evaluate the significance of arthropod abundance or forest type as predictors of larval mortality.

I compared average avian abundance, species richness and diversity (H'; defined in Appendix 1) between forest stand types using Wilcoxon's rank-sums tests. I further compared the composition of bird communities between stand types using permutations based MANOVA (Anderson 2001), and used indicator species analysis to identify bird species common in, and unique to, each stand type at α=0.1 (Dufrêne and Legendre 1997). Indicator species values (IV) are a measure both of the abundance of the species in, and its affinity to, a particular forest type (Dufrêne and Legendre 1997). To determine how the bird community contributed to predation of late instar larvae and pupae, I modelled larval and pupal predation in open and arthropod exclusion treatments as a function of bird abundance, or the abundance of indicator bird species with logistic regressions. A quasibinomial error distribution was assumed to account for overdispersion. Abundance as a significant predictor of larval or pupal predation was evaluated with a likelihood ratio test.

I compared larval parasitism rates between forest stand types with a Wilcoxon's rank-sums test. I further compared the larval and pupal parasitoid communities between stand types with separate MANOVAs. I excluded from the analysis one aspen forest site from which I collected only four larvae.

Unless otherwise indicated, all analyses were conducted with α=0.05 and were conducted in R v 2.12.1 (R Development Core Team 2010, Vienna, Austria) using functions available in the stats, nlme, gmodels and vegan packages.

Results

Male moth FTC abundance did not differ between aspen and mixedwood stands in either year (2009: t_{18}=0.46, P=0.645; 2010: t_{18}=1.83, P=0.083). Abundance did increase significantly between years (2009: 5.1 ± 0.9 moths/trap; 2010: 16.5 ± 2.1 moths/trap; t_{38}=-4.75, P<0.0001), despite being low in both years compared to outbreak levels (Roland 2005).

Experimental Assessment of Natural Enemy-Caused Mortality of FTC

Early-instar larval mortality

Enemy exclusion treatments reduced early-instar larval mortality in both years (2009: $F_{2, 34}$=24.36, P<0.0001; 2010: $F_{3, 45}$=15.05, P<0.0001; Fig. 2-3). In 2009, excluding all generalist predators reduced larval mortality from 66.5% ± 5.6 % (open trees) to 28.8 % ± 2.1 % (predator and enemy exclusions; t_{52}=6.79, P<0.0001). In 2010, excluding only arthropod predators reduced larval mortality from 62.1% ± 4.9% (open trees) to 37.9% ± 2.3 % (all other treatments; t_{63}=6.64, P<0.0001). Further excluding avian predators had no additional effect on early-instar larval mortality (t_{63}=0.39, P=0.698), indicating that arthropod predators are the primary source of natural enemy-caused mortality for early instar larvae. There was no difference in early instar mortality between forest types in either year (2009: $F_{1, 18}$=2.93, P=0.104; 2010: $F_{1, 18}$=0.73, P=0.403; Fig. 2-3). Despite differences in initial hatch rate and the duration of the early-instar larval period between years, there was no effect of year on the loss rate of early instar larvae from open trees ($F_{1, 242}$=0.71, P=0.401). Loss rates were no different between stand types, ($F_{1, 242}$=2.61, P=0.108), but the trend was towards higher loss rates in aspen stands (Fig. 2-4; Table 2-2).

Late-instar larval mortality

Total mortality of tethered larvae was higher in aspen forest than in mixedwood forest ($F_{1, 18}$=5.41, P=0.032), and differed among enemy

exclusion treatments ($F_{3, 114}$=2.97, P=0.035) because mortality was reduced in enemy exclosures compared to all other treatments (t_{152}=2.56, P=0.011; Fig. 2-5a). Differences in total mortality among exclusion treatments were somewhat obscured by unknown mortality, most of which was mortality related to the tethering protocol. Of the total observed mortality of late instars, unknown mortality comprised 45% in both the open and arthropod exclusion treatments, 77% in the predator exclusion treatment, and 100% in the enemy exclusion treatment.

Predation on late instar larvae was greater in aspen stands than in mixedwood stands (predation: $F_{1, 18}$=11.90, P=0.003; Fig. 2-5b), indicating that predation was the primary cause of differences in total mortality between forest types. A small amount of predation (9%) occurred in the predator exclusion treatment indicating that some predator was not successfully excluded (Fig. 2-5b). Predation rate was affected by exclusion treatment ($F_{2, 76}$=7.816, P=0.0008; Fig. 2-5b). Excluding arthropod predators had no effect on late-larval predation rates ($t_{114,}$ =0.484, P=0.629), but additionally excluding avian predators reduced predation on late instars (t_{114}=3.51, P=0.0006), indicating that birds were the primary source of late instar predation. Exclusion of bird predators reduced larval predation in aspen stands only, leading to a significant interaction between forest type and exclusion treatment (treatment x forest: $F_{2, 76}$=3.22, P=0.046; Fig. 2-5b).

Low rates of parasitism occurred (mean: 4.8 ± 1.5%) over 24 h in all except in the enemy exclusion treatment. There was no effect of exclusion treatment or forest type on the proportion of larvae parasitized (treatment: K_3=1.28, P=0.526; forest type: K_1=0.911, P=0.340; Fig. 2-5c). Marginal parasitism rates were also low in all treatments (mean: 6.5 ± 2.0%) and did not differ between exclusion treatments or forest stand types (treatment: K_2=1.310, P=0.520; forest type: K=0.901, P=0.342; Fig. 2-5d). Although parasitism rates were very low for tethered larvae, similar apparent

parasitism rates in the predator exclusion treatment and the open treatment suggests that parasitoids do not cause additional mortality in the absence of predators, and similar marginal parasitism rates between these treatments suggests that birds do not differentiate between parasitized and non-parasitized FTC larvae.

Pupal mortality

Pupal mortality did not differ between years ($F_{1, 37}$=2.71, P=0.108), and there was no difference in total mortality between forest stand types ($F_{1, 37}$=0.075, P=0.786; Fig. 2-6a). Pupal mortality differed among exclusion treatments ($F_{2, 71}$=55.21, P<0.0001). Predator exclusion significantly reduced pupal mortality from 75.3 ± 4.0% to 51.6 ± 4.6% (t_{103}=4.128, P<0.0001), and excluding parasitoids further reduced mortality to 14.3 ± 2.8% (t_{103}=6.31, P<0.0001), indicating that both predation and parasitism contribute to FTC pupal mortality. Pupal mortality from unknown causes was generally low in all treatments and forest types, but was higher in the predator exclusion treatment (26.4 ± 3.7% of pupae) than the open or enemy exclusion treatments (12.0 ± 1.7% of pupae).

Predation on pupae only occurred in the open treatment, and was consistently greater in aspen than mixedwood forests in both years (forest type: $F_{1, 36-}$=4.60, P=0.039; year: $F_{1, 36}$=1.00, P=0.323; Fig. 2-6b). Neither the apparent nor the marginal pupal parasitism rates differed between years or between forest stand types (apparent parasitism – year: $F_{1, 37}$=0.04, P=0.834; forest type: $F_{1, 37}$=0.13, P=0.720; marginal parasitism – year: $F_{1, 37}$=0.31, P=0.582; forest type: $F_{1, 37}$=0.002, P=0.966). However, the apparent parasitism rate of pupae was lower when predators were excluded compared to when both predators and parasitoids were present ($F_{1, 38}$=12.01, P=0.0013; Fig. 2-6c). If parasitoids were not affected by the presence of the predator exclusion treatment, apparent parasitism rates in both treatments should be equal. The reduced parasitism rate in the

predator exclosure relative to the open treatment suggests that parasitoids were negatively affected by the presence of the predator exclosure (a "cage effect"). Marginal parasitism rates were also lower in the predator exclosure than in the open treatment ($F_{1, 38}$=11.79, P=0.0015; Fig. 2-6d), indicating that predation was non-random with respect to parasitism and that predators preferentially attacked non-parasitized pupae in the open treatment. Between the two primary guilds of pupal parasitoids, marginal attack rates between exclusion treatments were only different for the ichneumonid parasitoids (K_1=6.17, P=0.013), not for *Arachnidomyia aldrichi* (K_1=2.01, P=0.157).

Generational mortality

Predation and parasitism during the late-instar larval and pupal life history stages contributed most to natural enemy-caused generational mortality (Fig. 2-7). Differences in late-instar larval predation between forest stand types caused total generational mortality of FTC to be greater in aspen stands than in mixedwood stands (open treatment: k_{aspen}= 3.1, k_{mixed}=1.6; Fig. 2-7). However, differences in *k* values between treatments and stand types may be exaggerated by exceptionally high predation rates on late instar larvae resulting from the tethering protocol.

Natural Enemy Abundance and Diversity: Effects on FTC Mortality

Generalist arthropod predators

The composition of the arthropod community during the early-larval period (May 2010) differed between aspen and mixedwood stands ($F_{3, 16}$=4.38, P=0.0196; Appendix 4), due to higher activity of both spiders and beetles in aspen stands (spiders: $F_{1, 18}$=4.72, P=0.0434; beetles: $F_{1, 18}$=6.562, P=0.0196). The carabid beetle communities in aspen and mixedwood forests in May 2010 were not different ($F_{7, 12}$=2.54, P=0.075), but the activity of *Pterostichus pennsylvanicus* was greater in aspen stands

($F_{1, 18}$=17.91, P=0.0005). The differences in arthropod community composition between forest stand types in May 2010 did not result in differences in mortality of early instar larvae between stand types (above). Furthermore, despite high early-instar larval mortality in the presence of arthropod predators, mortality of early instar larvae was not related to the abundance of ants or either of the two most abundant beetle species (Table 2-3), indicating that no arthropod group alone was responsible for predation on early instar larvae. However, at one aspen site with extremely high ant abundance, mortality of early instar larvae was 100% in both years. Early instar larvae interestingly suffered lower mortality at sites with higher spider abundance (Table 2-3; Fig. 2-8a), possibly indicating that spiders are antagonists of common predators of early instar FTC larvae.

The composition of the arthropod community during the pupal period (July 2009, 2010) did not differ between mixedwood and aspen forests in either year (2009: $F_{3, 16}$=1.87, P=0.175; 2010: $F_{3, 16}$=1.13, P=0.366; Appendix 4). Similarly, the carabid beetle communities in aspen and mixedwood forests in July of both years were not different (July 2009: $F_{5, 14}$=2.77, P=0.061; July 2010: $F_{5, 14}$=2.61, P=0.072), but in July 2009 activities of *Pterostichus pennsylvanicus* and *P. adstrictus* were greater in aspen stands (*P. pennsylvanicus*: $F_{1, 18}$=6.06, P=0.024; *P. adstrictus*: $F_{1, 18}$=8.72, P=0.0085). Although pupal predation was higher in aspen stands (above), it was not related to the abundance of arthropod predators more active in aspen stands (*P. pennsylvanicus* and *P. adstrictus*), nor was pupal predation related to spider abundance (Table 2-4). Pupal predation was significantly related to ant abundance, but this pattern was strongly influenced by the single site with very high ant abundance in both years (Table 2-4; Fig. 2-8b).

Avian predators

In both 2009 and 2010, avian abundance and species richness were not different between aspen and mixedwood stands (Table 2-5), although mixedwood stands typically had greater species richness than did aspen stands. Avian diversity, as measured by H', was higher in mixedwood stands in 2010, but not in 2009 (Table 2-5). These results suggest that higher pupal predation and late-instar larval predation by birds in aspen stands were not a result of differences in bird abundance or species richness between stand types. Indeed, late-instar larval predation in 2010 and pupal predation in both years was not related to the overall abundance of birds (larvae: χ^2_1=0.31, P=0.761; pupae 2009: χ^2_1=2,704, P=0.316; pupae 2010: χ^2_1=0.001, P=0.987).

The composition of avian communities differed between aspen and mixedwood stands (2009: $F_{1, 18}$=2.37, P=0.026; 2010: $F_{1, 18}$=3.04, P=0.002). Species that were more abundant in, and unique to, aspen stands included least flycatcher (2009: IV=0.723, P=0.0200; 2010: IV=0.44, P=0.0629), black and white warbler (2009: IV=0.300, P=0.0010), red-winged blackbird (2010: IV=0.1, P=0.0009), yellow warbler (2010: IV=0.4, P=0.0869) and Connecticut warbler (2010: IV=0.4, P=0.0929). Among bird species associated with aspen stands in 2010, predation on late instar larvae increased with the abundance of Connecticut warbler (χ^2_1=21.36, P=0.008), red-winged blackbird (χ^2_1=15.90, P=0.023), and least flycatcher (χ^2_1=15.25, P=0.028; Fig. 2-9a-c). Although these relationships are all strongly influenced by a few sites with high predation rates and high bird abundance, they do indicate that these three bird species in particular may have contributed to the higher late-instar larval predation rates in aspen forest stands. Among bird species associated with aspen stands in 2009 or 2010, pupal predation was only positively related to yellow warbler abundance in 2010 (χ^2_1=8.072, P=0.032). Again, this relationship was

strongly influenced by a single site with complete pupal predation and high yellow warbler abundance (Fig. 2-9d).

Parasitoids

Late-instar larval parasitism rates in 2010 were highly variable, ranging from 0% to 66% among all sites. Across all sites, the most widespread parasitoids were the tachinid fly *Carcelia malacosomae* and the ichneumonid wasp *Agrypon anale*. *Leschenaultia exul (*Tachinidae) and *Hyposoter fugitivus* (Ichneumonidae) caused high parasitism rates at some sites (Appendix 5). There was no difference in larval parasitism rates or the composition of the larval parasitoid community between forest stand types (parasitism: W=49.5, P=0.740; community: $F_{7, 11}$=0.65, P=0.71).

Apparent pupal parasitism rates, estimated from pupae recovered from the open treatments in 2009 and 2010, were also highly variable. Among all sites, pupal parasitism rates ranged from 0% to 100% in both years. There was however no difference in pupal parasitism rates between forest stand types (see above). Pupal parasitoid community composition also did not differ between forest stand types ($F_{7,31}$=1.21, P=0.327), although there was a difference in the community between years ($F_{7,31}$=3.26, P=0.0104) resulting from higher parasitism by *Arachnidomyia aldrichi* in 2009, and higher parasitism by the ichneumonids *Itoplectis quadricingulata* and *I. conquisitor* in 2010.

Discussion

Most natural enemy-caused mortality in low-density FTC populations identified in this study was from generalists, both generalist predators and generalist parasitoids. Among generalists however, the guilds that contributed most to FTC mortality differed across the life history of FTC larvae and pupae. Natural enemy-caused mortality of FTC larvae and pupae was generally similar in aspen and mixedwood stands,

but predation on late instar larvae and pupae was greater in aspen stands. As a result, generational mortality from natural enemies was higher in aspen than in mixedwood stands.

Natural Enemy Impacts Across FTC Life History

Predation

Early instar FTC larvae are vulnerable to predation by birds and arthropods including spiders, ants, beetles and pentatomids. In 2009, results from the exclusion treatments indicated that predators of early instar FTC larvae could reduce larval abundance by approximately 40%. Although, predation by pentatomids can contribute to mortality of early instar FTC larvae, especially if hatch is delayed, and birds have been implicated in the disappearance of entire colonies (Parry et al. 1998), I found no evidence that pentatomids or birds contribute to early instar larvae FTC mortality. The addition of the arthropod exclusion treatment in 2010 demonstrated that loss of early instar larvae was almost exclusively caused by arthropods accessing larval colonies from the ground.

Loss of early instar larvae from the open treatments was not related to the abundance of any specific arthropod group (ants, spiders, or beetles). Ants have been reported to decimate FTC colonies elsewhere in Alberta (Parry et al. 1997), and ant predation results in high larval mortality in other forest insects such as autumnal moth (*Epirrita autumnata*) and gypsy moth (*Lymantria dispar*; Riihimäki et al. 2005, Weseloh 1993, Campbell and Torgersen 1983). Ants were a locally important mortality source in this study, as larval mortality was 100% in both years at the site with the single highest ant abundance (Appendix 4). At sites with low ant abundance however, larval mortality was also higher in the open treatments compared to all other treatments. Despite the clear potential of ants to cause high mortality of FTC larvae, they are not likely

to contribute generally to FTC mortality because of their patchy distribution on the landscape. My results suggest that high mortality of early instar larvae from predation is the cumulative effect of many generalist arthropods opportunistically preying on FTC colonies

Late-instar larval mortality was high in all exclusion treatments in this study, but high unknown mortality obscured trends in total mortality between forest types and among exclusion treatments. Much of the unknown mortality occurred from the tethering protocol; many larvae died after re-collection from the effects of the tether adhesive. Tethers were necessary to estimate natural enemy-caused mortality of late instar larvae because they typically disperse from their natal colony and tree in their 4th and 5th instar (Fitzgerald 1995). This behaviour occurs at both high and low densities, indicating that it may be related to predator and parasitoid avoidance as opposed to foraging (Parry et al. 1997). Tethers ensured that larvae could be found after 24 h., but they prevented larvae from dispersing and also may have interfered with other anti-predation behaviours, such as anterior body "thrashing" (Fitzgerald 1995). Predation rates observed over 24 h. in this study are likely elevated above natural levels of predation on late instar FTC larvae, and cannot be directly scaled across the entire duration of the 4th and 5th instar stages. Similarly, the parasitism rate of tethered larvae exposed to parasitoids may also be elevated above natural parasitism rates. The tether effects were consistent among treatments and forest types, so inferences about effects of forest type and the presence of predators and parasitoids on late-instar larval mortality remain tenable.

In contrast to high arthropod predation on early instar larvae, predation on late instar larvae was largely due to avian predators, particularly in aspen stands. Although early instar larvae are considered palatable to birds (e.g. Pelech and Hannon 1995), later instars are physically defended and are therefore less palatable to avian predators

(Parry et al. 1997, Heinrich 1993, Heinrich 1979). The high incidence of bird predation (up to 55% per day) on late instar larvae in my study was therefore unexpected, given that unpalatable prey items are unlikely to be preferred and targeted by predators, especially at low densities. The timing of late-instar larval predation corresponds to the appearance of bird nestlings, increasing the demand for food resources among insectivorous birds (e.g. Remmel et al. 2009, Parry et al. 1997), and possibly making FTC larvae more appealing prey.

Because of the difficulty of quantitatively assessing predation on late instar larvae, it is only anecdotally reported in the literature from direct observation (Parry et al. 1997) and stomach contents (Witter and Kulman 1979). In this study, the first to quantitatively assess predation of late instar FTC larvae, late-instar larval predation was positively related to the abundance of Least flycatcher *Empydonax minimus*, Connecticut warbler *Oporornis agilis* and red-winged blackbird *Agelaius phoeniceus*, but not to overall bird abundance, suggesting that predation by birds on late instar larvae is by specific species. Of these species, only Least flycatcher has been directly observed preying on late instar larvae in Alberta (Parry et al. 1997). Bird predation is likely to be fairly stochastic among years and locations, depending on encounters between bird predators and FTC prey, unless bird predators of FTC are consistently associated with specific habitats (see below).

Although carabid beetles of the genus *Calosoma* are highly specialized predators of lepidopteran larvae (Larochelle 1990), these beetles were uncommon across all my sites (Appendix 4) and additional predation on late instar FTC larvae by epigaeic arthropods was minimal. Predation that occurred in the predator exclusion treatment was likely due to pentatomids, which I observed attacking tethered larvae on several occasions, and which left behind characteristically exsanguinated larval carcasses.

Generalist predation on FTC pupae ranged from 10 – 30 % in the endemic FTC populations of this study. During outbreaks, predation typically accounts for a similarly small amount (<10-20%) of pupal mortality (Stark and Harper 1982, Hodson 1943). In contrast, Parry *et al.* (1997) reported >90% predation on pupae by avian predators in endemic FTC populations in Alberta, and Glasgow (2006) observed pupal predation rates of 25-45% over two years at intermediate densities. Pupal predation could not be related specifically to the abundance of particular arthropod guilds, although predation rates were high at the single site with high ant abundance. In contrast to late instar larvae, I found only weak evidence that pupal predation was related to individual bird species. Therefore, predation on FTC pupae is similar to predation on early instar larvae in that it is the cumulative result of various guilds of generalist predators.

Parasitism

No mortality of early instar larvae was attributed to parasitism in this study. Only one species of parasitoid, *Aleiodes malacosomatus*, attacks FTC prior to the 4th instar in Alberta (Parry 1995). This braconid wasp typically attacks late 2nd to early 3rd instar FTC larvae, subsequently emerging from late 3rd instar and early 4th instars. My study design prevented quantitative assessment of parasitism by *A. malacosomatus*, because monitoring of early instar larvae ended prior to emergence of the parasitoid. Previous studies of FTC parasitism indicate that *A. malacosomatus* is a common parasitoid in low-density FTC populations and may exert density-dependent mortality (Roland 2000, Parry et al. 1997, Parry 1995, Harmsen and Rose 1983).

Parasitism of late instar larvae, as determined from the broader collections, was highly stochastic, and each species of parasitoid was common at only a few sites. High parasitism rates by the ichneumonids

Agrypon anale and *Hyposoter fugitivus* at several sites were unexpected, considering previous studies have found only very low or no larval parasitism by ichneumonid wasps (Parry 1995, Roth et al. 2006, Witter and Kulman 1979). In contrast, the tachinids *Leschenaultia exul* and *Carcelia malacosomae* are commonly recovered from low-density FTC populations (Parry 1995, Witter and Kulman 1979). Hymenopteran parasitoids of FTC larvae are at least oligophagous, if not broad generalists (Goulet and Huber 1993) and, although they are more specialized (Mondor and Roland 1998, Parry 1995), the tachinid parasitoids may persist in low-density host populations by attacking FTC while larvae are still foraging as a colony (Parry 1995, Witter and Kulman 1979). Thus, generalist parasitoids, or those that are adapted to and common in low-density host populations, were common parasitoids of late instar larvae in this study.

Apparent pupal parasitism was high (45%) in the open treatment, and considerably more consistent across sites than was parasitism of FTC larvae. Ichneumonid parasitoids attack FTC pupae in outbreaking populations, but have not been previously reported in high numbers from endemic populations (Parry 1995, Witter and Kulman 1979). In contrast, *A. aldrichi* is a common facultative parasitoid of FTC pupae in Alberta and elsewhere across a range of host densities (Parry 1995, Witter and Kulman 1979, Hodson 1939). Similar to late-larval parasitoids, pupal parasitoids were comprised exclusively of generalists whose populations can be buffered against low densities of FTC by the presence of alternative prey (Schmidt and Roland 2006). Differences in apparent parasitism between the open and predator exclusion treatments (Fig. 2-6c) suggest that parasitoids were negatively affected by the presence of the predator exclusion treatment. To my knowledge, this has not been reported in any other exclusion experiments on forest defoliators.

Interactions between predators and parasitoids as mortality agents

Neither apparent nor marginal parasitism rates of late instar larvae differed in the presence or absence of predators, suggesting that generalist predators do not or cannot differentiate between parasitized and un-parasitized larvae. FTC larvae parasitized by tachinid flies typically show no external physical evidence of parasitism so birds, as visual predators, may not be able to distinguish between those parasitized and un-parasitized. In contrast to FTC larvae, predators of FTC pupae do appear to distinguish between parasitized and un-parasitized individuals, resulting in a higher marginal parasitism rate of pupae exposed to predators. In a similar study examining bird and beetle predation on FTC pupae Glasgow (2006) also found evidence that predators, especially birds, avoided parasitized FTC pupae. Selective predation on non-parasitized prey is not limited to interactions between birds and FTC pupae; beetle larvae prey on un-parasitized winter moth (*Operophtera brumata*) pupae in the soil more frequently than on pupae parasitized by the tachinid fly *Cyzenis albicans* (Roland 1990).

Selective predation on un-parasitized pupae may reflect lower palatability of parasitized pupae to generalist predators. Pupae parasitized by *Arachnidomyia aldrichi* may be easily detectable by, and unpalatable to, birds because larvae of *A. aldrichi* break down tissues of FTC pupae upon host penetration (Hodson 1939), and selective predation against pupae parasitized by *A. aldrichi* has been observed (Parry et al. 1997). However, differences in the response of marginal parasitism rates of ichneumonids and of *A. aldrichi* to predator exclusion observed in this study suggest that predators were avoiding pupae parasitized by ichneumonids, but not those parasitized by *A. aldrichi.* Predator selectivity for prey not parasitized previously can lead to compensatory mortality among natural enemy guilds (Campbell and Torgersen 1983). Parasitoids did not attack more FTC pupae when predators were excluded, suggesting that

parasitoids do not compensate for the absence of predators. In contrast, avoidance of parasitized pupae by bird predators suggests that birds may attack relatively more pupae in the absence of parasitoids, thus compensating for the absence of parasitoids, although I was unable to test this directly.

Forest Composition and Natural Enemy-Caused Mortality of FTC

According to the natural enemies hypothesis, natural enemy-caused mortality of insects should be greater in more vegetatively diverse habitats as a result of the capacity of these habitats to support a greater diversity or abundance of those natural enemies (Andow 1991, Russell 1989, Root 1973). Contrary to my expectations, I found no evidence that natural enemy-caused mortality of FTC was greater in mixedwood stands compared to aspen stands. In fact, the only differences in FTC mortality between stand types were higher bird predation on late instar larvae and higher generalist predation on pupae in aspen stands. Rather than differences in bird species diversity, it was the presence of specific species in aspen forest stands that was related to high predation rates on late instar larvae in those stands. Natural enemy-caused mortality of late instar FTC does vary with forest composition, but more as a consequence of specific differences in the composition of the avian community rather than species richness or diversity *per se*. This outcome suggests the possibility that some avian predators that are aspen habitat specialists are also able to capitalize on abundant FTC populations when they occur. High avian predation in aspen stands may reflect the specialization of FTC on aspen host-plants in the boreal region, which would result in the frequent spatial and temporal overlap between aspen habitat specialists and abundant FTC populations.

The otherwise similar natural enemy-caused mortality of FTC in aspen and mixedwood forest stands reflects the general similarity of

arthropod predator and parasitoid communities between forest stand types. Forest characteristics besides canopy diversity may have more influence over the natural enemy community. For example, epigaeic arthropods may respond to ground cover characteristics and soil moisture rather than the composition of the overstory (Work et al. 2004, Niemelä et al. 1992) or by the presence of specific tree species than by stand diversity (Vehviläinen et al. 2008, Riihimäki et al. 2005), with consequences for insect herbivore mortality (Riihimäki et al. 2005). The distribution parasitoids may also be determined more strongly by factors other than stand vegetation diversity, such as nectar for adult parasitoids (Cappuccino et al. 1998, Leiuss 1967).

In agroecosystems, plot size affects natural enemy responses to vegetation diversity, largely because natural enemies can respond to vegetation diversity and redistribute themselves more easily among smaller plots (Bommarco and Banks 2003). The lack of a forest stand type effect on the natural enemy communities and subsequent FTC mortality in my study may similarly reflect the scale of forest stands. Although the stands used in this experiment were much larger than plots typically used in agricultural experiments, the arthropods considered in this study are dominated by highly mobile species, especially the carabid beetles (Lindroth 1969), and inter-stand mobility may homogenize carabid beetle communities among stand types (Work et al. 2004). Both avian predators and parasitoids are also highly mobile and would easily move between stands selected for this study and adjacent stands (e.g. Roth et al. 2006). The influence of natural enemies from adjacent stands and the homogeneity of natural enemy communities between stand types may also reflect the proximity of study sites to the edges of stands.

There is accumulating evidence that landscape-scale heterogeneity may have a greater influence than local vegetation diversity on the diversity of natural enemy communities and their impacts on prey

populations (Barbaro et al. 2005, Cronin and Reeve 2005, Jactel et al. 2005, Roland 2000). For example, the bird species diversity responds positively to both the presence of deciduous stands surrounding pine plantations (Barbaro et al. 2005) and heterogeneity in forest cover measured over 100 ha (Drolet et al. 1999). Parasitoids also respond to landscape-scale vegetation diversity. In many agricultural studies, parasitoid diversity and parasitism rates of pests in diverse agricultural landscapes are greater (Cronin and Reeve 2005, Marino and Landis 1996 but see Menalled et al. 1999). Although evidence from forest systems is scarce, spruce budworm (*Choristoneura fumiferana*) parasitism rates are higher in more diverse forest landscapes (Cappuccino et al. 1998), and landscape-scale diversity is associated with reduced infestations of forest insect pests (Koricheva et al. 2006, Jactel et al. 2002). It may therefore also be useful to consider parasitism and predation on FTC in a landscape-diversity, rather than a stand-diversity, context (Roland 2000).

Implications for Endemic FTC Populations

This is the first study to estimate natural enemy-caused mortality across all larval and pupal stages of the forest tent caterpillar. Enemy exclusion treatments targeting specific guilds of enemies (parasitoids, arthropod predators and bird predators in this study) allowed for inferences regarding sources of mortality even when the result of enemy attack was simply the disappearance of the prey. The difficulty of locating FTC larvae and pupae in endemic populations and the use of exclusion treatments in this study necessitated planting FTC egg masses, larvae and pupae on saplings in the understory. In endemic FTC populations, early instar larvae are found high in the canopy where females laid egg masses the previous summer (Batzer et al. 1995). Late instar larvae and pupae are progressively more dispersed vertically through the forest strata, including in the understory, but especially at low densities, the majority

remain in the canopy (Batzer et al. 1995). Experimenting with FTC at endemic densities in the understory has several consequences. First, my observations of early-instar larval mortality in the understory may exaggerate the contribution of epigaeic arthropod predators, which are less likely to locate and attack FTC colonies located high in the canopy. Also, parasitism of FTC by both tachinid and sarcophagid flies is higher in the understory than in the canopy (Parry 1995, Witter and Kulman 1979), so parasitism rates of both larvae and pupae in this study are likely to be slightly overestimated. Conducting a similar experiment on FTC larvae and pupae in the canopy however, would be impractical if not impossible.

In forest insect populations, natural enemies may function both as regulators of low-density endemic populations and drivers behind periodic outbreaks of others (e.g. Klemola et al. 2010, Heisswolf et al. 2009, Klemola et al. 2002, Tanhuanpää et al. 2001, Kidd and Jervis 1997, Parry et al. 1997, Berryman 1996, Gould et al. 1990, Hassell and May 1986, Southwood and Comins 1976). Observations and theory suggest that density-dependent mortality from generalist natural enemies can maintain low-density prey populations, in part because their persistence does not depend on the abundance of a single prey species (Tanhuanpää et al. 1999, Gould et al. 1990, Hassell and May 1986, Southwood and Comins 1976). The contribution of generalist enemies to insect mortality is frequently studied in only one life history stage of the prey (e.g. Glasgow 2006, Elkinton et al. 2004, Tanhuanpää et al. 2001, Teder et al. 2000, Tanhuanpää et al. 1999, Gould et al. 1990). Understanding the full implications of natural enemy-caused mortality on insect populations however requires consideration of impacts across all life-history stages, and the nature of the density-dependence of each mortality factor.

My study was limited to FTC populations at low density, so no inferences can be made about the density-dependence of natural enemy

mortality. However, the results provide the first complete study of natural enemy-caused mortality in low-density FTC populations and provide direction for future studies concerning density dependence. I found that most natural enemy-caused mortality in low-density FTC populations was from generalists, both generalist predators and generalist parasitoids. Among generalists, the guilds that contributed most to mortality changed across the life-history of FTC larvae and pupae; arthropod predators caused high mortality in early instar larvae, mortality of late instar larvae was predominantly from avian predators and predators and generalist parasitoids each contributed to mortality of pupae. This outcome highlights the advantages of examining natural enemy-prey interactions across all life-history stages. Moreover, my results allow comparisons of the relative impacts of mortality during different life stages on total generational mortality and suggest that natural enemy-caused mortality during the late instar larval and pupal stages may have a greater impact on generational mortality than does mortality in the early instars. Natural enemy-caused mortality of FTC varied with forest composition, but contrary to expectation, mortality was higher in the less-diverse aspen habitat. This difference occurred because of specific differences in the enemy community between forest types rather than species diversity or enemy abundance, as implied by the natural enemies hypothesis. Because generalist predation on FTC was greater in aspen forest stands, FTC populations may be slower to reach outbreak levels in aspen compared to mixedwood stands, but this expectation remains to be tested.

Table 2-1. Average temperature and average daily maximum temperature (± standard error) recorded between May 11 and June 13, 2010 in the enemy exclusion, predator exclusion and open treatments at one aspen and one mixedwood site.

Treatment	Average Temperature (°C)		Average Daily Maximum Temperature (°C)	
	Aspen	Mixedwood	Aspen	Mixedwood
Enemy Exclusion	12.4 ± 0.1	12.7 ± 0.2	20.4 ± 1.3	24.9 ± 1.5
Predator Exclusion	11.8 ± 0.1	12.0 ± 0.2	19.2 ± 1.2	21.9 ± 1.3
Open	12.1 ± 0.1	11.6 ± 0.2	20.0 ± 1.3	19.7 ± 1.1

Table 2-2. Model estimates of loss of early instar forest tent caterpillar larvae from open trees in aspen and mixedwood forest stands. ln(survival) was modelled as a linear function of days since hatch, year and forest type. Estimates are the per-day change in ln(# FTC remaining) (slope ± s.e.).

Year	Forest Type	Estimate
2009	Aspen	-0.123 ± 0.035
	Mixedwood	-0.055 ± 0.035
2010	Aspen	-0.086 ± 0.037
	Mixedwood	-0.030 ±0.041

Table 2-3. Effects of arthropod predator abundances and stand type on early instar larval mortality in May 2010. Bold values indicate a significant effect based on logistic regression. All χ^2 tests had one degree of freedom.

Taxon		Effect	χ^2_1	P
Spiders		Abundance	**186.84**	**0.030**
(Araneae)		Forest Type	10.30	0.610
Ants		log(Abundance)	30.92	0.440
(Formicidae)		Forest Type	7.16	0.710
Beetles	*Pterostichus adstrictus*	Abundance	0.06	0.970
(Carabidae)		Forest Type	4.40	0.770
	Pterostichus	Abundance	28.79	0.450
	pennsylvanicus	Forest Type	2.77	0.810

Table 2-4. Effects of arthropod predator abundances and stand type on pupal mortality July 2009 and 2010. Bold values indicate a significant effect based on logistic regression. All χ^2 tests had one degree of freedom.

Taxon		Effect	χ^2_1	P
Spiders		Abundance	0.70	0.561
(Araneae)		Forest Type	5.79	0.094
Ants		log(Abundance)	**28.50**	**<0.0001**
(Formicidae)		Forest Type	1.60	0.320
Beetles	*Pterostichus adstrictus*	Abundance	2.81	0.244
(Carabidae)		Forest Type	4.01	0.164
	Pterostichus	Abundance	3.39	0.201
	pennsylvanicus	Forest Type	3.95	0.168

Table 2-5. Mean (± standard error) total bird abundance, species richness and diversity in aspen and mixedwood forest stands in 2009 and 2010. Means were compared between forest stand types using Wilcoxon's rank-sums tests. Bold values indicate a significant difference between forest stand types.

Year		Aspen	Mixedwood	Wilcoxon's W	P
2009	Abundance	4.9 ± 0.7	4.8 ± 0.4	48.5	0.940
	Species Richness	8.3 ± 0.9	8.9 ± 0.6	55.5	0.702
	Diversity (H′)	1.9 ± 0.1	2.0 ± 0.1	60	0.481
2010	Abundance	2.8 ± 0.7	3.3 ± 0.3	65.5	0.254
	Species Richness	4.6 ± 0.8	6.6 ± 0.4	76	0.051
	Diversity (H′)	1.2 ± 0.2	1.8 ± 0.1	**87.5**	**0.005**

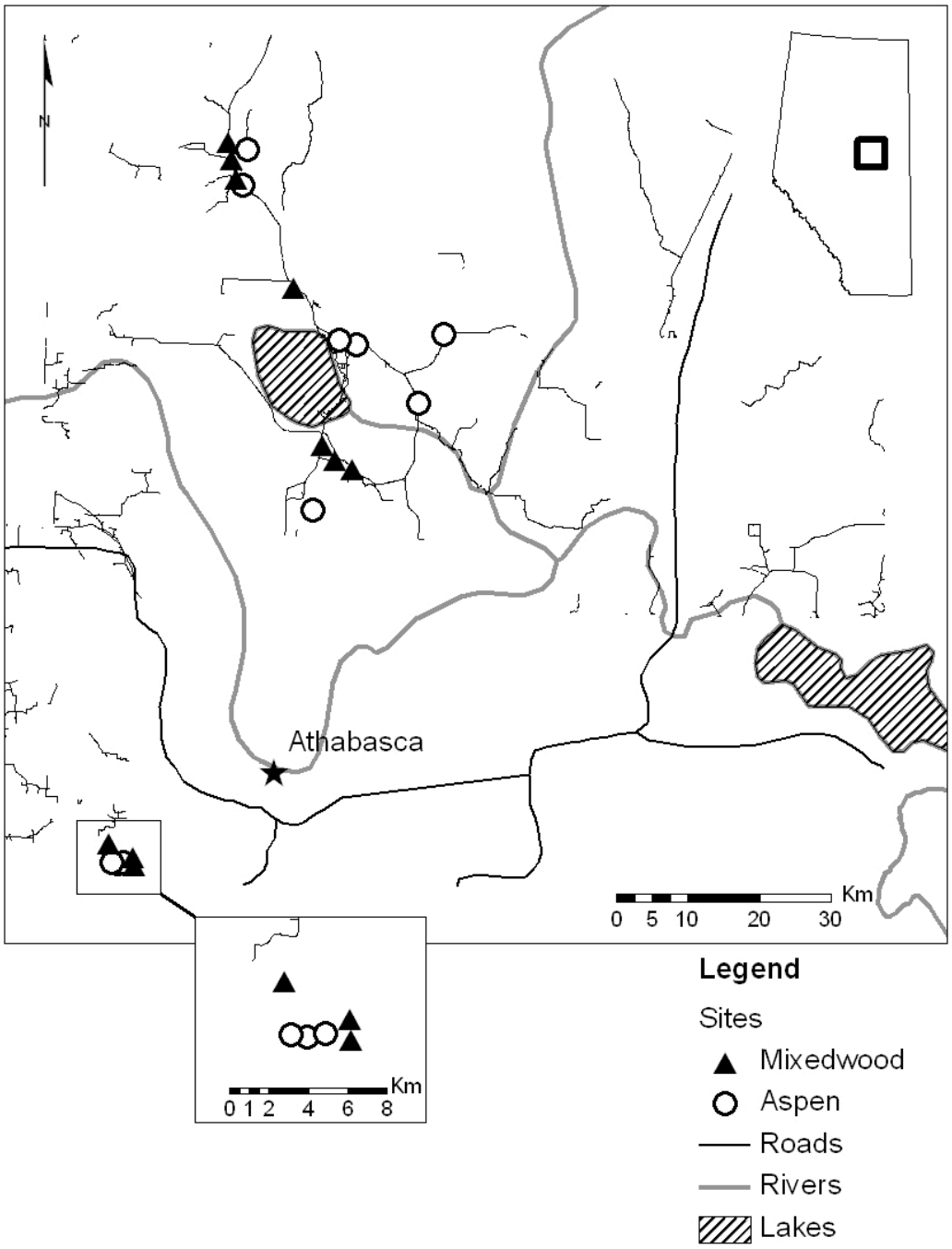

Figure 2-1. Study site locations in north-central Alberta.

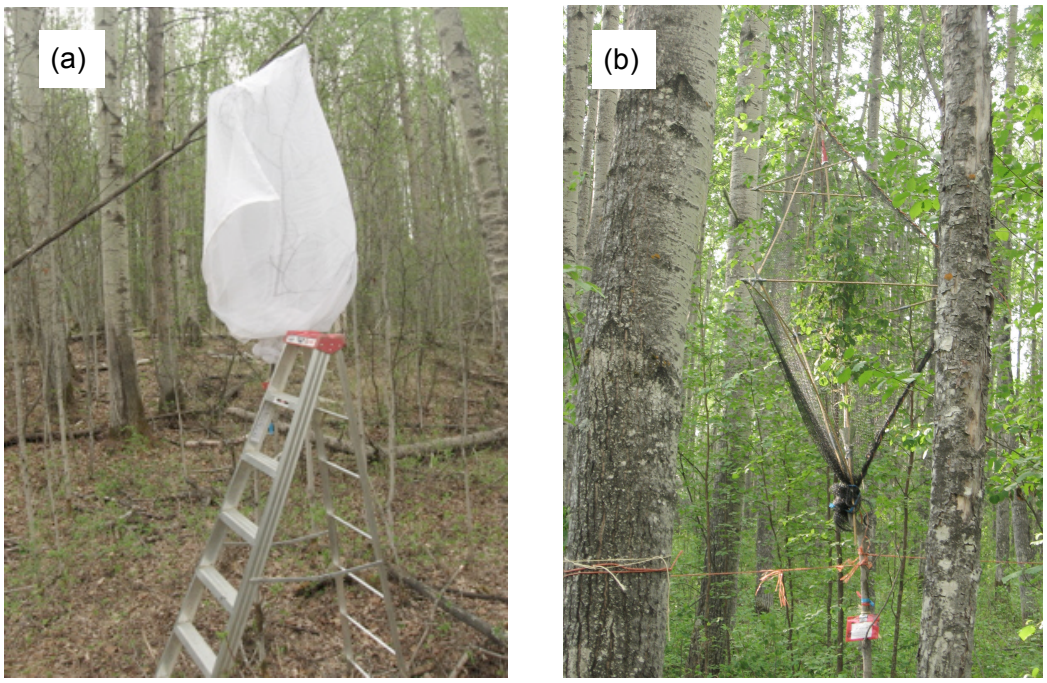

Figure 2-2. Examples of the (a) enemy exclusion treatment, and (b) predator exclusion treatment on aspen saplings in aspen forest stands.

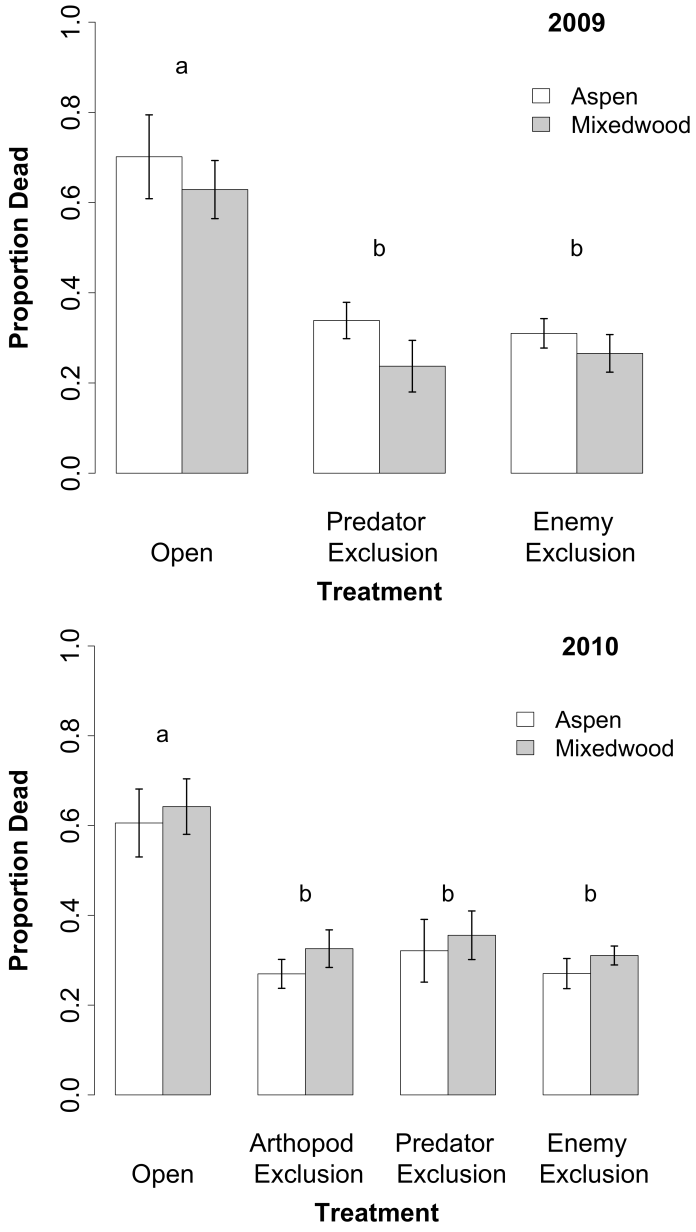

Figure 2-3. Mortality of early instar forest tent caterpillar larvae in aspen and mixedwood forest stands in 2009 and 2010. Exclusion treatments were designed to exclude all natural enemies (enemy exclusion), exclude avian and arthropod generalist predators (predator exclusion) or to exclude arthropod predators (arthropod exclusion; 2010 only). Larvae in the open treatment were exposed to all sources of natural enemy-caused mortality. Letters indicate homogeneous groups within years.

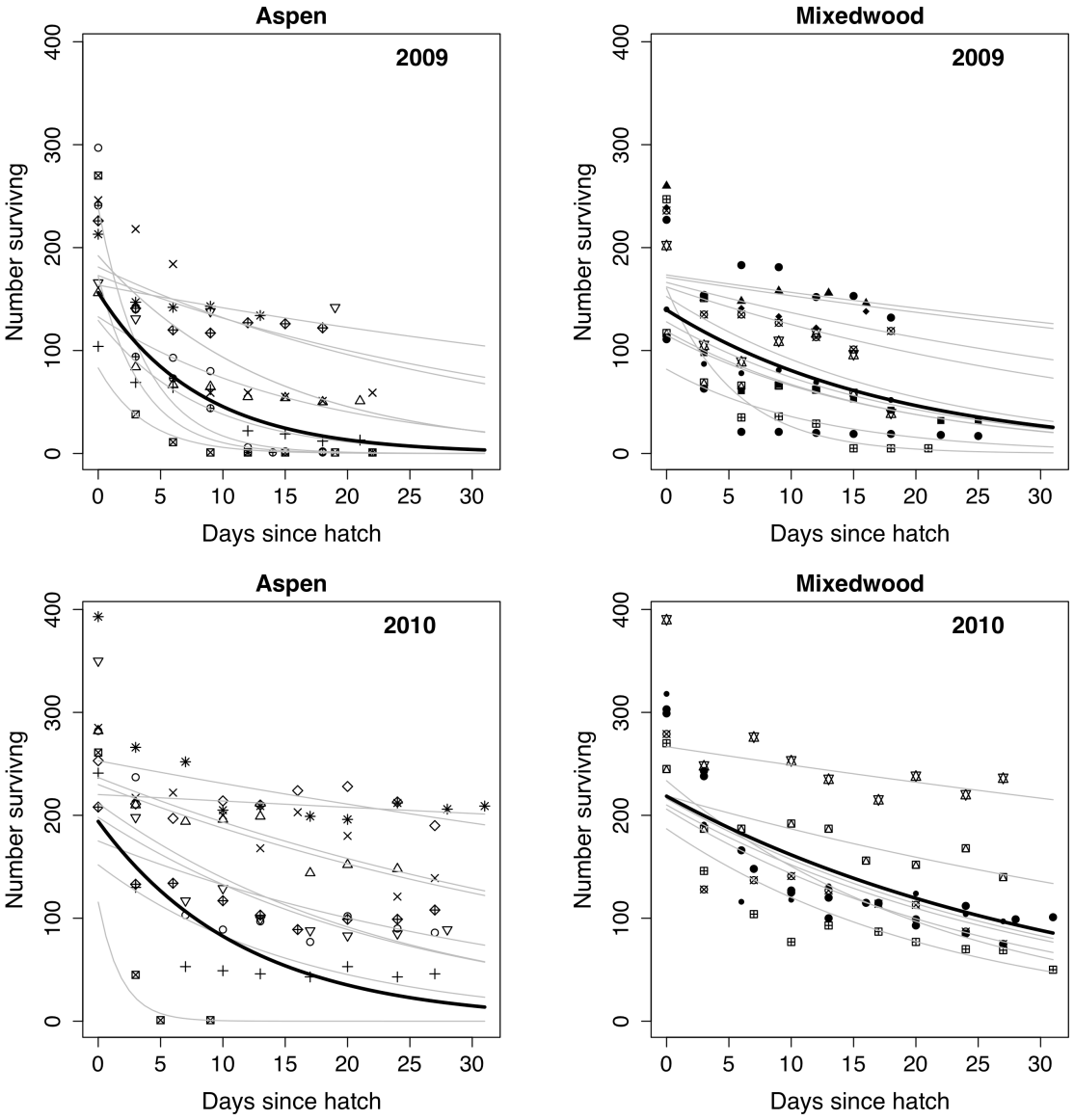

Figure 2-4. Loss of early instar forest tent caterpillar larvae from open trees in aspen and mixedwood forest stands in 2009 and 2010. Symbols fit by grey lines highlight the variation in slopes and intercepts attributable to individual trees. Model predictions of the fixed effects of year and forest stand type are in black. Slope estimates are provided in Table 2-2. Loss rates were not significantly different between years or between forest types.

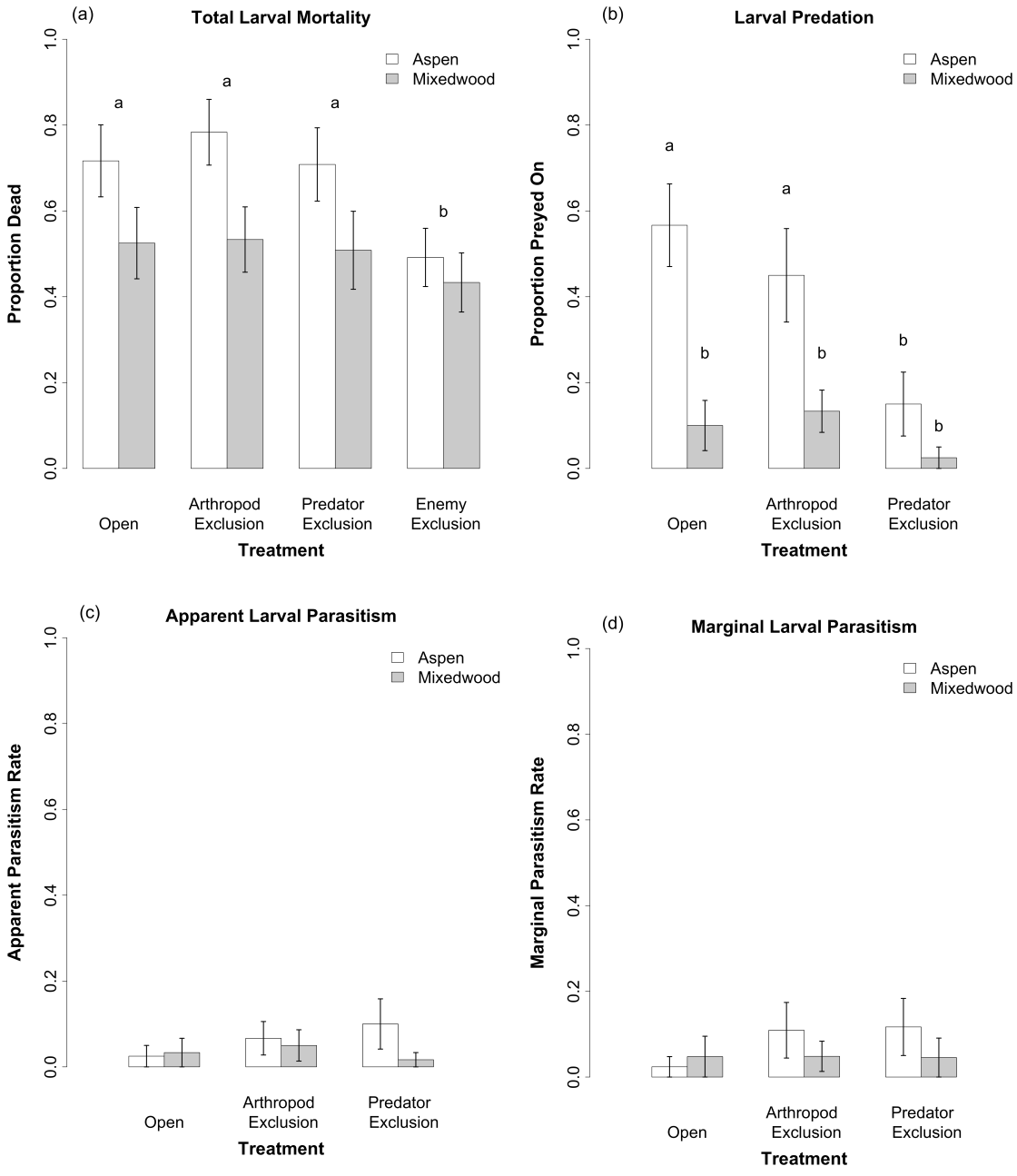

Figure 2-5. (a) Total mortality, (b) predation and the (c) apparent and (d) marginal parasitism rates of tethered late instar forest tent caterpillar larvae in aspen and mixedwood forest stands. Treatments as in Fig. 2-3. Letters indicate homogeneous groups where there are significant differences among treatments or between forest stand types.

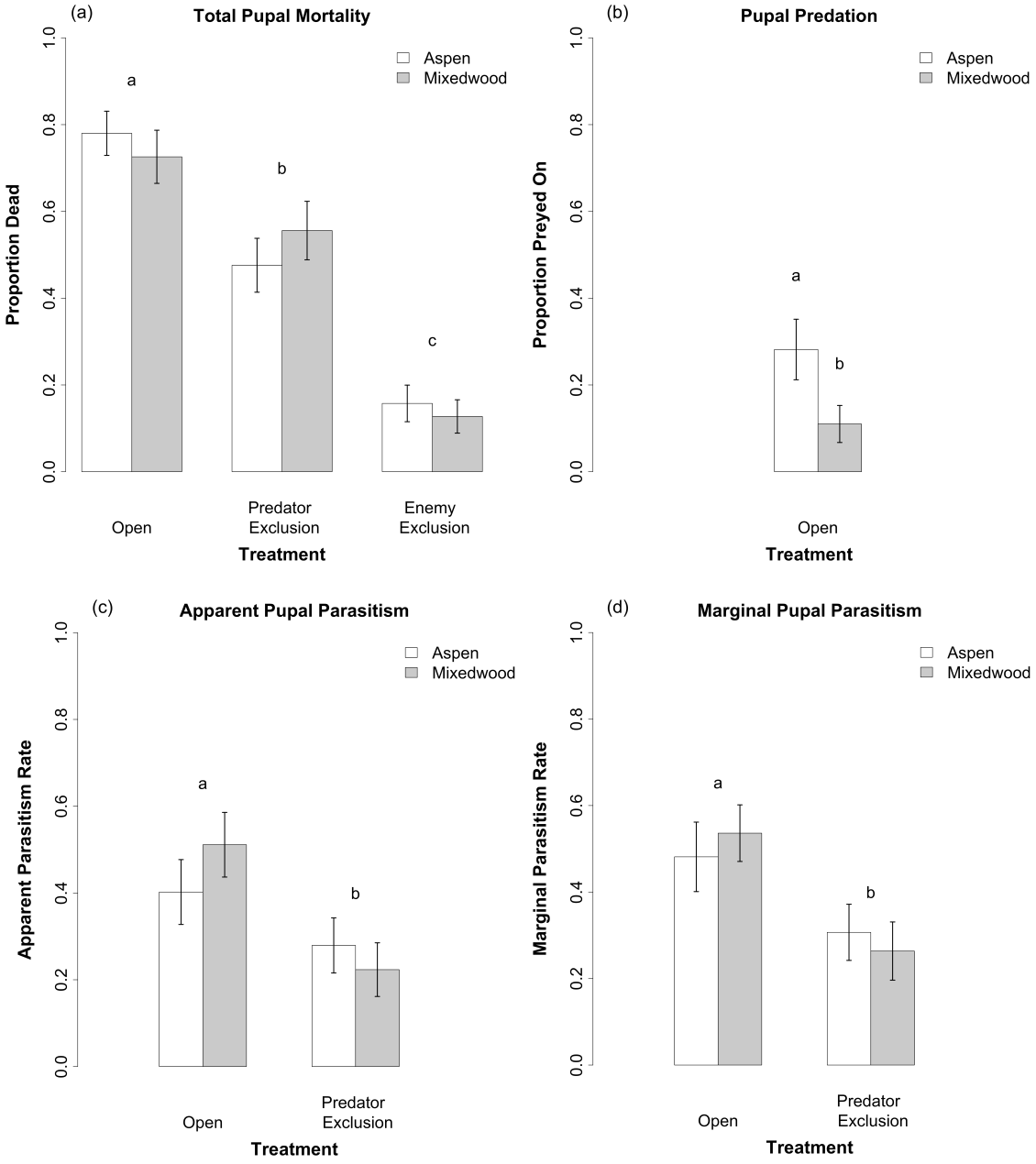

Figure 2-6. a) Total mortality, (b) predation and the (c) apparent and (d) marginal parasitism rates of forest tent caterpillar pupae in aspen and mixedwood forest stands. Treatments as in Fig. 2-3. Letters indicate homogeneous groups where there are significant differences among treatments or forest types.

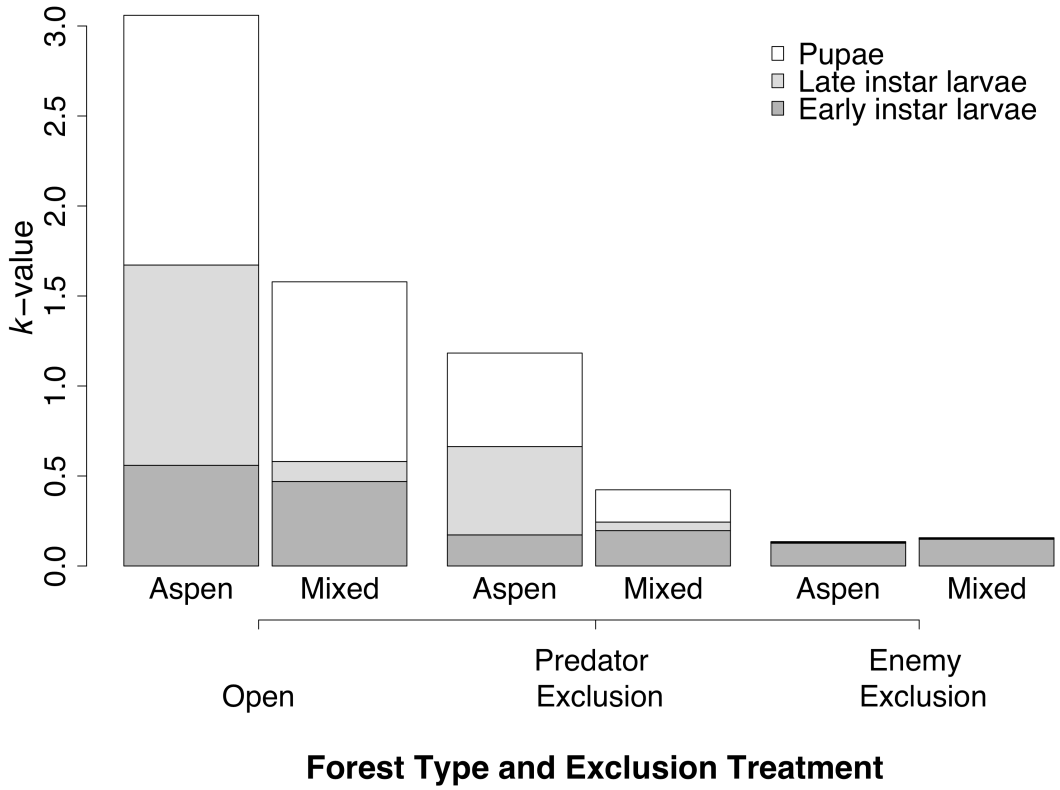

Figure 2-7. *k*-values of mortality of early and late instar larvae and pupae in aspen and mixedwood forest stands in 2010. Differences in *k*-values among life stages (shades of grey) within a bar reflect the relative contribution of mortality in each stage to generational mortality. Differences in *k*-values among exclusion treatments reflect the relative contribution of predators and parasitoids to total natural enemy caused mortality. *k*-values for late instar larvae and pupae include only mortality attributed to natural enemies (predators and parasitoids), and exclude mortality of larvae or pupae from unknown sources. In contrast, *k*-values for early instar larvae include all mortality because specific sources of mortality could not be identified beyond the use of exclusion treatments. Exclusion treatments as in Fig. 2-3. All bars are means across sites in each forest stand type.

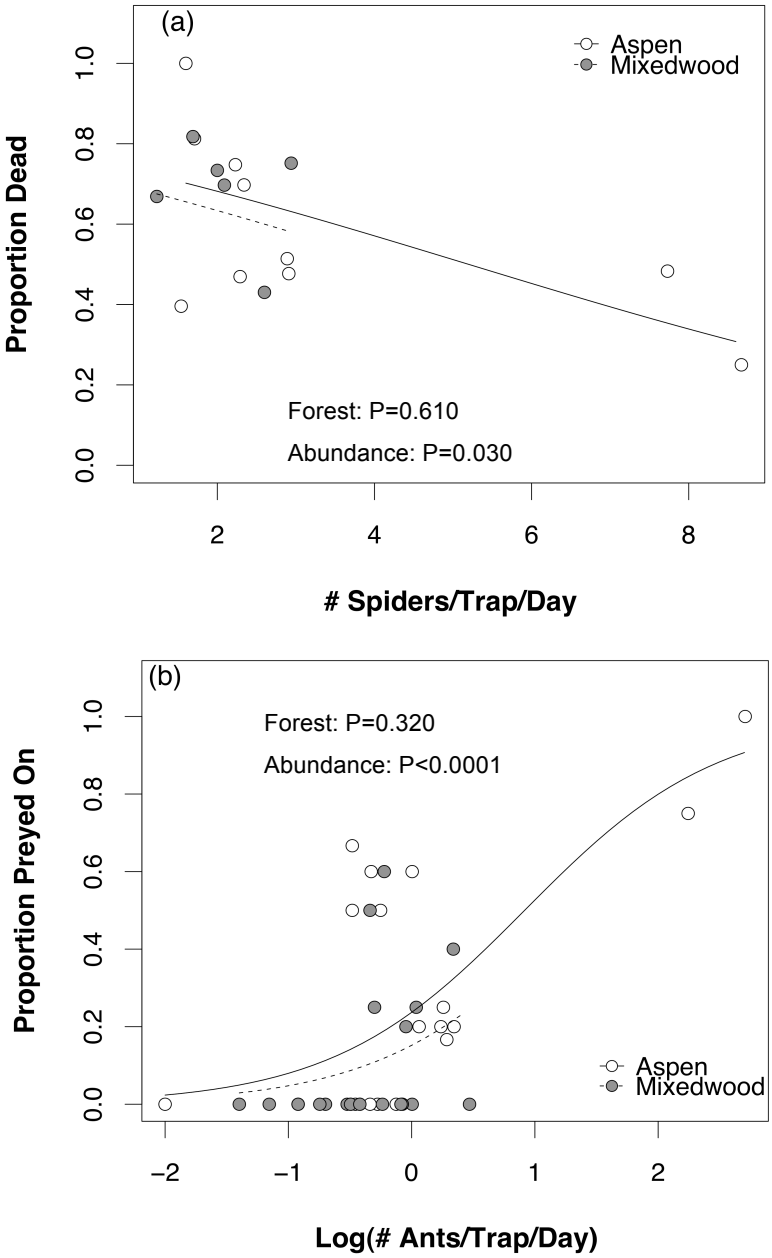

Figure 2-8. (a) Relationship between mortality of early instar FTC larvae on open trees in 2010 and spider abundance in aspen and mixedwood forest stands in May 2010. (b) Relationship between the proportion of depredated pupae on open trees in July 2009 and 2010 and ant abundance in aspen and mixedwood stands. Lines (solid: aspen; dashed: mixedwood) are model fits estimated by logistic regressions of larval mortality or pupal predation on spider and ant abundance.

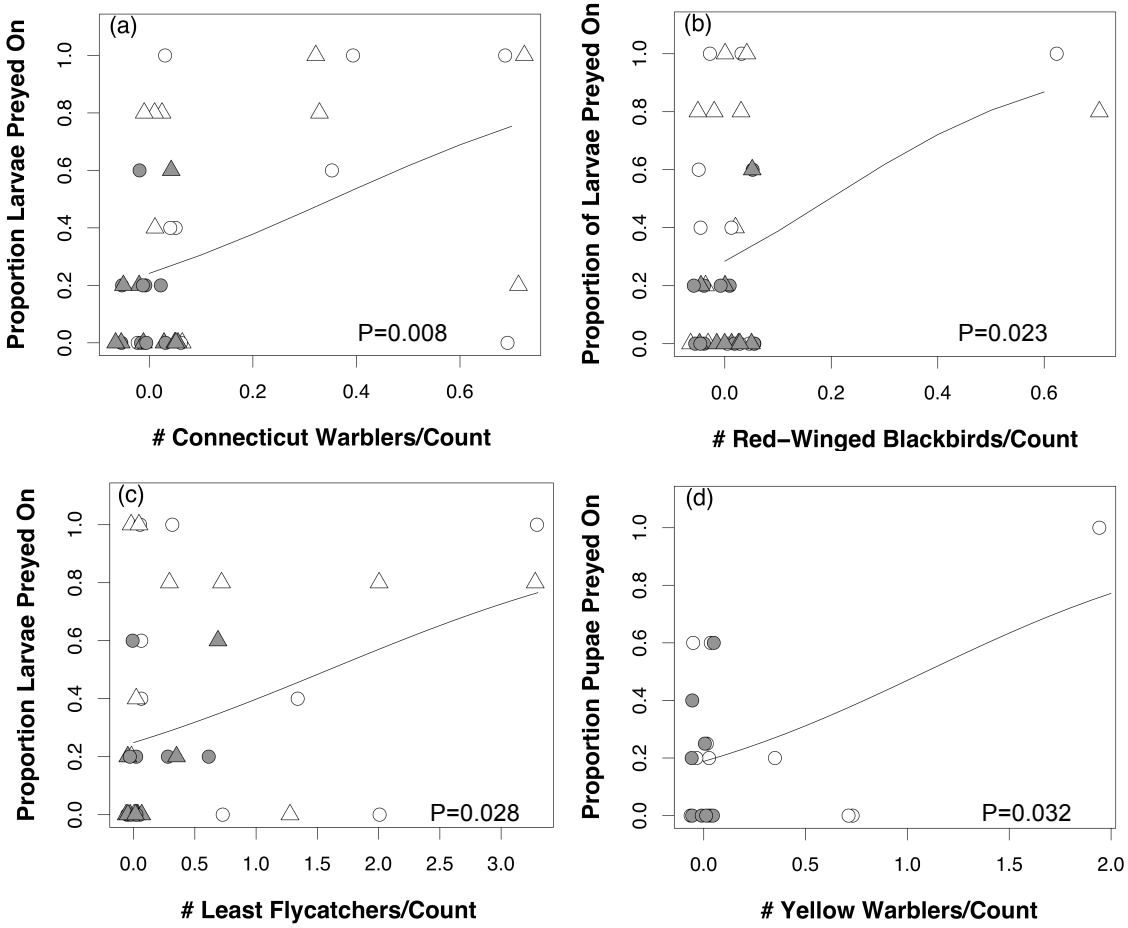

Figure 2-9. (a) – (c) Relationships between the proportions of late instar forest tent caterpillar (FTC) larvae on open trees (circles) and in arthropod exclusion treatments (triangles) preyed on , and the abundance of Connecticut warblers, red-winged blackbirds and Least flycatchers in aspen and mixedwood forest stands (open and grey symbols, respectively). (d) Relationship between the proportions of FTC pupae on open trees preyed on and the estimated abundance of yellow warblers at sites in aspen and mixedwood forest stands (open and grey symbols, respectively) in 2010. Horizontal jitter has been added in all plots to separate overlapping points. Lines are model fits estimated by logistic regressions of larval or pupal predation on bird abundance for each species.

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Chapter 3

The Role of Predation in Governing the Spread of Forest Tent Caterpillar (*Malacosoma disstria* Hübner) Outbreaks

Introduction

Many forest insect populations, of both native and introduced species, periodically outbreak and cause widespread defoliation (e.g. Bjørnstad et al. 2010, Tenow et al. 2007, Cooke and Lorenzetti 2006, Royama et al. 2005, Bjørnstad et al. 2002, Cooke and Roland 2000,). Outbreaks can be spatially dynamic, frequently spreading out or travelling across forested landscapes (Cooke et al. 2009, Tenow et al. 2007, Johnson et al. 2004, Bjørnstad et al. 2002). Among forest pests, the forest tent caterpillar (*Malacosoma disstria* Hübner; FTC) is a widespread cyclic defoliator of aspen (*Populus tremuloides*) in the boreal forest. Both the population cycle period and outbreak duration vary geographically, but typically FTC populations cycle with an approximately decadal periodicity and resulting local outbreaks can last 2-3 years (Cooke et al. 2009, Sippell 1962). In Canada, FTC outbreaks are spatially dynamic (e.g. Fig. 3-1, Cooke et al. 2009), but the processes governing the spatiotemporal patterns, especially the spread of a FTC outbreak, are not clear.

Population spread is a direct result of dispersal to, and population growth in, low-density populations (e.g. Hastings et al. 2005) so processes that prevent the increase of low-density populations may be crucial to governing the rate of spread of a FTC outbreak. Allee effects, negative effects of decreasing population density on individual fitness or per-capita

population growth rates, are a common dynamical mechanism in small populations (reviewed in Kramer et al. 2009, Courchamp et al. 2008, Courchamp et al. 1999, Stephens et al. 1999). Component Allee effects are negative effects of low population density on individual fitness and may be generated through several mechanisms relating to reproduction (e.g. mate finding, broadcast spawning) or survival (e.g. predator satiation; reviewed in Courchamp et al. 2008, Gascoigne and Lipcius 2004). A component Allee effect is necessary, but not always sufficient, to cause a population-level demographic Allee effect, which is a negative effect of low population density on the per-capita population growth rate (Courchamp et al. 2008, Berec et al. 2007). Demographic Allee effects may be strong, leading to declining population growth rates at low density, or weak, where per-capita population growth rates slow but remain positive as population density declines (Courchamp et al. 2008, Berec et al. 2007, Courchamp et al. 1999). In a population with a strong Allee effect, the Allee threshold is the population density below which the per-capita growth rate is negative (Courchamp et al. 2008, Courchamp et al. 1999).

Demographic Allee effects influence the spread rates of invading populations. Theoretical results indicate that the presence of a strong Allee effect in the dynamics of a spreading population can slow population spread (Taylor and Hastings 2005, Kot et al. 1996, Lewis and Karieva 1993). Population spread rates are also reduced in the presence of a weak Allee effect if it reduces population growth rates sufficiently (Wang and Kot 2001). Empirically, variation in the invasion speed of gypsy moth (*Lymantria dispar*) is associated with the strength of the local demographic Allee effect (Tobin et al. 2009, Tobin et al. 2007, Johnson et al. 2006a), which results from a mate-finding component Allee effect (Contarini et al. 2009). Although much research regarding the influence of Allee effects on spread rates has focused on invading populations (e.g.

Taylor and Hastings 2005), the same processes may be operating in the spatial dynamics of native insect outbreaks, like those of FTC.

Interactions between natural enemies and spreading populations can also govern the speed of population spread (e.g. Fagan et al. 2002). For example, depending on whether there is a demographic Allee effect in the prey population, predation can slow or even reverse the spread of an invading species (Fagan et al. 2002, Owen and Lewis 2001). Very mobile natural enemies can also restrict the spatial extent of an outbreak (Maron and Harrison 1997). However, predators themselves can also cause demographic Allee effects in prey populations, depending on the nature of their aggregative and functional responses to prey density (Gascoigne and Lipcius 2004). For example, a predator with a type II functional response to prey imposes high per-capita prey mortality in low-density populations, with declining per-capita prey mortality as prey population density increases (Kramer and Drake 2010, Gascoigne and Lipcius 2004). This positive relationship between survival and prey density represents a component Allee effect that can produce a demographic Allee effect in the prey population if predation is a major source of mortality (Kramer and Drake 2010, Courchamp et al. 2008, Gascoigne and Lipcius 2004). Predator-caused Allee effects have been proposed as the cause of population decline in several natural systems, including woodland caribou (*Rangifer tarandus caribou*; Wittmer et al. 2005) and Atlantic salmon (*Salmo salar*; Ward et al. 2008), and have been experimentally demonstrated to reduce growth rates and increase extinction probabilities of microcosm prey populations (Kramer and Drake 2010). Although the roles of both natural enemies and Allee effects in dictating spread rates of invading populations are widely appreciated separately, the degree to which predator-caused Allee effects can reduce the spread rate of prey populations has not been throuroughly considered; however, these predator-caused Allee effects may influence the spread of FTC outbreaks.

While the periodic outbreak dynamics of FTC populations are considered to be, in part, a result of interactions with a suite of specialist parasitoids (Roland 2005, Roland and Taylor 1997, Parry 1995), a variety of generalist arthropod and avian predators also attack FTC throughout their life cycle (Witter and Kulman 1972). Generalist predators can cause up to 30% pupal mortality in low-density FTC populations (Chapter 2), but the consequences of variation in generalist predation on FTC dynamics have not been explicitly considered. The objectives of this study are to: 1) Determine how generalist predation affects the local dynamics of a FTC population and the spread of a FTC outbreak; and 2) Determine to what degree a predator-caused Allee effect influences the outbreak spread rate.

I first describe the local dynamics of FTC populations with a set of difference equations that include the dynamics of the FTC population and a specialist parasitoid, including mortality from a generalist predator. Of particular interest in the non-spatial model is determining under what conditions the generalist predator can cause a demographic Allee effect in the FTC population. I then expand the non-spatial model to a set of integrodifference equations describing the spatiotemporal dynamics of a FTC outbreak and consider the impact of generalist predation, including potential Allee effects, on the outbreak spread rate.

Methods

Model Formulation

Non-spatial model

To model local interactions between FTC, a specialist parasitoid and a generalist predator, I use a Nicholson-Bailey host-parasitoid model (Nicholson and Bailey 1935), modified to include density-dependent growth in the host population and the effect of a generalist predator with a type II functional response (Holling 1965). Variations of modified

Nicholson-Bailey models are commonly used to describe host-parasitoid-predator interactions generally (Hassell 2000, Hassell and May 1986, Hassell 1978), but also specifically for the FTC system (Cobbold et al. 2009, Cobbold et al. 2005). This discrete-time model is appropriate for organisms with non-overlapping generations, such as univoltine insects like FTC (Turchin 2003, Hassell 2000, Hassell 1978). In the model, the FTC and parasitoid populations are coupled such that FTC abundance influences the reproduction and subsequent abundance of parasitoids in the next generation. In contrast, the abundance of the generalist predator is considered independent of the FTC population, so the generalist predator population is not modelled explicitly.

In field studies of FTC, populations are commonly censused using time-limited cocoon counts (counts of pupae collected during a 15 minute search; Roland and Taylor 1995), so model FTC populations are also censused at this life stage, with H_t describing the number of pupae present in the summer (beginning of year *t*; Fig. 3-2). Events occurring through the winter, spring and subsequent summer determine the final number of pupae surviving at the end of the next summer (beginning of year *t*+1; Fig. 3-2).

Adults emerge from pupae in mid-summer and females lay egg masses that overwinter on branches in the canopy. The number of first instar larvae (H_L) hatching in the spring of year *t* depends on the number of pupae surviving to adulthood in the previous summer (H_t) and the intrinsic growth rate of the FTC population (*r*):

$$H_{L_t} = H_t \cdot e^r \qquad (1)$$

After hatching, larvae proceed through five larval instars over the course of 8 – 10 weeks. During this time, a specialist parasitoid attacks larvae while scramble competition among larvae for resources occurs. The

number of larvae that survive competition and parasitism and subsequently pupate (H_P) in year *t* is modelled as:

$$H_{P_t} = H_{L_t} \cdot e^{\frac{-rH_{L_t}}{K}} \cdot e^{-aP_t} \qquad (2)$$

where H_{L_t} is given by Eq. 1. In equation 2, the fraction of FTC larvae surviving scramble competition ($e^{\frac{-rH_{L_t}}{K}}$) is determined by the Ricker equation for density dependence (Cobbold et al. 2009, Turchin 2003, Beddington et al. 1975) and depends on both the intrinsic growth rate (*r*) and the carrying capacity (*K*) of the FTC population. The fraction of larvae surviving parasitism is governed by a linear functional response of the parasitoid attacking FTC (e^{-aP_t}) that depends both on the abundance of parasitoids in the current year (P_t) and the searching efficiency of the parasitoid (*a*) and assumes a random encounter rate. Although FTC larvae are attacked by a suite of parasitoids that vary in their degree of specialization (Parry 1995, Witter and Kulman 1972), I make the simplifying assumption that parasitism is limited to a single, specialist parasitoid that attacks fourth instar larvae and emerges prior to FTC pupation. The dynamics of the parasitoid are coupled to the dynamics of FTC:

$$P_{t+1} = H_{L_t} \cdot (1 - e^{-aP_t}) \cdot e^{\frac{-rH_{L_t}}{K}} \qquad (3)$$

with the abundance of parasitoids in the subsequent year (P_{t+1}) depending on the abundance of 1st instar larvae in the current year (H_{L_t}; Eq. 1), and the fraction of those larvae that are parasitized ($1 - e^{-aP_t}$). In addition, because parasitoids attack FTC larvae before the onset of intraspecific competition, the abundance of parasitoids in the subsequent year also depends on the fraction of hosts that survive intraspecific competition ($e^{\frac{-rH_{L_t}}{K}}$).

The pattern of generalist predation on FTC pupae over a range of FTC abundances is consistent with a type II functional response (Glasgow 2006, *Predation Parameters* below). A type II functional response of a generalist predator causes a component Allee effect in the prey population, which may result in a demographic Allee affect if predation is sufficiently high, with consequences for outbreak spread rates. I model generalist predation on those FTC larvae surviving competition and parasitism and that subsequently pupate (H_P) according to a discrete-time type II functional response (Turchin 2003, Hassell 1978, Rogers 1972) that determines the final abundance of surviving pupae to begin year *t*+1 (H_{t+1}):

$$H_{t+1} = H_{P_t} \cdot e^{\frac{-bZT}{1+bhH_{P_t}}} \qquad (4)$$

The type II functional response is described by the 'random parasite equation' of Rogers (1972) where the proportion of FTC pupae surviving predation depends on predator abundance (*Z*), FTC pupal abundance (H_{P_t}; Eq. 2), predator handling time (per prey item; *h*), per-predator searching efficiency rate (*b*) and the duration of the predation period (*T*). This functional response equation assumes that predators search randomly and that prey may be re-encountered; it is used here to describe predation of FTC pupae by generalist predators because a successful attack and removal of a FTC pupa from a cocoon does not remove the cocoon from the pool of apparently available prey. To simplify parameter estimation (see *Predation Parameters* below), I let: $b'=bZT$ and $h'=h/ZT$. This manipulation assumes that the total predation period and the generalist predator population do not vary with the abundance of FTC pupae or with time.

Equation 4 then becomes:

$$H_{t+1} = H_{P_t} \cdot e^{\frac{-b'}{1+b'h'H_{P_t}}} \qquad (5)$$

where b' is the cumulative searching efficiency of all predators over the duration of the predation period and h' is the proportion of the total time spent handling prey items per predator per prey.

In the absence of dispersal, H_{t+1} and P_{t+1} are the local abundances of FTC pupae and parasitoid adults present at the beginning of year t+1.

In a discrete time model, the order of events can impact the model outcomes (e.g. Cobbold et al. 2009, Hassell and May 1986, May et al. 1981). For simplicity, I only consider one possible chronology that describes interactions among FTC, a specialist parasitoid and a generalist predator over a year. Other chronologies are certainly possible; for example, the addition of generalist predation on early instar larvae (Chapter 2).

Spatial model

To examine how predation influences the spatial dynamics of FTC outbreaks in one-dimensional space, I use a set of integrodifference equations in which the discrete time difference equations of the non-spatial model are integrated over continuous space. The integrodifference equation is comprised of two distinct phases: a dispersal stage and a sedentary stage where reproduction and mortality occurs (Kot 1992). The sedentary stage is described by the local dynamics in the non-spatial model (above). The dispersal of both adult hosts and parasitoids is represented by a continuous redistribution function, or dispersal kernel (Kot 1992) that describes the probability of an individual moving from any location, Y, to location X. The abundance of FTC or parasitoids at location X after dispersal is determined by integrating the dispersal kernel across all locations Y in the spatial domain, Ω, from which individuals may disperse. For simplicity, only the Laplace dispersal kernel is considered, with dispersal parameters that describe the average dispersal distance of adult FTC moths or adult parasitoids (d_H and d_P, respectively, Eq. 6a, b).

$$H_{t+\tau}(X) = \int_{\Omega} \frac{1}{2d_H} \cdot e^{\left(\frac{-|X-Y|}{d_H}\right)} \cdot H_t(Y)\ dY \qquad (6a)$$

$$P_{t+\tau}(X) = \int_{\Omega} \frac{1}{2d_P} \cdot e^{\left(\frac{-|X-Y|}{d_P}\right)} \cdot P_t(Y)\ dY \qquad (6b)$$

The Laplace dispersal kernel models a greater proportion of long distance dispersal events than would the Gaussian (random diffusion) redistribution function (Kot et al. 1996). In applications of dispersal models to biological systems, including some representation of long-distance dispersal provides better agreement with field observations than relying on random diffusion to model dispersal (Kot et al. 1996).

The spatial model for the FTC and parasitoid dynamics depends on dispersal of adult moths emerging from surviving pupae (H_t) or parasitoid adults (P_t) and is the first event to occur after populations are censused, before FTC reproduction, intraspecific competition and natural enemy-caused mortality (Fig. 3-2). Thus, in the spatial model, $H_{t+\tau}$ and $P_{t+\tau}$ replace H_t in Eq. 1 and P_t in Eq. 2 and Eq. 3, respectively.

Parameter Estimation

FTC population growth, parasitism and dispersal parameters

I relied on estimates of the parameters of FTC population growth (*r*, *K*) and parasitoid searching efficiency (*a*) from field data used by Cobbold et al. (2009; Table 3-1). Both *K* and *a* were estimated by Cobbold et al. (2009) from data obtained from time-limited cocoon counts. The value of *r* was estimated from the average size of egg masses produced by FTC females and estimates of generational mortality (not caused by natural enemies; Cobbold et al. 2009).

Dispersal of both FTC moths and parasitoid adults is difficult to characterize, so the dispersal parameters (d_H, d_P) could not be estimated directly from data. Observations made by Roland and Taylor (1997, 1995)

suggest that FTC moths are capable of dispersal farther than parasitoid adults, and that parasitoids may disperse 300 – 800 m, depending on the species. In these simulations, I assumed an average dispersal distance of 1 km for FTC adults (d_H) and 0.8 km for parasitoid adults (d_P). Given the uncertainty of these parameters, I examined the sensitivity of the model outcomes to variation in average dispersal distance and to the relative dispersal distances of moths and parasitoids.

Predation parameters

I relied on observations of predation on FTC pupae collected between 1993 and 2008 near Cooking Lake and Rocky Mountain House, AB (Roland unpubl.) to determine the shape and to estimate the parameters of the functional response of generalist predation on FTC pupae (b' and h'; Eq. 5). Data consisted of time limited cocoon counts (Roland and Taylor 1995) with collected pupae scored as healthy, parasitized or preyed on. Cocoon counts in which predation was not observed (i.e. zeros; 536 out of 967) were removed. The final data set contained 430 observations of FTC pupal abundance (cocoons/15 minutes) and number of pupae preyed on (cocoons/15 minutes) from 252 sites collected between 1993 and 2007.

I used a two-step approach to characterize the shape and to estimate the parameters of the functional response of generalist predation on FTC pupae (Juliano 2001). First, I used logistic regression to determine the shape of the functional response, and then used non-linear least squared regression to estimate the parameters of the most suitable functional response form. To confirm that the proportion of pupae preyed on across a range of FTC densities was consistent with a type II functional response (Holling 1965), I fit a cubic function of cocoon abundance to the logit-transformed proportion of available cocoons preyed on with a quasibinomial error distribution to account for overdispersion (Crawley

2007). In this polynomial model, a negative first order term is consistent with a type II functional response, whereas a positive first order term is consistent with a type III functional response (Juliano 2001).

I estimated the parameters of the type II functional response, b' and h' (Eq. 5), by fitting the following equation to the data using non-linear least squares regression:

$$H_e = H - H \cdot e^{\frac{-b'}{1+b'h'H}} \quad (7)$$

where H is the observed abundance of FTC pupae (cocoons/15 minutes) and H_e is the number of pupae preyed on. Non-linear regression to estimate the parameters of this model is preferred to estimating the parameters of a linearized version, as parameter fits from linearized models frequently provide biased estimates of parameters (Juliano 2001, Williams and Juliano 1985). To further confirm that a type II functional response accurately described generalist predation on pupae, I compared the fit of the estimated type II functional response to that of a type I functional response (i.e. h' =0) using AIC scores.

All statistical analyses were conducted with =0.05 in R v 2.12.1 (R Development Core Team 2010, Vienna, Austria) using functions available in the stats package.

Numerical Simulations

Non-spatial model

The behaviour of a similar non-spatial model, excluding the generalist predator, has been considered in depth by Cobbold et al. (2009) who found that the FTC population exhibits cycles as a result of the lagged density-dependent relationship between FTC abundance and parasitoid-caused mortality. I simulated the non-spatial model over 50 years to examine the effect of adding a generalist predator on the local dynamics. I investigated the effects of changes in both parasitoid and

predator searching efficiency (*a* and *b'*, respectively) on cycle period by calculating the average time between FTC population peaks in the last 500 years of a 1000-year simulation.

I examined the effect of stochasticity in *r*, the intrinsic growth rate of FTC populations, in generating peak abundances in the FTC population well above the fluctuations generated in the non-spatial deterministic model (*i.e.* in generating outbreaks). Because FTC populations are very sensitive to between year variation in weather, especially in winter and spring (Cooke and Roland 2003, Roland et al. 1998), including some stochasticity in the intrinsic growth rate may generate exceptionally abundant FTC populations with a frequency that mirrors the frequency of outbreaks observed across their range. In each time step of the simulated stochastic model, *r* was drawn from a normal distribution with a mean (μ=0.9), and a standard deviation (σ = 0.3, 0.5, 0.7 or 0.9) that describes the degree of variation in *r*. While Cobbold et al. (2009) suggested a mean estimate (*r*=0.9), they admitted that *r* could easily vary between 0.4 and 1.3 based on observed variation in egg mass size. A standard deviation of 0.5 produces a distribution of values of *r* in which approximately 65% of values are within this range.

I simulated the stochastic model, assuming each standard deviation separately, for 1000 iterations of 200 years. I examined the number of peaks exceeding a threshold abundance of 90 cocoons/15 min in the final 150 years of each iteration to determine the average outbreak interval (number of years between outbreaks) for each level of stochasticity. Although this threshold abundance is well below the carrying capacity (*K*=260), it corresponds to a 50% increase above the maximum abundance predicted by the deterministic model (Results), and to approximately 50% defoliation (Cobbold et al. 2009). Because stochasticity in *r* influenced the persistence of both the parasitoid and FTC populations, I also determined

whether these populations were extinct at the end of each 200-year iteration, with FTC populations considered extinct if $H_t<0.01$.

To determine if the component Allee effect in the FTC population caused by type II predation on pupae generates a demographic Allee effect, I quantified realized per-capita growth rates ($R_t = \ln(H_{t+1}/H_t)$) across a range of host abundances. Using simulations of the non-spatial model with values of b' and h' estimated from the predation data, I determined the slope of the relationship between FTC abundance and realized per-capita growth rate. A positive slope at low FTC abundance would indicate a demographic Allee effect. I further explored the roles of the parameters of the type II functional response (b' and h') in generating demographic Allee effects by considering the parameter space in which a demographic Allee effect is produced.

Spatial model

I relied on numerical simulations of the spatial integrodifference equations to determine the effects of variation in generalist predation on the spread of a FTC outbreak. However, analytical tools have been developed to determine the speed of similar travelling wave invasions in a variety of models, including single species reaction–diffusion or integrodifference models with and without Allee effects (e.g. Wang and Kot 2001, Kot 1992, Kot et al. 1996, Lewis and Karieva 1993) in two-species reaction-diffusion models with stable co-existence predator-prey waves (Fagan et al. 2002, Owen and Lewis 2001) and in reaction-diffusion models with oscillatory predator-prey dynamics (periodic travelling waves; e.g. Sherratt and Smith 2008).

I measured the spread of FTC outbreaks in two ways, each using a different feature to define the leading edge of the outbreak. First, I calculated the spread rate of the FTC population front (P_F), the point where $P_F=0.5(\max H)$, or the FTC abundance that is half the maximum

abundance observed in a given year. This measure differs slightly from previous measures of the position of a spreading front (e.g. Lewis and Karieva 1993), because FTC population abundance does not remain stable behind the spreading front. Instead, the location of the maximum FTC abundance is sensitive to the dynamics governing the collapse of the outbreak behind the spreading front. Second, I used a measure that reflects the spread of easily detectable defoliation, which may be more relevant for forest management than would the less obvious population front. I calculated the spread rate of a defoliation front (D_F), defined by D_F= 50 cocoons/15 min, which is a FTC pupal abundance that is consistent with 30-40% defoliation (Cobbold et al. 2009), and is approaching the maximum abundance observed in the deterministic non-spatial simulations (Results, below). These two measures permit description of the spreading population, and indicate whether the population is sufficiently large to cause meaningful defoliation.

Initial abundances of FTC and parasitoids were set to zero (H_0 =0; P_0 =0) across the spatial domain (Ω: -50 km ≤ X ≤ 50 km) except for a 3 km long 'outbreak' (H_0 =130; P_0 =13) at the centre (-1.5 km ≤ X ≤ 1.5 km), where FTC populations were at carrying capacity and parasitoid populations were one-tenth the FTC abundance. These initial conditions assume that the FTC population is spreading into an unoccupied landscape; there were no FTC or parasitoid populations initially outside of the outbreak. In contrast to the absence of FTC and parasitoids, the model assumes that the generalist predator is at a constant, non-zero abundance across the entire spatial domain. FTC outbreaks typically last only 1-2 years at a single location (Cooke et al. 2009), so I simulated the spatial model for 5 years, and calculated the spread rates of both P_F and D_F over this duration. I assumed periodic boundary conditions wherein any individuals dispersing outside the spatial domain re-enter at the opposite

side. Because the total spatial domain was much longer than the domain influenced by the outbreak over the duration of the simulation, this assumption should have little influence on the results. For simplicity, I present results from $X \geq 0$, but because the spatial domain is symmetrical the outcomes are the same for $X \leq 0$.

Because both predator searching efficiency (b') and handling time (h') influence the shape of the functional response, and therefore the presence and magnitude of a demographic Allee effect in the host population, I examined the effects of varying b' and h' on spread rates of both P_F and D_F.

Spread rates depend directly on both dispersal and the growth rate of the spreading population (Hastings et al. 2005). Given the uncertainty in the dispersal parameters and in the intrinsic growth rate, I examined the sensitivity of outbreak spread (spread rates of P_F and D_F) to the intrinsic growth rate, to the ratio of FTC and parasitoid dispersal distances (d_H and d_P, respectively), and to the distance dispersed by both FTC and parasitoids, assuming a constant ratio of FTC and parasitoid dispersal (0.80). Sensitivity (S) was calculated as the absolute value of the ratio of the proportion change in the response (spread rate) to the proportion change in the parameters (Haefner 2005).

All calculations were performed in R v 2.12.1 (R Development Core Team 2010, Vienna, Austria), using discrete fast Fourier transforms (available in the stats package) to solve the integrodifference equations.

Results

Parameter Estimation

Predation parameters

The proportion of pupae preyed on decreased with increasing cocoon abundance (Fig. 3-3a). The coefficient of the first order (linear)

term of the best-fit cubic logistic regression was significantly negative (Table 3-2), indicating that a type II functional response is a suitable description of generalist predation on FTC pupae. Non-linear least squares regression of Eq. 7 provided estimates (95 % CI) of b'=0.067 (0.052, 0.091) and h'=0.043 (0.022, 0.067) of the type II functional response (Table 3-1, Fig. 3-3b). The type II functional response (AIC=2508.1) modelled the data better than did a linear functional response where h'=0 (AIC=2524.0). The number of pupae preyed on as predicted by the type II functional response model approaches an asymptote of approximately 15 cocoons/ 15 min, despite abundances greater than 500 cocoons/15 min observed in the field (Fig. 3-3b). This model of generalist predation on FTC pupae therefore predicts very low predator-caused mortality for pupae in all FTC populations, but especially so for large populations.

Non-Spatial Model

Deterministic and stochastic outcomes

In the non-spatial deterministic model, FTC populations cycle with an approximately decadal periodicity (Fig. 3-4a), which is consistent with field observations of FTC population abundances and defoliation (Cooke et al. 2009, Cooke and Lorenzetti 2006, Roland 2005) and with results obtained from a similar model by Cobbold et al. (2009). Peaks in parasitoid-caused mortality lag behind peaks in host abundance because of the lagged density-dependence inherent in the host-parasitoid interaction. Mortality caused by generalist predators is low (< 10%; Fig. 3-4a), reflecting the low maximum number of pupae potentially preyed on (model asymptote in Fig. 3-3b), and exhibits subtle fluctuations driven by fluctuations in FTC abundance. Increasing predator searching efficiency (b') by an order of magnitude from 0.067 to 0.67 increases the maximum mortality of FTC caused by generalist predators to 42% (Fig. 3-4b).

The presence and period of cycles in the modelled FTC population depends on the values of both parasitoid searching efficiency (a) and predator searching efficiency (b'; Fig. 3-5a, b), with cycles occurring if $a > 0.023$. Parasitoids that are very efficient (high a) cause very high mortality in large FTC populations, resulting in dramatic declines from peak abundance of the FTC, and subsequently in the parasitoid population and thus producing very low parasitoid population abundances. As a result, the parasitoid population is slow to respond to eventual increases in the host population, thus prolonging the duration of the abundant phase and producing longer host population cycle periods when parasitoids are more efficient. Increases in predator searching efficiency (b') also lengthen the FTC population cycle period. However, in contrast to the interaction with parasitoids, high mortality from efficient generalist predators in small FTC populations prolongs the duration of the low-abundance phase and therefore the cycle period (compare Fig. 3-4a, b), rather than prolonging the duration of the high-abundance phase.

The non-spatial deterministic model predicts low FTC abundances (<60 cocoons/15 min; $<{}^1/_4 K$) throughout the cycle that, at maximum abundance, correspond to approximately 40% defoliation (Fig. 3-4a; Cobbold et al. 2009). This result is in direct contrast to frequent field observations of severe (100%) defoliation in many areas across the range of FTC (e.g. Cooke and Lorenzetti 2006, Cooke and Roland 2000, Roland 1993, Sippell 1962). Although the deterministic model never predicts outbreak abundances of FTC pupae, it may accurately represent the intrinsic population dynamics, which are then modulated by abiotic stochastic factors such as weather (Cooke and Roland 2003, Roland et al. 1998). In fact, when the intrinsic growth rate (r) is modelled as a stochastic parameter, the model can predict 'outbreak' FTC abundances (>90 coccons/15 min; >50% defoliation), depending on the level of stochasticity assumed (σ). When variation in r is low (σ=0.1), the stochastic model

never predicts outbreak FTC abundances (Table 3-3). However, as the standard deviation of *r* is increased from σ=0.3 to σ=0.7, the predicted outbreak interval decreases from 2344 y to 23 y (e.g. Fig. 3-6; Table 3-3), indicating FTC outbreaks become more frequent with increased variation in the intrinsic growth rate of the FTC population. The probability of both FTC and parasitoid extinction in the stochastic model also increases with σ (Table 3-3).

Demographic Allee effects

Although the type II functional response of generalist predation on FTC pupae causes a component Allee effect in FTC populations (Courchamp et al. 2008, Gascoigne and Lipcius 2004), predation does not cause a demographic Allee effect in the FTC population when modelled with parameters estimated from field data (cross in Fig. 3-7). However, increases in predator searching efficiency (b') and handling time (h') do produce a demographic Allee effect in the FTC population (black zone in Fig. 3-7). The demographic Allee effect is always weak, wherein the realized per-capita growth rate (R_t) of the FTC population is never below the Allee threshold and FTC populations always grow (e.g. Fig 3-8b, c).

Increased predator searching efficiency (b') has an overall negative effect on R_t and causes a demographic Allee effect (Fig. 3-7) because more efficient predators can cause greater FTC mortality, especially in small populations, thereby reducing the per-capita growth rate in these populations (compare Fig. 3-8a, b). In contrast, increased handling time (h') has an overall positive effect on R_t, despite inducing a demographic Allee effect (Fig. 3-7), because an increase in handling time reduces predator efficiency, especially at higher densities. Reduced predation pressure on large FTC populations increases the per-capita population growth rate in these populations (higher asymptote; compare Fig. 3-8b, c).

Spatial Model

Using the parameter estimates in Table 3-2, the deterministic spatial model predicts spreading rates of 1.76 km yr^{-1} and 1.52 km yr^{-1} for the FTC population front (P_F) and the defoliation front (D_F), respectively (Fig. 3-9). The FTC populations at the original location of the outbreak (X=0) collapse immediately because of mortality imposed by parasitism and intraspecific competition (Fig. 3-9). The spread rate of P_F is determined by the relative locations of peak FTC abundance and the forward spreading tail, and is sensitive to changes in the location of peak abundance and to changes in FTC population growth rates. In contrast, the spread rate of D_F is determined more by the maximum spatial extent of large FTC populations and is more directly sensitive to FTC population growth rates than is P_F. The spread rates of both P_F and D_F are much slower than that observed for spread of defoliation in both Alberta and Ontario, which indicate spreading rates of up to 40 km yr^{-1} (e.g. Fig. 3-1, Cooke et al. 2009).

Effects of generalist predation on spread rates

Variation in the spread rate of both P_F and D_F caused by manipulation of the predation parameters (b' and h') is within ± 30% of the spread rates obtained with estimated parameters with an order of magnitude change in both b' and h' (Fig. 3-10a, b). Increasing predator efficiency (b') reduces the spread rate of both P_F and D_F, regardless of the predator handling time and has a larger effect on the spread rate of D_F compared to P_F (Fig. 3-10a, b). Furthermore, when b' exceeds ~0.2 and h' is low (i.e. predators are very efficient), no defoliation front exists because FTC populations do not exceed the defoliation threshold abundance (50 cocoons/15 min) anywhere in the spatial domain after 5 years (Fig. 3-10b). Thus, the spread of FTC populations sufficiently large to cause severe defoliation is more sensitive to predation than the spread of FTC

populations themselves. Efficient predators not only decrease the rate of spread of an outbreak, but also quell the outbreak, reducing both its spatial extent and its severity.

The demographic Allee effect produced in the FTC population by increasing h' (described for the non-spatial model, Fig. 3-7) does not result in a consistent decrease in the spread rate of P_F and D_F as expected with a demographic Allee effect in the spreading population (e.g. Wang and Kot 2001, Lewis and Karieva 1993). When predators are efficient (high b'), initially increasing predator handling time (h') above zero causes a slight reduction in the spread rate of P_F (transect "a" in Fig. 3-10a). This reduction in spread rate reflects the demographic Allee effect in the FTC population caused by predation governed by these parameter values (compare Fig. 3-7 and Fig. 3-10a). The demographic Allee effect results in slower population growth in less abundant populations, reducing population spread. However, further increases in h' result in faster spread of P_F (transect "a" in Fig. 3-10a). The reduction in FTC mortality at all abundances because of less efficient predators (increased handling time; Fig. 3-8 b, c) and the resulting higher per-capita population growth rates overcomes any reduction in per-capita growth rates in low-density FTC populations caused by predation. When b' is low, increasing h' always results in faster spread of P_F (transect "b" in Fig. 3-10a), and the spread rate of D_F increases with h', regardless of the value of b' (Fig. 3-10b), reflecting the strong positive effect of reduced predation pressure caused by inefficient predators (high h' or low b') on per-capita population growth and outbreak spread.

Model sensitivity to dispersal and FTC intrinsic growth rate

The outcomes of the model are generally not sensitive to changes in the ratio of FTC and parasitoid average dispersal distances, d_H and d_P (Table 3-4), however the influence of the ratio of dispersal distances on the

spread rates of P_F and D_F differs (Table 3-4). As d_P is increased to equal or exceed d_H, the FTC population is less able to escape the parasitoid population in space, resulting in higher FTC mortality, lower per-capita growth rates and abundances and therefore reduced spread of D_F (Table 3-4). If parasitoids disperse sufficiently farther than FTC moths, FTC populations are held below the defoliation threshold by parasitoids ahead of the spreading FTC population (Table 3-4). In contrast, increasing d_P relative to d_H increases the spread rate of P_F because the location of P_F depends on the location of the peak FTC abundance relative to the tail. Increased parasitoid dispersal reduces FTC abundances across the spatial domain, including abundance near to the initial outbreak, such that peak FTC abundance is actually further from the initial outbreak and P_F spreads more quickly.

In contrast to the response of the outbreak spread rates to changes in the ratio of FTC and parasitoid dispersal, the response of outbreak spread is more sensitive to the magnitude of the dispersal parameters (Table 3-5) and of the intrinsic growth rate (Table 3-6). Increasing the intrinsic growth rate of the FTC population or the average dispersal distance of FTC and the parasitoid, while maintaining a constant dispersal ratio, causes large increases in the spread rates of P_F and D_F (Table 3-5, Table 3-6). Prediction of spread rates consistent with field observations (20-40 km yr^{-1}; Fig. 3-1, Cooke et al. 2009) requires $d_H \approx$ 20-40 km.

Discussion

The outcomes of the non-spatial model indicate predation by generalists is not a primary source of forest tent caterpillar pupal mortality and does not cause a demographic Allee effect in FTC populations. A more efficient generalist predator causes higher FTC mortality, extends the period of the FTC population cycles, and is able to cause an Allee

effect in the FTC population. More efficient generalist predators also reduce the spread rate of a FTC outbreak. However, when the predation parameters are modified to cause a demographic Allee effect, outbreak spread rates are influenced more by the resulting increased inefficiency of predators than by the predator-caused demographic Allee effect, such that the predator-caused Allee effect has little additional influence on the spread of the outbreak. Generalist predation on pupae that is more efficient than that described by the field data used for parameter estimation has the potential to reduce the spread rate of an FTC outbreak in the field, but Allee effects caused by generalist predation contribute very little to the effect of predation on the spread rate of an outbreak.

Allee Effects Caused by Predation

Predators can cause a component Allee effect in prey populations by reducing prey survival in low-density populations (Courchamp et al. 2009, Gascoigne and Lipcius 2004, Courchamp et al. 1999). This component Allee effect may result in a demographic Allee effect in prey populations, leading to reduced growth rates or extinction (Courchamp et al. 2009, Gascoigne and Lipcius 2004, Courchamp et al. 1999). For a predator with no aggregative response, as is assumed in my model, a type II functional response is sufficient to generate a component Allee effect in the prey population (Gascoigne and Lipcius 2004). Other combinations of predator aggregative and functional responses will also create Allee effects in prey populations (a type II functional response with a sigmoid aggregative response, for example), whereas some will not (Gascoigne and Lipcius 2004). Therefore, careful consideration was given to the shape of the functional response describing generalist predation on FTC pupae. A type II functional response of generalist predation on FTC pupae was well supported by observations of pupal predation in this study and is

consistent with detailed observations of generalist predation on FTC pupae (Glasgow 2006).

In my non-spatial model of FTC populations, the component Allee effect caused by the generalist predator had the potential to produce a demographic Allee effect in the FTC population, but did not do so with the parameters estimated from field data. Central to the theoretical production of demographic Allee effects caused by generalist predation is that predation must be a primary source of mortality (Courchamp et al. 2008, Gascoigne and Lipcius 2004). The shape of the type II functional response of generalist predation on FTC pupae predicts that predators consume few FTC pupae, even when FTC abundance is high. As a result, the non-spatial model predicted consistently low FTC mortality (<10%) from generalist predation. Relative to other density-dependent mortality factors, including up to 90% mortality from parasitism, predation was not a sufficiently large source of mortality to generate a demographic Allee effect in the FTC population. Ultimately, the release from the negative density-dependent factors of intraspecific competition and parasitism offsets the component Allee effect of increased predation risk in small FTC populations and mitigates the formation of the demographic Allee effect (Courchamp et al. 2008, Stephens et al. 1999).

Data used to estimate generalist pupal predation were obtained from pupae that were not only exposed to predation, but also to parasitism. Generalist predators of FTC pupae frequently avoid parasitized individuals, as evidenced by field observations of pupal mortality in the presence and absence of predators (Chapter 2, Glasgow 2006) and by direct observation of predator avoidance of parasitized FTC pupae (Parry et al. 1997). This additional natural interaction may limit accurate quantification of the unique impacts of generalist predators on pupal survival, which may be considerably higher in the complete absence of parasitoids. However, the low mortality due to predation predicted by

the non-spatial model is inconsistent with field observations of 10-40% pupal predation, even when parasitoids are present (Chapter 2, Glasgow 2006), indicating that predation is somewhat under-represented in my model.

Although no predation did not cause a demographic Allee effect in the model with the parameters estimated from field data, it was possible to generate a weak demographic Allee effect in the FTC populations by manipulating the parameters of the functional response. The weak demographic Allee effect produced slower population growth rates in small FTC populations in the non-spatial model, but, because growth rates remained above the Allee threshold, the FTC populations were not driven to extinction (Courchamp et al. 2008, Stephens et al. 1999, Courchamp et al. 1999). Weak Allee effects are consistent with field observations of regular captures of male moths in pheromone traps, even in years when FTC populations are very small, indicating that small FTC populations rarely go extinct between population peaks (Roland pers. comm.).

Predator-Caused Allee Effects and Outbreak Spread

Demographic Allee effects in spreading populations can markedly reduce spread rates (Taylor and Hastings 2005). Theoretical results indicate that including an Allee effect in a spreading population can slow the spread of an invading population (Lewis and Karieva 1993). Empirical results from gypsy moth (*Lymantria dispar*), for example, show that the local strength of the mate-finding Allee effect influences spread rates (Tobin et al. 2009, Tobin et al. 2007). Much work has also considered the role of natural enemies in governing the spread rates of prey populations. Owen and Lewis (2001) demonstrated that specialist enemies slow or reverse prey population spread rates if they disperse farther than the prey and if there is an Allee effect in the prey-only dynamics. The impact of a generalist predator on spreading prey populations has also been

considered theoretically in the context of biological control (Magal et al. 2008, Fagan et al. 2002). Generalist predators can reduce the spread rates of prey populations, regardless of the dynamics of the prey population, because the generalist predator can persist ahead of the spreading prey population and reduce growth rates of the prey population at the leading edge (Fagan et al. 2002). To my knowledge, mine is the first study to explicitly consider predator-caused Allee effects and their impact on prey population spread rates.

Consistent with the outcomes of previous models (Fagan et al. 2002), my spatial model predicted approximately 30% slower spread rates of both the population front and the defoliation front with more efficient predators (high b', low h'). Predators that were more efficient also reduced FTC populations below the defoliation threshold, effectively quelling the outbreak. The large increase in predator searching efficiency associated with this reduction in spread corresponds to an increase in predator-caused mortality of pupae from approximately 10% to approximately 40% (Fig 3-4a, b), which is within the range of pupal mortality observed in the field (Chapter 2, Glasgow 2006). Therefore, predators that are more efficient may meaningfully reduce FTC outbreak spread rates. However, the predator-caused Allee effect in the FTC population generated by manipulating the parameters of the functional response caused little additional reduction in FTC outbreak spread rates.

The generation of a demographic Allee effect depended on the values of predator handling time (h') and searching efficiency (b'), with higher h' inducing an Allee effect for a given b'. However, increases in h' caused predators to become less efficient, thereby reducing the overall mortality experienced by the prey population. This reduction in total prey mortality generally outweighed the effects of the demographic Allee effect, resulting in increased spread rates with increases in h'. The only exception was for very efficiently searching predators (very high b'),

where a slight increase in *h'* above zero caused a small reduction in the spread of the FTC outbreak. The lack of influence of the predator-caused Allee effect on FTC spread rates likely reflects that the Allee effect was very weak. However, determining the generality of this result and how the strength of the demographic Allee effect may influence the outcome requires analysis that is beyond the scope of this paper.

The Role of Stochasticity in FTC Outbreak Generation

FTC outbreaks are highly stochastic; even though the average outbreak interval is approximately decadal across much of its range (Cooke and Roland 2007, Cooke and Lorenzetti 2006, Sippell 1962), some regions may be free of detectable outbreaks for several decades (Cooke and Lorenzetti 2006). Environmental variation, both spatially and temporally, may contribute to variation in outbreak frequency and duration. In particular, FTC populations respond negatively to both low overwintering temperatures (Cooke and Roland 2003) and cool early spring temperatures (Roland et al. 1998), with local climate and weather proposed as partial determinants of FTC outbreak duration (Cooke and Roland 2000, Roland et al. 1998). The effects of local climate and weather on spatial and temporal outbreak patterns may reflect the effects of environmental stochasticity on FTC population growth rates.

In agreement with Cobbold et al. (2009), my non-spatial model predicts FTC population cycles with a period similar to that observed in the field. However, my non-spatial model predicted maximum FTC pupal abundances much lower than those observed during and outbreak. When I added stochasticity to the intrinsic growth rate (r; σ=0.7), the model predicted FTC 'outbreaks' (>90 cocoons/15 min; ~50% defoliation) once every 23 years. This result, which corresponds reasonably well with field observations of outbreak frequency in some locations (Roland unpubl., Cooke and Lorenzetti 2006), highlights the role of stochastic influences on

the intrinsic growth rate in producing FTC outbreaks. The stochastic model also predicted occasional extinction of FTC populations and frequent extinction of parasitoid populations. Although these extinctions are a direct result of the variation in *r*, they do not reflect an unreasonable degree of stochasticity because they occur on a local scale. In a field setting, both the FTC and parasitoid populations may be rescued by dispersal from neighbouring areas. Future spatial models of FTC outbreaks could consider incorporating stochasticity in the population growth rate, especially given the sensitivity of outbreak spread rates to the intrinsic growth rate.

Model Sensitivity to Dispersal

The spread rates estimated for FTC outbreaks are highly sensitive to the average dispersal distance of FTC adults. Although dispersal distances for FTC adults are difficult to estimate, model predictions of outbreak spread rates similar to those seen in the field (Fig. 3-1, Cooke et al. 2009) would require FTC adults to disperse unreasonably far (20-40 km, on average). One possible reason for the incongruence between predicted and observed spread rates is that dispersal was not accurately represented in the model. In particular, correct modelling of long-distance dispersal is required to accurately predict spread rates (Clark et al. 1998, Kot et al. 1996, Andow 1990). Although the Laplace dispersal kernel assumed in my model has fatter tails than a Gaussian dispersal kernel, and therefore models relatively more long-distance dispersal (Kot et al. 1996), it may still under-represent true long-distance dispersal by FTC adults.

A more precise representation of FTC adult dispersal may require that most adults disperse locally, while a few travel very long distances, via wind currents for example (e.g. Brown 1965). This stratified dispersal may be better represented by a dispersal function that is even more leptokurtic than the Laplace dispersal kernel (Clark et al. 1998, Kot et al.

1996). Alternatively, local and long-distance dispersal can be modelled separately, either with a mixed dispersal kernel as suggested for plants (Higgins and Richardson 1999, Clark et al. 1998) or with separate diffusion and advection terms as used to describe flying insect dispersal elsewhere (e.g. Takahashi et al. 2005, Allen et al. 2001). Improved modelling of occasional long distance dispersal by FTC moths may result in predicted spread rates that are more congruent with observations, but choosing between alternative model structures may depend on first obtaining more detailed FTC dispersal data.

Future Directions

A critical simplification in my spatial model is that the FTC outbreak of interest occurs in isolation and spreads into a landscape otherwise unoccupied by FTC populations. Analyses of long-term field observations of spatio-temporal patterns of insect outbreaks suggest that the spatial dynamics of outbreaks may result from lagged spatial synchrony among adjacent populations (Tenow et al. 2007, Johnson et al. 2006b, Johnson et al. 2004, Bjørnstad et al. 2002,). Lagged spatial synchrony can produce travelling waves of outbreaks that, in the case of the larch budmoth (*Zeiraphera diniana*), travel over 200 km yr^{-1} (Bjørnstad et al. 2002). Importantly, the speed and direction of these travelling waves is not a direct result of immigration subsidies through dispersal (Tenow et al. 2007, Johnson et al. 2006b). A spatial tri-trophic model of the larch budmoth indicates that landscape variation in habitat quality is sufficient to produce directional travelling waves that originate in areas of high connectivity because of stronger over-compensatory dynamics (Johnson et al. 2006b). Observations of the spatio-temporal dynamics of FTC are also suggestive of travelling waves (Cooke et al. 2009, Cooke and Lorenzetti 2006). FTC outbreak spread resulting from lagged synchrony among populations rather than dispersal to, and growth in, low-density

populations of a transient invasion wave may account for the discrepancy in outbreak spread rates between my model and field observations. Future models of the spatio-temporal dynamics of FTC outbreaks should consider spatially extended populations.

Table 3-1. Descriptions and estimates for parameters of the non-spatial and spatial models.

	Parameter	Estimate	95 % Confidence Interval	Range explored in simulations
	Forest Tent Caterpillar			
r	Intrinsic growth rate (year^{-1})	0.9†	-	-
σ	Standard deviation of r (stochastic model only)	-	-	0.3 – 0.9
K	Carrying capacity (cocoons/15 min)	260†	-	-
	Parasitism			
a	Searching efficiency (area searched during a 15 min cocoon count per parasitoid)	0.027†	0.017, 0.042	0.023 – 0.1
	Predation			
b'	Searching efficiency (cumulative area searched by all predators during a 15 min cocoon count)	0.067	0.052, 0.091	0 – 0.7
h'	Handling time (proportion of a 15 min cocoon count spent handling prey per predator per prey item)	0.043	0.022, 0.067	0 – 0.4
	Dispersal			
d_H	Mean forest tent caterpillar adult dispersal distance (km)	1.0	-	0.25 – 1.75
d_P	Mean parasitoid dispersal distance (km)	0.8	-	0.20 – 1.4

†obtained from Cobbold *et al.* 2009

Table 3-2. Parameters of the best-fit logistic model[a] of the proportion of forest tent caterpillar pupae preyed across a range of local pupal abundances. The negative first-order term (b_1, in bold) indicates a type II functional response describes generalist predation on forest tent caterpillar pupae.

Parameter	Estimate	Standard error	t_1	P
Intercept (b_0)	-2.658	0.055	-48.29	<0.001
b_1	**-8.785**	**1.021**	**-8.61**	**<0.001**
b_2	5.088	0.906	5.62	<0.001
b_3	-3.127	0.822	-3.81	<0.001

[a]Logit(p)=$b_0+b_1x+b_2x^2+b_3x^3$ where p is the proportion of pupae preyed on and x is the local pupal abundance

Table 3-3. Average forest tent caterpillar (FTC) outbreak interval (years between populations >90 cocoons/15 minutes) and FTC and parasitoid population extinction observed in the non-spatial stochastic model, with stochasticity in the intrinsic growth rate of FTC populations (r) described by the standard deviation of a normal distribution (σ) centred on μ=0.9. For each value of σ, the non-spatial model was simulated for 1000 200-year iterations, with r selected with replacement from the normal distribution each year. Average outbreak interval was determined from the average number of outbreaks in the final 150 years of each 200-year iteration. When σ =0.1, outbreaks do not occur. Other parameter values as given in Table 3-1.

σ	Average outbreak interval (years)	Percent runs with FTC extinction (%)	Percent runs with parasitoid extinction (%)
0.1	-	0	0
0.3	2344	0	0
0.5	152	0	2
0.7	23	2	54
0.9	31	9	99

Table 3-4. Influence of the ratio of forest tent caterpillar (FTC) to parasitoid average dispersal distance (d_H and d_P, respectively) on the spread rates of the population front, P_F, and the defoliation front, D_F. Percent changes are given relative to the default spatial model, which assumes d_H=1 km and d_P=0.8 km. Sensitivity is the absolute value of the proportional change in the spread rates to the proportional change in the dispersal distance ratio. When d_H:d_P=0.56, FTC populations do not exceed the threshold abundance that defines the defoliation front (D_F; 50 cocoons/15 min). Other parameter values as given in Table 3-1.

Parameters			**Population Front (P_F)**			**Defoliation Front (D_F)**		
d_H (km)	**d_P (km)**	**Ratio d_H:d_P**	**Spread rate (km y^{-1})**	**Percent change (%)**	**Sensitivity (*S*)**	**Spread rate (km y^{-1})**	**Percent change (%)**	**Sensitivity (*S*)**
1	0.20	5	1.63	-7	0.02	1.57	+3	0.01
1	0.5	2	1.69	-4	0.07	1.55	+2	0.03
1	0.80	1.25	1.76	0	0	1.52	0	0
1	1	1	1.79	+2	0.1	1.48	-3	0.15
1	1.2	0.83	1.82	+3	0.09	1.43	-6	0.18
1	1.5	0.67	1.83	+4	0.09	1.32	-13	0.28
1	1.8	0.56	1.83	+4	0.07	-	-	-

Table 3-5. Influence of the average distance dispersed by forest tent caterpillar (FTC) moths and parasitoids (d_H and d_P, respectively) on the spread rates of the population front, P_F, and the defoliation front, D_F, assuming a constant ratio (d_H:d_P=1.25). Percent changes are given relative to the default spatial model, which assumes d_H=1 km and d_P=0.8 km. Sensitivity is the absolute value of the proportional change in the spread rates to the proportional change in the dispersal distances. Other parameter values as given in Table 3-1.

Parameters			Population Front (P_F)			Defoliation Front (D_F)		
d_H (km)	d_P (km)	Percent change (%)	Spread rate (km y^{-1})	Percent change (%)	Sensitivity (S)	Spread rate (km y^{-1})	Percent change (%)	Sensitivity (S)
0.25	0.20	-75	0.67	-62	0.83	0.61	-60	0.80
0.90	0.72	-10	1.62	-8	0.80	1.40	-8	0.80
1	0.80	0	1.76	0	0	1.52	0	0
1.10	0.88	+10	1.90	+8	0.80	1.64	+8	0.80
1.75	1.40	+75	2.78	+58	0.77	2.40	+58	0.77

Table 3-6. Influence of the intrinsic growth rate of the forest tent caterpillar (FTC) population on the spread rates of the population front, P_F, and the defoliation front, D_F. Percent changes are given relative to the default spatial model, which assumes r=0.9. Sensitivity is the absolute value of the proportional change in the spread rates to the proportional change in the intrinsic growth rate. When r=0.225, FTC populations do not exceed the threshold abundance that defines the defoliation front (D_F; 50 cocoons/15 min). Other parameter values as given in Table 3-1.

Parameter		Population Front (P_F)			Defoliation Front (D_F)		
r	Percent change (%)	Spread rate (km y^{-1})	Percent change (%)	Sensitivity (S)	Spread rate (km y^{-1})	Percent change (%)	Sensitivity (S)
0.225	-75	1.22	-31	0.43	-	-	-
0.81	-10	1.68	-5	0.5	1.37	-10	1
0.9	0	1.76	0	0	1.52	0	0
0.99	+10	1.85	+5	0.5	1.65	+9	0.9
1.575	+75	2.67	+52	0.69	2.34	+54	0.72

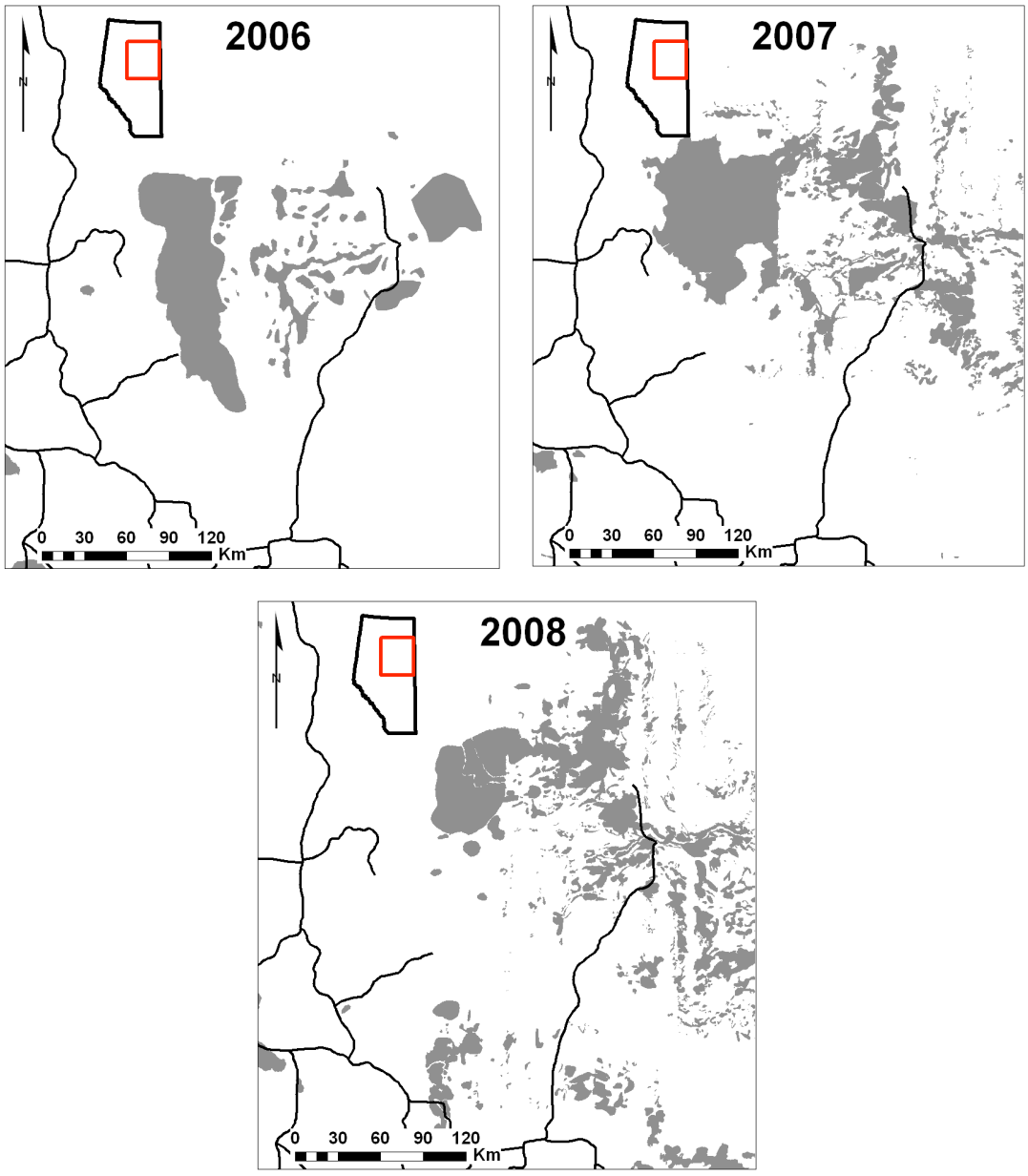

Figure 3-1. Maps displaying the spatial pattern of defoliation (grey) caused by the most recent forest tent caterpillar outbreak in northern Alberta (inset) between 2006 and 2008. Aerial defoliation mapping was used to identify the location and extent of aspen defoliation. Black lines indicate roads. Defoliation data obtained from Alberta Sustainable Resource Development.

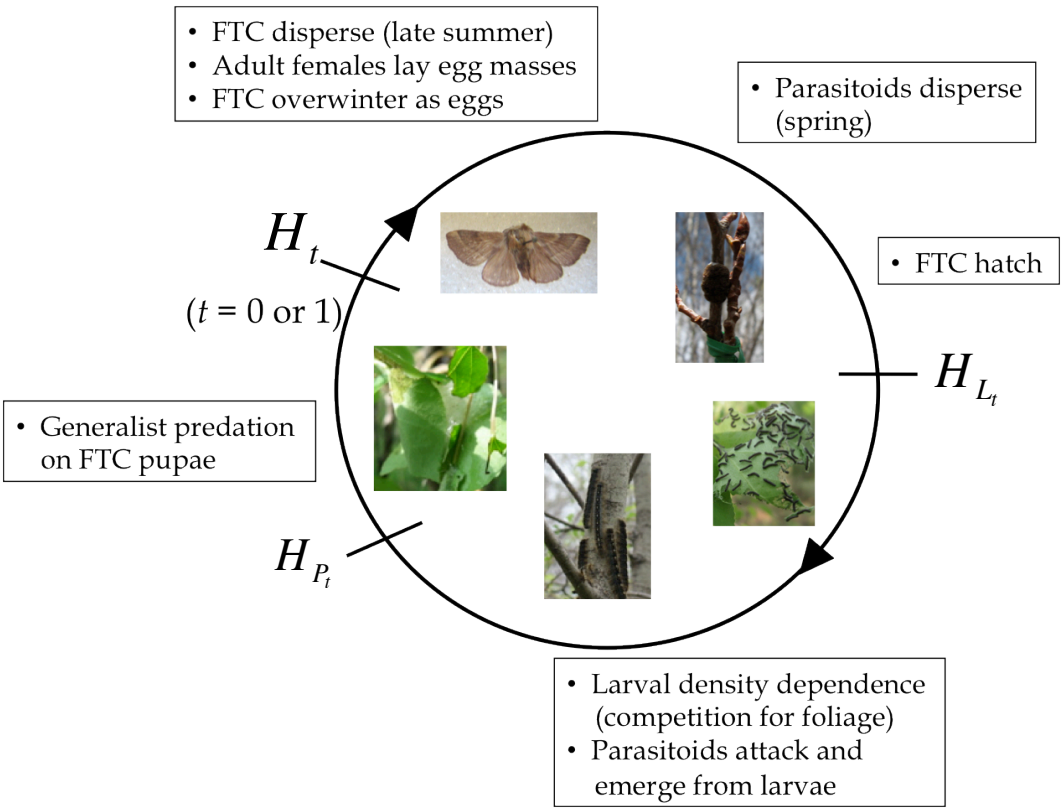

Figure 3-2. Life cycle of the forest tent caterpillar (FTC), highlighting life stages and indicating relative timing of all non-spatial and spatial model events.

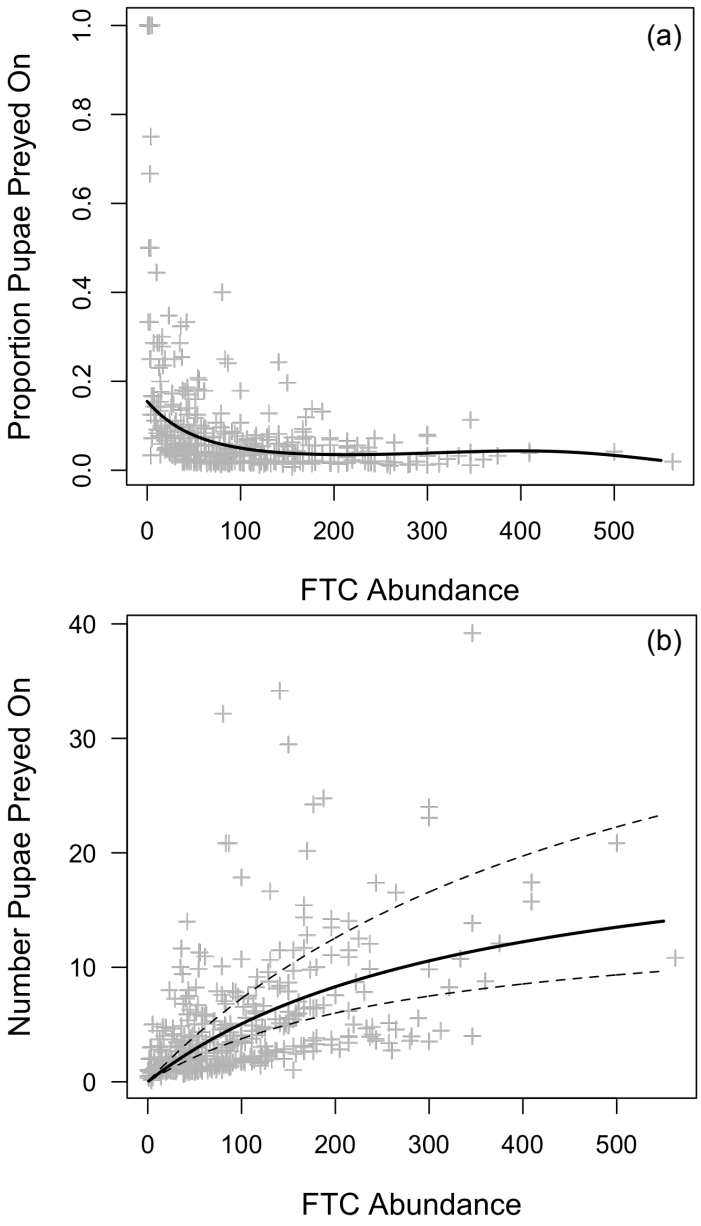

Figure 3-3. a) The proportion of forest tent caterpillar (FTC) pupae preyed on as a function of FTC abundance (cocoons/15 min). The best-fit cubic logistic regression (parameters in Table 3-2) is shown (black line); b) The number of FTC pupae preyed on (cocoons/15 min) as a function of FTC abundance (cocoons/15 min). The black line is Equation 7 fit by non-linear least squares regression with 95% confidence intervals (dashed).

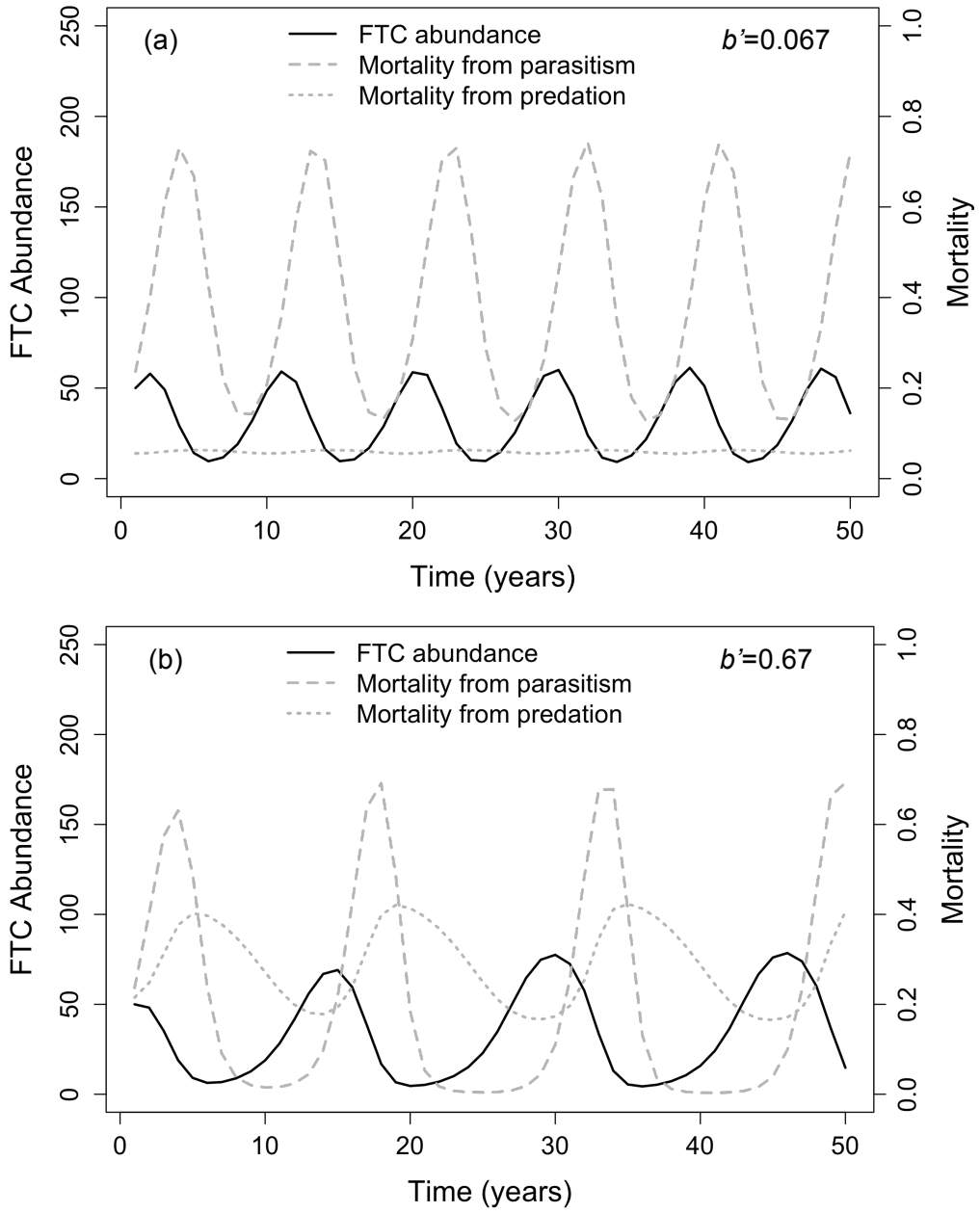

Figure 3-4. Forest tent caterpillar (FTC) abundance (cocoons/15 min; solid line) and proportional mortality from parasitism (dashed line) and predation (dotted line) as predicted by the non-spatial deterministic model. (a) All parameter values as given in Table 3-1. (b) Predator searching efficiency an order of magnitude higher than estimated from field data (b'=0.67).

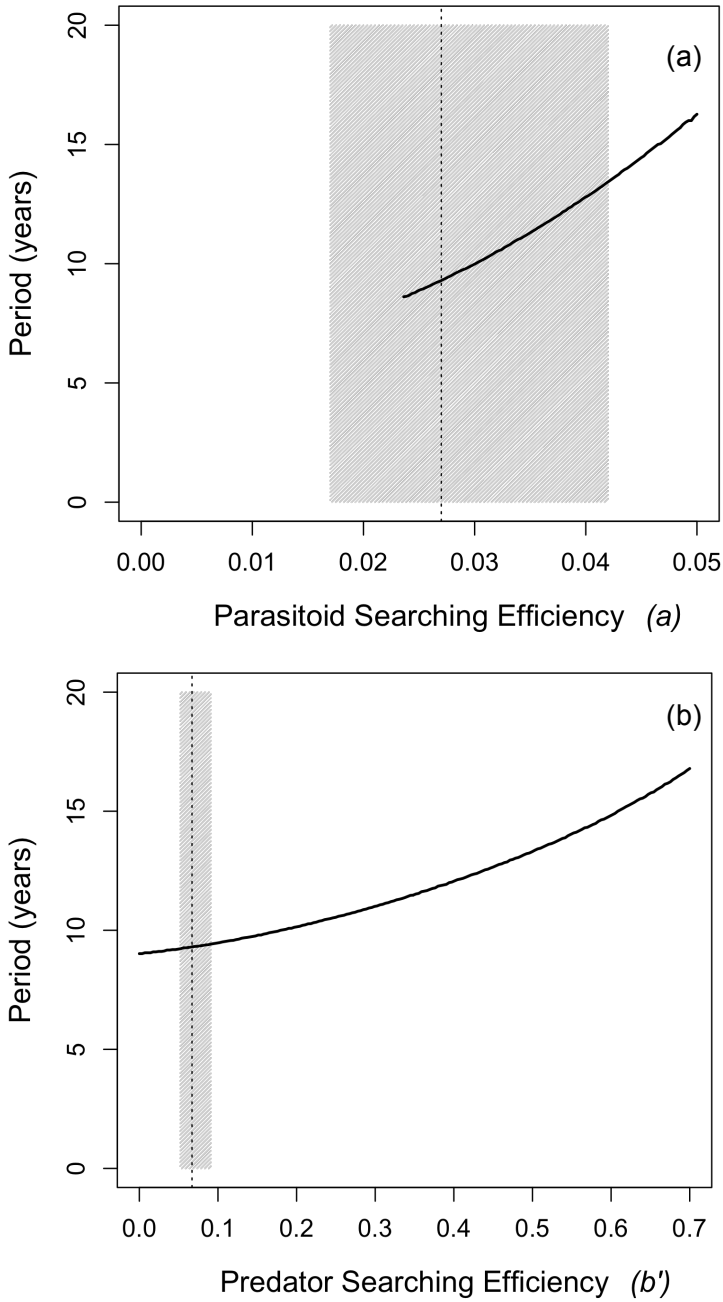

Figure 3-5. Change in the period of forest tent caterpillar (FTC) population cycles with changes in (a) parasitoid searching efficiency (a), and (b) predator searching efficiency (b'). Vertical dotted lines and grey boxes indicate the locations of the parameter estimates and 95% confidence intervals, respectively, provided in Table 3-1. Other parameter values as given in Table 3-1.

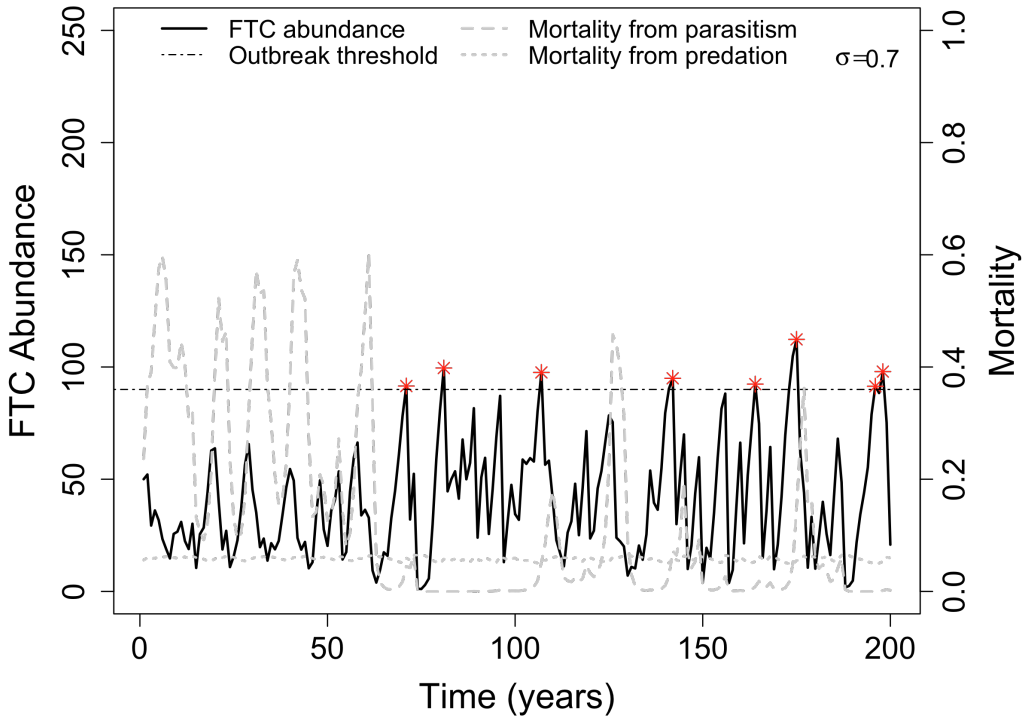

Figure 3-6. One example of forest tent caterpillar (FTC) abundance (cocoons/15 min; solid line) and proportional mortality from parasitism (dashed line) and predation (dotted line) predicted by the non-spatial stochastic model (μ=0.9, σ=0.7). Stars indicate FTC populations that exceed the outbreak threshold (90 cocoons/15 min; dash-dot line). Other parameter values as given in Table 3-1.

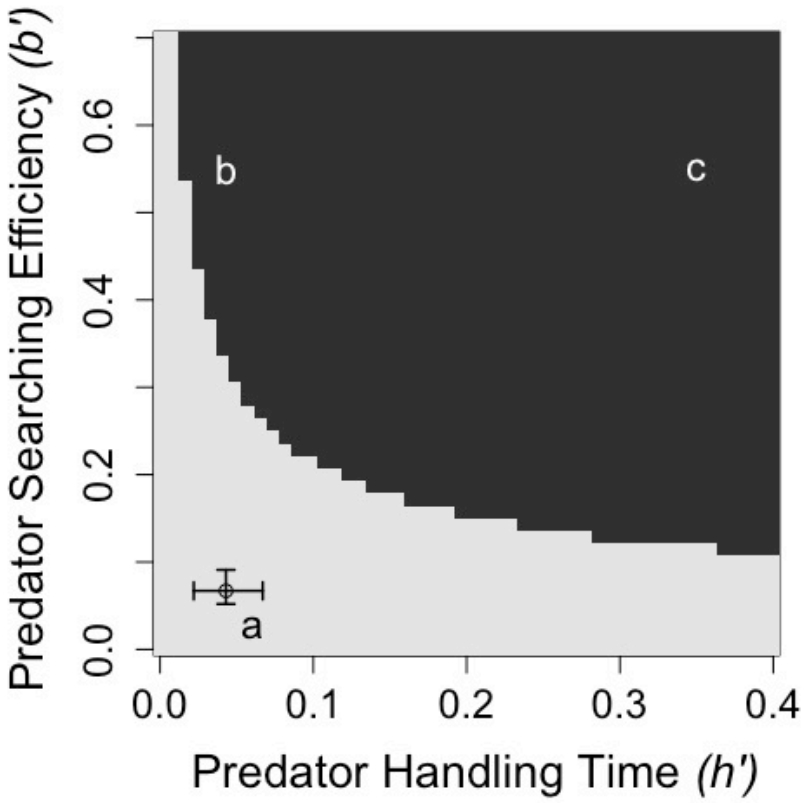

Figure 3-7. Presence (black) and absence (light grey) of a demographic Allee effect in the forest tent caterpillar (FTC) population resulting from the component Allee effect caused by predation according to generalist predator handling time (h') and searching efficiency (b'). Figure produced by simulations of the non-spatial deterministic model of the FTC population. Estimates of b' and h' from data are indicated by the point (± 95% confidence intervals). Letters indicate parameter values used to generate the corresponding panels in Figure 3-8 that display the relationship between FTC abundance and realized population growth rate, R_t. Other parameter values as given in Table 3-1.

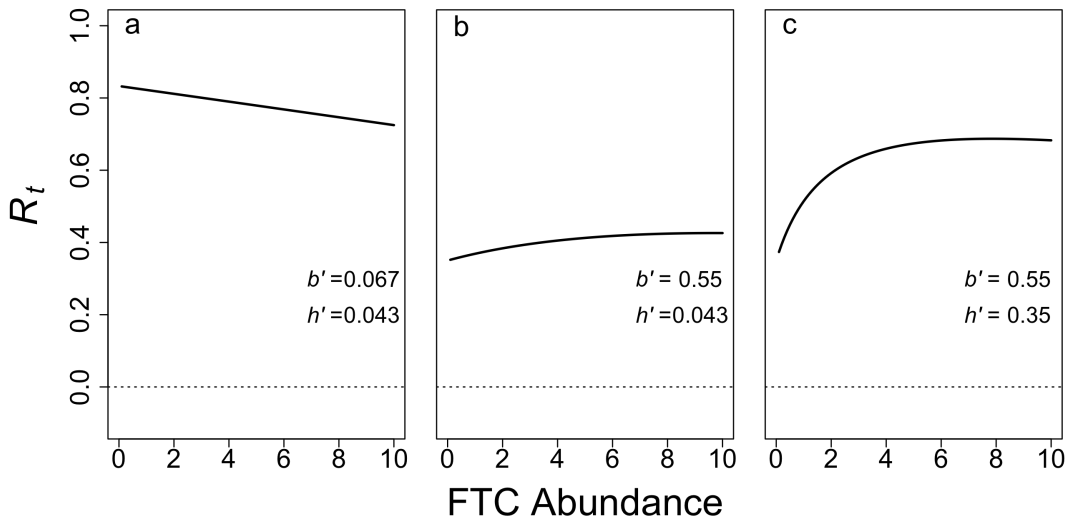

Figure 3-8. Relationship between the realized per-capita growth rate of the forest tent caterpillar (FTC) population (R_t) and FTC abundance as affected by generalist predator handling time (h') and searching efficiency (b') demonstrating the absence (a) or presence (b, c) of a weak demographic Allee effect in the FTC population resulting from the component Allee effect caused by predation. The Allee threshold is indicated by the dotted line (R_t=0). Panels (a-c) correspond to parameter values indicated in Figure 3-7. Figures produced by simulations of the non-spatial deterministic model of the FTC population. Other parameter values as given in Table 3-1.

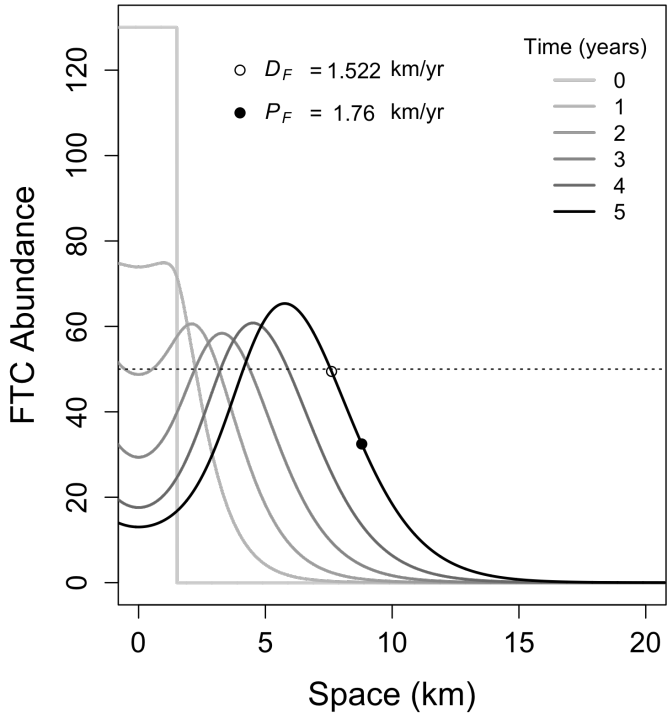

Figure 3-9. Outcome of the deterministic spatial model demonstrating the spatial progression of a FTC outbreak (FTC abundance in cocoons/15 min) over 5 years. The final position and calculated spread rates of the defoliation front (D_F; white) and the population front (P_F; black) are indicated. The threshold abundance defining the defoliation front is indicated by the dotted line. The FTC outbreak was initiated at t=0 with an outbreak population of H_0=130 at 0≤X≤1.5 and with H_0=0 at X>1.5. The initial conditions for the parasitoid population were P_0=13 at 0≤X≤1.5 and P_0=0 at X>1.5. All parameter values as given in Table 3-1.

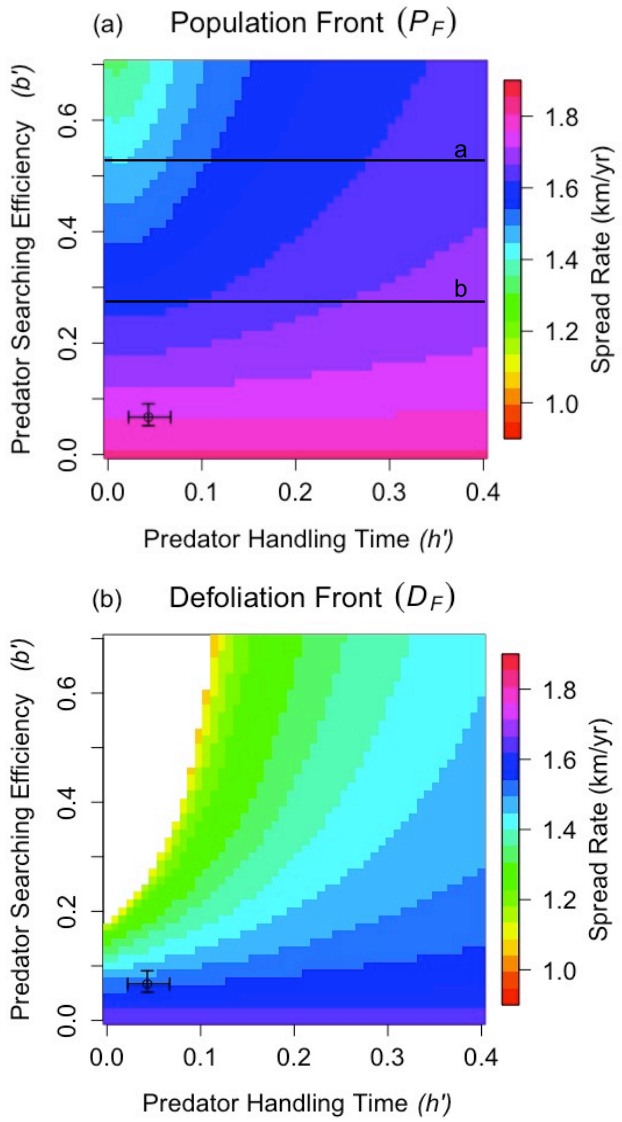

Figure 3-10. The effect of manipulating predator handling time (h') and searching efficiency (b') on the spread rate of (a) the population front (P_F), and (b) the defoliation front (D_F) of a forest tent caterpillar (FTC) outbreak. Estimates of b' and h' from data are indicated by the points (± 95% confidence intervals). Transects in (a) indicate values of b' for which an increase in h' does ("a") or does not ("b") reduce the population front spread rate. The white area in (b) corresponds to FTC populations that do not exceed the threshold abundance defining the defoliation front (D_F; 50 cocoons/15 min). Figures produced by simulations of the spatial deterministic model of the FTC population. Other parameter values as given in Table 3-1.

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Chapter 4

General Discussion and Conclusions

Outbreaking forest insects are ubiquitous but very little is understood about the factors that govern the spatial patterns of outbreaks, despite their widespread impacts on forest ecosystems. Because natural enemy interactions are an essential component of forest insect population dynamics (e.g. Klemola et al. 2010, Royama et al. 2005, Turchin et al. 2003, Berryman 1996), my objective was to determine whether variation in interactions with natural enemies mediated by forest composition could influence the spread of a forest tent caterpillar (*Malacosoma disstria* Hbn.; FTC) outbreak. I addressed two specific questions: 1) What are the relative effects of specialist and generalist natural enemies on mortality in low density FTC populations, and does forest composition affect natural enemy-caused mortality of FTC (Chapter 2)? and 2) Does generalist predation alter the spread rate of a FTC outbreak, especially through an Allee effect (Chapter 3)?

Overview

In contrast to the prediction that natural enemy-caused mortality of FTC would be higher in the more diverse mixedwood stands than in aspen stands, mortality of FTC in low-density populations differed very little between stand types, especially for early instar larvae and pupae. Generalists, both predators and parasitoids, caused most of the FTC mortality, and similar mortality rates between the forest stand types reflected the similar abundance and diversity of these natural enemies in aspen and mixedwood forest stands. The only observed difference in

natural enemy-caused mortality between stand types was predation on late instar larvae and pupae, which was higher in aspen stands than in mixedwood stands. This difference in predation was apparently mediated by the presence of specific bird species unique to aspen stands, rather than the diversity of the avian community generally.

Mortality caused by generalist predation of FTC pupae, with parameters estimated from field data, was not sufficiently severe to induce an Allee effect in the FTC population in my non-spatial model. However, for generalist predators that are more efficient and that impose higher mortality in the FTC population, the model indicates the potential for predation to both extend the period of FTC population cycles and to induce an Allee effect in the FTC population. The strength of generalist predation influences the spread rate of a FTC outbreak, with an order of magnitude increase in predator efficiency reducing the spread rate by nearly 30% or 0.5 km yr^{-1}. Although the change in predator efficiency associated with this change in spread rate is large, it corresponds to a change in predator-caused mortality consistent with the range of predation rates observed in the field. Thus, variation in the strength of generalist predation may have meaningful consequences for the spread of FTC outbreaks. In contrast, predator-induced Allee effects have little additional consequence for the spread rates of FTC outbreaks in my model.

Implications for the Effects of Stand Composition on FTC Outbreaks

More generally, the combined results from the field experiments (Chapter 2) and the FTC population models (Chapter 3) suggest the potential for FTC populations in forest stands of different composition to exhibit different dynamics, with possible consequences for the spatial pattern of spread of an outbreak. Despite being formulated for a generalist predator on pupae, the outcomes of my non-spatial model suggest that the

observed higher generalist predation on late instar larvae in low-density FTC populations in aspen stands compared to mixedwood stands may slow the increase of FTC populations in aspen stands. Similarly, my spatial model suggests that the higher generalist predation observed in aspen stands would also reduce the spread rate of an outbreak through those stands. The change in predator searching efficiency necessary for the population cycle period or outbreak spread to be appreciably impacted corresponds to a difference in predator-caused mortality rates of more than 30%. This difference is reasonably consistent with the difference in late-instar larval predation rates observed between aspen and mixedwood stands (approximately 40%; Figure 2-5b). Therefore, if predation on late-instar larvae has consequences for FTC outbreak dynamics similar to those of pupal predation, forest stand composition may impact the spatial pattern of FTC outbreaks in the field. From a forest management perspective, my results suggest the composition of forest stands and their arrangement on the landscape may have consequences for the spatial dynamics of FTC outbreaks, with outbreaks possibly occurring less frequently in, and spreading more slowly through, aspen stands than mixedwood stands.

Future Directions

As outlined in Chapter 2, landscape-scale forest heterogeneity, in addition to local vegetation diversity, may influence the composition of natural enemy communities and their impacts on prey populations (Barbaro et al. 2005, Cronin and Reeve 2005, Jactel et al. 2005, Roland 2000). Fragmentation of aspen forest by agriculture and urban development results in FTC outbreaks that appear earlier and last longer in fragmented stands compared to larger continuous forest tracts (Roland 2005, Roland 1993) as a result of the effects of fragmentation on natural enemies (Roland 2005, Roland and Taylor 1997). Similarly, landscape

composition in the mixedwood boreal forest may have a stronger influence over natural enemy communities and their impacts on FTC populations than stand composition itself. For example, both birds and parasitoids have been shown to respond positively to landscape heterogeneity, in terms of the diversity of land cover types (Drolet et al. 1999, Cappuccino et al. 1998). Similar experiments to the ones described in Chapter 2 could be conducted considering landscape-scale diversity rather than forest stand diversity to test this hypothesis.

Given the large spatio-temporal scale of FTC population dynamics and outbreaks, additional modelling studies will be necessary to refine our understanding of the factors affecting both the spatial and temporal dynamics of FTC populations throughout their range. Field studies will play a crucial role in providing data suitable for both model parameterization and validation. Many fine-scale field experiments on predation and parasitism of FTC have been conducted (e.g. Chapter 2, Glasgow 2006, Roth et al. 2006, Parry et al. 1997, Parry 1995), but future experiments could be planned in conjunction with modelling studies to provide data to estimate parameters of specific models (e.g. Cobbold et al. 2009). Although measuring dispersal, especially of short-lived adults like FTC moths, is extremely difficult in the field (e.g. Roland and Taylor 1995), the current lack of accurate information about dispersal is a particularly conspicuous gap in the available data.

In Chapter 3 I suggested the spatial dynamics of a spreading outbreak might be better described by models incorporating spatially extended FTC populations than a model of a transient invasion wave into unoccupied space. In this alternative framework, patterns of spatial synchrony among adjacent populations in stands of different composition may respond differently, and possibly more strongly, to variation in natural enemy impacts between forest stand types than the spread rate of an invasion wave. Consequently, differences in natural enemy impacts

may affect spatial synchrony among FTC populations and perhaps the characteristics of, or the occurrence of, a travelling wave of a FTC outbreak.

Previous work considering the spatial dynamics of FTC outbreaks has generally relied on abundant, but coarse-scale, defoliation data (e.g. Wood et al. 2010, Cooke et al. 2009, Cooke and Lorenzetti 2006, Cooke and Roland 2000), which describes FTC abundance in terms of the presence or absence of defoliation detectable from the air and which are frequently aggregated into the proportion of cells defoliated in a pre-determined area (e.g. Peltonen et al. 2002). At the finest scale (1 km^2; Cooke et al. 2009), these defoliation data provide useful insight into regional patterns of outbreak synchrony, however many forest characteristics, including stand composition, vary on a finer scale (hectares rather than square kilometres). Dendrochronological reconstruction, where historical FTC abundances are inferred from the width and type of aspen tree rings (Cooke and Roland 2007, Sutton and Tardiff 2007), can be used to obtain very spatially specific historic records of FTC population patterns, including records of abundant populations that were not detected through defoliation mapping (Cooke and Roland 2007). It may be possible to take advantage of this type of data to aid investigations of lagged spatial synchrony and the effects of landscape variables, including fragmentation and forest composition on both the local temporal and broader spatial dynamics of FTC populations (Cooke and Roland 2007, Sutton and Tardiff 2007, Cooke 2001). These fine-scale spatially referenced FTC abundance data may also be useful for validation of future population models.

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Appendix 1. Site Vegetation Characteristics

Methods

I characterized forest composition at all sites, including both canopy and understory variables. I used the point-centred quarter method (Krebs 1999) to estimate canopy tree species composition, diversity and density. At each site, six transects of five random points were used. Random points were between 1 and 10 m apart, such that the same tree was never measured twice. At each point, I measured the distance to and circumference at 130 cm (CCH) of the nearest tree (CCH >12 cm) in each of four quarters. Because random point locations determined transect length, transects ranged in length between 16 and 63 m with a median length of 33 m. Shrub species composition, density and diversity were also determined for each site. Shrubs >30 cm tall were identified and counted in 5, 2 x 10 m randomly placed quadrats. Shrubs > 1.5 m tall and saplings (trees < 12 cm CCH) in each quadrat were recorded separately.

I calculated overall stand density (trees/ha), basal area (cover; m^2/ha), the absolute and relative density and cover of aspen, tree species richness and tree species diversity as measured by the Shannon diversity index (*H'*; Krebs 1999):

$$H' = -\sum_{i=1}^{S} p_i \ln p_i \qquad (1)$$

where *S* is the total number of species (species richness) and p_i is the proportion of species *i* relative to the total number of species. I also calculated the density (#/100 m^2), species richness and species diversity (*H'*) of shrubs and saplings.

I used a MANOVA to confirm that local stand characteristics differed between stands classified as aspen and those classified as mixedwood. I used principle components analysis (Everitt 2005) to

determine which variables were responsible for differences among sites and to create a reduced set of vegetation variables. Data were standardized (scaled and centred) prior to performing the PCA to account for differences in variance among variables. K-means cluster analysis (Everitt 2005) was performed on the first three principle components to determine whether aspen and mixedwood sites fell into two separate clusters based on their vegetation characteristics.

All analyses were conducted with $\alpha=0.05$ in R v 2.12.1 (R Development Core Team 2010, Vienna, Austria) using functions available in the stats package.

Results and Discussion

Vegetation characteristics of aspen and mixedwood forest stands in my study were significantly different ($F_{14,5}=12.97$, $P<0.01$). Differennces were caused by both the amount of aspen in the stands and the diversity of tree species (Table A1-1). There were no significant differences in variables related to understory vegetation (Table A1-1).

The first three principle components explained 63% of the variation in vegetation characteristics among sites. Variables related to tree species composition, diversity and density are strongly loaded on the first principle component axis, and variables related to understory characteristics are strongly loaded on the second principle component axis (Fig. A1-1a). Aspen and mixedwood stands clearly map out with limited overlap according to these two principle components, in particular the variation in tree characteristics (Fig. A1-1a). The K-means clustering algorithm identified two clusters of 9 sites and 11 sites. Three sites were misclassified (grouped with the stands of the other type): two mixedwood stands were grouped with the aspen stands, and one aspen stand was grouped with the mixedwood stands (Fig. A1-1b), indicating aspen and mixedwood stands in my study were not entirely distinct.

The lack of distinction results primarily from two sources. First, some aspen stands had higher tree diversity and lower aspen content because of the presence of a higher percentage of balsam poplar, I could not easily identify visually in the spring before leaf flush. Second, some mixedwood stands had greater than anticipated aspen cover because they contained a high density of young aspen trees that dominated the sample in the point-centred quarter method, which is slightly biased towards sampling small trees if they are closely packed.

Table A1-1. Effect of stand-type on vegetation characteristics based on MANOVA. All F-tests had the same degrees of freedom. Bold values indicate significant differences between aspen and mixedwood stands.

Variable		$F_{1,18}$	P
Trees	Density	2.165	0.159
	Absolute aspen density	**15.17**	**0.001**
	Relative aspen density	**28.08**	**<0.001**
	Total cover	0.005	0.947
	Absolute aspen cover	**7.755**	**0.012**
	Relative aspen cover	**18.66**	**<0.001**
	Species richness	**11.44**	**0.003**
	Species diversity	**20.97**	**<0.001**
Shrubs	Density	0.008	0.929
	Species richness	0.144	0.708
	Species diversity	0.373	0.549
Shrubs and saplings	Density	0.387	0.542
(>1m)	Species richness	0.385	0.543
	Species diversity	0.188	0.670

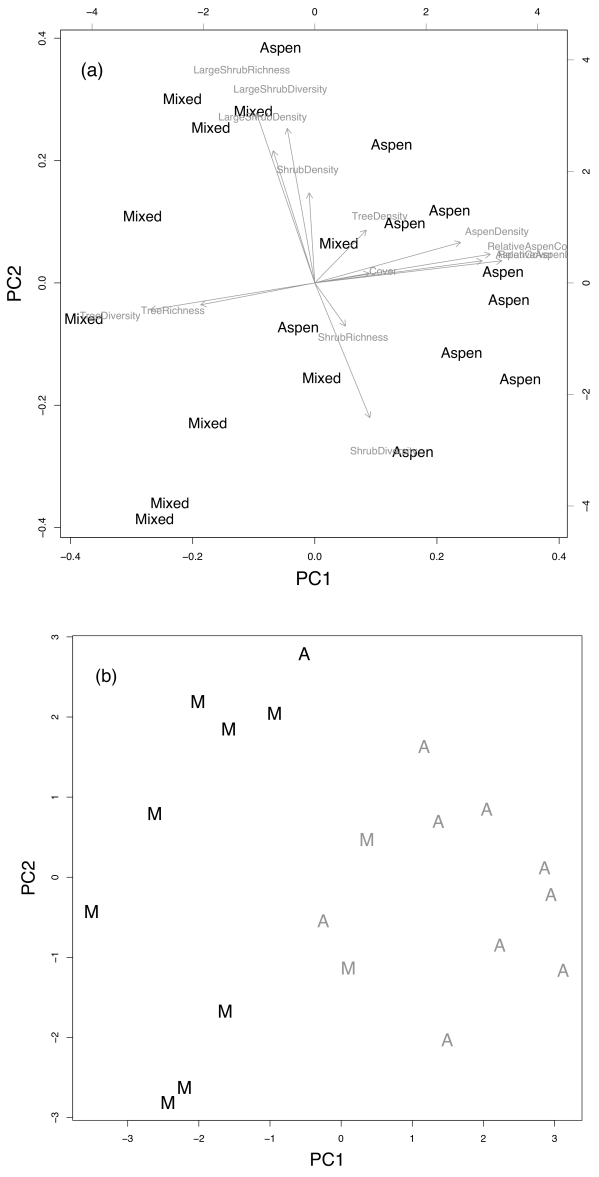

Figure A1-1. (a) Relative loadings of vegetation variables and the locations of the 20 sites on the first and second principle component axes. Mixed: mixedwood stands; Aspen: aspen stands. (b) Classification of study sites according to the first three principle components of site vegetation data using K-means clustering. Shades (black, grey) indicate the groups determined by the clustering algorithm. Letter symbols indicate the original classification of stands in the field (M: mixedwood stands; A: aspen stands).

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Krebs, C. J. 1999. Ecological methodology. Benjamin Cummings, Menlo Park, California.

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Appendix 2. Preliminary Analyses of Pupal Mortality

Methods

In 2009, the duration that pupae were exposed to natural enemies in the open and predator exclusion treatments was variable (range: 12-26 days, median: 17 days). I conducted logistic regressions to determine whether duration of exposure to natural enemies affected the probability of parasitism, predation or unknown mortality in pupae recovered from these treatments. To account for overdispersion where necessary, I assumed a quasibinomial rather than a binomial error distribution, and used an F-test rather than a χ^2 test to evaluate significance of the terms (Crawley 2007).

In 2010, three saplings with enemy exclusion treatments and two with predator exclusion treatments were broken or damaged during the experiment. I used a g-test to determine if tree-breakage affected the probability of pupal parasitism of pupae in the predator exclusion treatment. All analyses were conducted with α=0.05 in R v 2.12.1 (R Development Core Team 2010, Vienna, Austria) using functions available in the stats package.

Results and Discussion

Exposure time had no significant effect on the probability of predation ($F_{1,172}$=0.527, P=0.469; Fig. A2-2a) or parasitism ($F_{1,172}$=0.727, P=0.395; Fig. A2-2b) of pupae in the open and predator exclusion treatments in 2009. Pupae were exposed for a minimum of 12 days, and the duration of the FTC pupal stage is approximately 12-14 days (Fitzgerald 1995). Exposure duration did not affect predation and parasitism rates of FTC pupae because much of the additional exposure

time occurred after the eclosion of healthy moths, when there was no opportunity for predation or parasitism to occur.

There was a significant decrease in the probability of unknown pupal death with exposure time in the 2009 open and predator exclusion treatments (χ^2_1=6.762, P=0.009; Fig. A2-2c). This effect of exposure time on unknown mortality may have resulted from earlier-collected pupae being further from eclosion at the time of collection and being stored alive for longer. Pupae stored alive may have experienced higher death rates due to fungal or other pathogens. Tree-breakage did not affect the probability of pupal parasitism (G_1=1.314, P=0.256)

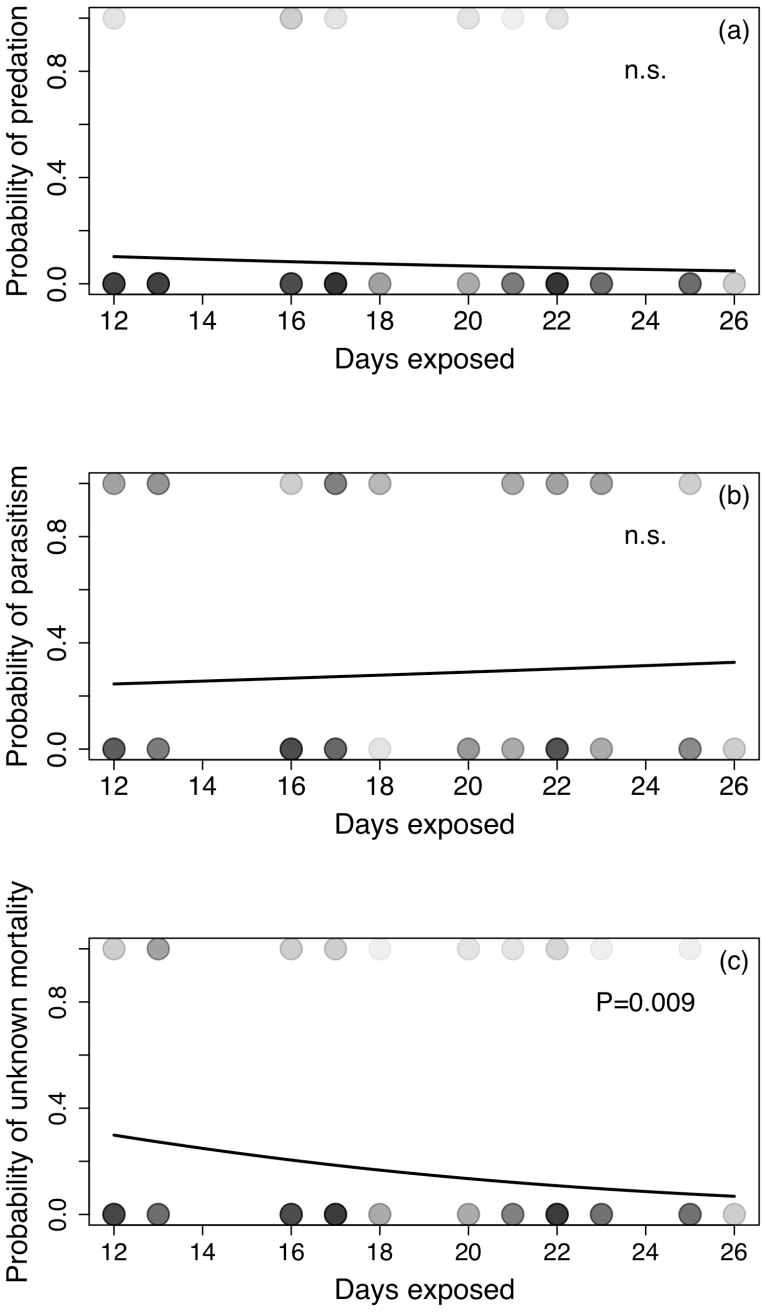

Figure A2-1. Probability of pupal (a) predation, (b) parasitism, or (c) unknown mortality in the open and predator exclusion treatments in 2009. Point intensity indicates overlapping points. Lines are best-fit logistic regression lines for each fate.

Literature Cited

Crawley, M. J. 2007. The R book. John Wiley & Sons Ltd., Mississauga Ontario.

Fitzgerald, T. D. 1995. The tent caterpillars. Cornell University Press, Ithica, NY.

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Appendix 3. Bird Species List

Table A3-1. Bird species recorded at aspen and mixedwood sites in 2009 and 2010. Numbers are the total observations over three point counts.

		2009		2010	
Common Name	**Scientific Name**	**Aspen**	**Mixed**	**Aspen**	**Mixed**
Alder flycatcher	*Empidionax alnorum*	0	1	0	0
American redstart ‡	*Setophaga ruticilla*	1	3	0	2
American robin ‡	*Turdus migratorius*	6	4	0	5
Black and white warbler ‡	*Mniotilta varia*	3	0	2	1
Black-capped chickadee ‡	*Poecile atricapillus*	1	5	0	2
Black-throated green warbler	*Dendroica virens*	0	4	0	3
Blackburnian warbler	*Dendroica fusca*	0	0	0	1
Brown-headed cowbird ‡	*Molothrus ater*	4	2	0	3
Cape May warbler	*Dendroica tigrina*	0	0	0	1
Chipping sparrow ‡	*Spizella passerina*	2	4	1	2
Connecticut warbler	*Oporornis agilis*	2	2	6	0
Dark-eyed junco	*Junco hyemalis*	0	3	0	4
Hermit thrush	*Catharus guttatus*	4	1	0	2
House wren ‡	*Troglodytes aedon*	3	0	0	2
Least flycatcher ‡	*Empidionax minimus*	28	3	23	3
Northern flicker ‡	*Colaptes auratus*	1	1	0	0
Ovenbird ‡	*Seiurus aurocapillus*	13	18	12	11
Pileated woodpecker	*Dryocopus pileatus*	1	0	0	0
Pine siskin ‡	*Carduelis pinus*	0	2	0	0
Raven	*Corvus corax*	0	2	0	0
Red-breasted nuthatch	*Sitta canadensis*	0	4	0	4
Red-eye vireo ‡	*Vireo olivaceus*	5	5	5	3
Red-winged blackbird ‡	*Agelaius phoeniceus*	3	0	2	0
Rose-breasted grosbeak ‡	*Pheucticus ludovicianus*	3	2	2	1
Ruby crowned kinglet	*Regulus calendula*	0	7	0	0
Solitary vireo ‡	*Vireo solitarius*	6	1	2	3
Swainson's thrush	*Catharus ustulatus*	2	5	0	0
Tennessee warbler	*Oreothlypis peregrina*	16	26	2	16
Warbling vireo	*Vireo gilvus*	7	1	3	2
Western wood peewee	*Contopus sordidulus*	3	0	0	0
White-throated sparrow ‡	*Zonotrichia albicollis*	3	6	2	2
Winter wren	*Troglodytes hiemalis*	0	3	0	2
Yellow warbler ‡	*Dendroica petechia*	17	4	11	0
Yellow-bellied sapsucker ‡	*Sphyrapicus varius*	3	6	5	6
Yellow-rumped warbler ‡	*Dendroica coronata*	9	18	7	17

‡ previously considered a predator of forest tent caterpillar larvae or pupae (Glasgow 2006, Witter and Kulman 1972, Hodson 1943).

Literature Cited

Glasgow, M. A. 2006. The effects of forest fragmentation on generalist predation on forest tent caterpillars (*Malacosoma disstria*). M.Sc. Thesis. University of Alberta, Edmonton.

Hodson, A. C. 1943. Birds feeding on forest insects. The Flicker 15:50-51.

Witter, J. A., and H. M. Kulman. 1972. A review of the parasites and predators of tent caterpillars (*Malacosoma* spp.) in North America. Agricultural experiment station technical bulletin 289. University of Minnesota.

Appendix 4. Arthropod Predators

Table A4-1. Number of potential arthropod predators of forest tent caterpillar in aspen and mixedwood forest stands in July 2009. Trap days is the total number of traps from which samples were collected over the course of the sample period.

Forest Type	Site	Trap days	Araneae	Formicidae	*Calosoma frigidum*	*Carabus chamissonis*	*Pterostichus adstrictus*	*Pterostichus pennsylvanicus*
Aspen	1	62	33	34	1	0	13	46
	2	56	27	18	0	0	2	4
	3	65	37	34	1	0	6	11
	4	65	46	29	1	1	1	7
	5	49	26	94	0	0	11	9
	6	55	14	0	0	0	19	5
	7	65	41	11386	0	0	1	4
	8	35	11	4	0	0	7	1
	9	65	50	48	0	0	12	12
	10	62	47	20	0	0	8	10
Mixedwood	11	62	19	19	1	0	4	4
	12	65	27	29	0	0	1	3
	13	65	35	70	0	0	7	1
	14	56	20	32	0	0	1	0
	15	62	35	4	0	0	0	0
	16	47	24	9	0	0	0	1
	17	65	32	7	0	1	3	0
	18	65	13	11	0	0	3	1
	19	59	50	48	0	0	3	0
	20	65	15	24	0	0	4	1

Table A4-2. Number of potential arthropod predators of forest tent caterpillar in aspen and mixedwood forest stands in May 2010. Trap days is the total number of traps from which samples were collected over the course of the sample period.

Forest Type	Site	Trap Days	Araneae	Formicidae	*Calosoma frigidum*	*Carabus chamissonis*	*Pterostichus adstrictus*	*Pterostichus melanarius*	*Pterostichus pennsylvanicus*	*Pterostichus punctatissimus*
Aspen	1	35	82	75	0	0	12	1	10	0
	2	35	102	12	0	0	2	0	13	0
	3	35	60	22	0	0	5	0	4	0
	4	35	101	30	2	2	9	0	21	0
	5	15	130	83	1	1	6	0	4	0
	6	35	78	1	0	1	25	0	0	0
	7	35	56	7893	0	0	0	0	8	0
	8	35	80	5	0	1	30	0	4	0
	9	15	116	105	0	1	6	0	6	1
	10	35	121	9	1	0	13	0	10	0
Mixedwood	11	35	54	27	0	0	6	1	0	0
	12	35	59	67	0	2	19	0	4	0
	13	35	70	42	0	4	9	0	3	0
	14	35	91	21	0	0	1	0	0	0
	15	35	40	0	0	4	9	0	0	0
	16	35	43	1	0	0	16	0	1	0
	17	35	14	1	0	0	3	0	0	0
	18	35	78	9	0	0	7	0	2	0
	19	35	103	77	0	0	23	0	0	0
	20	35	73	21	0	0	13	0	1	0

Table A4-3. Number of potential arthropod predators of forest tent caterpillar in aspen and mixedwood forest stands in July 2010. Trap days is the total number of traps from which samples were collected over the course of the sample period.

Forest Type	Site	Trap Days	Araneae	Formicidae	*Calosoma frigidum*	*Pterostichus adstrictus*	*Pterostichus melanarius*	*Pterostichus pennsylvanicus*
Aspen	1	22	16	22	0	7	8	1
	2	35	47	29	1	1	0	0
	3	35	55	63	0	12	0	11
	4	35	31	40	0	2	0	1
	5	25	55	43	0	5	0	3
	6	35	32	11	0	2	0	0
	7	35	40	17785	0	0	0	1
	8	35	30	12	0	2	0	1
	9	35	36	77	0	8	0	4
	10	35	17	16	0	12	3	3
Mixedwood	11	35	32	35	1	3	1	1
	12	35	38	76	0	4	3	4
	13	35	39	103	0	7	0	0
	14	35	13	31	0	2	0	0
	15	35	26	1	0	1	0	1
	16	35	29	17	0	2	0	0
	17	35	43	13	0	0	0	0
	18	35	17	10	0	0	0	0
	19	35	31	29	0	1	0	0
	20	32	14	19	0	6	0	0

Appendix 5. Parasitoids Recovered from FTC Larvae and Pupae

Table A5-1. Number of dipteran and hymenopteran parasitoids recovered from collections of 4th and 5th instar forest tent caterpillar larvae at aspen and mixedwood sites in 2010. At aspen site 5, the collection of larvae was too small to include.

Forest	Site	Total FTC Larvae	Recovered Parasitoids						
			Hymenoptera				Diptera		
			Agrypon anale	*Aleiodes malacosomatos*	*Hyposoter fugivitus*	*Unknown wasp**	*Leschenaultia exul*	*Carcelia malacosomae*	*Achaetoneura frenchii*
Aspen	1	76	6	1	0	0	0	0	0
	2	115	0	0	0	0	0	42	0
	3	102	0	0	0	0	19	1	0
	4	35	0	0	0	0	0	2	0
	5	-	-	-	-	-	-	-	-
	6	62	0	0	0	0	0	0	0
	7	54	0	1	13	0	0	0	0
	8	53	0	0	0	0	0	13	1
	9	93	2	0	0	0	0	16	0
	10	69	0	0	0	0	0	0	0
Mixedwood	11	102	0	8	0	1	0	0	0
	12	57	22	0	0	1	0	0	0
	13	31	0	0	0	0	0	6	0
	14	55	0	0	0	0	0	0	0
	15	81	0	0	0	0	0	0	0
	16	77	0	0	0	0	0	0	0
	17	78	3	0	0	1	0	0	0
	18	65	0	2	0	0	0	7	0
	19	105	0	0	0	0	37	31	0
	20	98	3	0	0	0	0	0	0

*died as larvae or adults and could not be identified to species

Table A5-2. Number of dipteran and hymenopteran parasitoids recovered from forest tent caterpillar pupae in aspen and mixedwood stands in 2009. Parasitoids were recovered from pupae planted in the open and predator exclusion treatments in both years.

Forest	Site	Total FTC Pupae	Recovered Parasitoids					
			2009					
			Diptera	Hymenoptera				
			Arachnidomyia aldrichi	*Itoplectis quadricingulata*	*Theronia atalantae fulvescens*	*Pimpla pedalis*	*Gambrus canadensis canadensis*	*Unknown wasp**
Aspen	1	9	0	0	0	0	0	1
	2	9	1	0	0	0	1	0
	3	9	5	0	1	0	0	0
	4	13	3	0	0	0	0	3
	5	10	1	0	2	0	0	1
	6	8	1	1	0	1	0	0
	7	10	0	0	0	0	0	0
	8	15	5	0	0	0	0	1
	9	14	1	0	2	0	1	1
	10	8	0	0	0	0	0	2
Mixedwood	11	10	0	0	0	0	0	0
	12	8	0	0	0	0	0	0
	13	7	0	0	0	0	0	2
	14	8	4	0	0	0	0	0
	15	10	3	0	4	0	0	2
	16	7	2	0	0	0	0	2
	17	12	2	0	0	0	0	0
	18	9	4	0	0	0	0	1
	19	7	3	0	0	0	0	1
	20	13	4	0	0	0	0	1

*died as larvae or adults and could not be identified to species

Table A5-3. Number of dipteran and hymenopteran parasitoids recovered from forest tent caterpillar pupae in aspen and mixedwood stands in 2010. Parasitoids were recovered from pupae planted in the open and predator exclusion treatments in both years.

Forest	Site	Total FTC Pupae	Recovered Parasitoids 2010						
			Diptera	Hymenoptera					
			Arachnidomyia aldrichi	*Itoplectis quadricingulata*	*Itoplectis conquisitor*	*Theronia atalantae fulvescens*	*Pimpla pedalis*	*Gambrus canadensis canadensis*	*Unknown wasp**
Aspen	1	10	0	0	0	2	0	2	0
	2	10	1	1	2	1	0	2	1
	3	9	2	0	0	0	0	0	1
	4	9	0	0	1	1	0	0	0
	5	10	0	0	0	0	0	0	0
	6	9	3	0	0	1	0	0	1
	7	10	0	0	0	0	0	0	0
	8	10	0	2	1	1	0	0	3
	9	10	0	0	0	2	0	3	2
	10	10	0	1	2	0	0	0	0
Mixedwood	11	10	0	1	1	0	0	0	0
	12	10	0	1	0	1	0	0	1
	13	10	1	1	0	1	0	0	1
	14	9	0	1	0	0	0	0	0
	15	7	0	3	0	0	0	0	2
	16	6	0	0	0	1	0	0	1
	17	8	0	0	1	0	0	0	1
	18	10	0	0	0	1	0	0	3
	19	4	1	0	0	0	0	0	1
	20	10	1	1	0	0	0	0	1

*died as larvae or adults and could not be identified to species