A MODEL FOR THE BIOLOGICAL CONTROL OF *ORYCTES RHINOCEROS* (COLEOPTERA: SCARABAEIDAE) BY MEANS OF PATHOGENS

BY M.E. HOCHBERG AND J.K. WAAGE

Centre for Population Biology and Department of Biology, Imperial College at Silwood Park, Ascot, Berkshire SL5 7PY; and International Institute of Biological Control, Silwood Park, Ascot, Berkshire SL5 7TA, U.K.

SUMMARY

(1) We develop a mathematical population model for *Oryctes rhinoceros*, where it is limited both by density-dependent larval mortality and a baculovirus.

(2) The model divides the phenology of the female beetle into three distinct life-stages: juveniles (egg through young adult), feeders (young to middle-aged adults which are either mating or initiating attacks on palms), and breeders (older adults which lay their eggs at breeding sites).

(3) The rates of six different transmission pathways of the baculovirus are esti-mated using a least squares minimization criterion based on field prevalences of disease. This technique reveals that transmission from infected to susceptible feeding adults is the dominant route. This is in agreement with the literature on the subject (Zelazny 1976).

(4) The numerical simulation of the system, although differing from field obser-vations in the first 6 months of the simulation, is in accord with longer-term field observations on the population densities, prevalence of disease, and depression of the pre-baculovirus release equilibrium of *Oryctes*. The reason for the short-term differ-ences is thought to be the omission from the model of spatial aspects of the spread of the disease.

(5) Inundation by the fungus *Metarhizium anisopliae* var. *majus* is modelled as a long-residual, density-independent, 'biopesticide' which kills both juveniles and breeding adults.

(6) Numerical simulations show that increasing fungus inundation rates result in substantial decreases in the juvenile beetles. However, the fungus has no perceptible effect on the economically important populations of healthy (i.e. uninfected) feeding adults, and at high rates of application leads to an amplification in population cycles (i.e. instability) and the eventual elimination of the baculovirus from the system.

INTRODUCTION

The rhinoceros beetle, *Oryctes rhinoceros* (L.), is one of the most serious pests of coconut and oil palms in South-East Asia (Catley 1969; Bedford 1980, 1986; Young 1986; Mawikere *et al.* 1989). Effective control measures have been developed which involve both cultural and biological methods (for reviews, see Swan 1974; Bedford 1980; Young 1986), the latter concentrating on the use of pathogens. A specific baculovirus of *Oryctes* has been successfully established on islands where it has previously been absent, usually reducing beetle populations well below pre-intro-duction levels (Marschall 1970; Young 1974; Zelazny 1974, 1977; Hammes 1978; Monty 1978; Gorick 1980; Bedford 1986). Although local outbreaks of the pest have been known to occur in areas where the baculovirus is established, beetle populations on most islands of the South Pacific appear to be regulated at low densities by the

pathogen (Young 1986).

Many aspects of the ecology of *Oryctes* and the epidemiology of its viral disease are difficult to measure under field conditions. Factors important to the degree of depression of the pest population and its population dynamics before and after control are, consequently, hard to identify. In this context, a mathematical population model, constructed from information at hand, is of value in predicting key parameters influencing the pattern of biological control. This information can be used to focus limited research capability on the collection of the most relevant information. It can also provide a preliminary insight into the possible effect of other mortality factors on a beetle population regulated by the baculovirus.

In this paper, we construct a population model for the beetle−baculovirus interaction, based on estimated life-history parameter values and documented population trajectories in the field. By means of a sensitivity analysis, we then examine what parameters influence most strongly the level of depression caused by the baculovirus, and compare this to existing hypotheses regarding its effect. We then examine the potential effect of additional control measures involving the application of a second pathogen, the fungus, *Metarhizium anisopliae* var. *majus*, which differs markedly from the baculovirus in the nature of its interaction with rhinoceros beetle. Present concerns about the complementarity of the two pathogens in a single control system are examined.

BIOLOGY OF *ORYCTES RHINOCEROS* AND ITS BACULOVIRUS

Detailed descriptions of the biology of *Oryctes* can be found in Peterson (1977), Bedford, (1980) and Young (1986). Adult female rhinoceros beetles lay their eggs in rotting palm trunks and decomposing vegetation, where larvae develop slowly to pupation over a period of about 5 months. Male and female adults emerging from logs disperse to feed on meristems at the tops of palms, boring into the young buds and causing damage to leaves which is visible when they expand. Mating probably occurs at their feeding sites and possibly at emergence sites as well. Laboratory studies have shown that adult males live about 6 months and females about 9 months (Bedford 1976). It is thought that the beetles spend the first 3 weeks post-emergence at breeding sites, followed by approximately 1 month of feeding and then alternate visits to feeding and breeding sites for the remainder of their lives (Zelazny & Alfiler 1987). However, a recent study suggests that the feeding period may be as long as 5−6 months and the breeding time about 2 months (Zelazny & Pattang 1987; Zelazny & Lolong 1988). The fecundity of the females is about 50−60 eggs (Hurpin & Fresneau 1973; Bedford 1976), and laboratory studies suggest that they are deposited at a gradually decreasing rate over several weeks (Zelazny 1973a; but see Schipper 1976).

The baculovirus of *Oryctes* affects all life-stages with the exception of the eggs and possibly the pupae (Zelazny 1973a). Infection occurs when the pathogen is ingested. Larvae may become infected when they come into contact with neighbouring larvae or infected adults in breeding sites (Zelazny 1976). Young adults are typically infected whilst mating or feeding on live palms. This probably occurs when the susceptible adult ingests food contaminated by the infective faeces of a mate or other feeding adult (Monserrat & Veyrunes 1976; Zelazny 1976). The virus develops and persists in the midgut epithelium (Huger 1973; Monty 1974) and the infected adult begins to excrete infective virus about 3−9 days post-infection (Mohan, Jayapal &

Pillai 1986). Infected adults can live several weeks or more in an infective state.

The fact that the baculovirus has a low persistence outside of the host, with over 90% inactivation after only 1 week (Zelazny 1972), means that transmission can only occur shortly after excretion of the baculovirus by adults or release due to the death of larvae. Although the relative importance of these infection pathways are not known, it is generally thought that most infections occur at mating or whilst the adults are feeding (Zelazny 1976).

The persistence of the virus in the system is a result of its maintenance within the midgut of infected adult *Oryctes*. These adults may live 4–5 weeks in an infective state (Zelazny 1973a), thereby serving as flying 'reservoirs'. A recent theoretical model, incorporating an idealized pathogen reservoir as a part of the pathogen population, demonstrates that the storage capacity of the reservoir (e.g. the life-span of infected beetles) may be pivotal in determining the persistence of the pathogen and the dynamics of the host (Hochberg 1989).

DEVELOPMENT OF A HOST–PATHOGEN POPULATION MODEL

A flow diagram for the beetle–baculovirus model is shown in Fig. 1. For the sake of simplicity, only female *Oryctes* are considered, and three age groups of these are recognized. (i) Juveniles: this includes eggs, larvae, pupae and immature adults (density J). (ii) Feeding adults: young adult beetles that are mating or feeding, but not yet egg-laying at breeding sites (density F). (iii) Breeding adults: older adults which are already mated, making frequent visits to breeding sites to lay eggs, and occasionally feeding (density B).

Recruitment and development of healthy and infected hosts

Both infected and healthy breeders can give birth to new individuals, which enter the population of healthy juveniles. The recruitment rates of healthy and infected breeding adult females are represented by the parameters a and b, respectively. For simplicity, it is assumed that the egg-laying rate of breeding adult females is constant through time and does not differ from female to female. Estimates of birth rates have been obtained from field and laboratory studies of Zelazny (1973a) and from studies of Hurpin & Fresneau (1973) which give estimates of total fecundity. Healthy breeder adults are assumed to have a complement of 60 eggs which are laid continuously over the breeding period (i.e. 53.5 days), or, assuming a 1:1 sex ratio, 0.56 female eggs day^{-1}. This estimation is reinforced by the results of Kapadia & Valia (1986) who found that females could lay as many as 58 eggs in 10 days.

Infected breeders have been found to stop laying eggs 1 week after infection, and lay an estimated 2.8 eggs per female during the first week, and about 0.9 eggs per female week^{-1} over the first 5 weeks of their lives (Zelazny 1973a). This latter figure is used to arrive at an estimate of 0.64 female eggs laid per 10 days.

Healthy juveniles may either develop into healthy feeders or become infected. Infected juveniles will either die (α_j) or become healthy feeders if originally exposed to the baculovirus late enough in their larval period (i.e. during the invulnerable stages of the pupa and immature adult). Data on survival of infected juveniles indicates considerable variation between instars, with first instar larvae living for 8·5
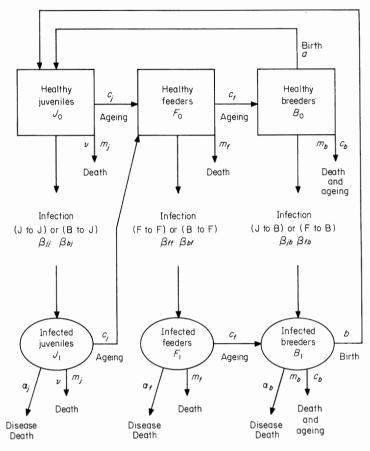
FIG. 1. Flow diagram of the interactions in the *Oryctes rhinoceros*−baculovirus model (see text for explanations of pathways and symbols).

days and larvae infected late in the third instar surviving for 25 days at average temperatures of 27 °C (Zelazny 1972). Here a value of 18 days is adopted (roughly equal to the life-span of an infected young third instar larva). c_j, c_f and c_b are the development rates for surviving juveniles, feeders and breeders. For survivors, the simplifying assumption is made that there are no differences in development rate between individuals within a given age-class, and that the development rates are constant through time. The average length of the juvenile stage is taken as 168 days, the feeding period is 154 days, and the breeding period 53·5 days (Zelazny & Alfiler 1986; Zelazny & Pattang 1987; Zelazny & Lolong 1988).

For juveniles, feeders or breeders dying from the baculovirus, it is assumed that there are no differences between individuals within any given age-class in terms of death rates due to the baculovirus. Feeders and breeders die at rates α_f, and α_b, respectively. The estimation of the life-span of an infected adult is based on survival of field collected adults (Zelazny 1973a). The average survival, 38·5 days, does not take into account the period of infection before collection, and is therefore probably an underestimate. For want of more accurate data, feeders and breeders are assumed

to live for the same average time when infected by the baculovirus.

Two kinds of mortality, besides that caused by infection, are assumed to affect beetle populations, and to contribute to the death terms in Fig. 1.

Density-independent mortality is known to affect all stages of *Oryctes*, but estimates are few. Density-independent mortality in Fig. 1 is indicated by the parameters m_j, m_f, and m_b for juveniles, feeders and breeders, respectively. Feeder and breeder mortalities were assumed to be equal, and were estimated from laboratory studies (Zelazny 1977) which gave an overall adult mortality of 7·2%. Assuming that mortality was constant over time, this results in a constant rate of 0·0152.

Density-dependent mortality in *Oryctes* is not well understood. Zelazny & Lolong (1988) posit that, in the absence of the baculovirus, populations of the beetle are limited by the availability of breeding sites. Such competition could influence (i) the oviposition rate of adults, (ii) the size of larvae, and hence of ensuing adults, or (iii) the survival of the larvae. For the purposes of this study, therefore, it is assumed that density-dependent mortality (v) only affects the juvenile age-class and that there is no time delay between intraspecific competition and the death of juvenile *Oryctes*.

Unfortunately, parameter estimates for density-independent and density-dependent mortalities in the juvenile age-class were impossible to make directly from the published data.

An estimate of m_j was possible by assuming that density-independent and density-dependent mortalities both contributed to the generational mortality of the juvenile age-class (97·1% based on study of Zelazny & Alfiler 1986). For any given estimate of m_j, a simple differential equation model ($dJ/dt = -m_j J - vJJ$) was simulated for the 168 days of the juvenile period, with an initial equilibrium density of J_o' juveniles (see eqn (8.) below). (The parameter v only acts to scale J_o' and therefore changing its value had no effect on the generational mortality). After the simulation, the final density of juveniles was noted and the generational mortality calculated ([initial density − final density]/initial density). This procedure was reiterated until the generational mortality equalled the one recorded by Zelazny & Alfiler (1986). This resulted in an estimate of 0·0474 for m_j.

Using the estimate of m_j, the value of v could then be determined from eqn (8), based on observed equilibrium levels of juveniles in the region of 200 − 300 (e.g. Young & Longworth 1981). The value of v chosen (0·005) resulted in an equilibrium level of 251 juveniles ha^{-1}.

Baculovirus transmission

Healthy individuals in all three stages can become infected and thereby enter the population of infected beetles. Densities of *Oryctes* susceptible to infection are subscripted by 0 (i.e. J_0, F_0, B_0), whereas the densities of infected hosts are subscripted by 1 (i.e. J_1, F_1, B_1). As the pathogen is short-lived outside of the host, it thus need not be explicitly modelled.

The transmission rate of the baculovirus between and within beetle stages is an extremely difficult parameter to estimate, and this has not been done under laboratory or field conditions. In principle, such measurement would require explicit knowledge of (i) the density of susceptible and infecting individuals, (ii) the probability of one or more contacts between a given infected individual and the population of susceptibles per unit time, and (iii) the probability of a given contact resulting in a

successful infection. Furthermore, the transmission rate is likely to depend upon the spatial aspects of the system, which are not considered here (e.g. densities of feeding and breeding sites, flight distances of adults, etc.).

For the purpose of the model, individuals within a particular age-class are assumed to be equally susceptible to the baculovirus. This assumption is made even though differences in susceptibility are known to exist between various instars of the larva (Mohan, Jayapal & Pillai 1985); in addition, eggs, pupae and immature adults are virtually immune to infection (Zelazny 1972, 1973a). This fact is taken into account by appropriate transmission rates in the model. Little is known about age-dependent susceptibility in feeding and breeding adults.

For all stages, per capita transmission rate is assumed to be directly proportional to the density of infected hosts. This is the simplest way of representing a density-dependent process in continuous time models, and has been used extensively in analytical host–pathogen models (e.g. Anderson 1982; Anderson & May 1981; Hochberg 1989).

On the basis of previous research, six different virus transmission pathways are considered (Fig. 1) (Zelazny 1973b; Zelazny 1976; Zelazny & Alfiler 1986): (i) breeders to juveniles (β_{bj}), (ii) juveniles to juveniles (β_{jj}), (iii) feeders to feeders (β_{ff}), (iv) breeders to feeders (β_{bf}), (v) feeders to breeders (β_{fb}), and (vi) juveniles to breeders (β_{jb}). Given what little is known about the transmission biology of the baculovirus in the field, estimates for the six transmission interactions were virtually impossible to make.

Transmission constants were estimated by minimizing the sum of squared differences of percentage infection between the model and field observations made by Zelazny (1973b). Approximately 3–4 years after the baculovirus was introduced into an *Oryctes* population on Western Samoa the levels of infection were: 3·1% in larvae, 54% in adults collected in palm trees, and 29% in adults found at breeding sites. For the purposes of this study these categories were taken as juveniles, feeders and breeders, respectively.

The criterion to be minimized, M, is given by

$$M = \frac{(O_j - E_j)^2}{E_j} + \frac{(O_f - E_f)^2}{E_f} + \frac{(O_b - E_b)^2}{E_b} \tag{1}$$

where O and E are the observed (model) and expected (empirical) percentages of infection, respectively. Although it could not be concluded that all combinations of initial conditions, or other minimization criteria (e.g. arcsine transform of the percentages), would result in the same final parameter values, several different initial conditions for the transmission constants gave the same final values. The final observed percentages of infection were the same as the expected ones to three-digit accuracy ($M = 7·5x\ 10^{-6}$). The transmission constants are presented in Table 1.

The values used for the parameters indicated in Fig. 1 are summarized in Table 1. The rates of change for susceptibles and infecteds in the three age-groups are determined by the following differential equations:

$$dJ_0/dt = aB_0 + bB_1 - \beta_{bj}B_1 J_0 - \beta_{jj}J_1 J_0 - [c_j + m_j + v(J_0 + J_1)]J_0 \tag{2}$$

$$dJ_1/dt = \beta_{bj}B_1 J_0 + \beta_{jj}J_1 J_0 - (c_j + m_j + v(J_0 + J_1) + \alpha_j)J_1 \tag{3}$$

$$dF_0/dt = c_j(J_0 + J_1) - \beta_{ff}F_1 F_0 - \beta_{bf}B_1 F_0 - (c_f + m_f)F_0 \tag{4}$$

$$dF_1/dt = \beta_{ff}F_1 F_0 + \beta_{bf}B_1 F_0 - (c_f + m_f + \alpha_f)F_1 \tag{5}$$

$$dB_0/dt = c_f F_0 - \beta_{fb}F_1 B_0 - \beta_{jb}J_1 B_0 - (c_b + m_b)B_0 \tag{6}$$

$$dB_1/dt = c_f F_1 + \beta_{fb} F_1 B_0 + \beta_{jb} J_1 B_0 - (c_b + m_b + \alpha_b) B_1 \tag{7}$$

Model simulations

All simulations were conducted using a Runge−Kutta differential equation solving algorithm. Unless otherwise indicated, the simulations were run for 2 years post-introduction of the baculovirus. The time-step used was 0·1 days, and the rate parameters represent the events taking place over a 10-day period (Table 1).

The initial densities of infected juveniles and breeders are assumed to be zero, and that of the (introduced) infected feeders is arbitrarily set equal to one beetle ha^{-1}. Unless otherwise specified, the initial densities of the healthy juveniles, breeders and feeders are set to their equilibrium densities (J_0', F_0', B_0') in the absence of the baculovirus, given by

$$J_0' = \frac{1}{\nu} \left[\frac{a c_j c_f}{(m_f + c_f)(m_b + c_b)} - m_j - c_j \right] \tag{8}$$

$$F_0' = \frac{c_j J_0'}{m_f + c_f} \tag{9}$$

$$B_0' = \frac{c_f F_0'}{m_b + c_b} \tag{10}$$

In conducting numerical simulations of the model, only a single equilibrium state

TABLE 1. Biological interpretations, numerical values, sensitivities, and sources of the parameters used in the simulation model[†]

	Biological Interpretation	Value	Units	Sen[‡]	Ref[1]§
a	Eggs laid per healthy breeder	5·60	10 days^{-1}	***	a,d
b	Eggs laid per infected breeder	0·64	10 days^{-1}	*	b
c_j	Development: juveniles	0·0595	10 days^{-1}	***	c
c_f	Development: feeders	0·0649	10 days^{-1}	**	d
c_b	Development: breeders	0·187	10 days^{-1}	**	d
m_j	DI mortality: juveniles	0·0474	10 days^{-1}	*	c
m_f	DI mortality: feeders	0·0152	10 days^{-1}	*	e
m_b	DI mortality: breeders	0·0152	10 days^{-1}	*	e
α_j	Virus mortality: juveniles	0·556	10 days^{-1}	*	f
α_f	Virus mortality: feeders	0·260	10 days^{-1}	**	b
α_b	Virus mortality: breeders	0·260	10 days^{-1}	*	b
β_{bj}	Trans: breeders ⇒ juveniles	0·0869	i^{-1} 10 days^{-1}	*	g
β_{jj}	Trans: juveniles ⇒ juveniles	0·0019	i^{-1} 10 days^{-1}	$\overline{*}$	g
β_{ff}	Trans: feeders ⇒ feeders	0·1501	i^{-1} 10 days^{-1}	$\overline{***}$	g
β_{bf}	Trans: breeders ⇒ feeders	0·0330	i^{-1} 10 days^{-1}	$\overline{*}$	g
β_{fb}	Trans: feeders ⇒ breeders	0·0070	i^{-1} 10 days^{-1}	$\overline{*}$	g
β_{jb}	Trans: juveniles ⇒ breeders	0·0024	i^{-1} 10 days^{-1}	$\overline{*}$	g
ν	DD mortality: juveniles	0·005	j^{-1} 10 days^{-1}	$\overline{***}$	g

† Abbreviations: DI=density-independent; DD=density-dependent; Trans=transmission; i=infected donor; j=juvenile competitor.

‡ Sensitivity, or relative importance in the accuracy of parameter estimation according to sensitivity analysis: *** very important; ** important; * unimportant. Underline indicates that parameter estimates are based on least squares estimation (see text for explanation).

§ References: (a) Hurpin & Fresneau 1973; (b) Zelazny 1973a; (c) Zelazny & Alfiler 1986; (d) Zelazny & Lolong 1988; (e) Zelazny 1977; (f) Zelazny 1972; (g) see text for estimation procedure.

was observed under a variety of initial conditions. We cannot conclude, however, that additional stable states of the host and pathogen did not in fact exist.

The hypothetical baculovirus introduction was made 50 days after the start of the numerical simulation, and the populations were simulated for an additional 2 years. At the end of the simulation the q-value (Beddington, Free & Lawton 1978), or amount of depression of the initial *Oryctes* population afforded by the introduction of the baculovirus, was calculated for each life-stage of *Oryctes* (the final observed host density divided by the initial host density, given by eqns (8), (9) and (10)).

RESULTS

The construction of the model relied heavily on the limited information available on *Oryctes* and its baculovirus. Critical information on the key transmission pathways for the baculovirus was lacking, due to the difficulty of measuring transmission rates in the field. Therefore, the model itself was used to identify these pathways. Different patterns of transmission involving the six possible pathways would generate different levels of infection between life-stages. Data existed on the distribution of infection between these stages (Zelazny 1973b). The least squares minimization (eqn (1)) identified transmission between 'feeders' as the key pathway. It is satisfying that this is in agreement with the views of experts based on accumulated field experience (Zelazny 1976).

Dynamics of the model

The dynamics of the *Oryctes*−baculovirus model are shown in Fig. 2. The introduction of the disease into the *Oryctes* population limited only by intraspecific density dependence is met by an immediate epidemic, which is rapidly damped, leading to more constant populations of healthy and infected beetles. The epidemics in the juvenile, feeder and breeder subpopulations peak to near 100% infection approximately 1 week (juveniles, feeders) to 2 months (breeders) after the baculovirus release. This epidemic is followed by a resurgence of healthy beetles, lowering the prevalence of infection to levels of about 15−25% in the adults and near zero in the juveniles. This occurs at about 10 months post-release. A second, smaller, epidemic wave then occurs, peaking at about 15 months post-release, followed by more or less constant prevalences of infection. By 2 years post-release the populations are effectively equilibrated, with infected prevalences of c. 54%, 29% and 3% for the feeders, breeders and juveniles, respectively.

Also pictured in Fig. 2 are the effects of the disease on the total populations of the beetles. Clearly, the introduction of the baculovirus in the model results in drastic reductions in *Oryctes* density. After only 6 months post-release, the populations of juveniles, feeders and breeders are depressed to their minimum levels. A slight resurgence occurs between 6 and 12 months post-release, but this is quickly checked by the disease. At 4 years post-release, the populations of juveniles, feeders and breeders are depressed to 6·5%, 2·3%, and 1·4% of their pre-disease levels, respectively.

The model has been run using parameter estimates from a number of different sources. Hence its behaviour, while partly governed by the laboratory and field data put into it, is novel and may usefully be compared to patterns observed in the
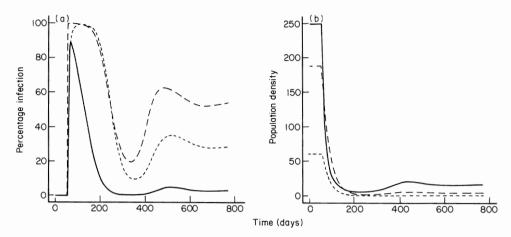
FIG. 2. Population dynamics of the basic model for juveniles $(-)$, feeders $(--)$ and breeders $(---)$ with parameter values as described in Table 1: (a) percentage infection; (b) population density. Baculovirus is introduced 50 days after the simulation starts. Initial healthy densities of *Oryctes rhinoceros*: $J_0=249$, $F_0=188$, and $B_0=61$.

field. This is particularly true of the trajectories predicted for beetle numbers and virus prevalence.

There are very few field studies which have conducted extensive long-term (i.e. >1 year) population studies of *Oryctes* both pre- and post-release. One such study is that of Zelazny & Lolong (1988), which examined the release of baculovirus on a number of different islands in the Maldives. In general, they found a maximum prevalence of infection about 9 months post-release, with a maximum reduction of the number of attacks on live palms (an indirect estimate of the density of 'feeder' populations) between 6 and 18 months post-release. This is in broad agreement with the results of the models shown in Fig. 2.

The initial levels of virus prevalence, however, are rather different from those observed in real baculovirus introductions. Thus, the first epidemic after release in the study of Zelazny & Lolong (1988) reached 45% infection of 'feeders' (in contrast to nearly 100% in the model), with the peak at 9 months being only 65%.

However, the effect of introduction of the baculovirus on the equilibrium prevalence of disease is in broad agreement with a number of field studies. Zelazny & Lolong (1988) estimated an equilibrium prevalence in 'breeders' of 40−50% (samples from palms) and 35% (samples from baited traps). Variability in such estimates is likely to arise not only from the sampling method (Zelazny 1976, 1977; Marschall & Ioane 1982) but also the type of site surveyed (Zelazny & Alfiler 1986). In general, estimates from both palm samples and bait traps are in the range of 20−50% prevalence in 'feeders' (Zelazny 1973b, 1977; Bedford 1976; Marschall & Ioane 1982; Zelazny &Alfiler 1986; Zelazny & Lolong 1988).

With respect to changes in beetle populations, the model predicts a more dramatic fall in beetle numbers following the introduction of baculovirus than is commonly observed in the field in the period following release. By the achievement of equilibrium, populations of juveniles, feeders and breeders are about 6·5%, 2·3% and 1·4% of their pre-baculovirus levels. In contrast, maximum observed reductions in

feeding damage to palms after baculovirus release (as an indicator of feeder density) can approach 95% (Hammes 1978; Zelazny & Lolong 1988) but is more typically in the range of 50−85% (references as in our study).

It is possible that these differences reflect spatial dynamics of the baculovirus in the field, which are not explicitly considered in the model. Field data such as that of Zelazny & Lolong represent averages of samples over a broad area, across which baculovirus may not spread evenly and quickly. This argument may apply as well to discrepancies in initial prevalence of infection between the model and field data. Where it has been studied, the spatial spread of insect virus epidemics appears to follow a characteristic pattern through time (Entwistle et al. 1983).

Finally, the model appears to overestimate substantially the ratio of adults to juveniles when compared to field studies, both before and after the introduction of the baculovirus. Zelazny & Lolong found a 1:50 ratio of breeders to juveniles before release and a 1:130 ratio after release of the virus, in contrast to the ratios of 1:4 and 1:18 predicted, respectively, by the model. This discrepancy may reflect the difficulty of accurately sampling breeders in the field, which spend a portion of their time in palms (perhaps as low as 30%; Zelazny & Alfiler 1987). However, it could also be caused by errors in estimation of development times or death rates.

Overall, the model shows a qualitative fit to the majority of field observations of Oryctes-baculovirus interactions, with similar trajectories following introduction, although different initial and, in some cases, equilibrium levels for key parameters. Here it is important to note that the densities of healthy and infected hosts change drastically over the first 24 months after the baculovirus is introduced. This is verified by analyses of field studies in which populations of Oryctes and the baculovirus took several years to equilibrate (Young 1974; Young & Longworth 1981; Entwistle et al. 1983). This means that tentative conclusions about the effectiveness of an introduction cannot be made reliably before 2 or more years post-release.

Sensitivity analysis

In the analysis conducted here, the very simple approach is taken of changing the value of one parameter at a time, adding or subtracting 10% or 30% of its value, and simulating the model for a period of 4 years (to ensure the achievement of an equilibrium). Larger deviations from the parameters could have been considered, but the purpose here is to reveal the relative effects of changes in the parameters on the levels of population depression (in terms of q-values, defined as the ratio of population density after release of the baculovirus to that before release, see Beddington et al. 1978) and prevalence of infection. Of course, parameter estimates which differ by more than 30% of the values quoted in this study would require further numerical simulations in order to assess their sensitivity. Table 1 presents a ranking of priorities in parameter estimation, according to the results of the sensitivity analysis.

As shown in Figs 3−5, the model is insensitive to the moderate changes in parameter values considered here. Some sensitivity occurred for the q-values of each age-class (Figs 3a, 4a, 5a); however, this still meant that the q-values only once exceeded 0.1. Sensitivity was low for the prevalence of disease in the feeders and breeders, but somewhat high for the juveniles.

Changes in certain parameters consistently have either large or small effects on the q-values and percentage infection of different age-classes of the host. The most
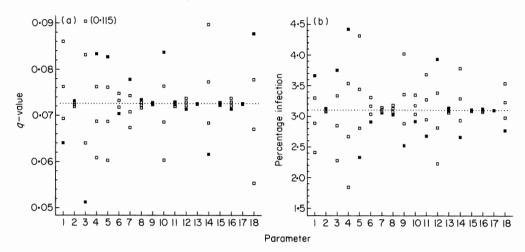
FIG. 3. Sensitivity analysis of the effects of parameter changes on (a) q-values and (b) percentage infection in the juvenile subpopulation. A solid square refers to the parameter, less 30% of its value as presented in Table 1. Empty squares indicate further changes in parameters -10%, $+10\%$, and $+30\%$), and always proceed one another in terms of the effects on q-values and prevalence of infection. For example, the solid square of parameter 1 (i.e. the egg-laying rate of healthy breeders) refers to the parameter, less 30% of its value. The next point down refers to the parameter, less 10% of its values, and so on. The broken line refers to the parameter values with no change (i.e. as per Table 1). Key to parameters: 1, a; 2, b; 3, c_j; 4, c_f; 5, c_b; 6, m_j; 7, m_f; 8, m_b; 9, α_j; 10, α_f; 11, α_b; 12, β_{bj}; 13, β_{jj}; 14, β_{ff}; 15, β_{bf}; 16, β_{fb}; 17, β_{jb}; 18, ν.
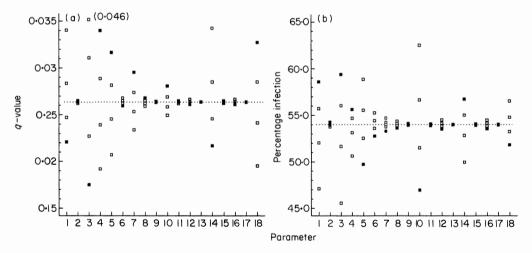

FIG. 4. Sensitivity analysis of the effects of parameter changes on (a) q-values and (b) percentage infection in the feeder subpopulation. Otherwise, as explained in Fig. 3.

sensitive parameters are (i) the egg-laying rate of healthy breeders, (ii) the development rates of all stages (especially juveniles), (iii) the death rate due to the baculovirus of the feeding adults, (iv) the transmission rate from feeder to feeder, and (v) the density-dependent mortality rate. Figures 3−5 clearly show that the
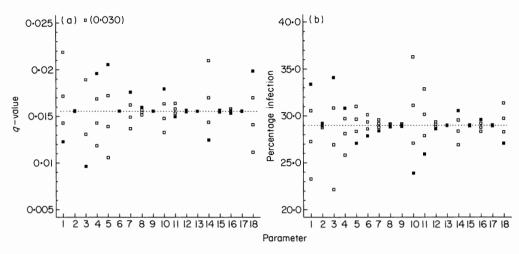
FIG. 5. Sensitivity analysis of the effects of parameter changes on (a) q-values and (b) percentage infection in the breeder subpopulation. Otherwise, as explained in Fig. 3.

sensitivity of transmission rate from feeder to feeder is much larger than the sensitivities of each of the five other transmission terms. For example, changes in breeder mortality had almost no effect on juvenile and feeder numbers. Notice, in particular, that transmission affects the equilibrium prevalence of infection, a result which is contrary to simple analytical models of directly transmitted diseases (Anderson & May 1981). (The prevalence of the disease within the whole beetle population is affected less than in each age-class, see the directions of sensitivity effects in Figs 3b, 4b, 5b). On the other hand, the model is consistently insensitive to changes in several other parameters. These include (i) the egg-laying rate of infected breeders, (ii) the density-independent mortality rates of all stages, and (iii) all of the transmission rates, with the exception of the dominant one (i.e. feeders to feeders).

Of course, there are a number of exceptions to the generalizations made above. First, as expected, the transmission rates involving juveniles have substantial effects on the prevalence of infection in that subpopulation, but no apparent effect on their q-values. Second, the effect on q-values of changes in the average development time of juveniles is consistently more sensitive than the life-spans of feeders and breeders, especially as juvenile life-spans increase. However, the effects of changes in development rate of the various age-classes on the percentage infection are less consistent. Third, the density-independent and baculovirus-caused death rates of the feeders are much more important to estimate, in terms of effects on q-values, than those of the juveniles or breeders. In general, deviations in death due to baculovirus have a much more substantial effect on prevalence of infection than does death from density-independent causes. Virus-induced death in juveniles only affects the prevalence of infection in juveniles.

Also note from Figs 3–5 that deviations in parameter estimates change q-values and prevalences of the disease in opposite directions. For example, reducing juvenile development rate (resulting in juveniles taking longer to develop into feeders) increases q-values whereas it decreases prevalences of infection. An exception to this is the development rate of feeders, where reducing feeder development lowers

q-values (corresponding to greater depression of the total population), but also reduces the prevalence of infection. A similar curious relationship exists in the effects of transmission from feeder to feeder on q-values and prevalence of disease (Fig. 4b). The reasons for these relationships are not understood, but may be linked with the fact that the feeders are the principal transmission agents in both circumstances.

Introduction of Metarhizium anisopliae

The fungus, *Metarhizium anisopliae* var. *majus*, can infect *Oryctes* through spores germinating on or penetrating through the cuticle and into the insect, where the fungus proliferates (Ferron, Hurpin & Robert 1972; Robert & Deotte 1975; Latch & Falloon 1976). Because of their protected, moist habitat, which favours the persistence of spores, it is the larval stage which is most frequently infected. The LD_{50} of third instar larvae has been estimated at 416 fungal spores, and infected larvae survive an average of about 3 weeks (Sundara-Babu, Balasubramanian & Jayaraj 1983). After succumbing to the fungus, dead larvae create local pockets of infective spores which may be picked up by other larvae or egg-laying female adults moving through logs. However, unlike the case of the virus, infected adults only release the fungus for transmission after death. The dispersal of *Metarhizium* is thus dependent upon the movement of adults shortly after infection.

Past attempts at biological control with *Metarhizium* have met with limited success or, more often, failure (Young 1986). This has been attributed to the low transmission rates of the fungus in populations of the beetle (C. Prior, pers. comm.). Recently however, increased attention has been paid to the use of *Metarhizium* as an inundative biological control agent against *Oryctes* (e.g. Beichle 1980; Sundara-Babu *et al.* 1983; Pillai 1987).

The effects of *Metarhizium* on the populations of *Oryctes* are considered by modelling it as a density-independent mortality factor, or a sort of 'biopesticide'. This assumption is justified on the grounds that in the initial months following inundation, infections would occur primarily through contact with the artificially applied spores; comparatively few infections are likely to arise by contacts between susceptible beetles and inoculum produced by fungus-killed beetles. Unfortunately, little is known about the density-dependent relationship between *Metarhizium* infection and host density. Future experimental (and modelling) studies should consider the extent to which the fungus is transmitted between hosts.

In the numerical simulations presented here, the fungus is assumed to be introduced in large quantities into breeding sites 2 years after the baculovirus has been introduced, and is assumed to remain completely active there for at least 2 years (Latch & Falloon 1976). Since the fungus is assumed not to be transmitted in a density-dependent manner (i.e. transmission is not a function of host density) only beetles in breeding sites, the juveniles and breeding adults, are assumed to be susceptible. For simplicity, juveniles and breeders, healthy and infected alike, are assumed to be equally susceptible to the disease.

Figures 6 and 7 illustrate the effect of increasing inundation rates of *Metarhizium* on the depression of the beetles afforded by the baculovirus alone (Fig. 6a,c) and on the prevalence of the baculovirus (Fig. 6b). Clearly, increasing inundation rates tend to lower both the q-values (indicating greater depression of the host population) and the prevalence of the baculovirus (Fig. 7). If the rate of inundation is sufficiently
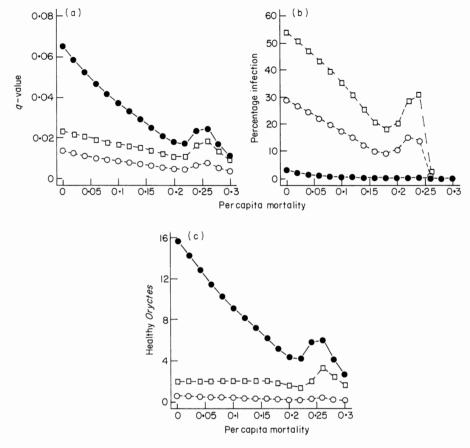
FIG. 6. Biopesticide effects on juveniles (●), feeders (□) and breeders (○) of the inundation of breeding sites by *Metarhizium anisopliae*. Effects on (a) *q*-values, (b) percentage infection by the baculovirus, and (c) the density of healthy *Oryctes rhinoceros*. The initial population densities were set at the values recorded at the end of the simulation presented in Fig. 2 (i.e. the *Oryctes*−baculovirus equilibrium). The fungus was then released, imposing a constant per capita mortality for a period of 2 years (tabulated on horizontal axis). This 'blip' that occurs at mortality rates of 0·20−0·25 corresponds to oscillations (and hence less settled populations after 2 years) induced by the approaching extinction of the baculovirus. If the simulations had been run for sufficient time, then the blips would have disappeared.

high, such that the mortality imposed by the fungus (in isolation from other density-independent mortality factors) results in 30% mortality per 10 days, the baculovirus is excluded from the system (corresponding to the density of hosts falling below the invasion threshold of the baculovirus) (Fig. 7e,f). Further increases in the inundation rate of the fungus results in ever-smaller *q*-values.

DISCUSSION

The interactions between *Oryctes* and its baculovirus pose some particularly difficult problems for practical investigation. The broad dispersion and relative inaccessibility of the host stages make difficult the accurate determination of beetle population parameters. This further complicates the sampling of beetles to determine
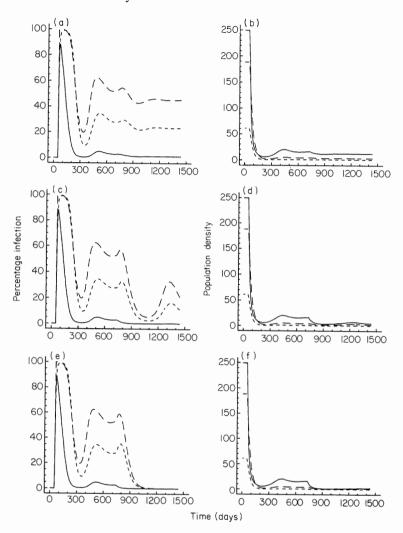
FIG. 7. The consequences for juveniles (—), feeders (— —) and breeders (— — —) of three different levels of inundation of *Metarhizium anisopliae* on the prevalence (a, c, e) and population densities (b, d, f) of the various life-stages of *Oryctes rhinoceros*. (a,b) Mortality rate of 0·10 per 10 days; (c,d) mortality rate of 0·20 per 10 days; (e,f) mortality rate of 0·30 per 10 days. The baculovirus is introduced 50 days into the simulation. Inundation of *Metarhizium* begins 2 years after the start of the simulation (on day 730).

baculovirus distribution and transmission pathways and rates. In this context, population models, based on available data, can help to suggest possible relationships which cannot be easily measured, and to focus limited resources on the most profitable areas for further research.

In the case of the present model, a consideration of the possible pathways of the virus transmission in relation to known levels of baculovirus prevalence has led to the prediction of the feeder–feeder pathway as the primary one in the system. This supports conjecture based on field experience and strengthens this hypothesis in

the absence of measured transmission rates. It also suggests that this transmission pathway is the most deserving for future research.

On a more practical level, the model predicts short-term trajectories for beetle numbers and baculovirus prevalence which may aid practitioners in interpreting samples after releases. Thus, changes in prevalence of infection, achieved as equilibrium is approached (Fig. 2a), might not be seen as indicative of a biological control success without the aid of such understanding.

Oryctes is not always effectively controlled by the baculovirus. Outbreaks sometimes occur after tropical storms and have been associated with the creation of more larval breeding sites in damaged and felled palms (e.g. Zelazny & Alfiler 1986). This, in turn, has led to the empirical prediction that the virus is not effective above a particular density of dead palms (Zelazny & Alfiler 1986). In the context of the model, it is clear to see how a reduction in larval density dependence as a result of an increase in larval resources could lead to a higher beetle population. However, the model also predicts that this increase is of limited duration and, in the absence of any change in pathogen transmission, populations of damaging feeder stages should return to pre-storm levels after a period of predictable duration. If this prediction is borne out, it may prove valuable in long-term management strategies for *Oryctes*.

In the shorter term, chemical pesticides and more recently the fungus *Metarhizium anisopliae* have been investigated for the control of the juvenile stage. Inclusion of this kind of control into the model as a simple density-independent juvenile mortality reveals important potential interactions with the contribution of the baculovirus. First, because of the strong density dependence of mortality caused by the baculovirus, a very large proportion of the juvenile population can be killed by *Metarhizium* before there is any observable effect on the damaging feeder population. Secondly, increases in the use of *Metarhizium* decrease the contribution of the virus to mortality until transmission breaks down and the virus is lost from the system. At best the expensive application of a biopesticide only replaces the free contribution of the baculovirus. At worst, at high levels of biopesticide use, baculovirus transmission breaks down and the virus is lost from the system. In the absence of regular biopesticide application, the *Oryctes* population would resurge to outbreak levels limited by larval competition.

It is not suggested that these model predictions will be of direct use in decision-making in specific contexts, but they do indicate a need for caution in programmes to control the juvenile stage of *Oryctes* and they identify parameters which should be measured in such programmes (see Table 1). The interference of a density-independent mortality in a pest life system with an important density dependence is a general prediction of a number of recent population models directed at pest management problems (e.g. Barclay & van den Driessche 1977; Carpenter 1981; Barclay 1982; Waage, Hassell & Godfray 1985).

The model could be suitably modified to incorporate a number of other important processes. These include spatial structure (e.g. adult dispersal, see Onstad *et al.* 1990), the development of resistance to the baculovirus (Zelazny, Alfiler & Lolong 1989), temperature-dependent development rates, environmental stochasticity and catastrophies (e.g. monsoons), and explicit account of breeding sites and feeding sites. Furthermore, *Metarhizium* could be modelled as a density-dependent mortality factor, rather than the biopesticide assumed here.

ACKNOWLEDGMENTS

REFERENCES

Anderson, R.M. (1982). Theoretical basis for the use of pathogens as biological control agents of pest species. *Parasitology*, **84**, 3–33.

Anderson, R.M. & May, R.M. (1981). The population dynamics of microparasites and their invertebrate hosts. *Philosophical Transactions of the Royal Society, London, B*, **291**, 451–524.

Barclay, H.J. (1982). Models for pest control using predator release, habitat management and pesticide release in combination. *Journal of Applied Ecology*, **19**, 337–348.

Barclay, H.J. & van den Driessche, P. (1977). Predator–prey models with added mortality. *Canadian Entomologist*, **109**, 763–768.

Beddington, J.R., Free, C.A. & Lawton, J.H. (1978). Characteristics of successful natural enemies in models of biological control of insect pests. *Nature*, **273**, 513–519.

Bedford, G.O. (1976). Use of a virus against the coconut palm rhinoceros beetle in Fiji. *PANS*, **22**, 11–25.

Bedford, G.O. (1980). Biology, ecology and control of palm rhinoceros beetles. *Annual Review of Entomology*, **25**, 309–339.

Bedford, G.O. (1986). Biological control of the rhinoceros beetle (*Oryctes rhinoceros*) in the South Pacific by baculovirus. *Agricultural Ecosystems and Environments*, **15**, 141–147.

Beichle, U. (1980). Rhinoceros beetle control in Western Samoa. *Alafua Agricultural Bulletin*, **5**, 52–54.

Carpenter, S.R. (1981). Effect of control measures on pest populations subject to regulation by parasites and pathogens. *Journal of Theoretical Biology*, **32**, 181–184.

Catley, A. (1969). The coconut rhinoceros beetle *Oryctes rhinoceros* (L.) *PANS*, **15**, 18–30.

Entwistle, P.F., Adams, P.H.W., Evans, H.F. & Rivers, C.F. (1983). Epizootiology of a nuclear polyhedrosis virus (Baculoviridae) in European spruce sawfly (*Gilpinia hercyniae*): spread of disease from small epicentres in comparison with spread of baculovirus diseases in other hosts. *Journal of Applied Ecology*, **20**, 473–487.

Ferron, P., Hurpin, B. & Robert, P.H. (1972). Sur la specificité de *Metarhizium anisopliae* (Metsch.) Sorokin. *Entomophaga*, **17**, 165–175.

Ferron, P., Robert, P.H. & Deotte, A. (1975). Susceptibility of *Oryctes rhinoceros* adults to *Metarhizium anisopliae*. *Journal of Invertebrate Pathology*, **25**, 313–319.

Gorick. B.D. (1980). Release and establishment of the baculovirus disease of *Oryctes rhinoceros* (L.) (Coleoptera: Scarabaeidae) in Papua New Guinea. *Bulletin of Entomological Research*, **70**, 445–453.

Hammes, C. (1978). Estimation de l'efficacité du control exercé par *Rhabdionvirus oryctes* (Huger) sur *Oryctes rhinoceros* (L.) par l'étude des variations de dégats sur les cocotiers de l'ile Maurice. *Revue Agricole et Sucrière de l'Ile Maurice*, **57**, 4–18.

Hochberg, M.E. (1989). The potential role of pathogens in biological control. *Nature*, **337**, 272–275.

Huger, A.M. (1973). Grundlagen zur biologischen Bekämpfung des Indischen Nashornkäfers *Oryctes rhinoceros* (L.) mit *Rhabdionvirus oryctes*: Histopathologie der Virose bei Käfern. *Zeitschrift für Angewandte Entomologie*, **72**, 309–319.

Hurpin, B. & Fresneau, M. (1973). Etude en laboratoire des facteurs de fécondité de *Oryctes monoceros* Ol. and *Oryctes rhinoceros* L. *Annales de la Société Entomologique de France*, **9**, 89–117.

Kapadia, M.N. & Valia, R.Z. (1986). A note on the biology of *Oryctes rhinoceros* L. as a pest of coconut palm. *Gujarat Agricultural University Research Journal*, **12**, 53–55.

Latch, G.C.M. & Falloon, R.E. (1976). Studies on the use of *Metarhizium anisopliae* to control *Oryctes rhinoceros*. *Entomophaga*, **21**, 39–48.

Marschall, K.J. (1970). Introduction of a new virus disease of the coconut rhinoceros beetle in Western Samoa. *Nature*, **225**, 288–289.

Marschall, K.J. & Ioane, I. (1982). The effect of re-release of *Oryctes rhinoceros* baculovirus in the biological control of rhinoceros beetles in Western Samoa. *Journal of Invertebrate Pathology*, **39**, 267–276.

Mawikere, J., Lolong, A.A., Sabbatoellah, S., Crawford, A.M., Zelazny, B., Maliangkay, R.B. & Mangindaan, H.F. (1989). Control of *Oryctes* during replanting. *Integrated Coconut Pest Control Project, Annual Report 1989*, pp.122–125. Coconut Research Institute, Manado, North Sulawesi, Indonesia.

Mohan, K.S., Jayapal, S.P. & Pillai, C.B. (1985). Response of *Oryctes rhinoceros* larvae to infection by *Oryctes* baculovirus. *Journal of Plantation Crops*, **13** 116–124.

Mohan, K.S., Jayapal, S.P. & Pillai, C.B. (1986). Diagnosis of baculovirus infection in coconut rhinoceros beetles by examination of excreta. *Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz*, **93**, 379–383.

Monserrat, P. & Veyrunes, J.C. (1976). Evidence of *Oryctes* virus in adult faeces and new data for virus characterization. *Journal of Invertebrate Pathology*, **27**, 387–389.

Monty, J. (1974). Teratological effects of the virus *Rhabdionvirus orgyctes* on the coconut palm rhinoceros beetle *Oryctes rhinoceros* (L.). *Bulletin of Entomological Research*, **64**, 633–636.

Monty, J. (1978). The coconut palm rhinoceros beetle, *Oryctes rhinoceros* L., in Mauritius and its control. *Revue Agricole et Sucrière de l'Ile Maurice*, 57: 60–76.

Onstad, D.W., Maddox, J.V., Cox, D.J. & Kornkven, E.A. (1990). Spatial and temporal dynamics of animals and the host-density threshold in epizootiology. *Journal of Invertebrate Pathology*, 55, 76–84.

Peterson, G.D. (1977). *Research on the control of the coconut palm rhinoceros beetle. Fiji, Tonga, Western Samoa. Review of project results.* UNDP/FAO, Rome, 109pp.

Pillai, C.B. (1987). Integrated IPM in plantation crops. *Journal of Coffee Research*, **17**, 150–153.

Schipper, C.M. (1976) Mass rearing the coconut rhinoceros beetle, *Oryctes rhinoceros* L. (Scarab., Dynastinae). *Zeitschrift für Angewandte Entomologie*, **81**, 21–25.

Sundara-Babu, P.C., Balasubramanian, M. & Jayaraj, S. (1983). Studies on the pathogenicity of *Metarhizium anisopliae* (Metschnikoff) Sorokin var. *major* Tulloch in *Oryctes rhinoceros* L. *Research Publication of the Tamil Nadu Agricultural University*, **1**.

Swan, D.I. (1974). A review of the work on predators, parasites and pathogens for the control of *Oryctes rhinoceros* (L.) in the Pacific area. *Commonwealth Institute Biological Control Miscellaneous Publication No. 7.*

Waage, J.K., Hassell, M.P. & Godfray, H.C.J. (1985). The dynamics of pest–parasitoid–insecticide interactions. *Journal of Applied Ecology*, **22**, 825–838.

Young, E.C. (1974). The epizootiology of two pathogens of the coconut rhinoceros beetle. *Journal of Invertebrate Pathology*, **24**, 82–92.

Young, E.C. (1986). The rhinoceros beetle project: history and review of the research programs. *Agricultural Ecosystems and Environments*, **15**, 149–166.

Young, E.C. & Longworth, J.F. (1981). The epizootiology of the baculovirus of the coconut palm rhinoceros beetle (*Oryctes rhinoceros*) in Tonga. *Journal of Invertebrate Pathology*, **38**, 362–369.

Zelazny, B. (1972). Studies on *Rhabidionvirus oryctes*. I. Effects on larvae of *Oryctes rhinoceros* and inactivation of virus. *Journal of Invertebrate Pathology*, **20**, 235–241.

Zelazny, B. (1973a). Studies on *Rhabdionvirus oryctes*. II. Effect on adults of *Oryctes rhinoceros*. *Journal of Invertebrate Pathology*, **22**, 122–126.

Zelazny, B. (1973b). Studies on *Rhabdionvirus oryctes*. III. Incidence in the *Oryctes rhinoceros* population in Western Samoa. *Journal of Invertebrate Pathology*, **22**, 359–363.

Zelazny, B. (1974). *UNDP/FAO/SPC Rhinoceros Beetle Project Technical Document No. 108.* 4pp.

Zelazny, B. (1976). Transmission of a baculovirus in populations of *Oryctes rhinoceros*. *Journal of Invertebrate Pathology*, **27**, 221–227.

Zelazny, B. (1977). *Oryctes rhinoceros* populations and behaviour influenced by a baculovirus. *Journal of Invertebrate Pathology*, **29**, 210–215.

Zelazny, B. & Alfiler, A. (1986). Oryctes rhinoceros (Coleoptera: Scarabaeidae) larval abundance and mortality factors in the Philippines. *Environmental Entomology*, **15**, 84–87.

Zelazny, B. & Alfiler, A.R. (1987). Ecological methods for adult populations of *Oryctes rhinoceros* (Coleoptera: Scarabaeidae). *Ecological Entomology*, **12**, 227–238.

Zelazny, B., Alfiler, A.R. & Lolong, A. (1989). Possibility of resistance to a baculovirus in populations of the coconut rhinoceros beetle (*Oryctes rhinoceros*). *FAO Plant Protection Bulletin*, **37**, 77–82.

Zelazny, B. & Lolong, A. (1988). *Oryctes rhinoceros* survey in the Maldives. *Integrated Coconut Pest Control Project, Annual Report 1988.* pp.142–184. Coconut Research Institute, Manado, North Sulawesi, Indonesia.

Zelazny, B. & Pattang, B. (1987). *Oryctes* survey in South Sulawesi. *Integrated Coconut Pest Control Project, Annual Report 1987.* pp.18–30. Coconut Research Institute, Manado, North Sulawesi, Indonesia.