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MINIREVIEW

Debilitating Effects of Viral Diseases on Host Lepidoptera

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While viral diseases of Lepidoptera are usually recognized for their ability to kill infected hosts, they may also reduce the fitness of individuals which survive infection. We have surveyed studies in the literature in which qualitative characteristics of individuals following treatment with virus have been evaluated. Most of these studies have involved nuclear polyhedrosis virus (NPV) and cytoplasmic polyhedrosis virus (CPV), but several have used granulosis virus (GV), entomopox virus (EPV), and small RNA viruses. Debilitating effects of viral diseases of Lepidoptera include slower development rates, lower pupal and adult weights, reduced reproductive capacity, and shorter adult longevity. These effects were observed more frequently in studies of CPV- than NPV-treated hosts. We evaluate the potential impact of debilitating effects of viral disease by comparing the net reproductive rate (R_0) of control and treated samples of the host. Studies using CPV and NPV showed significant reductions in R_0 from debilitating effects as well as from mortality, when compared to R_0 based on mortality alone. Sublethal effects were tested for and confirmed more frequently in studies using CPVs than those using NPVs. Little evidence exists for dose-dependent sublethal effects, but in studies involving NPV later instars tended to show stronger effects.

INTRODUCTION

Most studies of pathogens in populations of Lepidoptera focus on mortality and its direct causes, but disease may also reduce reproductive capacity of the host and increase host susceptibility to other mortality agents.

Debilitating effects of disease may be important in the population dynamics of lepidopteran hosts (Anderson and May, 1981; Munster-Swendsen, 1991; Myers, 1993; Ginzburg and Taneyhill, 1994) and are often overlooked when assessing the potential of disease to control populations of insect pests. An important group of pathogens of Lepidoptera is the baculoviruses or BVs. BVs are generally highly pathogenic and induce lethal infections (Entwistle and Evans, 1985). Because of this, BVs such as nuclear polyhedrosis viruses or NPVs (Baculoviridae: Subgroup A), and to a lesser extent, granulosis viruses or GVs (Baculoviridae: Subgroup B), have received considerable attention as candidates for biological control and bioinsecticides. Transmission of NPV is generally dependent on the death of the host and thus NPVs are not expected to affect individual quality. Infected individuals should die, and caterpillars which are exposed to virus but avoid infection should be uninfluenced by the virus. By contrast, less pathogenic diseases such as cytoplasmic polyhedrosis viruses [CPV (Reoviridae)] may be transmitted without death of the host, horizontally through fecal material and vertically through transovum or transovarian routes. Debilitating effects are therefore expected to be more common following exposure to CPVs than to NPVs because death is not necessary for transmission (Myers and Rothman, 1995).

There is evidence that NPVs can affect host quality despite their general pathogenicity. Reduced reproductive potential has been reported following NPV treatment in laboratory studies (Rothman and Myers, 1994, and references therein). Further, NPV epizootics and reductions in host fecundity may be observed following high densities of the host in populations of Lepidoptera (Myers, 1988; see references in Rothman and Myers, 1994). In this review we survey the literature to examine debilitating effects of viral disease on host Lepidoptera, and more specifically to examine the importance of these effects for pathogenic diseases such as NPVs compared to more benign CPVs.

METHODS

Survey of Literature

Only experiments that involved measurement of debilitating effects were included in the survey. One study by Klein and Podoler (1978) reporting reduced

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fecundity in *Spodoptera littoralis* was not included, as no debilitating effects were quantified. The following information was gathered from each study: host species, host instar at time of inoculation, inoculation method, pathogen dose, survival, debilitating effects examined and observed either by using statistics or when trends were emphasized by the author(s), magnitude of effects, and presence of pathogen (inclusion bodies) in pupae or adults following treatment. Only trials where some individuals survived treatment were considered. The magnitude of debilitating effects, when available, were calculated as: 100 × [1 – (mean value in treated sample/mean value in controls)], and scored as small (<15%), moderate (15–30%), or large (>30%).

Frequency and Magnitude of Debilitating Effects

The frequency of debilitating effects was examined for studies involving NPVs and CPVs. Frequency (percentage) was calculated by: 100 × (number of times effect was observed/total number of trials). For example, if two studies examined fecundity in one species, and each study examined three doses for each of two instars, the denominator for the calculation of frequency would be 12 for that species. Frequency data were summarized graphically by combining specific traits into general categories (development rate, weight, adult longevity, and reproduction). For example, if fecundity reduction was observed in 4 of 12 trials while fertility reduction was observed in 8 of 12 trials for a given species, the frequency of effects on reproduction would be 12 of 24 or 50%. Each host species was given equal weighting when calculating mean frequencies across all species, regardless of the number of studies or trials within each species. Magnitude of effects was also included in the summary of frequency data. Virus treatments resulting in increased weight or development rate of the host were scored as "no effect" for the summary graph.

Net Reproductive Rates

For each host species, we calculated net reproductive rates or the number of times a population could multiply per generation (R_0) (Southwood, 1991) for each trial (dose, instar, study) for control and treated samples. Sex ratios were assumed to be 50:50, and mortality values given for the pupal stage were assumed to indicate apparent mortality (Southwood, 1991) unless otherwise indicated. For example, a control sample with 40% survival to adult emergence, mean fecundity of 100, and mean percentage viable eggs of 80 would result in a calculated net reproductive rate of 0.4 × (100/2) × 0.8 or 16 (fecundity is halved to estimate the number of new females). Percentage reduction in R_0 due to treatment with pathogens was calculated by: 100 × [1 – (R_0 treated/R_0 control)]. Treatment effects were examined in three ways to assess percentage reduction in R_0: (1) due to mortality (m), (2) mortality and debilitating effects ($m + d$), and (3) debilitating effects alone (d). R_0 (m) was calculated based on survival in the treated group and values for "qualitative" traits (e.g., fecundity, egg viability, mating success, etc.) observed in the controls. R_0 ($m + d$) was calculated based on both survival and values for qualitative traits observed in the treated sample. R_0 (d) was calculated using control survival and values for qualitative traits observed in the treated group. For example, if treatment with a pathogen resulted in 20% survival, a fecundity of 50 and 60% viable eggs, then using values for the control example above, R_0 (m) = 0.2 × (100/2) × 0.8 = 8, R_0 ($m + d$) = 0.2 × (50/2) × 0.6 = 3, and R_0 (d) = 0.4 × (50/2) × 0.6 = 6. Percentage reduction in potential growth for the three examples would then be 50, 81, and 63%. Mean percentage reduction in R_0 was calculated within each species across all doses, instars, and studies (equal weighting given to reach trial). Similarly, mean percentage reduction in R_0 was calculated across all species per pathogen, giving equal weighting to each species. In studies where mean fecundity was not examined, mean weight of female pupae or mean pupal weight (unsexed) was used in calculations as pupal weight and fecundity are correlated in Lepidoptera. Although this does not yield a true R_0 value, it allows one to calculate percentage reduction in R_0 when comparing treated and control samples.

RESULTS

Frequency and Magnitude of Debilitating Effects

Results of the literature survey are given in Tables 1 to 3. The majority of studies show that mortality from disease is accompanied by qualitative changes in the surviving hosts (Tables 2 and 3). Pathogen treatment can reduce adult longevity, mating and oviposition success, fertility, weight, fecundity and egg viability, and rate of development, although cases of increased development rates and pupal size have been observed. Magnoler (1974b) reported a decrease in the duration of the pupal stage following treatment of *Lymantria dispar* with CPV, while Magnoler (1974a) reported a small increase in male pupal weight following NPV treatment of the same host species.

The frequency and magnitude of debilitating effects were generally greater for CPV- than NPV-treated hosts, although frequency of effects on reproduction approached 50% for NPV-treated hosts (Fig. 1). A Mann-Whitney U test showed a significantly greater frequency of cases of reduced weight (pupal and/or adult) in CPV- than in NPV-treated hosts ($P < 0.01$, one tailed test, $n = 8$ (CPV) and 11 (NPV)). No other comparisons yielded significant differences in frequencies although

TABLE 1
Explanation of Abbreviations Used in Tables 2 and 3

	Development rate
dt	Total development time (inoculation to adult emergence).
dl,dlm,dlf,	Duration of larval stage (unsexed), males, females.
dlmf	As above, both sexes.
dp,dpm,dpf	Duration of pupal stage (unsexed), males, females.
dpmf	As above, both sexes.
	Weight
wp,wpm,wpf	Pupal weight (unsexed), males, females.
wpmf	As above, both sexes.
wa,wam,waf	Adult weight (unsexed), males, females.
wamf	As above, both sexes.
we	Egg mass weight.
	Adult longevity
la,lam,laf	Adult longevity (unsexed), males, females.
lamf	As above, both sexes.
	Reproduction
m	Percentage mated females.
o	Percentage ovipositing females.
of	Percentage females ovipositing fertile eggs.
f	Fecundity or egg production.
v	Percentage viable eggs or percentage hatch.
	Test for presence of inclusion bodies (infection) in pupal or adult tissue
ni	No test performed.
i-	Test performed—infection not found.
i?	Test performed—results not given.
i*	Test performed—infection confirmed.

all tests had limited power due to small sample sizes. In general, weight and variables directly influencing reproduction were examined more frequently than developmental rates or adult longevity in species treated with NPVs and CPVs (Fig. 1).

Testing for Viral Infection

Surviving pupae or adults were surveyed for inclusion bodies in a greater proportion of studies using CPVs (10/14 or 71%) than of those using NPVs (5/19 or 26%). A greater proportion of these tests confirmed the presence of inclusion bodies in at least some trials for CPVs (9/10 or 90%) than for NPVs (2/5 or 40%). In the remaining studies of CPVs, pupae or adults were not tested, but some evidence for infection was reported. In their study of *Pectinophora gossypiella,* Ignoffo and Adams (1966) inoculated larvae using CPV obtained from previously infected adults. Bullock *et al.* (1970) tested adults but did not report the results although a sample of larvae were tested and found to contain virus. Mohamed *et al.* (1989) screened a sample of larvae only and reported 96% infection. Bird (1969) observed 100% infection in treated *Choristoneura fumiferana* larvae, but in a separate experiment involving debilitating effects of virus did not establish the presence of infection in survivors. In the remaining studies of NPVs, Young (1990) looked for inclusion bodies but did not report the results. Among studies of GV, Melamed-Madjar and Raccah (1979) tested for and confirmed infection in 60% of the pupae, but no test for infection was carried out by Sait *et al.* (1994).

Disease Effects on Net Reproductive Rates

In most trials examined in this survey, net reproductive rates (R_0) were reduced when debilitating effects were included in our calculations compared to R_0 based on mortality alone (110/126 or 87% of all trials for all pathogens groups). This effect is illustrated for NPV- and CPV-treated hosts in Fig. 2. In the remaining 13%, increases in R_0 may be attributed to chance events although selection for more vigorous individuals following treatment with virus is a plausible mechanism. Considering NPVs and CPVs, for which there was sufficient replication (i.e., number of species tested), the difference between mortality effects and combined mortality and debilitating effects on R_0 (i.e., percentage reduction in R_0 $(m + d)$-percentage reduction in R_0 (m)) was calculated for each host species. Parametric and nonparametric paired tests on these data showed that debilitating effects significantly reduced R_0 beyond the effects of mortality alone (NPV: parametric, $t = 3.33$, $P < 0.005$, nonparametric; $P < 0.01$, $n = 13$, CPV: parametric; $t = 3.17$, $P < 0.01$, nonparametric; $P = 0.01$, $n = 7$). Note that sample size is only seven for tests on CPV because mortality was not given for three of the host species treated with this pathogen. Surprisingly, the magnitude of reduction in R_0 was similar for species treated with NPV and CPV, when debilitating effects were considered in addition to mortality (i.e., $(m + d) - m$) (Fig. 2) (mean difference: NPV; 22 + 7% SE, $n = 13$, CPV; 19 ± 6% SE, $n = 7$) or alone (d) (NPV; 34 ± 8% SE, $n = 13$, CPV; 32 ± 10% SE, $n = 10$). For hosts treated with GVs, EPV, and small RNA viruses, reductions in R_0 from debilitating effects in addition to mortality were 11 ± 6% ($n = 2$ species), 25% ($n = 1$), and 5% ($n = 1$), respectively.

Instar Dependence

To examine instar dependence, percentage reduction in R_0 due to debilitating effects alone (d) are plotted against larval instar at the time of inoculation with NPVs and CPVs regardless of host species (Fig. 3). Inoculations prior to hatch (i.e., virus treatment of eggs, progeny of virus-treated parents), until larvae were 2 days old, were scored as first instar. Studies involving older larvae (>2 days) which did not give larval instar were excluded. If more than one trial was performed per instar within a study (i.e., using different doses), percentage reduction in R_0 was calculated using a mean R_0 across doses. NPV-treated hosts showed in-

TABLE 2

Summary of Effects of NPV Treatment on Host Lepidoptera

Host	Inoculation method	Dose (No. IB)	Instar/age[a]	Effect(s) on survivors: Examined	Effect(s) on survivors: Observed	Reference
Lymantria dispar (i*)	Diet	0.69,6.9	I	waf	—	Doane (1967)
		0.69,6.9	II		—	
		0.69,6.9/mm^2	III		—	
(ni)	Diet	25–5000 (5 doses)	III	dlmf,wpmf	—	Magnoler (1974a)
		2.5 × 10^4/larva			dlm-S,wpm-S^{neg}	
(i*)	Diet	500-5 × 10^4/larva	II	wpmf,m,we	(we-S-M)	Shapiro and Robertson (1987)[b,c]
(i-)	Droplet feeding	2500/larva	IV	wpmf,f	—	Murray *et al.* (1991)
Stilpnotia (*Leucoma*) *salicis* (ni)	Field spray	2 × 10^9/tree	?	dlmf,wpmf, f,v	(dlm-M,dlf-L),wpm-L, wpf-M,(f-L,v-L)	Nef (1971)
Choristoneura fumiferana (ni)	Field spray	1 × 10^{11}/acre	IV	v	(v-L)	Morris *et.al.* (1974)
Epiphyas postvitana (ni)	Diet	1600/25 larvae	10	dt,wp,wa, f,v	wa-S,f-L	Geier and Oswald (1977)
Malacosoma neustria (ni)	Diet	3–3000 (4 doses)	III	wpmf	—	Magnoler (1975)
		3–3000/larva (4 doses)	IV		—	
M. californicum pluviale (ni)	Leaf disc	4000	V	wpmf,f	wpf-S,f-S	Rothman and Myers (1994)
		4000/larva			wpm-S, wpf-S,f-M	
Trichoplusia ni (i-)	Diet	1708–1.708 × 10^5/larva (3 doses)	I	dl,wpmf la,f,v	—	Vail and Hall (1969)[d]
(ni)	Diet	5 × 10^4-1.5 × 10^6/ml (7 doses)	II	wp	—	Ignoffo (1964)
Heliothis zea (ni)	Diet	0.14,0.92	III	dp,lamf,f,v	—	Luttrell *et al.* (1982)
		4.8/mm^2			f-L	
(ni)	Diet	3-292/mm^2 (7 doses)	II–III	wp	—	Ignoffo (1965)
Pseudoplusia includens (ni)	Diet	2000-5 × 10^4 (3 doses)	IV	wpmf,lamf f,v	—	Young and Yearian (1982)
		3500-1 × 10^5 (3 doses)	V		—	
		8600,8.6 × 10^4, 8.6 × 10^5/mm^2	VI		— f-L,v-L	
Spodoptera litoralis (ni)	Leaf disc	8 × 10^3	III	dl,dp,la	—	Vargas Osuna and Santiago-Alvarez (1988)[e]
		1.6 × 10^4	IV	f,v	v-L	
		8 × 10^4	V		v-L	
		4 × 10^5/larva	VI		v-L	
(ni)	Leaf disc	7.8 × 10$^{5-6}$	III	dp,wp,lamf o,f,v	(dp-M,o-L,f-M)	Abul-Nasr *et al.* (1979)[f]
		1.2 × 10$^{5-7}$/larva	III	dp,wp,lamf,	(wp-S,laf-M,f-M)	
	1.2 × 10$^{6-7}$/larva	V	f,v	(wp-S,f-L)		
S. ornithogalli (i?)	Diet	10^7/larva	IV	dpmf, lamf,f,v	f-L	Young (1990)
S. frugiperda (ni)	Diet	87,142/mm^2	2	wp,f,v	—	Perelle and Harper (1986)
Mythimna (Pseudaletia) separata	Diet	LC_{25} LC_{50}	8	wpmf,wamf, dl,dp,lamf, f,v	Both doses: wpmf-S,wamf-S, dl-S,dp-S,lamf-S-M,f-L	Patil *et al.* (1989)
(ni)			Progeny		Both doses: wpmf-S,wamf-S, dl-S,dp-S,lamf-S-M,f-L	

Note. For effects on survivors, disease causes reduced development rate, weight, adult longevity, and reproduction unless otherwise indicate (by superscript^{neg}). Symbols listed under "effects on survivors examined" are for all doses listed. Symbols in parentheses under "effects on survivors observed" indicate that no statistical tests were performed. Symbols in parentheses following species names indicate test for infection and results. Magnitude of effects are indicated by: S, small (<15%); M, moderate (15–30%); and L, (>30%). See Table 1 for explanation of all symbols.

[a] Instar or age denoted by roman or arabic numerals, respectively.

[b] R_0 based on midpoints of mortality classes.

[c] R_0 based on larval survival only. (No information on adult emergence given.)

[d] R_0 based on mean of experiment with and without formalin.

[e] Similar results given for 24- to 36-h-old fourth and fifth instars.

[f] Fecundity estimates incorporate adult longevity.

[g] Author measured pupal size rather than pupal weight.

[h] Vail and Gough (1970) also report effects on fecundity but results are not given in a form amenable to this analysis.

[i] R_0 based on mean larval survival for doses within each instar.

TABLE 3

Summary of Effects of CPV, GV, EPV, and Small RNA Viral Treatment on Host Lepidoptera

Host	Inoculation method	Dose (No. IB)	Instar/age[a]	Effect(s) on survivors: Examined	Effect(s) on survivors: Observed	Reference
CPV						
Lymantria dispar (i*)	Diet	4-4 × 10^7 (8 doses)	III	wpmf dlmf,dpmf	All doses: dlm-L,dlf-L, dpmf-S^{neg},wpmf-L	Magnoler (1974b)
		4-4 × 10^7/larva (8 doses)	IV		All doses: dlmf-L,dpm-S-M^{neg} dpf-0-S,wpmf-L	
Choristoneura funiferana (ni)	Sprayed diet	200-2 × 10^6	II	wpmf,dlmf	(dlm-L,dlf-M,wpmf-M)	Bird (1969)[c,g]
	Sprayed foliage	larva	III		(dlmf-M,wpmf-S)	
			IV			(wpmf-S)
Trichoplusia ni (i*)	Diet	0.5–10 (4 doses)	I	dt,wpmf	(wpmf-S)	Vail et al. (1969)
		1	IV		(wpmf-M,dt)	
		10			(wpm-S,wpf-M,dt)	
		100			(wpm-M,wpf-L,dt)	
		1000/mm^2			(wpmf-M,dt)	
Heliothis virescens (i*)	Egg mass immersion	10^4/ml	Egg	dl,dp,wp, la,f	dl-M,wp-M, la-M,f-L	Simmons and Sikorowski (1973)
(i*)	Egg mass immersion	6.6 × 10^6/ml	Egg	dl,lamf,f	(dl-L),f-L	Sikorowski and Thompson (1979)
(ni)	Diet	8 × 10^5/larva	III	wpmf,f,v	wpm-M,f-L	Mohamed *et al.* (1989)
H. zea (ni)	Leaf disc	2 × 10^4/larva	5	dt,wp,wa	dt-S,wa-S	Bong and Sikorowski (1991)
Pseudaletia unipuncta (i*)	Dipped leaves	"high cone."	II–III	lamf,f	—	Tanada and Tanabe (1964)
Mamestra brassicae (i*)	Leaf disc	137/larva	III	lamf,f,v	v-M	Maleki-Milani (1970)[c]
Pectinophora gossypiella (ni)	Diet	73.6–7356.3/mm^2 (3 doses)	I	dl,dt,wp, la,f,v	All doses: (dl-M-L,dt-S-M	Ignoffo and Adams (1966) f,v,la),wp-S-M
(i?)	Diet	100/mm^2	I	lamf,wpmf, m,f,v	wpmf-M,lamf-L, (f-L)	Bullock *et al.* (1970)
(i*)	Diet	1000	I	dlmf,dpmf, wpmf	wpmf-M	Bell and Kanavel (1976)[c]
		10^4			dlmf-L,wpmf-M	
		10^5			dlmf-L,wpmf-M	
		10^5/ml			dlmf-L,wpmf-M	
Alsophila pometaria (i*)	Sprayed foliage		Late	wpmf,f,v	wpm-M,wpf-L (f,v)	Neilson (1965)
Nymphalis antiopa (i*)	Sprayed foliage		Late	wp,f,v	wp-M,(f,v)	Neilson (1965)
GV						
Plodia interpunctella (ni)	Droplet (treated females only)	2.12 × 10^{1-3}	I	dt,wpf,la,f,v	—	Sait et al. (1994)[c,i]
		6.3 × 10^{1-3}	II		f-M,v-S	
		2.98 × 10^{3-5}	III		f-M,v-S	
		4.83 × 10^{4-6}	IV		dt-S,v-S	
		6.36 × 10^{4-6}/larva	V		dt-S,f-M,v-S	
Sesamia nonagrioides (i*)	Diet	1.7 × 10^7/ml of diet	7–21	dp,of,f,v	(of)	Melamed-Madjar and Raccah (1979)
Small RNA viruses						
Amyelois transitella (ni)	Diet	5 ng virus/g of diet	III–IV	wpmf,of,f,v	wpmf-L, (of-L),f-L	Kellen and Hoffmann (1983)
EPV						
Choristoneura funiferana (ni)	Field spray	7.6 × 10^{10}/acre	IV	v	(v-L)	Morris *et al.* (1974)

Note. Columns, symbols, and footnotes are the same as in Table 2.

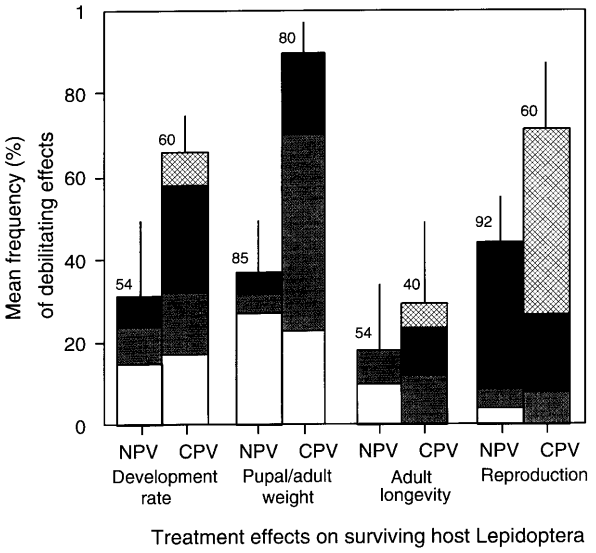

FIG. 1. Mean frequency (percentage) of debilitating effects/host species (+SE) in survivors of NPV- and CPV-treated Lepidoptera. Magnitude of effects are: small (<15%), unshaded bars; moderate (15–30%), shaded bars; large (>30%), black bars; magnitude unknown, hatched bars. Numbers above bars indicate percentage species in survey for which development rate, weight, adult longevity, and reproduction were examined in at least one study. See Table 1 for explanation of x-axis headings.

creased debilitating effects at later instars, while this trend was not evident for CPV-treated hosts (Fig. 3). Based on linear regression, larval instar explained 27 and 0.1% of the variation in reduction of R_0 for NPVs (n = 25) and CPVs (n = 15), respectively (significance levels are not reported as data are not independent). Similarly, if we consider reductions in weight of female or unsexed pupae and measures directly affecting reproduction (i.e., m, o, of, f, v; see Table 1), and score the presence of these effects in at least one dose per instar

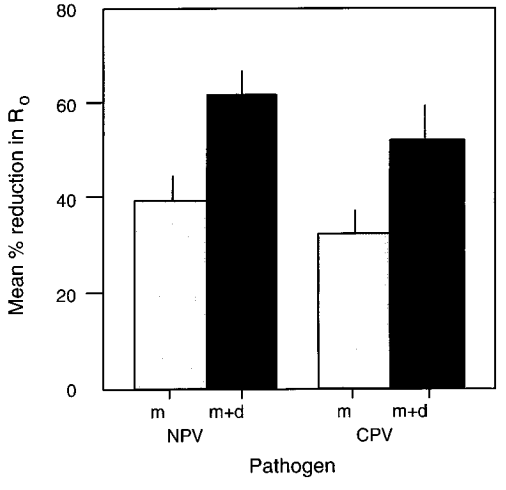

FIG. 2. Mean percentage reduction in net reproductive rate (R_0) (+SE) due to mortality alone (m) and mortality and debilitating effects (m + d) following treatment of host samples with NPVs and CPVs.

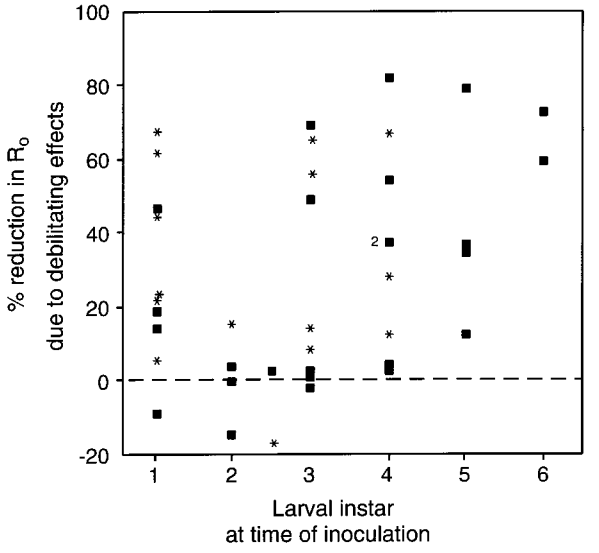

FIG. 3. Effects of larval instar on percentage reduction in net reproductive rate (R_0) due to debilitating effects alone in survivors of NPV (squares)- and CPV (asterisks)-treated hosts.

as 1, debilitating effects were more frequent in late instars, IV–VI (8/12 or 67%) than in early instars, I–III (3/13 trials or 23%) for NPV-treated hosts. This trend was not evident for CPV-treated hosts (instars I–III; 11/12 or 92%, instars IV–VI; 5/5 or 100%).

Dose Dependence

To examine dose dependence, the mean percentage reduction in R_0 from debilitating effects alone was calculated for the low(est) and high(est) dose from each instar and study within each species (equal weighting for all trials). Parametric and nonparametric paired tests on these data showed no consistent or significant effect of dose. The trend was toward greater debilitating effects at higher doses of NPV. The mean difference between reduction in R_0 at high and low doses was 11 ± 7% SE (n = 7). This was not the case for CPV-treated hosts (mean difference = −4 ± 6% SE, n = 4).

DISCUSSION

Frequency and Magnitude of Debilitating Effects

As predicted, debilitating effects are more common and more severe among CPV- than among NPV-treated hosts (Fig. 1) but are commonly observed among studies of NPVs. One potential problem with this result is the greater probability of publication of studies showing positive results. However, debilitating effects are rarely the sole focus of reported studies, and multiple studies showing negative results have been published (see *L. dispar* and *Trichoplusia ni,* Table 2). Further, a differential effect of this bias between NPV and CPV is not expected. Even so, a bias against studies showing negative results might still be expected

overall and should be considered when interpreting frequency of effects within a pathogen group.

Reductions in Net Reproductive Rates

Examination of the frequency and magnitude of debilitating effects alone (Tables 2–3, Fig. 1) cannot readily be related to potential growth of host populations. Statistically significant changes in individual quality do not necessarily imply biological significance, and consideration of each type of debilitating effect independently (e.g., fecundity, percentage hatch, percentage oviposition, etc.) can underestimate effects of pathogens. The use of net reproductive rates (R_0) provides a method by which a suite of individual characteristics can be considered in concert and related to potential growth of populations. Further, the use of R_0 allows one to consider debilitating effects in the context of mortality caused by the disease. This is the most meaningful approach since debilitating effects will be more important to the population dynamics of the host if the associated percentage mortality of the host population is low. For example, a 50% reduction in reproductive capacity in a population experiencing 90% mortality from disease will only reduce population growth by a further 5%. If the same population experiences 10% mortality, debilitating effects will reduce population growth by a further 45%. Across published studies on NPV, calculations of R_0 that include debilitating effects ($m + d$) led to significantly greater reductions in net reproductive rate when compared to calculations based on mortality alone (m). Inclusion of debilitating effects, on average, reduces potential population growth by a further 22% in hosts treated with NPV.

Surprisingly, the additional reduction in R_0 caused by debilitating effects ($(m + d) - m$) was similar for CPV- and NPV-treated hosts (Fig. 2) despite greater magnitude and frequency of effects following CPV treatment (Fig. 1). There are several reasons for this result. First, calculation of R_0 does not incorporate changes in development rate or adult longevity. Delayed development may influence population natality by increasing the individual's exposure and thus susceptibility to mortality agents. Rates of population growth may also be reduced by increased generation time for host species that do not have a fixed number of generations per year. However, without further study it is difficult to quantify effects of development rates on population growth. Longevity of the reproductive stage (adult) will also affect reproductive output. Individuals of species that oviposit only once during adult life may not survive long enough to mate and successfully lay eggs. Individuals of species that lay eggs throughout adult life may show reduced total reproductive output due to reduced lifespan. Although these factors may contribute to the reduced fecundity and egg viability reported in many studies, the effects of reduced adult lifespan are again difficult to quantify directly.

Second, although pupal weight and fecundity are often highly correlated, using pupal weights to calculate R_0 may underestimate effects of treatment with pathogens. In studies that examine both pupal weight and fecundity, the magnitude of treatment effects on fecundity are often greater than on female pupal weight, or pupal weight of both sexes combined (e.g., see Geier and Oswald, 1977; Rothman and Myers, 1994). All studies finding debilitating effects and examining both pupal (or adult) weights and fecundity reported greater magnitude of effect on fecundity for both NPVs (6/6) and CPVs (3/3). Therefore, disease may directly affect host reproductive organs, eggs, or processes involved in conversion of energy stores to eggs (Rothman and Myers, 1994) in addition to the indirect effect of disease on fecundity via reduced weight (pupal or adult). Studies of CPVs examined fecundity less frequently, and in some cases did not quantify a reduction in fecundity (and so were not used for estimates of population growth) (e.g., Neilson, 1965; Ignoffo and Adams, 1966). This made it impossible to assess the potential effects of fecundity reduction on population growth. Six of 14 studies involving CPVs (43%) examined and quantified changes in mean fecundity compared to 13 of 19 (68%) studies involving NPVs.

Finally, examination of the reproductive characteristics of adults, in general, was less frequent among host species when larvae were treated with CPVs than with NPVs (Fig. 1). Further, reproductive characteristics other than fecundity were more frequently examined and quantified following NPV treatment (12/19 or 63% of studies) than CPVs treatment (3/14 or 21% of studies) (and see Tables 2 and 3). Examination of measures such as mating and oviposition success and particularly egg viability allow greater potential for observing reductions in net reproductive rates. In order to fully quantify potential effects of pathogens on host populations, it is therefore important to examine effects on adults such as mating success, oviposition success, and particular fecundity and percentage hatch of offspring.

Mechanisms of Debilitating Effects

Sublethal infection is the most likely mechanism for debilitating effects of viral disease reported in this survey, particularly among the more benign CPVs. Sublethal effects may be due to the diversion of host energy reserves to support or combat the pathogen (Sikorowski and Thompson, 1979; Wiygul and Sikorowski, 1978, 1991), disruption of oocyte development (Neilson, 1965), or hormonal changes induced by the pathogen (O'Reilly and Miller, 1989; Burand and Park, 1992; Park *et al.,* 1993). For CPVs, infection is limited to particular tissues rather than being spread through the whole body as occurs with NPV. For example, CPV replicates in the epithelial cells lining the

midgut and new cells continue to differentiate (Evans and Entwistle, 1987). Although the efficiency of digestion is reduced with infection, death of caterpillars usually only occurs with infection at a very young stage, or if cross infected with another pathogen such as bacteria (Sikorowski and Lawrence, 1994).

Sublethal effects remain a contentious issue particularly with respect to more pathogenic baculoviruses such as NPV. Sait *et al.* (1994) have criticized previous studies which used the contaminated diet method for host inoculation, the most common method of inoculation in the survey (Tables 2 and 3). By allowing individuals to feed on contaminated diet for extended periods, smaller and more slowly developing individuals may survive by ingesting the pathogen more slowly than more vigorous individuals feeding at faster rates (Sait *et al.,* 1994). Selection rather than sublethal infection could be a cause of reported changes in host quality. In their study of sublethal GV infection of *Plodia interpunctella,* Sait *et al.* (1994) observed GV effects on development rate and host reproductive capacity using a modified droplet technique where individuals ingest virus within a fixed and short time period (1–2 hr).

Regardless of the dosing technique, sublethal effects can only be absolutely confirmed by the examination of survivors for the presence of virus inclusion bodies or viral DNA or RNA. Thus the evidence for true sublethal effects is scant for NPVs but good for CPVs infecting Lepidoptera. This could reflect a difference in emphasis of research on the two virus groups. Because NPVs have received more attention as potential biological control agents, the emphasis may be on establishing the presence and magnitude of virus effects on field populations rather than the specific mechanisms causing these effects. This could also explain why the data on the impact of debilitating effects is more complete for studies of NPVs than CPVs. Confirmation of sublethal infection becomes important when considering the potential for vertical transmission of virus from parents to offspring within eggs or on egg surfaces. How important sublethal infection and vertical transmission may be to subsequent host generations will depend on the strength of other routes of virus transmission between generations such as via environmental contamination. Examination of this issue is beyond the scope of this study.

Disease Effects on Host Males

Pathogens could in some host species influence population growth through their effects on male survivors. The influence of reduced male weight on reproduction is not well understood but small males may have reduced capacity to inseminate females (Haukioja and Neuvonen, 1985). Matings with virus-treated males and untreated females can also lead to reduced reproductive output. Santiago-Alvarez and Vargas Osuna (1988) observed reduced egg fertility following matings with NPV-treated *S. littoralis* males and suggested higher production of empty spermatophores or introduction of improperly oriented spermatophores as possible explanations for this result. Similarly, Kellen and Hoffmann (1983) observed reductions in the proportion of females laying fertile eggs following matings with *Amyelois transitella* males treated with small RNA virus, while Sait *et al.* (1994) observed reductions in the number of eggs laid and percentage hatch following mating with GV-treated *P. interpunctella* males. Tanada and Tanabe (1964) found no effect of CPV treatment on fecundity of *Pseudaletia unipuncta* when both males and females had been inoculated (Table 3), but observed a trend toward reduced number of eggs laid when treated males were mated with untreated females. Melamed-Madjar and Raccah (1979) found no effect of GV-treated males on oviposition of fertile eggs in *Sesamia nonagrioides.*

Instar Dependence

Results of the literature survey suggest that debilitating effects following NPV treatment are more likely to be observed late in larval development (e.g., Fig. 3). This trend is not evident for CPV-treated hosts. Last instar larvae should be the starting point for studies on debilitating effects of NPVs. Because NPVs are more pathogenic than CPVs, individuals infected earlier in development should succumb to disease more frequently. NPV epizootics are often characterized by heavy mortality of late instars, and the release of large amounts of inoculum into the environment due to within-generation dynamics of pathogen transmission (Woods and Elkinton, 1987; Dwyer and Elkinton, 1993). Exposure of larvae to this inoculum prior to pupation could result in partial infection, as viral replication may be suppressed at pupation (Watanabe, 1987). This could result in reduced individual vigor and reserves for egg provisioning without killing the host.

Of course, this examination of instar dependence may be confounded by host taxonomy as no distinction between species was made in Fig. 3 nor in our calculations of frequency of effects in early and late instars. For example, the absence of debilitating effects of NPV observed in *T. ni* may be because such effects do not occur in this species or because only early instars were examined. To examine instar dependence more rigorously, studies examining a range of instars are required. In the study by Sait *et al.* (1994), instar-dependent increase in development time was observed in *P. interpunctella* following GV treatment, but there was no instar effect on reproductive output (Table 3). Vargas Osuna and Santiago-Alvarez (1988) observed reduced egg viability of *S. littoralis* individuals treated with NPV in instars IV-VI but not in instar III. Young

and Yearian (1982) observed reductions in egg viability and fecundity following NPV treatment of *Pseudoplusia includens* at instar VI, not at instars IV and V (Table 2).

Dose Dependence

Strong evidence for dose-dependent debilitating effects among NPV-or CPV-infected hosts was not found in this study, although there was a trend toward greater effects at high doses following NPV treatment. However, unpublished examples of dose-related influence of NPV on pupal weight have occurred for *L. dispar* (Myers and Malakar, unpublished) and *M. c. pluviale* (Kukan, unpublished). In *L. dispar,* Shapiro and Robertson (1987) reported a decrease in egg mass weight with increasing doses of NPV while in the same species, Magnoler (1974b) observed a general increase in pupal weights with increasing doses following CPV treatment. Sait *et al.* (1994) reported decreases in percentage hatch, egg production, female pupal weight, and increases in development time with increasing dose of GV in at least one of five instars of *P. interpunctella* tested. Dose dependence was not examined in any other studies reporting debilitating effects of disease.

CONCLUSIONS

Debilitating effects in host Lepidoptera following pathogen treatment occur primarily with less pathogenic diseases, but may also be common with NPVs. Debilitating effects following exposure to NPV and CPV reduced the net reproductive rate of host samples. Effects of disease on host reproduction and development should be considered in the construction of models and collection and analyses of field data (e.g., life table studies) aimed at understanding the population dynamics of Lepidoptera and in the evaluation of the potential of viruses for biological control. The importance of viruses may be greatly underestimated by considering mortality alone.

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