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Mechanisms underlying spruce budworm outbreak processes as elucidated by a 14-year study in New Brunswick, Canada

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Abstract. We conducted a 14-yr intensive study of spruce budworm (*Choristoneura fumiferana* (Clem.)) survivorship at three study plots in largely balsam fir (*Abies balsamea* (L.) Mill.) stands in New Brunswick, Canada, to elucidate certain key mechanisms underlying spruce budworm outbreak cycles. The study covered a peak-to-declining phase (from 1981 and 1994) of the budworm outbreak cycle that had started in the early 1960s. Frequent sampling was carried out in each plot-year to construct a practically continuous survivorship curve, and the annual variation in population density was estimated. We found a high level of correlation between the studied phase of the outbreak cycle and annual variations in the survivorship over the postdiapause period, suggesting that postdiapause survivorship was the chief determinant of the cycle. We found the annual changes in population density in the present study to be closely similar in pattern to those from the provincial budworm surveys conducted in much larger areas. This implies that the mechanism underlying the population process found in the few study plots in largely balsam fir stands also applies to the process in much larger areas of diverse stand types. The main source of postdiapause mortality is found to be natural enemies. The impacts of parasitoids and disease are evaluated by rearing budworm samples in the laboratory. Hymenopteran and dipteran parasitoids are by far the major sources of mortality, and microsporidians are the most prevalent pathogen. Occurrences of other entomopathogenic fungi and viruses were insignificant throughout the study. Seasonal changes in laboratory survivorship are compared with the corresponding field survivorship to estimate the effect of predation. No major mortality factor is found to singly play a predominant role in determining the outbreak cycle. Conversely, some minor factors are shown to have played significant roles. Thus, the importance of recognizing the action of natural enemies as a complex is emphasized for understanding the budworm outbreak cycle. Finally, centered on the roles played by the chronological succession of natural enemies in the present study, the results of budworm research in New Brunswick since the mid-1940s are synthesized to outline basic mechanisms underlying the outbreak processes as a guide for further studies.

Key words: Choristoneura fumiferana; *density estimation; entomopathogenic fungi; insect parasitoids; natural-enemy complex; outbreak cycle; predation; succession of mortality factors; survivorship curve; viruses.*

Introduction

Historical account

It is well documented that spruce budworm (the univoltine insect *Choristoneura fumiferana* [Clem.], Lepidoptera: Tortricidae) populations in the spruce–fir forests of eastern Canada periodically increased to epidemic levels in the past few centuries (Blais 1965, Boulanger and Arseneault 2004). In the province of New Brunswick, the first reliable record goes back to around 1770 (Greenbank 1963). Since then, seven outbreaks (and another currently on its way) have occurred quite regularly, with an average interval of about 35 yr (Royama 1992: Fig. 9.4). The latest few outbreaks have been quantitatively documented by the province-wide egg-mass (later, larvae in diapause) surveys in relation to the budworm control program since 1952 (Royama et al. 2005).

To investigate the cause of these outbreaks, the Canadian Forest Service Atlantic Forestry Centre conducted a long-term study (1945–1959, partially extended to 1972) of budworm populations in northwestern New Brunswick as part of the well-known Green River Project (Morris 1963). Although this pioneering work provided basic knowledge of budworm ecology, the reanalysis and reinterpretation of its results (Royama 1984) suggested a need for further investigations to elucidate certain key issues. The reanalysis revealed that the observed budworm population process consisted of two major components: (1) a low-frequency, near periodic principal cycle, commonly recognized as an outbreak cycle; and (2) high-frequency, haphazard deviations

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about the principal cycle, acting as random disturbances (in the statistical sense) to the cycle. In particular, the annual variation in egg-to-adult (i.e., over entire generation) survival was found to have dictated the principal (outbreak) cycle, whereas the variation in the rate of egg recruitment (to a new generation) was the chief source of the deviations about the cycle.

However, a further examination of these earlier data revealed that annual changes in survival from eggs to larvae emerging from winter diapause did not show a consistent trend and, thus, could not be a cause of the principal cycle. This led to a conjecture that postdiapause larval survival, rather than survival over the entire span of a generation, was the effective cause of the principal cycle. Because the dominant factor that determined the postdiapause survival appeared to be the complex of natural enemies (parasitoids, predators, and diseases), the near-periodic outbreak cycle was almost certainly a natural enemy–host interaction cycle (Royama 1992). Also apparent in the early study was a methodological problem in evaluating the impact of mortality factors: in general, it was found to be substantially underestimated (Royama 2001).

To test the above conjecture, and to evaluate the impacts of mortality factors accurately, the Atlantic Forestry Centre launched the present study in 1981 when budworm populations across New Brunswick were extremely high. The present study differs in approaches from those in the Green River study. In the earlier study, life tables were constructed on the basis of intensive but sporadic sampling (i.e., more or less once every instar change) in as many as 19 study plots selected across comparatively large areas to include diverse forest stand types. As a result, the information on population processes obtained from each plot was limited in detail, and the impact of mortality factors could not be adequately evaluated. In contrast, the present study focused on only three study plots in largely balsam fir (*Abies balsamea* (L.) Mill.) stands. Fewer samples were collected at a time, but as frequently (daily) as possible, to produce a practically continuous survivorship curve over the postdiapause period in each season. The frequent field sampling and laboratory rearing scheme in the present study allowed a more precise evaluation of the impact of mortality factors on budworm population processes.

Soon after the study began, however, the budworm populations in New Brunswick started to decline and, by the mid-1990s, had declined so low that field sampling became impractical. The 1993 samples barely provided usable pieces of information, and the field work was terminated after 1994. Thus, the present study covered a major part of the declining phase of the cycle that spanned from the trough around 1960 to the following trough in the middle of the 1990s.

Outline of approaches in the present paper

This paper is divided into three major parts. Part I deals with the estimation and analysis of survivorship in the field. We first construct a survivorship curve, a series of changes in population density over each plot-season, estimated by field sampling, from which we estimate the rates of changes at three key periods: survival of the newly hatched larvae over their winter diapause to spring emergence; their subsequent postdiapause survival to adult eclosion; and the recruitment of eggs to the following generation. We show that the postdiapause survivorship is the chief source of a principal (outbreak) cycle.

Part II deals with the mortality factors that determine the postdiapause survivorship. First, we identify major factors to be a complex of natural enemies, comprising parasitoids, entomopathogenic microbes, and invertebrate and vertebrate predators. We identify the parasitoids and diseases by rearing sampled specimens in the laboratory, and construct a laboratory survivorship curve to compare with the corresponding field survivorship curve. The discrepancy between the two curves evaluates the impact of field mortality other than parasitism and disease, the chief source of which is shown to be predation.

Part III synthesizes the available pieces of information from all budworm research since the mid-1940s in New Brunswick with the view to outlining the mechanisms underlying the budworm outbreak processes as a guide for further studies. In particular, we discuss the roles played by a chronological (seasonal and annual) succession of mortality factors in determining an outbreak cycle.

Life Cycle

General accounts

Spruce budworm in eastern Canada has one generation per year. Eggs are laid in early July to early August in masses, each containing about 20 eggs attached to a needle of host trees (fir or spruce). Eggs hatch in about 10 d. The first-instar larvae (L_1) immediately disperse within the tree crown or beyond by wind, spin hibernacula in suitable sites (e.g., inside old staminate flower bracts, under bark, behind lichen, or anywhere within the stand, not necessarily on the host trees), molt to second instar (L_2), and enter diapause to overwinter. The L_2 emerge from the hibernacula in late April to early May (when an average of 46 degree days above 5.6°C have accumulated), immediately disperse again to find foliage of the host trees, and enter the postdiapause stadia (from final part of L_2 through L_6, pupa, and adult). Before settling on the foliage to feed, the L_2 tend to move within the tree crown. By the time they have settled, the overall abundance of the larvae tends to be highest in mid-crown levels (Eveleigh and Johns 2014).

Having settled on the foliage, the L_2 mine into needles of previous years to feed. After feeding inside a few of these needles, an L_2 molts to L_3 and moves out to feed on a newly developing bud on the periphery of the branch. As new shoots grow, a mature larva webs

together needles around itself to form a "feeding tunnel." Movements within or beyond the tree crown rarely occur among larvae after the L_3 stadium unless the needles (including those of previous years) have been exhausted on the branch or tree. We never saw beyond-tree dispersal in the present study: it was observed once in one plot during the Green River study but was documented as an extreme and rare event (Morris 1963:185). Pupation occurs about late June on the foliage where the L_6 have been feeding.

Adults eclose in about 10 d. The pupal exuviae left by the adults tend to remain on the foliage for a while. Therefore, the number of exuviae estimates the number eclosed. After having laid a portion of their eggs, the majority of females (as well as males) disperse long distances on the wing (depending on weather conditions) from their natal sites to lay the remainder of their eggs elsewhere (Greenbank et al. 1980). Spruce budworm completes its life cycle within tree crowns, except for the comparatively short periods of L_1, L_2, and adult dispersal. The sex ratio in adults is practically 1:1.

Terminology

In the present paper, we divide the life cycle into two major periods: from the laying of eggs to spring emergence of larvae after diapause, designated the "pre-emergence" period and the subsequent "postemergence" or "postdiapause" period to adult eclosion. At the end of each generation, eggs are recruited to a new generation. The ratio (eggs in a new generation)/(adults of preceding generation) is designated "egg-recruitment rate" or simply "recruitment rate." The "adults" include both sexes to represent the rate of population recruitment. Some of the eggs could have been laid by immigrant females, whereas some eggs could have been carried away by emigrating local females. Therefore, the realized recruitment rate at a given local population is the net result of adult dispersal, i.e., the ratio (total number of eggs laid [including those of immigrants] at the locality)/(total number of moths [both sexes] eclosed locally).

Because one generation spans over two calendar years, we distinguish between generation and calendar years. A generation year (designated t, say) starts with eggs laid in calendar year $t - 1$ and ends with adults in calendar year t. In particular, the pre-emergence period in generation t spans over calendar years $t - 1$ to t, whereas the postdiapause period in generation t is the same as the calendar year.

Part I: Estimation and Analysis of Survivorship and Recruitment Rate in Relation to Principal Population Cycle

The aim of this part is to demonstrate that annual changes in postdiapause survivorship are the chief determinants of the principal (outbreak) cycle. We first describe the selection of study plots in the field, sampling routines, and the method of measuring population density. These are followed by the graphical presentation of seasonal changes in population (i.e., field survivorship curves) at all plot-years. From these graphs, we estimate densities at three key stages (i.e., emergence of larvae from diapause, eclosion of adults, and deposit of eggs). Using these estimates, we in turn estimate the postdiapause survivorship, the recruitment rate, and the subsequent pre-emergence survivorship. Finally, we correlate the annual rate of change in principal cycle with postdiapause survivorship.

Study plots

Three plots were selected in largely balsam fir forests (Fig. 1), mixed with spruces, *Picea* spp., and miscellaneous hardwood species (Eveleigh et al. 2007: Table 1). Plot 1 (1981–1990; a 30-yr-old stand around 1980 with codominant trees up to 15 m tall; 98% balsam fir in basal area) was selected in the Acadia Research Forest near the main laboratory in Fredericton. By the mid-1980s, this plot became heavily defoliated, causing more than 60% tree mortality. Therefore, we added Plot 2 (1986–1994; an ~25-yr-old stand with trees up to 12 m tall; 77% balsam fir and 8% *Picea* spp.), also within the Acadia Forest about 10 km west of Plot 1. Toward the beginning of the 1990s, the populations in Acadia, and in general in the middle to southern regions of the province, had declined to such low levels (cf. Royama et al. 2005: Fig. 3) that a detailed survivorship study became difficult. Thus, Plot 3 (1988–1994; 50-yr-old stand, up to 18 m tall; 50% balsam fir and 36% *Picea* spp.) was established in a stand in the northern region of the province near Saint Quentin for a few more years until the population there also declined to a low level. Each plot was sufficiently outside the provincial zones of aerial application of insecticides for spruce budworm control.

Sampling procedures

Prior to the beginning of each plot-year, we marked a group of several codominant balsam fir trees at each of 20 (more or less evenly spaced but readily accessible) locations within each plot. Before larval emergence from winter diapause in each plot-year, 20 sample branches (one from each of the 20 locations) were collected. These were wrapped in paper towel, kept moist, and hung in the rearing room (off the floor) of the laboratory to collect the emerging L_2 until no more larvae emerged. These larvae were reared individually on artificial diet (to be described in *Part II*) to determine parasitism and disease already incurred before and during the diapause.

Soon after the L_2 in the field had emerged and settled on foliage for feeding after dispersal, we began regular sampling and continued until the end of the oviposition period. Sampling was conducted as frequently as possible (mostly daily; otherwise, within the budgetary

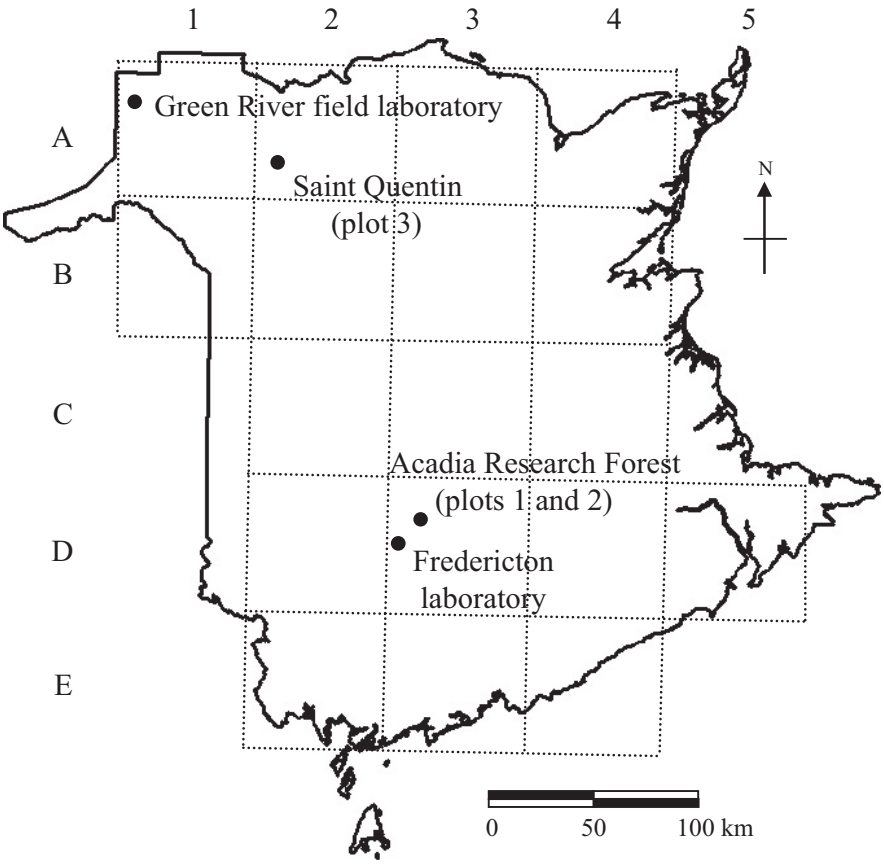

FIG. 1. Outline of the province of New Brunswick, Canada, divided into 18 blocks (after Royama et al. 2005) and the locations of the study plots and laboratories; Green River field laboratory is no longer in use for budworm research.

restriction). On each sampling day during the earlier years when the budworm populations were at their epidemic state, one branch was collected at a mid-crown level of one tree from each of the 20 locations, i.e., 20 branches each day/plot altogether. In the later years of low populations, extra branches were collected at each location. Within each group of marked trees, a different one was used for sampling at a time so as not to deplete the foliage of any single tree throughout the season. We collected the entire section of a foliated branch to count all budworm individuals therein in order to eliminate the effect of seasonal redistribution of budworm individuals within the branch, e.g., from old needles to new buds and to growing shoots.

The sample branches were collected in the morning and were immediately transported to the laboratory for same-day processing, mainly to count eggs, larvae, pupae, and pupal exuviae, and to transfer larvae and pupae individually into rearing vials. As Plot 3 was located in a remote area, same-day processing at the laboratory was impossible, and the sampled branches were stored in a cold room (at 4°C) until the following morning. Details of laboratory rearing of individuals will be given at the beginning of *Part II*.

Method of measuring population density

Density can be expressed in several different ways: number per unit area of a forest stand; number per tree; or number per unit area of sample branches. Variations in forest type, e.g., species composition, tree ages or the health status, would make it difficult to standardize the first two of those expressions. Thus, we use the number per unit area of the foliated section of whole branches (cut near bole) on apparently healthy codominant trees.

Sample branches were placed on a flat surface to measure the length and average width of the foliated area (in meters): the foliated section of a sample branch was on average about 1 m in length. The product (length × width), so-called "branch surface area" or BSA, was used as the budworm's effective habitat space. The total counts of eggs, larvae, pupae, or pupal exuviae (as an estimate of the number of adults) on the sample branches were divided by the total BSA as the unit of density, i.e., number/m^2 BSA. Since its origin in the Green River Project (Morris 1963), this unit has been widely used as a standard, with minor variations, for its practicality in budworm research in New Brunswick and elsewhere. Therefore, many aspects of the results from our study are

readily compared with those from the Green River study and the provincial egg-mass surveys.

Results and Analyses of Part I

Seasonal changes in population density

Fig. 2 shows the series of daily population densities in Plot 1 plotted against sampling days; the results from Plots 2 and 3 are collectively shown in Fig. 3. The series of solid circles in each graph constitutes a survivorship curve for the entire postdiapause period from the emergent L_2 to adults (pupal exuviae), and the series of open circles forms an oviposition curve. These curves serve two different purposes. One is to provide a baseline for the estimation and analyses of postdiapause mortality, the main topic in Part II. The immediate purpose here is to estimate densities at three key points over a generation, already mentioned in the section *Outline of approaches in the present paper*: (1) initial density of postemergence larvae, (2) adult density, and (3) egg density.

Estimation of densities at three key points in a generation

The data points in a series of population densities fluctuate considerably from one sampling day to another, which makes a single-value estimation difficult. This fluctuation in sampling is minimized by smoothing the data series by five- or three-point moving-average transformation, henceforth designated Ma(5) or Ma(3) smoothing, depending on the length of the series concerned. We use the simplest, unweighted transformation.

Initial density of the postdiapause larvae.—Ideally, an average of the earliest few points in the postdiapause (solid circle) series in Figs. 2, 3 would estimate the initial density. Often, however, the early section of the solid-circle series tended to increase before declining due to mortality. This tendency was quite consistent from year to year, especially in Plots 1 and 2 where peaks of survivorship curves tended to occur around ordinal day 150 when the majority of larvae were L_3 to L_4. This tendency is shown in an example from Plot 1, 1985 (Fig. 4a) in which the data points (open

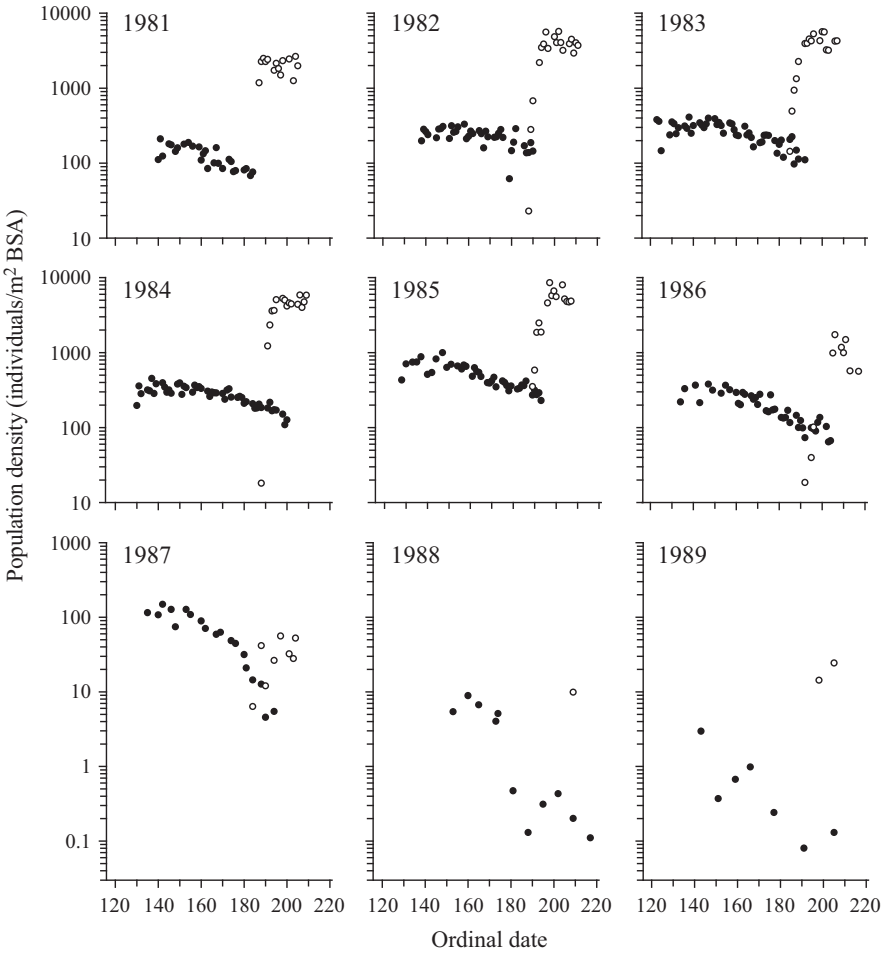

Fig. 2. Seasonal changes in budworm populations in Plot 1 from 1981 to 1989 calendar years, as depicted by foliage sampling. Solid circle: total number of larvae, pupae, and pupal exuviae on each sampling day. Open circle: number of eggs for the generation year following the calendar year marked in each graph. BSA, branch surface area.

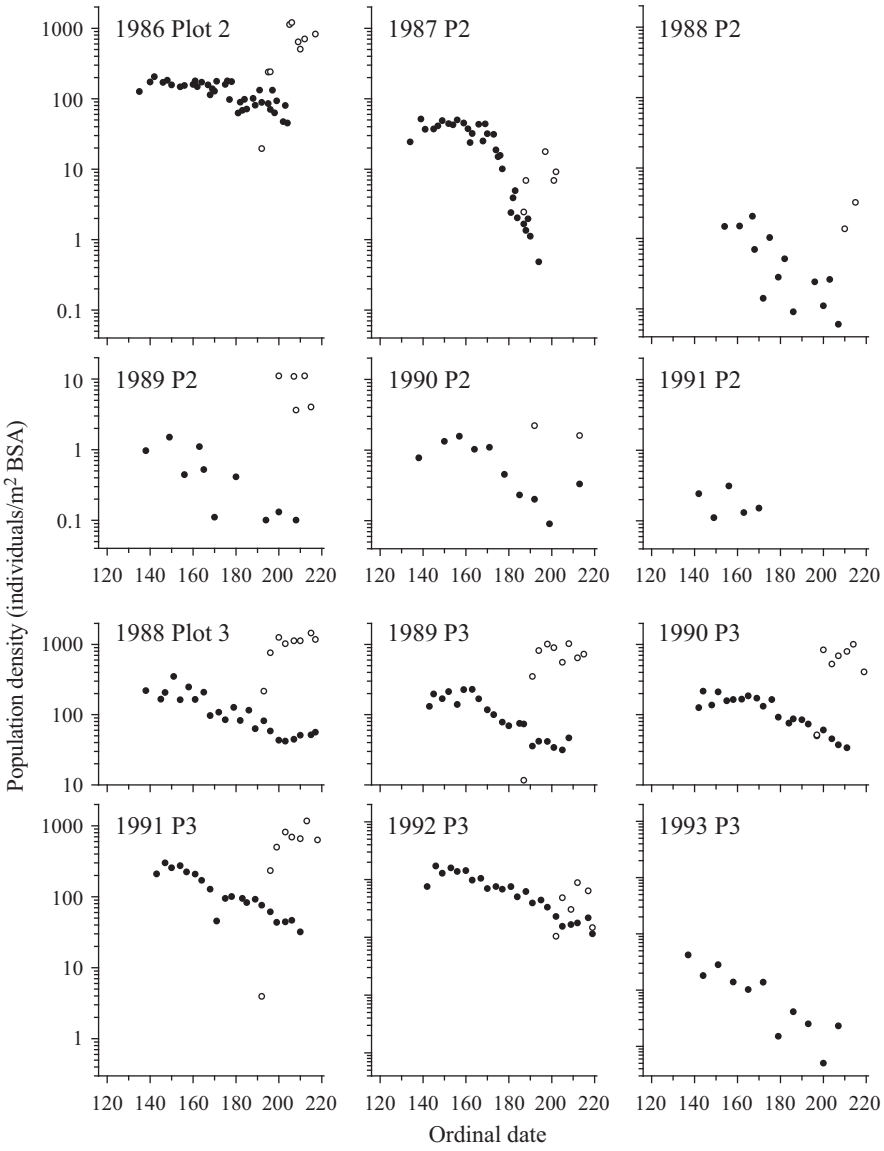

FIG. 3. Same as Fig. 2 but for Plot 2 (1986–1991) and Plot 3 (1988–1993) calendar years.

circles) are plotted in a linear scale and are Ma(5)-smoothed (solid-circle series). It is also apparent in Fig. 4a that over the initial increasing period, the unsmoothed data points (open circles) tended to fluctuate in much larger amplitude than those after the peak. The most likely cause of the increase, associated with the large variation in the samples, was the movement of larvae within the tree crown: as already mentioned in *Life cycle*, the emerging L_2 larvae tended to move within the tree crown during the early period of emergence from diapause.

As little mortality occurred during this early stage (to be shown in *Part II*), we take the peak of the smoothed curve as an optimal (from the field study point of view) estimate of the density of initial postdiapause larvae, henceforth designated "initial larvae ($L_{3\text{-}4}$)" as most of the larvae were then in these instars (Fig. 4b). When the population became extremely low toward the end of the study, we used a simple average of the first few data points, excluding those that were judged by eye to be exceptionally low.

Adult density.—We use the number of pupal exuviae as that of adults successfully eclosed. The exuviae tend to remain on the foliage for a period of time, and plotting their counts against the sampling days forms a cumulative eclosion curve. Ideally, the curve should reach a plateau after eclosion has ended, and the plateau estimates the number of adults eclosed for the season. However, we often observed a field eclosion curve to decrease after a peak had been reached (Fig. 5). This decrease was most likely due to some of the exuviae falling off the foliage. Thus, we used the highest point of an Ma(3)-smoothed

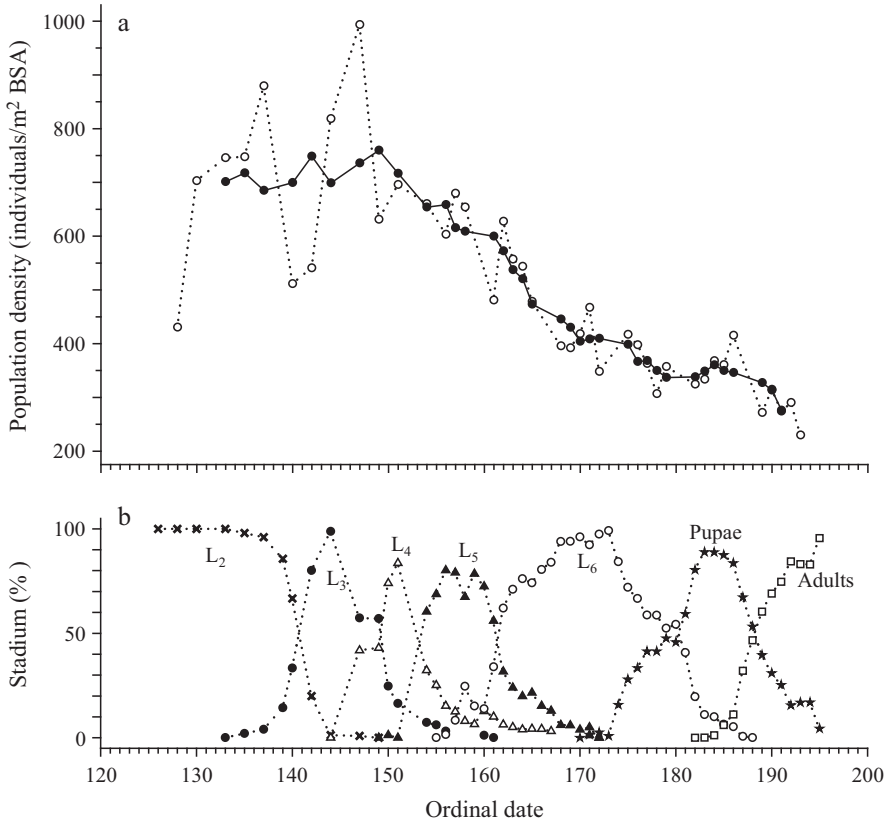

FIG. 4. (a) An example of seasonal changes in postdiapause budworm population at Plot 1, 1985 (open circles: same as the solid-circle series in Fig. 2 but in linear scale), smoothed by a five-point moving-average transformation (solid circles). (b) Seasonal changes in budworm developmental stadia (%) in the same plot-year. L_2–L_6 represent instars.

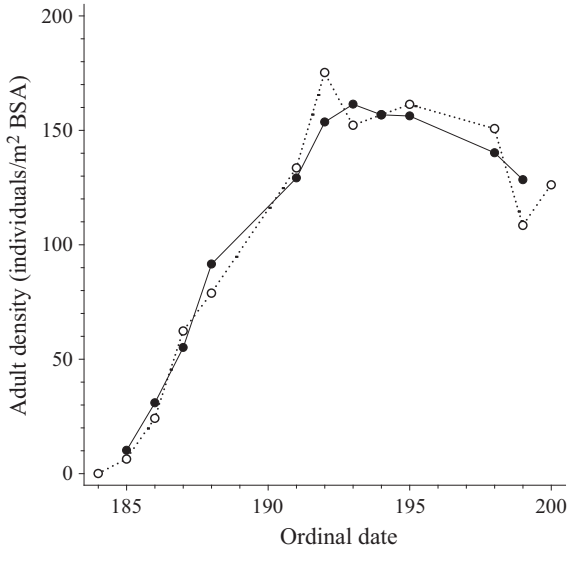

FIG. 5. An example of adult eclosion curve from Plot 1, 1984 (open circles), smoothed by an Ma(3) transformation (solid circles).

curve as an optimal estimate of adult density, permitting slight underestimation.

Egg density.—Eggs were counted by the (number of egg masses) × (20 eggs/mass) (19.7 ± 0.60 [mean ± SE], an average of 12 plot-years). In Figs. 2 and 3, there is a tendency for an oviposition (open circle) series to decline slightly after reaching a peak, as exemplified in Fig. 6. This was most certainly due to the loss of the needles to which egg masses were attached. We use the highest point of the Ma(3)-smoothed curve as an optimal estimate of the total eggs laid. In years with very low populations, there frequently were days with zero counts, yet larvae were subsequently found. A simple average (rather than a moving average) of the nonzero data points in the cluster (excluding those at much lower levels by eye) was used as an estimate.

Analyses of annual variations in populations, survivorship, and recruitment rate

Annual variation in population densities.—Table 1 shows estimated densities of the initial postdiapause larvae

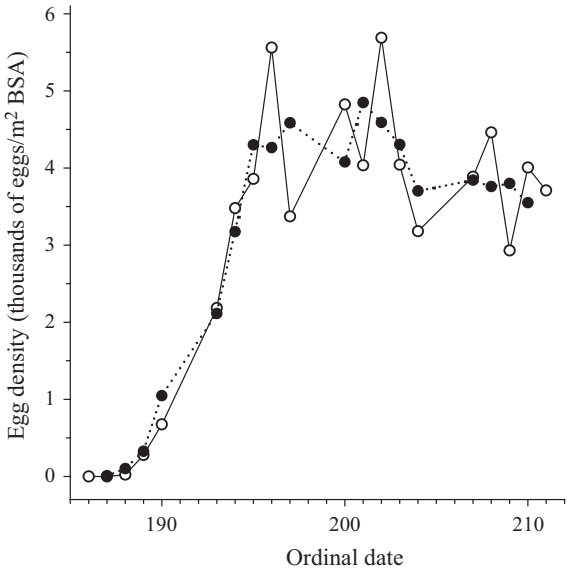

FIG. 6. An example of oviposition curve (open circles) observed at Plot 1, 1982 (copy of open-circle series in Fig. 2 but in linear scale), smoothed by an Ma(3) transformation (solid circles).

($L_{3\text{-}4}$), adults, and eggs. A close examination of the data reveals that the population trend is nearly identical between Plots 1 and 2, which were closely located within the Acadia Research Forest. Therefore, we combined the data from these plots as Plot (1 + 2) to increase the length of observations: Appendix S1 shows how we combined them. After transforming to common logarithms, the annual variations in Plot (1 + 2) and Plot 3 are graphically summarized in Fig. 7 to depict the pattern of population changes from the peak-to-declining phase of the outbreak cycle. We now attempt to evaluate the contribution of postdiapause survivorship to the principal (outbreak) cycle. Yet, density is the number of individuals at a given point in time, whereas survivorship is a rate of change in the number over a given interval of time. Therefore, to make these two measurements directly comparable, we transform the annual variation in population density into its rate of change from one year to the next. The annual rate of change (henceforth just rate of change) can then be decomposed into survivorships and recruitment rate.

Decomposition of the rate of change in population into its components.—Let l_t, a_t, and e_t stand, respectively, for the densities of the initial ($L_{3\text{-}4}$) larvae, adults, and eggs in generation t. Then, selecting the (longest) $L_{3\text{-}4}$ series, its rate of change from generation t to $t + 1$ (i.e., l_{t+1}/l_t) can be decomposed into the postdiapause ($L_{3\text{-}4}$-to-adult) survivorship (i.e., a_t/l_t); the rate at which eggs are recruited to generation $t + 1$ (i.e., e_{t+1}/a_t); and the subsequent pre-emergence (egg-to-$L_{3\text{-}4}$) survival (i.e., l_{t+1}/e_{t+1}). The exact relationship is

$$l_{t+1}/l_t = (a_t/l_t)(e_{t+1}/a_t)(l_{t+1}/e_{t+1}).$$

Fig. 8 shows the annual variations in $\log(l_{t+1}/l_t)$, $\log(a_t/l_t)$, $\log(e_{t+1}/a_t)$, and $\log(l_{t+1}/e_{t+1})$ at Plot (1 + 2) on the left and Plot 3 on the right. The series $\{\log(l_{t+1}/l_t)\}$ in particular are smoothed by an Ma(3) transformation (open-circle series) as an estimate of the rate of changes in principal cycle in each plot. All rates (in solid circles) are plotted against the calendar year t such that, given t, the log rates in panels b to d sum up to panel a. (Note 1: Although the pre-emergence survival $[l_{t+1}/e_{t+1}]$ occurred in generation year $t + 1$, it is plotted against calendar year t to conform to the above

TABLE 1. Estimates of the initial number of postdiapause larvae ($L_{3\text{-}4}$), total number of adults eclosed, and total number of eggs laid (all numbers/m^2 BSA) at three study plots in New Brunswick, Canada.

	Plot 1			Plot 2			Plot 3		
Year	$L_{3\text{-}4}$	Adult	Egg	$L_{3\text{-}4}$	Adult	Egg	$L_{3\text{-}4}$	Adult	Egg
1981	171.6	80.6							
1982	293.6	168.9	2381.4						
1983	358.7	145.1	4848.4						
1984	372.0	171.9	4985.6						
1985	759.8	247.0	5105.6						
1986	332.6	68.8	6960.7	176.4	45.7				
1987	148.2	4.6	1353.1	42.8	3.0	1160.2			
1988	7.0	0.3	38.7	1.7	0.0	11.2	225.6	49.7	
1989	3.0	0.1	9.9	1.1	0.0	1.5	194.3	31.3	1245.8
1990			24.3	1.3	0.1	8.5	176.0	38.0	903.1
1991				0.2	0.0	0.7	251.3	17.6	826.4
1992				0.4	0.0	0.0	170.8	13.1	835.0
1993				0.2	0.0	0.0	2.9	0.0	60.4
1994				0.1	0.0	0.0	0.1	0.0	0.0

Notes: Year is generation year. Blank spaces indicate no sampling.

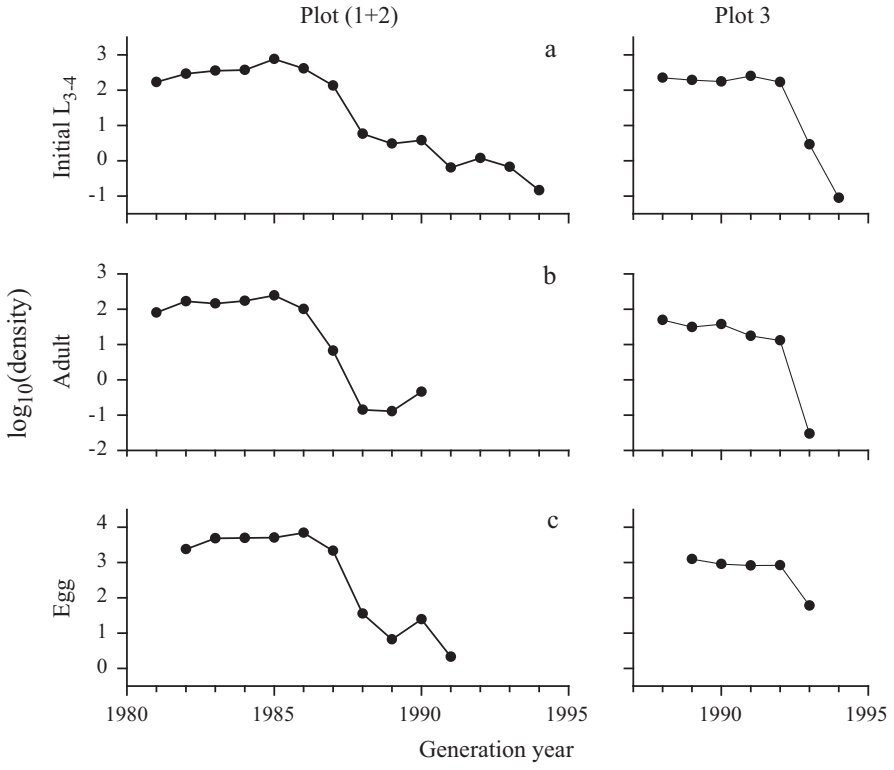

FIG. 7. Annual variations in log10(individuals/m^2 BSA) of (a) initial postdiapause larvae (L_{3-4}); (b) adults; and (c) eggs, in Plot (1 + 2) on the left, and those in Plot 3 on the right.

summation. Note 2: An open circle [Fig. 8a], e.g., average of 3 yr t, $t + 1$, and $t + 2$, is plotted against $t + 2$.)

Demonstration of postdiapause survivorship as chief source of principal cycle.—The open-circle series in Fig. 8a (i.e., estimated rate of change in principal cycle) and the series in Fig. 8b (i.e., observed postdiapause survivorship) in each plot are reproduced in Fig. 9a as open-circle and solid-circle series, respectively. We see a high degree of congruence between the two series. The rate of change in principal cycle is regressed on the postdiapause survivorship (Fig. 9b) after pooling the results from Plot (1 + 2) and Plot 3, yielding a 0.92 correlation (r). It suggests that 85% ($100 \times r^2$) of the variation in the rate of change in principal cycle is attributable to the variation in postdiapause survivorship.

In each plot, we combine the egg-recruitment series, i.e., $\{\log(e_{t+1}/a_t)\}$ (Fig. 8c), and the subsequent pre-emergence survivorship series, i.e., $\{\log(l_{t+1}/e_{t+1})\}$ (Fig. 8d). The combined series, i.e., $\{\log(l_{t+1}/a_t)\} = \{\log(e_{t+1}/a_t)\} + \{\log(l_{t+1}/e_{t+1})\}$, plotted in Fig. 9c (solid circles), represents the series of all events occurring during the pre-emergence period, and is designated "pre-emergence series." In the meantime, we calculated deviations of the solid-circle series $\{\log(l_{t+1}/l_t\}$ in Fig. 8a about the open-circle series of the estimated rates of change in principal cycle. These deviations are the residuals after smoothing the $\{\log(l_{t+1}/l_t\}$ series. The series of the residuals is plotted in Fig. 9c (open-circle series) to be compared with the foregoing pre-emergence series $\{\log(l_{t+1}/a_t)\}$. We see a high degree of congruence between the two series, quite naturally as a corollary of the congruence in Fig. 9a. The residuals are regressed in Fig. 9d on the corresponding pre-emergence events, $\log(l_{t+1}/a_t)$, after pooling Plots (1 + 2) and 3, yielding a 0.95 correlation (r). This suggests that 90% ($100 \times r^2$) of the deviations about the rate of change in principal cycle are attributable to events during the pre-emergence period. Thus, these results support the conjecture, posed in *Introduction*, that the annual variations in postdiapause survivorship are the chief source of the principal outbreak cycles in these study plots.

To conclude *Part I*, we show that the foregoing results, obtained in local areas that were limited in size and stand diversity, in fact apply to much larger areas and diverse stand types.

Universality of the population process.—The New Brunswick Department of Natural Resources annually conducts province-wide budworm population surveys (counting egg masses in earlier years and, since 1985, the L_2 in diapause) as part of its pest-control program. It provides independent information to compare with certain aspects of the population changes depicted in the

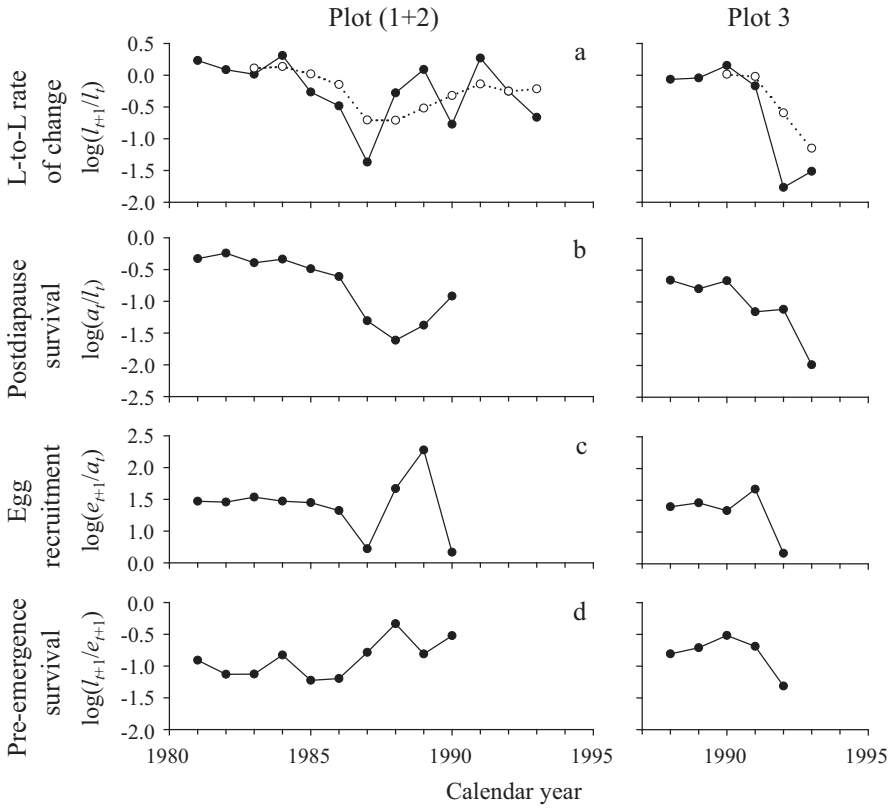

FIG. 8. Annual variations in Plot (1 + 2) and Plot 3 in (a) log10($L_{3\text{-}4}$-to-$L_{3\text{-}4}$ rate of change, l_{t+1}/l_t), decomposed into (b) log ($L_{3\text{-}4}$-to-adult survival, a_t/l_t); (c) log(egg recruitment, e_{t+1}/a_t); and (d) log(egg-to- $L_{3\text{-}4}$ survival, l_{t+1}/e_{t+1}), all plotted against the calendar year t. The solid-circle series in each plot in panel a is smoothed by an Ma(3) transformation and plotted as open-circle series lagging 2 yr behind the solid-circle series. The variables l_t, a_t, and e_t stand, respectively, for the densities of the initial ($L_{3\text{-}4}$) larvae, adults, and eggs in generation t.

present study. The province is divided into 18 blocks (Fig. 1) of about 66 × 66 km^2, and the annual changes in the mean (egg masses or L_2) density in each block were estimated. The numerical data are provided in *Ecological Archives* E086-067-S1 as a supplement to Royama et al. (2005). Appendix S2 provides additional notes.

Fig. 10 compares the temporal variations in four key attributes between each plot and its respective block. We see a high degree of congruence in the pattern of annual variation in each attribute especially between Plot (1 + 2) and Block D3, although the degree of congruence is much less between Plot 3 and Block A2. This lesser degree of congruence is attributable largely to differences in the pre-emergence processes in Plot 3 substantially deviating from the corresponding (average) processes in Block A2. However, as shown in Appendix S2, the postdiapause processes are much the same as in Block A2.

The above findings imply that the mechanism underlying the postdiapause population process as revealed in the present study basically applies to those populations in the surrounding much larger areas with diverse stand types.

PART II: EVALUATION AND ANALYSIS OF MORTALITY FACTORS IN POSTDIAPAUSE PERIOD

Outline of procedures

Given that postdiapause survivorship is the chief source of the principal outbreak cycle, our aim here is to identify the major mortality factors involved and to evaluate their impacts. There are three groups of factors as the chief sources of budworm mortality: parasitism, disease, and predation. There is a fourth, conceivable factor: defoliation-caused stand conditions that might influence the supply of quality food. As will be discussed shortly, we find little sign that the fourth is a significant factor determining the postdiapause mortality.

We first construct, in each plot-year, a survivorship curve in laboratory rearing. The curve is constructed so as to "simulate" what would be expected in an environment in which the combined effect of parasitism and disease is the chief source of mortality. The curve is then compared with the corresponding field survivorship curve already constructed (Figs. 2, 3) for similarity and dissimilarity. The latter implies the involvement of a factor other than parasitism and disease, the most likely being predation.

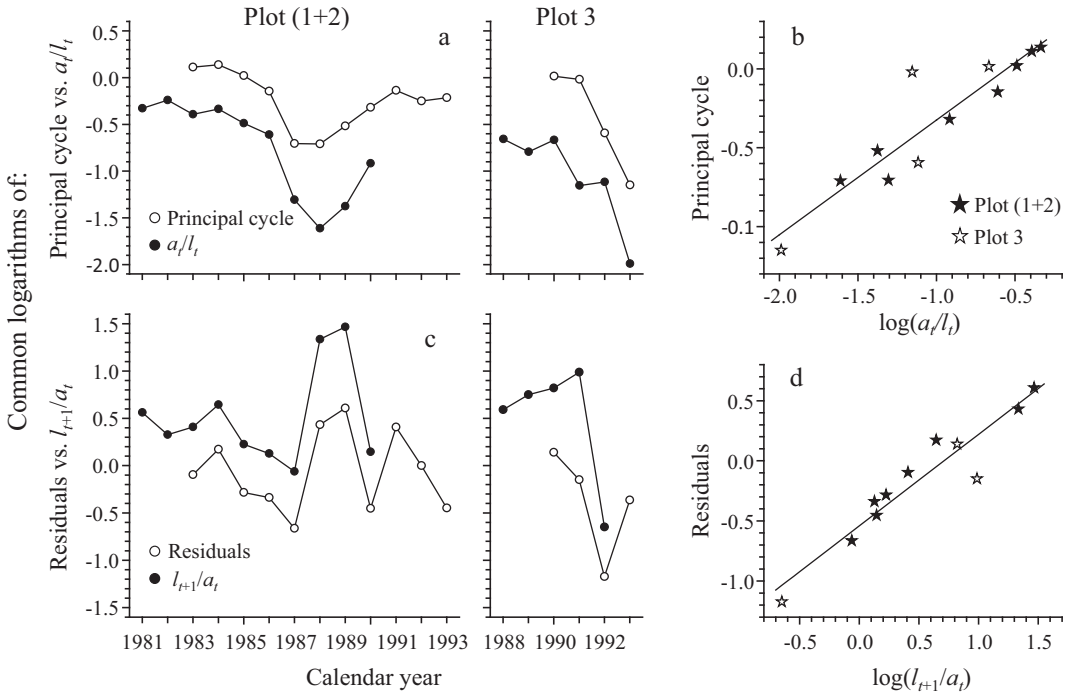

FIG. 9. (a) Open-circle series: a copy of the same in Fig. 8a, i.e., the series of $\log(l_{t+1}/l_t)$ smoothed by an Ma(3) transformation as an estimate of the (rate of change in) principal cycle, compared with the solid-circle series of postdiapause survivorships $\{\log(a_t/l_t)\}$, a copy of the same in Fig. 8b. (b) Regression of the (rate of change in) principal cycle on the postdiapause survivorship, $\log(a_t/l_t)$, yielding the Pearson correlation coefficient $r = 0.921$, $P < 0.001$. (c) Open circles: the series of residuals, after smoothing the series $\{\log(l_{t+1}/l_t)\}$ (Fig. 8a), as estimates of the deviations about the rate of change in principal cycle, compared with the solid-circle series $\{\log(l_{t+1}/a_t)$, i.e., the sum of egg recruitment, $\log(e_{t+1}/a_t)$, and the subsequent pre-emergence survivorship, $\log(l_{t+1}/e_{t+1})\}$. (d) Regression of the residuals on $\log(l_{t+1}/a_t)$, yielding the correlation coefficient $r = 0.951$, $P < 0.001$.

We then attempt to evaluate the relative impacts of parasitism and disease as separate sources of mortality. First, we describe the laboratory rearing procedures.

Laboratory rearing

Sample branches from Plots 1 and 2 were collected in the morning and brought to the laboratory as soon as possible for same-day processing. The samples from Plot 3 (which required full-day return trips) were kept at 4°C and were processed the following morning. The processing involved counting budworm individuals and transferring them individually into glass vials for rearing. We reared a minimum of 100 individuals on each sampling day. To avoid selecting easy-to-find individuals, we reared all specimens from one whole branch even if there were more than 100 on the branch; if fewer than 100, all from another branch were added to rearing. However, securing the minimum 100 specimens became difficult at extremely low populations toward the end of the study.

Each of the 7.4-ml vials was partially filled with artificial diet (McMorran 1965) and was placed upside-down with the opening (plugged with non-airtight foam plastic) at the bottom. The amount of diet in each vial was enough for the L_2 larva within to complete its development. The rearing room was kept in the low 20s°C with 50–60% relative humidity and 16:8 light-to-dark hours. Every individual was inspected as frequently as possible for its status, e.g., dead or alive, development, parasitized, or diseased. Dead individuals were kept either at 4°C for diagnosis within a few days, or at −20°C if diagnosis was delayed. Unidentifiable parasitoids were either pinned and kept at laboratory temperature, or kept unpinned at −20°C for later identification. Daily inspections were maintained until 1985 but, from then on, weekends had to be curtailed for budgetary reasons. For other aspects of rearing, see Lucarotti et al. (2004) and Eveleigh et al. (2012).

Construction of laboratory survivorship curves

The principle of construction is as follows. Ideally, a fresh batch of field-collected larvae and pupae on a given day are reared, and the number of survivors by the following day is counted. The ratio (survivors)/(number reared) estimates the survivorship of the batch over the 1-d interval, designated "interval survivorship" in rearing. The successive multiplications of the interval survivorships over a given period (in days) produce the cohort survivorship curve over the period. Keeping the

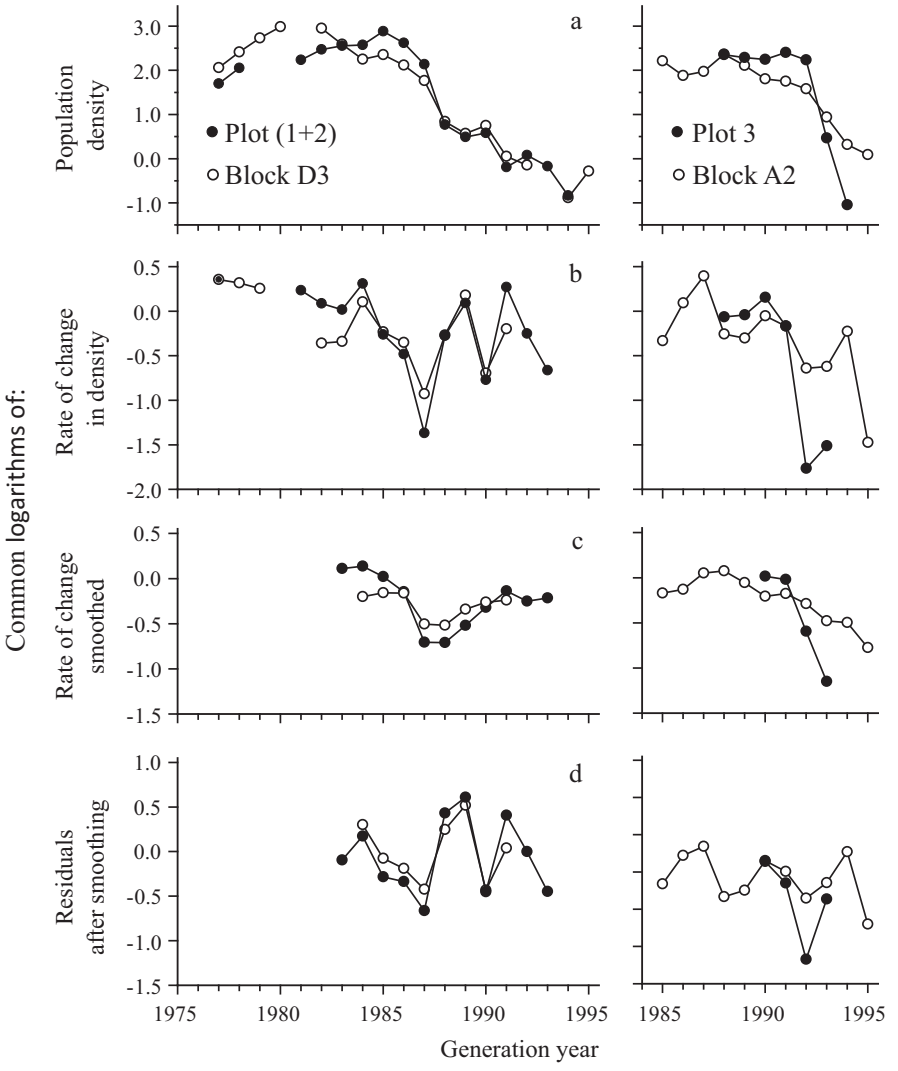

FIG. 10. Comparisons of annual variations in budworm populations between the Provincial budworm surveys (open circles) and the present study (solid circles); locations of Blocks D3 and A2 are in Fig. 1. (a) Population densities: individuals/10 m^2 BSA in the Provincial survey; individuals/m^2 in the present study. (b) Inter-generation rate of change in population. (c) Ma(3)-smoothed series of the rate-of-change in (b). (d) Residual series after Ma-smoothing in (c). Extra data points (1977 and 1978) in (a) for Plot (1 + 2) are from the preliminary sampling.

interval of estimation as short as 1 d has the advantage of minimizing a potential discrepancy between field and laboratory conditions. We found, however, that this created practical problems. First, immediately following a collection, the physical or physiological status of the samples could be unsettled, resulting in a probable discrepancy between the field population and laboratory samples. Second, a mortality estimate based on 1-d collection (batch) tended to fluctuate considerably from batch to batch. Therefore, we pooled the batches of samples collected over 3 d to minimize these undesirable effects. The actual procedures involved are as follows.

On a given inspection day (say, day i), we select three batches consecutively collected over the past three sampling days (i.e., $i - 3$ to $i - 1$). Among these batches, we count the total number survived to day $i - 1$, as well as those survived to day i. Then, we use the following ratio

$$\frac{[\text{Counts of survivors on day } i]}{[\text{Counts of survivors on day } (i-1)]}$$

as an estimate of the survivorship over the interval between day (i –1) and i, designated "the ith interval survivorship" or S_i. Then, we move on to calculate the (i + 1)th interval survivorship, S_{i+1}, using the following group of the three batches from day ($i - 2$) to ($i + 1$) and so on, an idea similar to calculating moving averages. Successive multiplications of interval survivorships from day 1 of rearing to inspection day i yield the cohort survivorship over the period, designated $Cs_i = S_1 \times$

$S_2 \times \ldots \times S_i$. Successively plotting Cs_i against inspection days 1, 2, 3,. . ., i constructs a survivorship curve of a laboratory cohort, starting with its initial survivorship S_1 that is set nominally at 100 such that a Cs_i is standardized as a percentage of the initial cohort size.

The calculation is straightforward until adult eclosion begins. Thenceforth, the adults that eclosed in a given interval are counted as (eternal) survivors, and hence their numbers accumulate in the calculations for all subsequent days. Accordingly, the ith interval survivorship S_i should eventually converge to 1 when all survivors have eclosed; or the cohort survivorship curve (Cs curve) should eventually level off. However, we often found some parasitized larvae or pupae, albeit destined to die, still alive for a while after adult eclosion had practically been over; often they exhibited peculiar symptoms and were readily identified. The calculation of the survivorship curve was terminated at this moment, even if it had not yet leveled off.

We encountered some technical problems in the process of constructing the laboratory survivorship curves. Major problems and practical remedies are explained in Appendix S3.

Comparison between laboratory and field survivorships

The laboratory survivorship curves are superimposed upon the corresponding field curves to reveal their similarities and dissimilarities (Fig. 11 for Plot 1; Fig. 12 for Plots 2 and 3). The method of superimposition is as follows. In an ideal situation, it could have been done by matching their initial values, i.e., 100% in the laboratory curve and the initial postdiapause density in the field curve. However, as already exemplified in Fig. 4, the very early part of a field curve tended to increase (because of the within-crown movement of the emerging larvae) only to have begun to consistently decrease after a while. Thus, the earliest part of the declining section in the field curve (e.g., after around day 150 in Fig. 4) is matched (by eye) with the corresponding section of the laboratory curve. We see two anomalous cases, 1982 in Plot 1 and 1986 in Plot 2, in which there were more deaths in the laboratory than in the field: these were likely because the parasitoids somehow developed faster in rearing. No such anomalies occurred in other plot-years.

Generally, in years of high population density, the two curves tend to match well for the early part of the

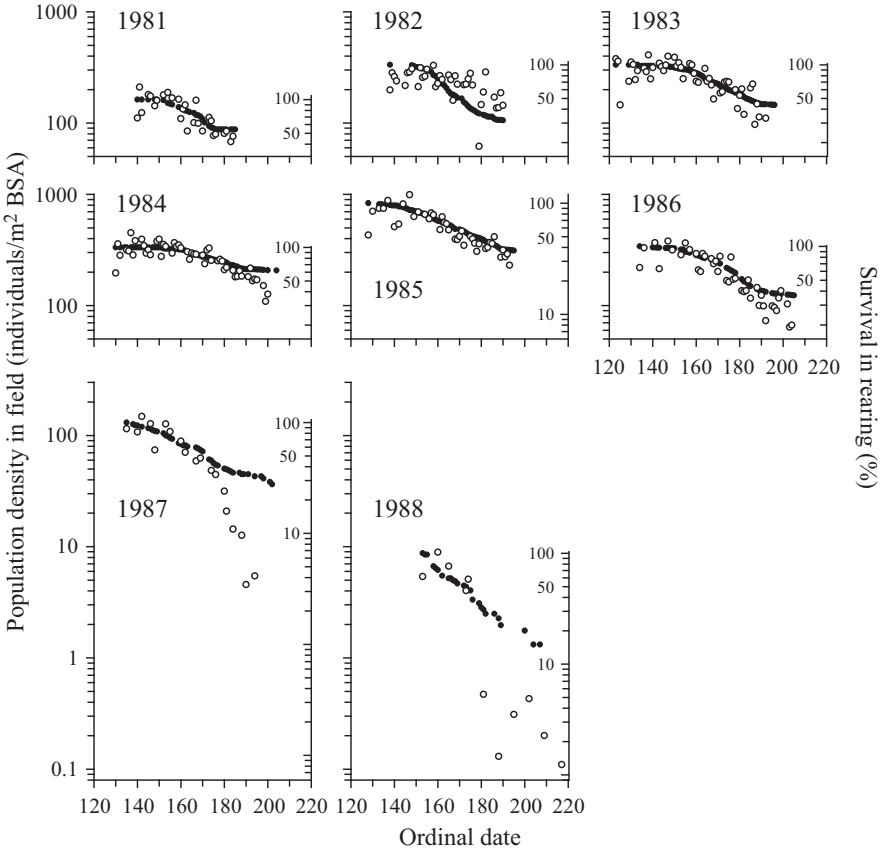

FIG. 11. Comparison between the field and laboratory survivorship curves in Plot 1 in the 1981–1988 postdiapause seasons. Open-circle series: Survivorship curves in field as depicted by series of population densities (individuals/m^2 BSA; copies of solid-circle series in Fig. 2), scaled on the left-axis. Solid-circle series: survivorship curves in laboratory rearing scaled on the right.

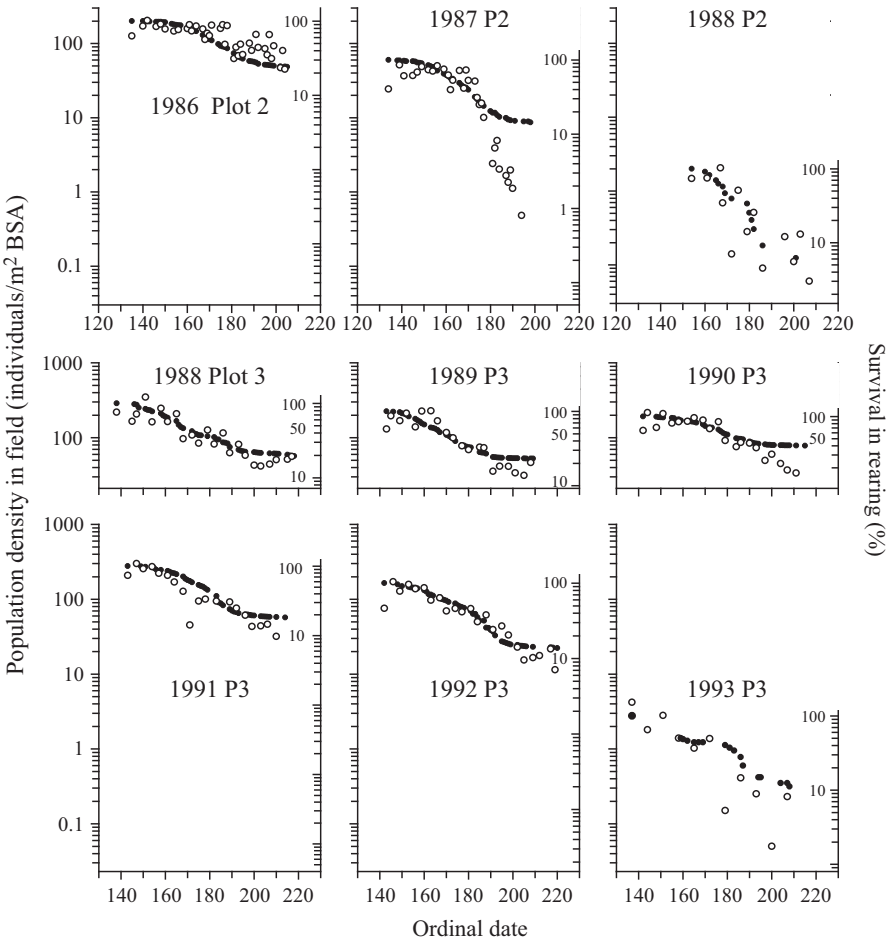

FIG. 12. Same as Fig. 11 but for Plot 2 (1986–1988) and Plot 3 (1988–1993); open-circle series are copies of the solid-circle series in Fig. 3.

season, suggesting that parasitism and disease were the chief sources of field mortality.

On the other hand, a field curve often became noticeably lower than the laboratory curve later in the season. Also, the deviation became particularly noticeable when the field density of budworm became as low as or below 50 individuals/m^2 BSA.

Two conceivable sources of the discrepancy are: predation and, as noted at the beginning of *Part II*, a defoliation-caused decrease in the quality of food supply. We reject the latter to be a likely cause, however. First, as will be shown shortly, the downward deviation of the field curve from the laboratory curve occurred after a majority of the larvae had pupated or had virtually finished feeding. If these were due to malnutrition from feeding on low quality needles, we should have seen some symptoms among the larvae in rearing. Virtually all deaths among the mature larvae and pupae in rearing were diagnosed as parasitized or diseased by pathogens. Second, as already shown in Appendix S1, the rate of change in population was virtually identical between the heavily defoliated Plot 1 and only lightly defoliated Plot 2. Third, we see no sign that the occurrence of a noticeable discrepancy (Figs. 11, 12) was consistently associated with differences in defoliation level (cf. Table in Appendix S4). Thus, we consider predation to be the chief source of the discrepancy between the laboratory and field survivorships. However, the degree of discrepancy varies from barely noticeable to extremely high. To comprehend such variations, we examine probable sources of predation in the following.

Probable sources of predation

Although no quantitative investigation was systematically made in the present study, many invertebrates were found on sample branches, e.g., spiders, pentatomids, adult carabid and elaterid beetles, and a few ants, all of them being potential predators of early instar budworm larvae. However, these predators were never sufficiently abundant to be the likely cause of the large difference between the field and laboratory survivorship curves,

especially in those years, or in the early part of a season, when the larval density was sufficiently high. Therefore, we attribute a substantial deviation (e.g., one in Fig. 13) to larger vertebrate predators. The following circumstantial evidence suggests that insectivorous birds were the major source.

Birds as major predators in later part of season.—An example in Fig. 13 typically shows that the field curve began to deviate from the laboratory curve around day 175 when a majority of budworm individuals were late L_6 to pupae; by day 180, the proportion of L_6 had declined substantially. This chronological pattern fits the following observations. During the previous outbreaks, many warblers of the family Parulidae were reported to be major vertebrate predators. Their stomach contents were mostly fully mature larvae and pupae but rarely younger-instar larvae (Mitchell 1952, Dowden et al. 1953, Mook 1963). It was also reported, albeit unrelated to spruce budworm, that tortricoid pupae in particular were extremely vulnerable to bird predation (Royama 1970).

Also noticeable is the fact that a substantial difference between the field and laboratory curves occurred only sporadically over all plot-years. This also makes sense for the following reason. The number of the breeding birds in a forest compartment tends to be limited because of their territoriality. However, their consumption of budworm could increase substantially after the successful nests had produced many fledglings. Thus, the impact of bird predation on budworm would depend on the rate of breeding success, which could vary greatly in time and space.

In general, the abundance of predators is always limited compared with budworm populations at an epidemic level. Consequently, even if a large number (in absolute value) was actually eaten, the rate of predation stayed negligible until the budworm populations declined to a sufficiently low level. (Note: the impact of predation can be quantitatively evaluated as shown in Appendix S5.) In conclusion, it is unlikely that predation would consistently exert a significant impact on a budworm population until it is brought down to a sufficiently low level by parasitism and disease.

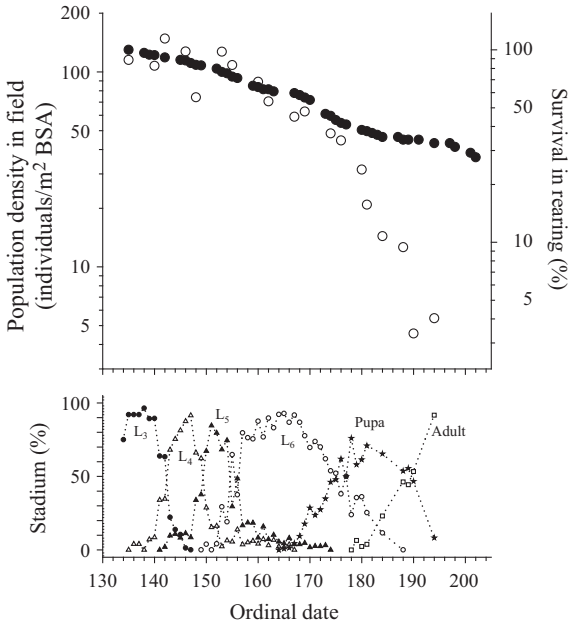

FIG. 13. Example from Plot 1, 1987 (singled out from Fig. 11), showing the timing of the field survivorship (open circles) deviating downward from the laboratory curve (solid circles) around day 175 when a majority of budworm was in pupal stage.

Evaluation of parasitism and disease as separate units

In the preceding section, we treated the union of parasitism and disease as a single unit of mortality factors as against predation. Here, we attempt to evaluate the relative impacts of parasitism and disease as separate units. However, there is a technical problem that needs to be resolved. First, we conceptually recognize four categories of field-collected individuals to be reared: (1) those that are parasitized but not "diseased" (see note); (2) those that are diseased but not parasitized; (3) those that are parasitized as well as diseased; and (4) those that are neither parasitized nor diseased. (Note: the "diseased" category excludes those individuals that are infected but can complete their development.)

The problem lies in the evaluations of the first three categories, as we can only observe the proximate cause of a death in rearing. Thus, those were nominally tallied as either killed by parasitism or by disease, depending on whichever factor killed them first. Therefore, the nominal value of mortality is an unreliable measure of the impact of each unit because it would be affected by the presence or action of the other unit. This is awkward, especially when the frequency of deaths due to either unit varies inconsistently from time to time or from place to place. To avoid the problem, we use the latent status of each unit as an appropriate measure of its impact. However, the latency of each unit could not be determined by observation. Therefore, we opted for finding an approximate measure of the latency by calculation, using the nominal frequency of mortality in rearing. How to derive the approximation is shown in the following schema.

Schematic representation of latent parasitism and disease.—Fig. 14 represents the aforementioned four categories of individuals in rearing over a given interval of inspections, e.g., the ith interval between inspection days $i - 1$ and i. The unit square [aceg] represents the initial cohort of individuals collected in the field at the beginning of the interval. Among the cohort, there is a set (say, **V**) of those individuals belonging to category 1 or 3. Their mortality occurring over the ith interval is represented by the shaded rectangle [acdh], designated $M_i(\mathbf{V})$, which is equal in value to side [cd].

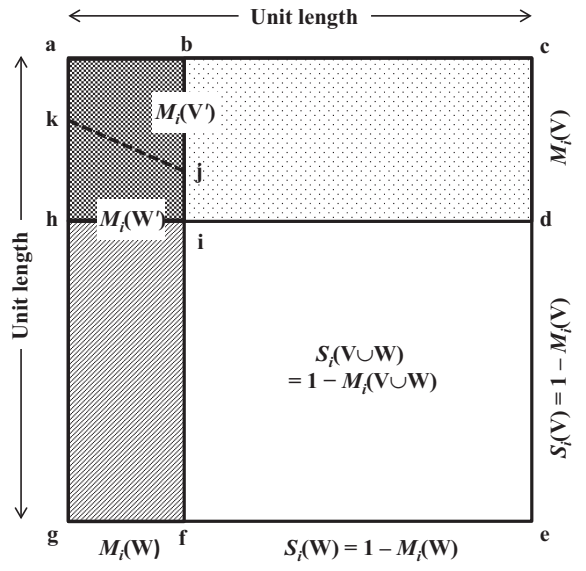

FIG. 14. Schematic representation of mortality (M_i) and survivorship (S_i) = 1 − M_i in rearing during the ith interval of inspection. The square of unit size represents the initial size of the host cohort at the beginning of the ith interval. **V** or **W** is the set of host individuals destined to die of parasitism or disease, and $M_i(\mathbf{V})$ or $M_i(\mathbf{W})$ is their latent mortality, during the interval. **V**′ or **W**′ is the set of individuals nominally tallied to have been killed by parasitism or disease during the interval, $M_i(\mathbf{V}')$ or $M_i(\mathbf{W}')$ being their nominal mortality. In particular, $M_i(\mathbf{V}') + M_i(\mathbf{W}') = M_i(\mathbf{V}\cup\mathbf{W}) = 1 - S_i(\mathbf{V}\cup\mathbf{W})$.

Likewise, there is a set (**W**) of those individuals belonging to category 2 or 3. The rectangle [abfg] represents their mortality over the interval, designated $M_i(\mathbf{W})$, equal to side [fg]. Then, there is the set of all individuals belonging to category 1, 2, or 3, i.e., the union of sets **V** and **W**, designated **V**∪**W**. The total shaded area [acdifg] represents the mortality of these individuals, designated $M_i(\mathbf{V}\cup\mathbf{W})$.

Now, assume that there is an equal chance of parasitism (or disease infection) occurring among healthy and diseased (or parasitized) individuals. Then, the mortality of those individuals in category 3 is represented by the area of intersection [abih]. The intersection is further divided into two parts: the area [abjk] is the part killed by parasitism first; and the area [kjih] is the part killed by disease first. Under these circumstances, the area [acdijk] represents the nominal mortality due to parasitism, designated $M_i(\mathbf{V}')$. Likewise, the area [kjfg] represents the nominal mortality due to disease, i.e., $M_i(\mathbf{W}')$. The remaining (unshaded) rectangle [defi] represents the survivorship of the individuals in category 4. As they survive parasitism or disease or both, the unshaded area is designated $S_i(\mathbf{V}\cup\mathbf{W})$, equal to $1 - M_i(\mathbf{V}\cup\mathbf{W})$. By the same token, the survivorship $S_i(\mathbf{V}) = 1 - M_i(\mathbf{V})$, defined by the side [de], is expected to be realized when **W** is an empty set (i.e., in an idealized space void of disease). The survivorship $S_i(\mathbf{W}) = 1 - M_i(\mathbf{W})$ is likewise defined by side [ef] when **V** is an empty set. We now show that the latent mortality $M_i(\mathbf{V})$ or $M_i(\mathbf{W})$ can be approximately estimated in terms of its nominal value $M_i(\mathbf{V}')$ or $M_i(\mathbf{W}')$.

Approximation of a latent mortality in terms of its nominal value.—Consider (in Fig. 14) the space within the unit square void of **W**′, i.e., the area [acefjk] = $M_i(\mathbf{V}') + S_i(\mathbf{V}\cup\mathbf{W})$. Then, the ratio [acdijk]/[acefjk] = $M_i(\mathbf{V}')/[S_i(\mathbf{V}\cup\mathbf{W}) + M_i(\mathbf{V}')]$ approximates the latent value $M_i(\mathbf{V})$, i.e., letting $M_i^*(\mathbf{V})$ be the approximation

$$M_i(\mathbf{V}')/[S_i(\mathbf{V}\cup\mathbf{W}) + M_i(\mathbf{V}')] \equiv M_i^*(\mathbf{V}). \qquad (1a)$$

Conversely, the corresponding survivorship in a **W**′-void space is given approximately by $S_i^*(\mathbf{V}) = 1 - M_i^*(\mathbf{V})$, i.e.,

$$S_i(\mathbf{V}\cup\mathbf{W})/[S_i(\mathbf{V}\cup\mathbf{W}) + M_i(\mathbf{V}')] \equiv S_i^*(\mathbf{V}). \qquad (1b)$$

Likewise, the area [kjideg] = $M_i(\mathbf{W}') + S_i(\mathbf{V}\cup\mathbf{W})$ is the **V**′-void space such that $M_i^*(\mathbf{W})$ and $S_i^*(\mathbf{W})$ are given by the left-hand side of the above formulae in which **V**′ is replaced by **W**′. Then, the successive multiplications of the $S_i^*(\mathbf{V})$, or $S_i^*(\mathbf{W})$, over inspection day 1 to i simulate a cohort survivorship curve to day i in an idealized disease-void, or parasitism-void, environment.

Construction of survivorship curves in rearing with respect to the estimated latent mortalities.—We use a procedure similar to the one employed in the previous *Construction of laboratory survivorship curves* with respect to parasitism and disease as a single unit. On inspection day i in rearing, we selected three batches of larvae and pupae that were consecutively field collected over the past three sampling days (i.e., $i - 3$ to $i - 1$). Among those that have survived to day $i - 1$, we counted, on day i, the number survived and the (nominal) number dead of parasitism. Then, by Formula 1b, the following ratio (realized on day i) gives an estimate of the approximate interval survivorship $S_i^*(\mathbf{V})$:

$$\frac{(\text{Counts of survivors})}{(\text{Counts of survivors} + \text{those dead of parasitism})} = S_i^*(\mathbf{V}).$$

The successive multiplications of the $S_i^*(\mathbf{V})$ from day 1 of rearing (as 100% survival) to the ith inspection day yield the cohort survivorship to day i, designated $\text{Cs}_i(\mathbf{V})$. Then, the plot of $\text{Cs}_i(\mathbf{V})$ against $i = 1, 2, 3, \ldots$ constitutes a cohort survivorship curve (to day i) in a disease-void environment, or simply Cs(**V**) curve. Likewise, the interval survivorship $S_i^*(\mathbf{W})$ is calculated by replacing "parasitism" with "disease" in the above ratio, and the Cs(**W**) curve can be constructed.

Fig. 15 shows the Cs(**V**) curve (solid circles; percentage of the initial cohort) and the Cs(**W**) curve (open triangles) for each of the three study plots for years from 1985 onward; the identification of diseases was incomplete in

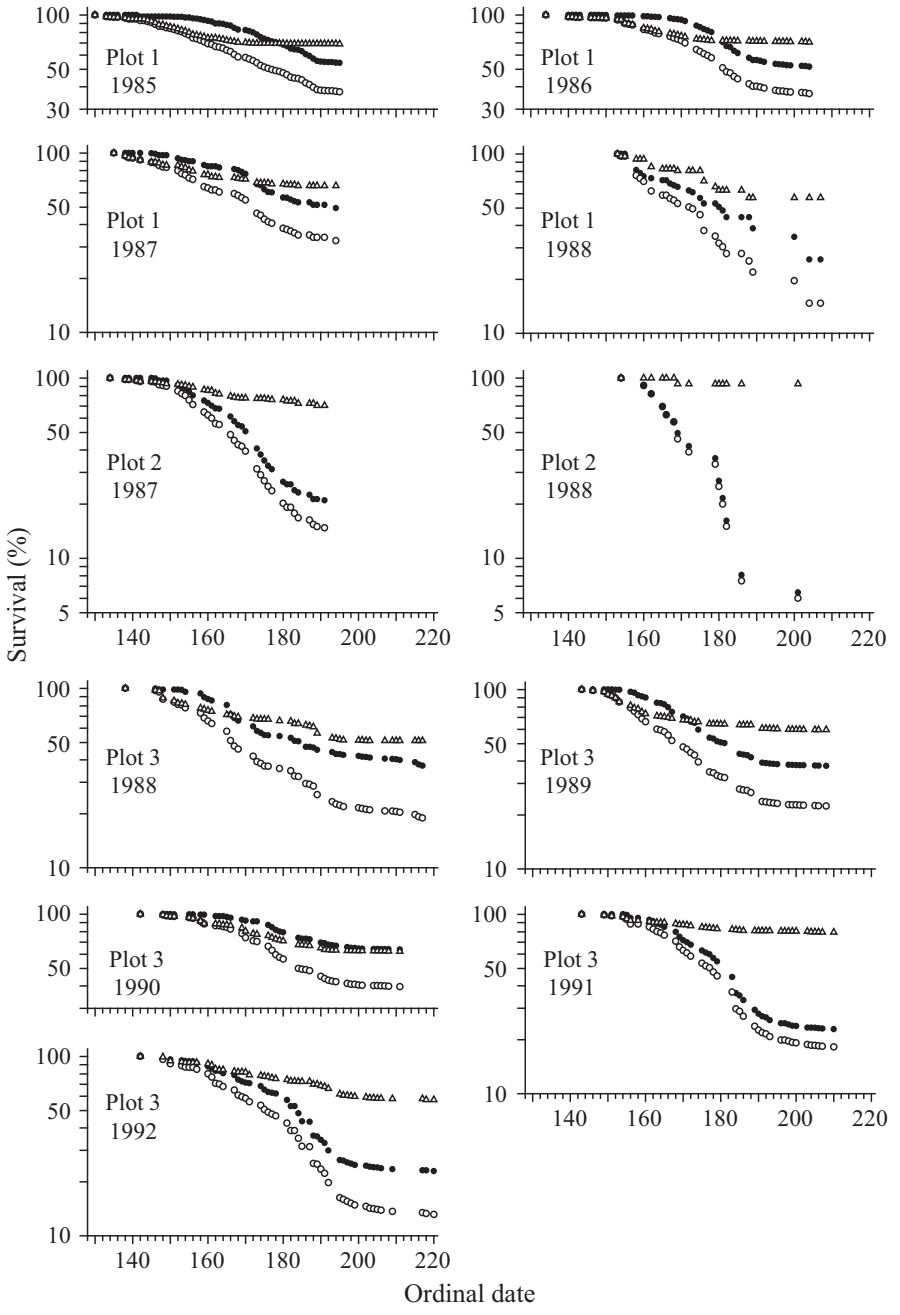

FIG. 15. Cohort survivorship (Cs) curves in rearing of the samples from the three study plots. Solid-circle series: Cs(**V**) curves (percentage of initial cohort surviving parasitism). Open-triangle series: Cs(**W**) curves (percent surviving disease). Open-circle series: Cs(**V**∪**W**) curves (percent surviving the union of parasitism and disease), copies of solid circle-series in Figs. 11, 12.

the earlier years. Also shown as a reference in each graph is the Cs(**V**∪**W**) curve (open circles), i.e., overall laboratory survivorship curve, a copy of the solid-circle curve in the corresponding plot-year (Fig. 11 or 12).

We see that, in general, the mortality due to disease tended to occur in an early part of the season. However, by the time the host larvae had reached L_5 to L_6, they tended to survive disease to become adults, even if still infected (to be discussed in Analysis of disease). Thus, a Cs(**W**) curve tended to decrease comparatively fast earlier and then to level off. In contrast, parasitism did not actually kill the hosts until about L_4 but, thenceforth, consistently killed them at all stadia until adult eclosion. Thus, a Cs(**V**) curve tended to stay more or less level earlier before it began to decrease. The curve continued to decrease until it leveled off when adults began to eclose.

We now look into details of specific mortality factors to evaluate their relative contributions to the Cs(**V**) or Cs(**W**) curve: first into specific parasitism.

Analysis of species-specific parasitism

Here, we attempt to assess the impact of parasitism attributable to a given species (or group) of parasitoids. But, first, we list every species recovered in rearing and its frequency of occurrence.

Comprehensive list of parasitoid species recovered in rearing.—All of the 65,608 budworm larvae and pupae that were put in rearing were reared through until their deaths or eclosion as adults, and the relative frequency of a given parasitoid species recovered was recorded (Tables 2 and 3). The species are grouped in accordance with the host stadia at which parasitization (oviposition on the hosts) occurred. There are three major groups. Group 1 comprises those species that parasitize the hosts during their pre-emergence period as L_1 or L_2, but actually kill them only at their postdiapause stadia usually after L_4. The species are listed in order of the average frequency of recovery (percentage of the total number of all species recovered in each plot-year). All parasitoids in this group were hymenopterans: nine braconid and four ichneumonid species. *Apanteles fumiferanae* and *Glypta fumiferanae* were consistently the major species (in frequency of recovery) in this group. Group 2 comprises those parasitizing postemergence larvae. Group 2a includes nine hymenopterans (three braconid, five ichneumonid, and one chalcidoid species) and Group 2b, nine dipterans (seven tachinid and two sarcophagid species). Among the hymenopterans, *Meteorus trachynotus* was consistently the dominant species. *Tranosema tenuifemur* was also high in its average frequency. However, it had been a minor species for most plot-years until the host populations declined to an extremely low level: 1988 and 1989 in Plot 1 and 1993 in Plot 3. Among the dipterans, *Lypha fumipennis* and *Smidtia fumiferanae* (*Winthemia fumiferanae*) were consistently the two major species.

Group 3 comprises those parasitizing the hosts invariably at their pupal stage. All were hymenopterans (two chalcidoid and three ichneumonid species). Most of the parasitoid individuals, especially those of the chalcidoids, issued from the hosts after a majority of nonparasitized pupae had eclosed as adults.

Tables 2 and 3 provide a comprehensive guide of the parasitoid complex and a crude idea of species ranks (in frequency of recovery) among the constituents; it even detected an extremely rare species. However, Tables 2 and 3 do not provide quantitative information about the actual contribution by each species to the host cohort mortality attributable to parasitism. This is because the frequency of a species in the table depends on the timing of collection and the total number of host individuals reared through. In the following, we investigate the actual contributions by the aforementioned major parasitoid species (singly or collectively).

Evaluation of contributions by major parasitoids to the host cohort mortality.—Note first that the difference between $Cs_1(\mathbf{V})$ and $Cs_i(\mathbf{V})$ of each Cs(**V**) curve in Fig. 15 is a percent reduction in the cohort size from day 1 to day i due to total (latent) parasitism in the disease-void space. We attempt to partition the reduction into parts contributed by species-specific (or group-specific) parasitism. We do this in terms of the nominal values of the specific parasitism because the evaluation of a latent value at the specific level is not as practically feasible as at the higher levels. Actual procedures are the following.

First, recall Formula 1a, which provides an approximate estimate of the latent parasitism (i.e., mortality due to parasitism in a disease-void space): $M_i^*(\mathbf{V}) = M_i(\mathbf{V}')/[S_i(\mathbf{V} \cup \mathbf{W}) + M_i(\mathbf{V}')]$, in which $M_i(\mathbf{V}')$ is the nominal value of the total interval mortality due to parasitism. Now, let $\mathbf{V}'_a$, $\mathbf{V}'_b$,..., $\mathbf{V}'_p$ be the sets of hosts nominally killed by parasitoid species a, b,..., p (or q as a generic designation), and let $M_i(\mathbf{V}'_q)$ be the nominal mortality due to q realized over the ith (inspection) interval. Then, $M_i(\mathbf{V}'_a) + M_i(\mathbf{V}'_b) + \ldots + M_i(\mathbf{V}'_p) = M_i(\mathbf{V}')$. It follows from Formula 1a that

$$M_i^*(\mathbf{V}) = [M_i(\mathbf{V}'_a) + M_i(\mathbf{V}'_b) + \ldots + M_i(\mathbf{V}'_p)]/[S_i(\mathbf{V} \cup \mathbf{W}) + M_i(\mathbf{V}')].$$

Thus, the individual quotient $M_i(\mathbf{V}'_q)/[S_i(\mathbf{V} \cup \mathbf{W}) + M_i(\mathbf{V}')]$ gives the nominal contribution of species q (for: a, b,..., p) to the latent mortality $M_i^*(\mathbf{V})$.

Actual calculation.—The calculation, using observations in rearing, is much the same as calculating the cohort survivorship $Cs_i(\mathrm{V})$ in Fig. 15. We pooled the batches of hosts collected on days $i - 3$ to $i - 1$. Then, among the surviving hosts by day $i - 1$, we counted (HK) the nominal number of hosts killed by parasitoid q by day i; and (HS) the hosts surviving on day i plus the total number of host killed by all parasitoid species by day i. The counts of HK estimate $M_i(\mathbf{V}'_q)$ and those of HS estimate $[S_i(\mathbf{V} \cup \mathbf{W}) + M_i(\mathbf{V}')]$. Thus, the ratio HK/HS estimates the contribution by parasitoid q over the ith interval to the total parasitism $M_i^*(\mathbf{V})$.

Furthermore, we standardize the contribution by q as the proportion (percent killed by q) of the initial size of the host cohort. Referring to a Cs(**V**) curve in Fig. 15, recall that the initial size $Cs_1(\mathbf{V})$ is set equal to 100, which, by the beginning of the ith interval (from day $i - 1$ to i), has been reduced to $Cs_{i-1}(\mathbf{V})$. Hence, the product (HK/HS) $\times$ $Cs_{i-1}(\mathbf{V})$ gives the ith interval mortality due to parasitoid q in terms of the proportion (%) of the initial host cohort.

Results and interpretations.—Fig. 16 shows results from Plot 1, 1986, as an example. The top six graphs are the sequential occurrences of three single species (on the

TABLE 2. Frequency occurrence of parasitoid species in rearing (percentage of total number of individual parasitoids recovered) from Plot 1 in the present study in New Brunswick, Canada, with parasitoids grouped by host stadium at which parasitization takes place.

	Plot 1									
	Generation year									
Parasitoid group	1981	1982	1983	1984	1985	1986	1987	1988	1989	Mean (sum)
Group 1: pre-emergence larvae										
Apanteles fumiferanae Viereck (Br)	52.40	68.39	60.33	49.48	28.26	11.87	24.47	15.94	9.09	35.58
Glypta fumiferanae (Viereck) (Ich)	13.90	11.35	18.43	23.26	28.63	19.42	15.76	1.45		14.69
Apanteles milleri Mason (Br)				0.09						0.01
Apanteles morrisi Mason (Br)				0.17		0.14			9.09	1.05
Unidentifiable Apanteles (Br)						0.65	2.59	4.35		0.84
Charmon extensor (L.) (Br)		0.20	0.06	0.09						0.04
Chelonus sp. (Br)										
Diadegma sp. (Ich)										
Orgilus sp. (Br)										
Macrocentrus linearis (Nees) (Br)				0.09						0.01
Bassus binominata (Muesebeck) (Br)		0.20				0.07	0.24			0.06
Bassus dimidiator (Nees) (Br)†						+				
Campoplex sp. (Ich)†						+				
Pristomerus sp. (Ich)†						+				
Group 1 total	66.31	80.14	78.82	73.18	56.90	32.16	43.26	21.74	18.18	52.25
Group 2: postemergence larvae										
(a) Hymenoptera										
Meteorus trachynotus Viereck (Br)	2.14	7.40	9.87	2.78	1.92	7.12	12.24	7.25		5.63
Tranosema tenuifemer (Walley) (Ich)			0.40	0.52			0.24	28.99	45.45	8.40
Dolichogenidea absona (Muesebeck) (Br)				0.09				5.80	27.27	3.68
Apanteles petrovae Walley (Br)					0.06		0.24			0.03
Stictopisthus sp. (Ich)‡					0.06		0.24			0.03
Enytus montanus (Ashmead) (Ich)								1.45		0.16
Scambus sp. (Ich)‡										
Tranosema rostrale (Brischke) (Ich)			0.06				0.24			0.03
Elachertus cacoeciae (Howard) (Ch)‡										
Subtotal a	2.14	7.40	10.33	3.39	2.04	7.12	13.17	43.48	72.73	17.97
(b) Diptera										
Lypha fumipennis Brooks (Tach)		9.42	0.52	0.26	0.25	1.08	8.47	24.64		4.96
Smidtia fumiferanae (Tothill) (Tach)	4.81		7.29	13.19	20.53	13.02	6.35			7.24
Phryxe pecosensis (Townsend) (Tach)		0.41	0.52	0.17	1.92	1.94	0.71			0.63
Eumea caesar (Aldrich) (Tach)		0.30	0.17	0.09	0.19	0.36				0.12
Actia interrupta Curran (Tach)			0.17			0.14				0.04

TABLE 2. (Continued)

	Plot 1									
	Generation year									
Parasitoid group	1981	1982	1983	1984	1985	1986	1987	1988	1989	Mean (sum)
Agria affinis (Fallén) (Sarc)				0.61		0.14				0.08
Madremyia saundersii (Williston) (Tach)			0.06	0.17						0.03
Pseudoperichaeta erecta (Coquillett) (Tach)		0.10								0.01
Sarcophaga aldrichi Parker (Sarc)				0.09						0.01
Unidentified Diptera		0.71	0.29	2.34	1.18	4.53	2.59			1.29
Subtotal b	4.81	10.94	9.01	16.93	24.06	21.22	18.12	24.64		14.41
Group 2 total	6.95	18.34	19.35	20.32	26.10	28.35	31.29	68.12	72.73	32.39
Group 3: Pupae										
Mesopolobus verditer (Norton) (Ch)‡			0.17	0.78	2.91	10.43	1.18			1.72
Mesopolobus tortricis (Brues) (Ch)	3.74	0.91	0.63	1.04	5.19	15.47	10.59			4.18
Dirophanes hariolus (Cresson) (Ich)			0.06	3.13	2.35	1.44				0.77
Apechthis ontario (Cresson) (Ich)	0.53	0.20	0.63	0.87	4.82	3.24	5.41			1.75
Itoplectis conquisitor (Say) (Ich)		0.20	0.17	0.17	0.87	2.81	6.59			1.20
Unidentifiable Ichneumonidae					0.68	5.97	1.65			0.92
Group 3 total	4.28	1.32	1.66	5.99	16.82	39.35	25.41			10.54
Unidentifiable: host stadium uncertain										
Chalcidoidae			0.06	0.26	0.19	0.07	0.24	1.45		0.25
Ichneumonidae			0.06	0.17		0.07				0.03
Braconidae			0.06	0.09						0.02
Totally unidentifiable	22.46	0.20	0.06					8.70	9.09	4.50
Number of parasitoids recovered	145	1012	1742	1152	1617	1390	425	69	11	(7563)
Number of budworm reared§	1547	4700	7809	7683	10233	6309	2109	165	23	(40578)
Parasitoids recovered (%)	9.4	21.5	22.3	15.0	15.8	22.0	20.2	41.8	47.8	18.6

Notes: In Group column, (Br) Braconidae; (Ch) Chalcidoidae; (Ich) Ichneumonidae; (Tach) Tachinidae; (Sarc) Sarcophagidae. In data columns, blank (empty) cells indicate a zero occurrence; +, present but unquantified.

†Recovered only from pre-emergence samples.

‡Possibly both primary and secondary parasitoid (Eveleigh et al. 2007).

§Excluding pre-emergence samples.

TABLE 3. Frequency occurrence of parasitoid species in rearing (percentage of total number of individual parasitoids recovered) from Plots 2 and 3 in the present study in New Brunswick, Canada, with parasitoids grouped by host stadium at which parasitization takes place, as well as mean sum from all three plots.

	Plot 2					Plot 3							All plots
	Generation year					Generation year							
Parasitoid group	1986	1987	1988	1989	Mean (sum)	1988	1989	1990	1991	1992	1993	Mean (sum)	Mean (sum)
Group 1: pre-emergence larvae													
Apanteles fumiferanae	29.86	33.15	34.38		24.34	35.33	30.88	16.93	13.88	11.88	7.69	19.43	28.12
Glypta fumiferanae	9.90	9.03		12.50	7.86	1.83	3.60	5.82	5.28	6.28		3.80	9.81
Apanteles milleri				12.50	3.13								0.66
Apanteles morrisi	0.07				0.02					0.27		0.05	0.51
Unidentifiable Apanteles	1.34	3.81	6.25		2.85	3.06	1.65	1.23	1.45	2.94	3.85	2.36	1.74
Charmon extensor									0.09			0.01	0.02
Chelonus sp.							0.15					0.03	0.01
Diadegma sp.							0.15		0.09			0.04	0.01
Orgilus sp.									0.09			0.01	0.0+
Macrocentrus linearis													0.0+
Bassus binominata	0.22				0.06		0.15		0.09	0.13	3.85	0.70	0.26
Bassus dimidiator†	+												
Campoplex sp.†	+												
Pristomerus sp.†	+												
Group 1 total	41.40	45.98	40.63	25.00	38.26	40.22	36.58	23.99	20.96	21.49	15.39	25.81	41.15
Group 2: postemergence larvae													
(a) Hymenoptera													
Meteorus trachynotus	16.38	21.58	3.13	12.50	13.40	3.55	5.85	1.59	1.11	3.34		2.57	6.30
Tranosema tenuifemer		0.71	12.50		3.30	2.93	2.10	0.53			76.92	13.75	9.02
Dolichogenidea absona							0.15					0.03	1.75
Apanteles petrovae	0.07		6.25	12.50	4.71								1.01
Stictopisthus sp.‡	0.07	0.85	3.13		1.01	0.12	0.30			0.67		0.18	0.29
Enytus montanus		0.14	3.13		0.82	0.24						0.04	0.26
Scambus sp.‡						0.24		0.53		0.13		0.15	0.05
Tranosema rostrale		0.14			0.04					0.27		0.05	0.04
Elachertus cacoeciae‡									0.09	0.13		0.04	0.01
Subtotal a	16.53	23.41	28.13	25.00	23.26	7.09	8.40	2.65	1.19	4.54	76.92	17.43	18.72
(b) Diptera													
Lypha fumipennis	0.60	21.16	21.88	25.00	17.16	1.34	3.30	1.59	0.43	4.27	3.85	2.46	6.74
Smidtia fumiferanae	5.14	3.10			2.06	1.47	5.55	3.17	3.75	4.94		3.15	4.86
Phryxe pecosensis	0.37				0.09	2.93	1.20	3.35	8.86	10.55		4.48	1.73
Eumea caesar	0.15				0.04	0.86	0.45	1.41	7.67	12.02		3.73	1.25
Actia interrupta		0.14	3.13		0.82	0.12	1.05	0.53	0.17	0.13	3.85	0.98	0.50

TABLE 3. (Continued)

Parasitoid group	Plot 2					Plot 3							All plots
	Generation year					Generation year							
	1986	1987	1988	1989	Mean (sum)	1988	1989	1990	1991	1992	1993	Mean (sum)	Mean (sum)
Agria affinis								4.06	0.94	0.13		0.85	0.31
Madremyia saundersii	0.07				0.02			0.18	0.17			0.06	0.03
Pseudoperichaeta erecta													0.01
Sarcophaga aldrichi													0.0+
Unidentified Diptera	2.16	1.55			0.93	2.08	5.10	26.63	3.83	10.55		8.03	3.34
Subtotal b	8.49	25.95	25.00	25.00	21.11	8.80	16.64	40.92	25.81	42.59	7.69	23.74	18.76
Group 2 total	25.02	49.37	53.13	50.00	44.37	15.89	25.04	43.56	27.00	47.13	84.61	41.17	37.48
Group 3: Pupae													
Mesopolobus verditer‡	9.83	0.56	3.13		3.38	19.19	18.59	11.99	15.59	1.07		11.07	5.02
Mesopolobus tortricis	14.97	3.10			4.52	4.16	5.40	1.94	3.49	3.87		3.14	3.92
Dirophanes hariolus	0.15	0.00			0.04	3.91		3.00	0.43			1.22	0.76
Apechthis ontario	2.61	0.71			0.83	10.15	6.30	8.99	15.16	6.01		7.77	3.45
Itoplectis conquisitor	3.43	0.14			0.89	0.24	0.15	0.35	1.70	4.01		1.08	1.10
Unidentifiable Ichneumonidae	2.61	0.14		12.50	3.81	5.99	7.80	3.53	10.82	8.28		6.07	3.16
Group 3 total	33.58	4.65	3.13	12.50	13.47	43.64	38.23	29.81	47.19	23.23		30.35	17.41
Group 4: host stadium uncertain													
Pimpla pedalis Cresson										0.0+			0.0+
Bathythrix migripalpis Townes								0.0+					0.0+
Euderus sp.‡										0.0+			0.0+
Sympiesis sp.											0.04	0.01	0.0+
Unidentifiable: host stadium uncertain													
Chalcidoidae						0.12	0.15	0.35		0.27		0.15	0.17
Ichneumonidae										2.14		0.36	0.13
Braconidae													0.01
Totally unidentifiable			3.13	12.50	3.91	0.12	0.15	2.29	4.94	5.74		2.21	3.65
Number of parasitoids recovered	1343	709	32	8	(2092)	818	667	567	1174	749	26	(4001)	(13656)
Number of budworm reared§	4255	2023	61	26	(6365)	4924	3673	3455	3484	3020	109	(18665)	(65608)
Parasitoids recovered (%)	31.6	35.0	52.5	30.8	32.9	16.6	18.2	16.4	33.7	24.8	23.9	21.4	20.8

Notes: In data columns, blank (empty) cells indicate a zero occurrence; +, present but unquantified; 0.0+, positive but <0.01.
†Recovered only from pre-emergence samples.
‡Possibly both primary and secondary parasitoid (Eveleigh et al. 2007).
§Excluding pre-emergence samples.

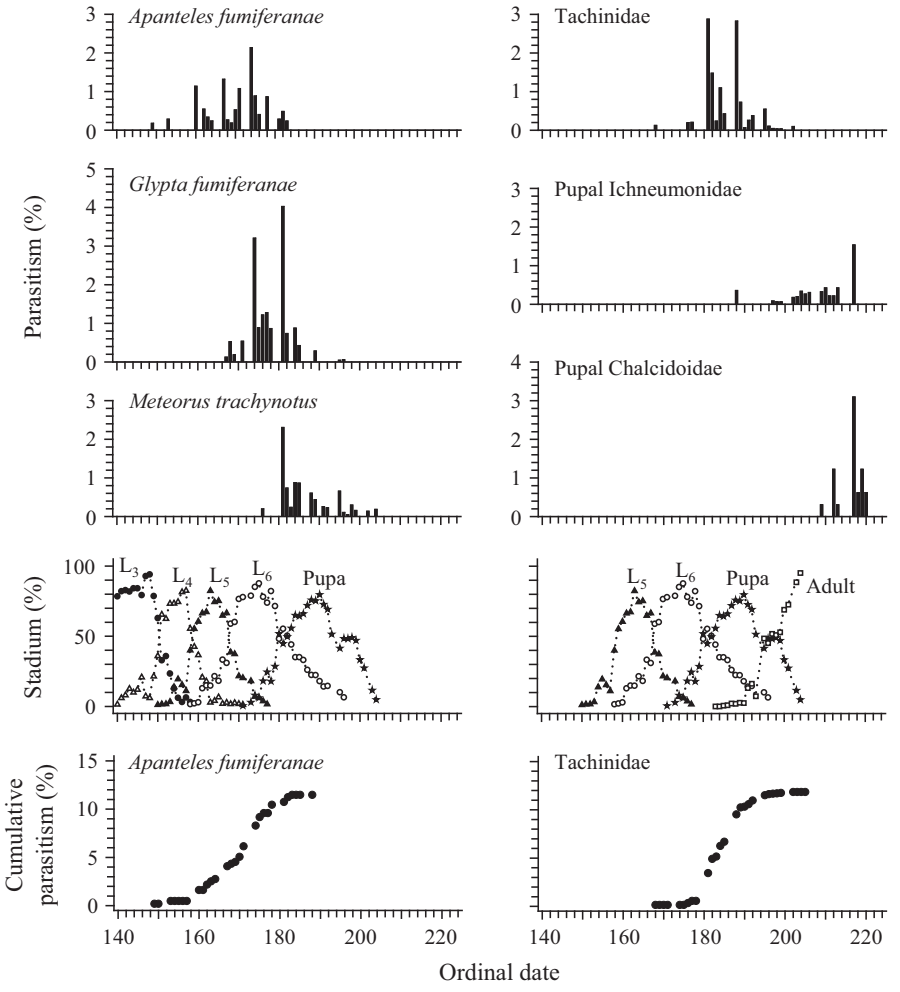

FIG. 16. Examples (from Plot 1, 1986) of the daily occurrence of nominal parasitism (percentage of initial size of host cohort) by selected (single or family of) species in rearing. Additional graphs at bottom two rows: Frequency (%) distribution of host developmental stadia and the cumulative sums of the parasitism by *Apanteles fumiferanae* and the Tachinidae in the top graphs.

left-hand side) and three families (on the right). Each bar represents the daily occurrence of the hosts killed (i.e., when the parasitoids issued from them) in terms of the proportion (%) of the initial cohort size. (Note: when an inspection in rearing was skipped, the bar tended to spike on the day the inspection was resumed. This is because the number killed on the days of no inspection was carried over to the day the inspection was resumed.)

The bar graphs depict the timing of kill by each species (or family) of parasitoids in relation to the chronological changes in the developmental stadia of all living host individuals. The earliest parasitoid species was *A. fumiferanae*, which had parasitized pre-emergence host larvae (Group 1 in Tables 2 and 3) and issued from those at the postdiapause L_4 and L_5 instars. (Note that a parasitized host tended to be retarded in its development, and it was actually killed by the parasitoid when a majority of host individuals was already at L_6.) *G. fumiferanae* (Group 1) issued mostly from the hosts at L_5 and a few at L_6, although the majority of healthy hosts were L_6 or pupae. *M. trachynotus* (Group 2) parasitized postdiapause larvae at L_5 and early L_6 and issued from them at late L_6; those parasitized hosts were so retarded as to never attain the full size of the normal L_6. The tachinids had a similar timing as *M. trachynotus*, but certain of them (e.g., *Smidtia fumiferanae*) issued from the hosts at the pupal stadium. Many individuals of the pupal hymenopterans (Group 3) tended to stay within the host bodies and issued long after all non-parasitized pupae had eclosed as adults. Thus, the percent occurrences of those kills (later than ordinal day 220) were distributed proportionately among the earlier occurrences.

The two bottom graphs (Fig. 16) for *A. fumiferanae* and the Tachinidae show examples of the cumulative sums of their corresponding bar graphs at the top. The plateau of a cumulative sum estimates the total (seasonal) realization of the cohort mortality due

(nominally) to the parasitoid species concerned in the conceptualized absence of disease.

Table 4 gives the numerical results (the plateau values) for all parasitoid species and families in the three study plots, calculated to the end of the season, i.e., to the end of the Cs(**V**) curve for corresponding plot-year in Fig. 15. The nominal values for individual species or families are divided into the three major groups (G1, G2, and G3 in accordance with the timing of parasitization as in Tables 2 and 3). These individual values sum up to total latent parasitism at the bottom row of each plot. Total latent parasitism in Plot (1 + 2) and Plot 3 in Table 4 are reproduced in Table 5 to compare with the corresponding total postdiapause mortality (equal to 1 – postdiapause survival in Fig. 8b in linear scale). Then, the contribution of (the total latent) parasitism to the total postdiapause mortality is evaluated as the proportion (latent parasitism)/(postdiapause mortality) (reported as a percentage). We see that the total latent parasitism accounted for on the average 85% of the annual total postdiapause mortality. (Note: the latent parasitism for 1982 is omitted as it was anomalous in rearing [cf. Fig. 11]. Also omitted is the result for 1983, which also was somehow overestimated.]

Furthermore, we look into the annual variation in total latent parasitism (Table 4) in Plot 1 as an example. The variation is graphically partitioned into its three group-specific (nominal) totals (Fig. 17), omitting the aforementioned anomalous results for 1982. We see that Group 1 had contributed most in determining the total parasitism until 1984. Thenceforth, it leveled off, and the later Groups 2 and 3 in combination became significant contributors. Evidently, the overall pattern of annual variation in parasitism cannot be attributable to any single major group but to the totality of all major groups, a topic to be discussed in *Discussion of part II* after the following analysis of disease.

Analysis of disease

A similar analysis was applied to the disease complex. Table 6 gives the results from 1985 onward as a counterpart of Table 4 for parasitism; Fig. 18 gives the example from Plot 1, 1986, as a counterpart of Fig. 16. The quantitative investigation of disease is incomplete before 1985. However, the yearly prevalence of diseases in 1982–1984 was low in Plot 1: nominally, on average <10% for microsporidia, and <2% and 1% for fungi and viruses, respectively.

The most prevalent pathogen throughout the study was *Nosema fumiferanae* (Thomson) (Microsporidia: Nosematidae); a few other microsporidians were present but at a much lower frequency (Eveleigh et al. 2012). The host mortality attributed to microsporidians occurred mainly before ordinal day 180 (cf. Fig. 18). Thus, an early decline in many host survivorship curves (the triangle series in Fig. 15) is attributable to the microsporidians.

The entomopathogenic fungi, collectively listed as "fungus" in Table 6, are those belonging to the five genera of Hyphomycetes (*Beauvaria*, *Hirsutella*, *Paecilomyces*, *Verticillium*, and *Tolypocladium*), and two Zygomycetes (*Entomophaga* and *Erynia*) (cf. Eveleigh et al. 2007: Table 3). Albeit regularly found, their collective frequency of occurrence in most plot-years in the present study (except for Plot 3, 1988) was not much more than 5% (of the initial host cohort) by the end of the season. It should be noted, however, that there were sporadic and temporary occurrences of high mortality due to undetermined causes during the Green River study (Royama 1984: Fig. 5c). These were likely fungal epizootics similar to one observed in Ontario caused by *Entomophaga aulicae* that infected as much as 70% of the budworm samples (Perry and Régnière 1986). No such fungal epizootics occurred in the present study.

The prevalence of viruses was even less. Those that regularly occurred in our study are the two baculoviruses: nucleopolyhedrovirus (CfMNPV) and granulovirus (ChfuGV). The examinations of dead and live hosts at all stadia of development (collected separately from the regular sampling, including cadavers in drop trays) revealed only a low level of infection by these viruses throughout the study; cytoplasmic polyhedrosis and entomopox viruses were found but extremely rarely (Lucarotti et al. 2004).

On several occasions, especially in Plot 3, two or more different types of pathogens were found in a single host cadaver, providing direct evidence for multiple infections. Not listed in Table 6 are the pathogenic bacteria, *Bacillus thuringiensis* and *Bacillus cereus*. These were isolated only occasionally from budworm cadavers found on sample branches or in drop trays (Strongman et al. 1997).

Miscellaneous notes on pathogens.—It is known that infection by microsporidians tends to reduce the fecundity of the host females and the survival of the overwintering larvae (Bauer and Nordin 1989, Régnière and Nealis 2008). However, the pathogen is generally known to be low in virulence (van Frankenhuyzen et al. 2007, Eveleigh et al. 2012). Infection by the pathogen is known to occur in two different ways: female-to-offspring (transovarial or vertical) transmission and the ingestion of spores in the external environment by feeding (peroral or horizontal transmission). Because of its low virulence, the pathogen often allows an infected larva to become an adult that lays infected eggs, permitting a vertical transmission at a rate of 50–100% (van Frankenhuyzen et al. 2007). In horizontal transmission, the spores must be ingested through feeding on food that has been contaminated by other infected individuals or by decomposed cadavers (Campbell et al. 2007). Now, opportunities for ingesting spores by the prediapause (L_1) larvae would most likely be limited, as they only imbibe water and nibble on the cuticular waxes of the host plant needles before settling in hibernacula

TABLE 4. Species- (or group-) specific nominal parasitism (percentage of the initial host cohort), where nominal values add up to the total latent parasitism.

	Generation year												
Parasitoid group	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990	1991	1992	Mean
Plot 1													
G1													
Apanteles fumiferanae	34.0	56.0	42.7	23.0	20.5	11.4	17.9	28.2					29.21
Glypta fumiferanae	6.9	6.1	4.3	6.8	11.7	15.3	7.8	1.8					7.59
Other	0.0	0.0	0.0	0.2	0.0	0.3	0.0	0.0					0.06
G2													
Meteorus trachynotus	0.3	1.8	6.5	1.6	1.0	8.4	9.7	13.7					5.38
Other Braconidae	0.0	0.0	0.0	0.0	0.4	0.0	0.0	11.3					1.46
Ichneumonidae	0.0	0.0	0.0	0.0	0.0	0.0	0.0	14.1					1.76
Tachinidae	0.2	6.1	5.4	5.6	10.9	11.8	13.7	13.5					8.41
G3													
Ichneumonidae	1.2	0.3	0.0	2.4	4.1	5.1	9.0	0.0					2.76
Chalcidoidae	0.0	0.0	1.8	0.8	1.8	7.4	9.8	0.0					2.70
Unidentifiable	7.5	0.0	0.5	0.1	0.4	0.2	0.0	14.1					2.85
Total latent parasitism	50.1	70.3†	61.2	40.5	50.8	59.9	67.9	96.6					62.16
Plot 2													
G1													
Apanteles fumiferanae						42.7	43.2	63.2					49.70
Glypta fumiferanae						6.7	5.4	0.0					4.03
Other						0.0	0.0	0.0					0.00
G2													
Meteorus trachynotus						13.6	17.8	2.7					11.37
Other Braconidae						0.3	0.0	5.7					1.99
Ichneumonidae						0.3	1.7	11.5					4.47
Tachinidae						4.3	11.4	13.5					9.73
G3													
Ichneumonidae						3.4	1.5	0.0					1.63
Chalcidoidae						6.1	2.0	0.6					2.90
Unidentifiable						0.0	0.0	0.0					0.00
Total latent parasitism						77.3†	83.0	97.2					85.83
Plot (1 + 2)													
Total latent parasitism	50.1	†	61.2	40.5	50.8	59.9	75.5	96.9					64.24
Plot 3													
G1													
Apanteles fumiferanae								49.0	46.1	19.5	41.5	35.5	38.32
Glypta fumiferanae								0.4	2.8	3.3	6.3	7.9	4.14
Other								0.0	1.4	0.0	0.0	0.1	0.30
G2													
Meteorus trachynotus								2.1	2.6	0.3	0.4	2.1	1.50
Braconidae								0.0	0.0	0.0	0.0	0.2	0.04
Ichneumonidae								0.2	0.3	0.0	0.0	0.8	0.26
Tachinidae								7.8	9.7	13.6	28.7	30.5	18.06
G3													
Ichneumonidae								8.9	3.0	3.5	4.5	4.1	4.80
Chalcidoidae								5.6	4.0	2.8	4.1	1.4	3.57
Unidentifiable								0.0	0.3	2.3	0.8	3.6	1.41
Total latent parasitism								74.0	70.2	45.3	86.3	86.2	72.40

Notes: G1, G2, and G3 are the three major groups of parasitoid species in Tables 2 and 3 in order of their timing of parasitization. Total latent parasitism in Plot (1 + 2) for 1987 and 1988 generations are the averages of Plot 1 and Plot 2.

†Overestimation in rearing in Plot 1 in 1982, and in Plot 2 in 1986 (cf. those years in Figs. 11 and 12, respectively).

TABLE 5. Contribution of the total latent parasitism in Table 4 to the total postdiapause mortality (1 – survivorship in Fig. 8b in linear scale) in Plot (1 + 2) and Plot 3 in New Brunswick, Canada.

Generation year	Total postdiapause mortality (%)	Total latent parasitism (%)	Contribution of parasitism (%)
Plot (1 + 2)			
1981	53.0	50.1	94.5
1982	42.5	70.3†	
1983	59.5	61.2†	
1984	53.8	40.5	75.3
1985	67.5	50.8	75.3
1986	75.5	68.6	90.9
1987	95.1	75.5	79.4
1988	97.6	96.9	99.3
Plot 3			
1988	78	74	94.9
1989	83.9	70.2	83.7
1990	78.4	45.3	57.8
1991	93	86.3	92.8
1992	92.4	86.2	93.3
Mean			85.2
SE			3.74

†Overestimation to be omitted in calculating the contribution.

(Retnakaran et al. 1999). Furthermore, the *Nosema* spores present during winter in the field would not be viable by spring (Thomson 1958) when main feeding by budworm larvae begins. For these reasons, most of the postdiapause budworm that died of the diseases in a given year (especially those that died before about day 175 in Fig. 18) must have been those that had already been infected through transovarial transmission from the previous generation.

The identification of fungi and viruses often required time-consuming and expensive procedures. Certain fungi, e.g., *Hirsutella*, required culturing; granulovirus needed molecular probing to confirm its presence; nucleopolyhedrovirus was identifiable under a light microscope, but this is not as accurate as molecular probing. These identification impediments made a thorough quantitative analysis of pathogens in large samples impractical in the present study. However, except for the microsporidians and the sporadic occurrences of apparent epizootics, the impact of pathogens has certainly been insignificant on budworm populations in New Brunswick.

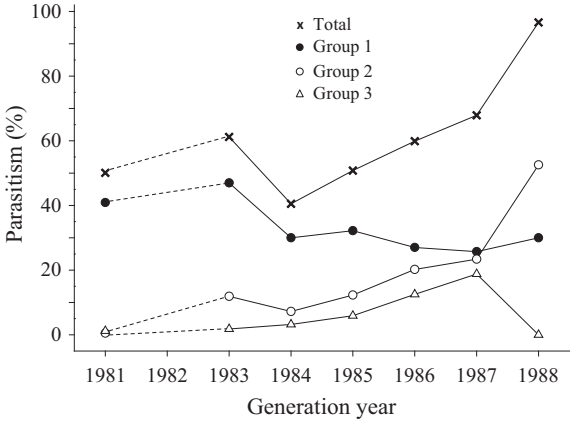

FIG. 17. Example of the annual variation in total latent parasitism in rearing as observed with samples from Plot 1 (Table 4), partitioned into variations in nominal parasitism by three major groups. The 1982 results are omitted as anomalous (cf. Fig. 11).

Discussion of part II

To conclude *Part II*, we discuss two issues arising from the foregoing analyses. First, none of the natural enemies singly, or even collectively, were predominant in determining the postdiapause budworm survivorship, and hence the principal outbreak cycle. The second issue concerns the ranking of mortality factors in determining the survivorship.

As for the lack of predominance among natural enemies, a likely reason is the movement (or allotment of search time) by each parasitoid or predator species among alternative hosts, seeking its own more profitable host species as budworm abundance changed relative to others. Thus, for example, the Group 1 parasitoids might have found budworm to be a profitable host when it was superabundant but, when less abundant, might have looked for alternative hosts with an increasing relative profitability; the concept of "profitability" is quantitatively defined in Royama (1970: 644). Conversely, the parasitoids of Group 2 or 3 might have found a low budworm density with a reduced action of the competitive species from Group 1 to have become profitable. Consequences of such movements manifest in the observed changes in food-web structures in accordance with changes in budworm population from high to low levels: cf. Fig. 1B, C in Eveleigh et al. (2007). At least 31 species were confirmed to have parasitized spruce budworm at an epidemic level, of which 14 species were confirmed to be present also at an endemic level, together with an additional five species that were not found at the epidemic level (Appendix S6). Furthermore, five lepidopteran species were found to be alternative hosts to many parasitoids of spruce budworm (Eveleigh et al. 2007: Fig. 1 and Table 3). These include two Gelechiids, *Coleotechnites atrupictella* and *Coleotechnites piceaella*, and three Tortricids, *Acleris variana*, *Choristoneura rosaceana*, and *Epinotia radicana* (Appendix S7).

It would be hard to comprehend the complexity of the budworm system in terms of the classical concept of a density-dependent interaction of single prey vs. single predator species. Understanding this requires a holistic approach to the system of natural-enemy complex vs. host complex, a topic to be discussed more fully in *Part III*.

TABLE 6. Group-specific disease rates as percentage of initial cohort with cases of multiple infections.

Group	Generation year								Mean
	1985	1986	1987	1988	1989	1990	1991	1992	
Plot 1									
Microsporidia	37.1	29.5	36.1						34.22
Fungus	0.2	1.1	5.2						2.15
Virus	0.1	1.4	2.1						1.21
Microsporidia + fungus	0.0	0.0	0.0						0.00
Fungus + virus	0.0	0.0	0.0						0.00
Microsporidia + virus	0.0	0.2	0.0						0.07
Microsporidia + fungus + virus	0.0	0.0	0.0						0.00
Total latent disease	37.3	32.2	43.5						37.66
Plot 2									
Microsporidia		22.14	27.1						24.59
Fungus		4.51	5.8						5.13
Virus		0.47	0.4						0.43
Microsporidia + fungus		0	0.0						0.00
Fungus + virus		0	0.0						0.00
Microsporidia + virus		0	0.0						0.00
Microsporidia + fungus + virus		0	0.0						0.00
Total latent disease		27.15	33.2						30.19
Plot 3									
Microsporidia				38.4	38.6	28.7	17.4	25.2	29.66
Fungus				9.4	4.0	5.7	2.8	3.5	5.10
Virus				0.0	0.0	3.0	1.2	5.7	1.98
Microsporidia + fungus				3.9	1.7	1.9	0.2	3.5	2.23
Fungus + virus				0.0	0.0	0.0	0.0	1.6	0.31
Microsporidia + virus				0.0	0.0	0.1	1.1	4.1	1.06
Microsporidia + fungus + virus				0.0	0.0	0.0	0.0	0.3	0.05
Total latent disease				51.7	44.3	39.5	22.8	43.8	40.40

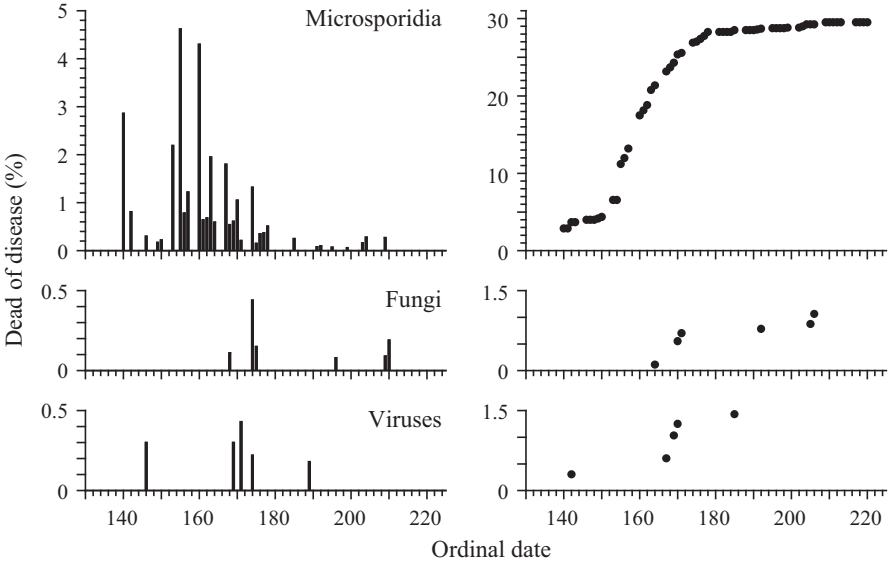

FIG. 18. Examples (from Plot 1, 1986) of the occurrence of diseases (percentage of initial size of host cohort) in rearing nominally attributed to the microsporidia, entomopathogenic fungi, and viruses. Graphs on the left: daily occurrences. Those on the right: cumulative sums. For changes in host developmental stadia, see Fig. 16.

As for the ranks among mortality factors, a certain theoretical consideration is in order. Consider that the set of parasitoids consists of species a, b,..., p, each of which has nominally killed y_a, y_b,..., y_p host individuals by the end of the season. Then, given the initial host density x_1, the survivorship of the host cohort over the season, i.e., surviving all parasitism or Cs(**V**) as in Fig. 15, is given by

$$Cs(\mathbf{V}) = 1 - (y_a + y_b + \ldots + y_p)/x_1.$$

Then, the parasitoid species a, b,..., or p can be ranked in terms of its y-value.

However, there is another way to express Cs(**V**) in terms of survivorship over the ith interval, say $(1 - z_i/x_i)$, in which z_i is the total number of host individuals killed by parasitism out of x_i hosts over the interval. The successive multiplication of the interval survivorships (over $i = 1, 2,\ldots$, to end of season) gives

$$Cs(\mathbf{V}) = (1 - z_1/x_1)(1 - z_2/x_2)\ldots(1 - z_i/x_i)\ldots.$$

Although exactly equivalent to each other (see Appendix S8), the two expressions may on occasion appear to contradict each other as in the following example. In general, a z includes a part or whole of several y's that occurred over the same interval. Also, the cohort size (x) monotonically decreases over the season, i.e., $x_1 \geq x_2 \geq \ldots \geq x_i \geq x_{i+1} \geq \ldots$. Thus, the ratio z_i/x_i could be large even though the absolute value of z_i is small. Then, a later-acting species (involved in determining later z_i), even if its rank in the first expression was low, might have a large impact in the second expression. Thus, one may have a dilemma in deciding the rank of the species concerned: this may be called "the Thomson-Bess dilemma" as these early ecologists expressed their concern in this matter (Royama 2001). No problem would arise if a given species killed a large (or small) number of hosts and, at the same time, caused high (or low) interval mortality. If not, the dilemma remains. The concept of joint (or conditional) contributions among the parasitoids involved eliminates it.

The reason why later-acting species could have a large impact by killing only a small number of hosts is that earlier-acting species collectively have already reduced the host cohort size by the time the later species begin to act. In this sense, the contribution by a later-acting species (or for that matter, any later factor, e.g., predation) is in general conditional on, and should be evaluated jointly with, the impacts of the earlier species (factors). This recognition, too, suggests the need for a holistic approach to the subject, to consider the entirety of the natural-enemy complex. Conversely, an attempt to single out an important or key factor would likely fail.

Moreover, it is important to recognize that the natural enemy complex is not an organized entity, as we see no particular sign of coevolution. Rather, the complex is an ensemble of species, each seeking its own benefit in an opportunistic manner, as it were. Nevertheless, the successive actions of the constituents of the ensemble would produce the near periodic cycles of the budworm populations as we have actually seen them. How to comprehend this apparent paradox is a topic to be discussed in the following synthesis.

Part III: Synthesis

We now attempt to synthesize various pieces of information available in New Brunswick since the mid-1940s to reveal the basic mechanisms underlying the budworm outbreak process.

We also discuss, for a deeper understanding, what needs to be studied and suggest a few plausible approaches.

Synopsis of core aspects

As mentioned in *Introduction*, spruce budworm populations in New Brunswick have exhibited near periodic outbreak cycles in the past few centuries. Results from the province-wide egg-mass and L_2 surveys since 1952 have revealed that populations in all parts of the province have cycled in unison (cf. Royama et al. 2005: Fig. 3). Appendix S9 shows the provincial average updated to 2013. It depicts the cyclic process comprising two distinct components: the principal outbreak cycle (estimated by a moving average smoothed line) and random deviations about the cycle. As demonstrated in Part I, the chief source of the principal cycle is the mortality due to natural enemies occurring in succession throughout the budworm postdiapause period. The annual variation in the deviations has two independent sources: variation in the egg-recruitment rate and variation in the subsequent survivorship to the beginning of the postdiapause period. The variation in the recruitment rate is dictated by the immigration and emigration of egg-carrying moths and is the major source of the deviations. Functionally, the interaction with natural enemies is the chief mechanism underlying the principal cycle of a local population. On the other hand, the correlated variation in egg-recruitment rate across the province synchronizes the local cycles (Royama et al. 2005). We begin by outlining the local process determining the principal cycle and conclude with the mechanism of synchronization.

Interaction between natural-enemy complex and host complex

As described in Part II, major sources of mortality during the postdiapause stadia are natural enemies, including parasitoids, predators, and pathogens. Thus, in principle, the budworm outbreak cycle must be a local, natural-enemy–host interaction cycle. However, the interaction is between the complex of these natural enemies and another complex of alternative and alternate host species (Eveleigh et al. 2007). In particular, we recognize that the

system centered on spruce budworm is an open system such that many constituents (especially parasitoids and predators) of the natural-enemy complex would move in and out of the system. More generally, each constituent would change its allotment of time spent within or outside the system. They would likely do so as a result of individuals of a species trying to optimize their fitness in accordance with the changing phases of budworm cycle. These activities of the natural enemies would, in turn, influence (determine) the budworm cycle phases. Thus, we see that the above process of interaction manifests in the succession of different species of natural enemies in certain chronological orders.

Roles played by the succession of mortality factors in determining a local budworm principal cycle

We recognize the succession occurring at two levels: within season and between seasons, the latter occurring along with the host cycle changing its phase (e.g., decreasing or increasing).

Seasonal succession.—The seasonal succession began with the microsporidians (Fig. 18), immediately followed by the Group 1 parasitoid species, represented by *Apanteles fumiferanae* and *Glypta fumiferanae* (Fig. 16). These were soon followed by the Group 2 parasitoids (led by *Metorus trachynotus* followed by several tachinid species), and finally at the host pupal stadia by the Group 3 (pupal Chalcidoidae and Ichneumonidae) parasitoids, as well as by birds (Fig. 13). It is important to note that we saw no single species of natural enemies playing a predominant role, but the succession of them, in determining the seasonal budworm survivorship. A notably significant attribute of the succession is the following. Even the combined effect of some early mortality factors caused only a fraction of the total seasonal mortality. Nonetheless, on average, their effect was large enough to reduce the host population to such a level that some later-acting factors (even if killing only a small number of host individuals) could have a significant impact to reduce the host population even further, a typical example being predation by birds on pupae.

Between-season succession in accordance with cycle phases.—As in the seasonal succession, we saw no single factor that predominantly determined the budworm dynamics throughout our study: a major part of the declining phase of the budworm cycle. Even the Group 1 parasitoids, albeit ranked high in their impact (Table 4), did not play a predominant role in setting the observed trend in total parasitism (Fig. 17). The impact of these parasitoids was even reduced after the host population had begun to decrease. In contrast, it was the lower ranked parasitoids of Groups 2 and 3 that increased their impact such that the host population kept decreasing.

The observed succession of mortality factors is characteristic of an open system of natural-enemy complex and host complex. In particular, we recognize that each constituent of the natural-enemy complex is adapted to optimize its own reproductive success. Then, a question arises as to how such an ensemble of natural enemies, each seeking its own opportunity, could act as an apparent unit to cause budworm populations to consistently and almost periodically cycle. A plausible mechanism we suggest is the following.

Mechanism promoting the cyclic pattern.—As described in *Discussion of part II*, the composition of constituents in the natural-enemy complex changes in accordance with changes in the phase of a budworm cycle: more parasitoid species were found parasitizing budworm when its population was high than when it was low (Eveleigh et al. 2007; Appendix S6). Such changes in composition can be viewed as a generalization of the classical concept of the numerical response of a single predator species (by reproduction) to its prey density. This promotes a pattern similar to the simpler classical predator–prey cycle. However, a further consideration is in order.

Note first that, in general, population density at a given point in time is determined by mortality that has occurred in the preceding interval of time. This creates a phase shift between the survivorship cycle and the resultant population cycle. In particular, the population cycle would be at its steepest descent (ascent) when the survivorship cycle is in its trough (peak), i.e., when the impact of mortality is at its peak (trough). As depicted in Fig. 8b for Plot (1 + 2), the budworm postdiapause survivorship had reached a trough around 1988. Correspondingly, its population was at its steepest descent from 1987 to 1988 (Fig. 7a for Plots 1 and 2). Thenceforth, budworm survivorship began to increase and, consequently, the rate of population decrease slowed down. This implies that, midway down the declining phase of the budworm cycle, the impact of the natural-enemy complex had reached a maximum and then began to slacken.

However, during the peak to declining phases of the budworm cycle under observation, there was a practically linear relationship between the diversity in the composition of the parasitoid complex and the budworm density (Eveleigh et al. 2007, their Fig. 2A). This relationship suggests that there is a close link between the diversity of the parasitoid complex and the phases of the budworm cycle. In particular, the diversity was highest when the budworm population was at its peak level. This implies that, midway down the declining phase of the budworm cycle (where the impact of the parasitoid complex was the highest), its diversity had already been reduced. It suggests the involvement of an additional mechanism in determining the impact, although we do not have observational materials to reveal it. The following is our perception that may provide a guide to further studies for better understanding of the nature of the complex.

The succession of the parasitoid species in the complex is most certainly a result of the evolutionary process of each species to find a suitable niche in the timing of

parasitization. In other words, the process is a result of each species adapting to the influences from the already existing constituents. This implies that the parasitoid complex is unlikely to be just a haphazard ensemble of species but is likely to be dynamically ordered through interactions within the complex. In other words, the impact of the parasitoid complex depends not only on its composition but also on the performance of each constituent occupying a particular niche. The latter aspect of the impact can be viewed as a generalization of the notion of functional response by an individual predator. Thus, subject to further studies, we suggest that the dynamically ordered complex of natural enemies behaved as though it was an organized unit and caused the budworm population to cycle as in a classical predator–prey interaction system.

Having passed the phase of steepest descent, the budworm population continued to decline, albeit at a reduced rate. By the mid-1990s, the population cycles in New Brunswick had on average reached the lowest level but began to increase without a pause (Appendix S9): in fact, this occurred also at the trough of the preceding cycle in around 1960. Evidently, the natural-enemy complex continued to weaken its impact, even past the trough of each population cycle, allowing the cycle to increase without a pause. Little is known about what factors were actually involved at the trough because of technical difficulties in studying a low population: Appendix S10 gathers some fragmental pieces of information. Neither did we actually study the subsequent increasing phase of the cycle. Nonetheless, the following perception logically follows.

Having passed a cycle trough, the impact of the natural enemy complex must have kept weakening because the budworm population kept increasing. The impact kept weakening until it reached a minimum midway up the budworm population cycle where the cycle was at its steepest ascent, as observed in the previous cycle, just past the mid-1960s. Thenceforth, the impact must have begun to increase again to a level around 1980 at which the budworm population reached a peak level, whence a new cycle and the present study began.

Length and amplitude of a cycle.—As documented in the province-wide egg mass (L_2) surveys, a budworm outbreak cycle is unusually long in length (on average 35 yr) and extremely large in the trough-to-peak amplitude (in the order of 10,000 times in many localities), a feature that appears to radically depart from the classical notion of a "single-predator–single-prey" interaction. Thus, to comprehend the trend, it again requires a consideration of the structure of the natural enemy complex. The long cycle length with high amplitude suggests that an annual rate of change in budworm density is small, which in turn means that, despite the vast array of parasitoids, their combined impact on budworm in each year stayed comparatively small throughout the cycle. Conceivable reasons are as follows. First, some of the parasitoids could be multivoltine, requiring an alternate host other than budworm. The braconid wasp *M. trachynotus* parasitizing *Choristoneura rosaceana* as its second host is a known example (Maltais et al. 1989). The impact of these parasitoids on budworm should be limited by the population size of their alternate hosts. Thus, when a budworm population increases beyond the levels of the alternate hosts (which seldom become as abundant), these parasitoids could not exert an impact high enough to prevent the budworm population from a further increase. Second, many of the parasitoids are attacked by a large set of hyperparasitoid species (Appendix S11; after Eveleigh et al. 2007). Although not actually measured, we logically infer that these hyperparasitoids must have reduced the rate of increase in the primary parasitoids, and have allowed the budworm population to continuously increase. In the meantime, as described in the section *Analysis of disease*, the impact of pathogens (save the microsporidia) stayed mostly at a low level at all phases of the budworm cycle. Lack of impact from those pathogens, together with the consistent but comparatively low annual impact of parasitoid complex, allowed the budworm population to increase steadily but slowly to reach an extremely high level, resulting in an unusually long cycle length.

Synchronization of outbreaks among local populations

The mechanisms considered so far are all local processes. Yet, budworm outbreaks tended to occur more or less simultaneously across a large part of northeastern North America. Up until the late 1970s, the commonly held view of the occurrence of large-scale outbreaks was that they spread out from a few epicenters by the dispersal of egg-carrying moths. However, the existence of epicenters had never been substantiated. Rather, a close look at the New Brunswick egg-mass survey data had revealed that budworm populations began to increase simultaneously everywhere in the province (Royama 1984). This prompted the application of the theory, coined "the Moran effect" (Royama 1992). The effect stipulates that: if the random deviations of annual population changes about the principal cycle are correlated among local populations, their cycles will be synchronized in phase. This has in fact been shown to be true in the analysis of the provincial (egg mass–L_2) survey data (Royama et al. 2005). However, the cause of the correlation should be made more specific in details.

Recall the open-circle series in Fig. 9c, i.e., residuals after smoothing the L-to-L rate of change in Fig. 8a (solid circles) as estimates of the random deviations about the rate of change in principal (outbreak) cycle. Recall also that the residual series is made up of two components: the recruitment of eggs to a new generation and their survivorship over the subsequent pre-emergence period (Fig. 8c, d). The pre-emergence survivorship is a local process, largely dependent on the loss of larvae during their pre- and postwinter dispersal, and we

have not found a positive sign that it is correlated among local populations in a wide area. On the other hand, the rate of egg recruitment is the net result of immigration and emigration of the egg-carrying moths (Greenbank et al. 1980) and has been found to be correlated across New Brunswick (Royama et al. 2005). The mechanism of the correlation is as follows.

On a given day, there would be a net emigration in some sites (source sites) and a net immigration in other sites (receiving sites). However, the study of moth flight by radar and aircraft by Greenbank et al. (1980) revealed that the flight paths were determined by prevailing wind and tended to be directional. Consequently, some areas tended to be source sites and other areas, more like receiving sites. Then, the recruitment rate could be negatively correlated between the source and receiving sites, which contradicts the Moran effect because it requires a positive correlation. The following interpretation eliminates the contradiction.

Moths, no matter whether they are in a source or receiving site, tend to take off whenever weather is favorable, but not in poor weather. In the meantime, at each site, the egg recruitment rate fluctuates about an average level, which tends to be lower in a source site but higher in a receiving site. However, at each site, the rate would be lower than the average level when weather is good and the other way round in poor weather. Then, in a given local population, the rate would vary about its average level in accordance with the variation in weather conditions, which tend to be regionally correlated. Thus, the pattern of the temporal variation in the egg recruitment rate tends to be positively correlated across New Brunswick.

To sum up our synthesis: a budworm outbreak is the manifestation of the epidemic phase of the continuous population cycle, governed primarily by the interactions between the local host and natural-enemy complexes, and independently cycling local populations are synchronized by the Moran effect of regionally correlated variations in the egg-recruitment rate.

Final remarks

The above view is an explorer's crude map of the vast area of unknowns, as it were. Nonetheless, on the basis of recognizing the importance of the hosts–natural-enemies interactions, we provided our view of the core mechanism underlying the spruce budworm outbreak cycles in New Brunswick. Although understanding the mechanisms does not necessarily lead to an improvement in pest management, we hope that it at least provides some guidelines or working hypotheses for future studies.

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Supporting Information

Additional supporting information may be found online at: http://onlinelibrary.wiley.com/doi/10.1002/ecm.1270/full

Data Availability

Data associated with this paper is available from the Dryad Digital Repository: http://dx.doi.org/10.5061/dryad.t175g