Chapter 8
The Use of *Oryctes* Virus for Control of Rhinoceros Beetle in the Pacific Islands

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Abstract The rhinoceros beetle (*Oryctes rhinoceros*) was accidentally introduced into Samoa in 1909 from where it spread to many islands in the south-west Pacific. A novel virus pathogenic to the beetle, originally designated as *Rhabdionvirus oryctes* and later *Oryctes* virus, was isolated from Malaysia and introduced into Samoa in 1963. Later releases took place in Tonga, Fiji and other Pacific Islands. The virus rapidly established and caused high levels of infection that spread as epizootics through the beetle populations. The virus killed larvae in breeding sites and caused adult beetles to cease feeding, leading to reduced damage and a decline in fecundity of the pest population. Researchers reported spectacular declines in the treated populations within 1–3 years of application. Reapplication has proven effective where there has been a resurgence of beetle damage. After 40 years from the initial releases, high palm damage has been reported from some areas suggesting a breakdown of control. Selection of more virulent strains and improved methods of application could overcome these problems.

8.1 Introduction

The rhinoceros beetle *Oryctes rhinoceros* (Scarabaeidae; Dynastinae) is a large horned beetle endemic to the Asia/West Pacific region. Its biology has been reviewed by Catley (1969) and Bedford (1980), and quantitative ecological data was reviewed and incorporated into a model by Hochberg & Waage (1991). The adult beetles feed and aggregate in the crowns of palm trees (Fig. 8.1A). The beetles enter the leaf axil and feed by boring into the unfurled tissue at the meristem of the palm. Male beetles produce an aggregation pheromone, attractive to both males and females, which results in a patchy distribution of the beetles within a stand of palms. Beetle presence is evidenced through notching, fanning and breaking of the emergent palm fronds (Fig. 8.1B). In cases of high beetle feeding pressure, the

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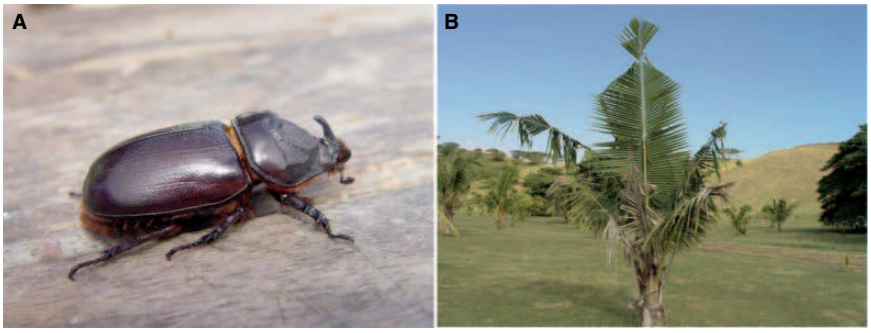

Fig. 8.1 **A**. Adult rhinoceros beetle. **B**. Young coconut palm with extensive leaf damage caused by adult rhinoceros beetle feeding (Photos courtesy of Sada Nand Lal)

growing tip of the palm will be killed leaving a dead standing stump. After mating, female beetles will fly to accumulations of dead organic matter, heaps of fronds, fallen logs and even standing dead palms, for oviposition, laying approximately 60 eggs into the decaying organic matter. The first instar larva will emerge from the egg after two weeks and begin to feed on the surrounding organic matter. Development of the three larval instars will take approximately 6 months in tropical conditions before transformation into the pupal stage, which lasts for about 1 month prior to emergence of the neonate adult. The adult beetles can live for about 6 months and are strong fliers over relatively short distances. A sibling pest species, *Oryctes monoceros*, is known from Africa and Islands of the Indian Ocean and the massive *Scapanes australis* occupies a similar niche damaging coconut palms in Asia and Papua New Guinea. Within its endemic zone the rhinoceros beetle can be a pest of coconut palm (*Cocos nucifera*) and oil palm (*Elaeis guineensis*), especially when there is an abundance of decaying organic matter suitable for breeding. Young palms on replant sites are particularly susceptible.

8.2 Rhinoceros Beetle in the Pacific

In the early part of the 20th century the major economic activity for most Pacific Islands was production of copra from coconuts in natural or managed plantations. Rhinoceros beetle is believed to have entered the Pacific concealed in rubber plant seedlings from Ceylon in 1909 (Catley 1969). The insect established rapidly in Samoa and subsequently spread to Tonga (1921) (since successfully eradicated); Wallis Island (1931); Palau, New Britain and West New Guinea (1942); Vavua, Tonga and New Ireland (1952); Pak Island and Manus Island, New Guinea (1960); Tongatapu, Tonga (1961) and the Tokelau Islands (1963). The beetle was first reported from Fiji in 1953 and, despite intensive attempts to implement quarantine procedures, had spread through most of the Fiji Island group by 1971 (Bedford 1976). Initial outbreaks of the pest were devastating; on Palau 50% of palms were killed within 10

years of *Oryctes* introduction (Gressit 1953). Establishment of quarantine systems, coupled with a reduction in beetle numbers through control measures, appears to have reduced the rate of spread of the insect with few new outbreaks reported since 1970. However, recent reports indicate that the beetle has been discovered on Guam and damage has been reported from Raratonga (SN Lal personal communication).

8.3 The Search for Controls

Damage by rhinoceros beetle, the effect on villagers' livelihoods and the threat to economic viability of the island communities, prompted regional bodies to investigate possible solutions. Initial responses included cultural control through destruction of breeding sites and treating palms with chemical pesticides, which had limited success (Catley 1969). Biological control was attempted and gained momentum with the initiation of the UNDP/SPC Project for Research on the Control of the Coconut Rhinoceros Beetle in 1965 (Young 1986), resulting in the release of a large number of natural enemies of rhinoceros beetle into the Pacific Islands. Most releases of parasites and predators had little success (Waterhouse & Norris 1987) but the search for pathogens in the region of origin of the pest was to prove more successful. Dr Alois Huger of the Institute for Biological Control, Darmstadt, Germany, was contracted to search for pathogens of the beetle by UNDP/SPC (Huger 2005). He concentrated his survey studies on the Malay peninsular working closely with staff of the oil palm plantations where large numbers of beetles were found in decaying logs in the replanting areas. Unusual larvae were identified displaying a lethargic, translucent condition indicative of dissolution of the fat body. Feeding macerates of putative diseased larvae to healthy larvae from a laboratory colony resulted in similar symptoms and strong indications of an infective agent, which was revealed as a virus by histological and electron microscopy studies.

8.4 The *Oryctes* Virus

The *Oryctes* virus is a non-occluded dsDNA virus that was first described as *Rhabdionvirus oryctes* (Huger 1966) and later defined as *Oryctes* virus, the type species of Subgroup C of the Baculoviridae by the International Committee on Taxonomy of Viruses (ICTV). It has recently been suggested that *Oryctes* virus be incorporated into a new virus genus and designated as *Oryctes rhinoceros* nudivirus (OrNV) (Wang *et al*. 2007). Characteristics of the virus and pathology of the infected beetles are described by Huger (1966, 2005) and Huger & Krieg (1991). Following ingestion by larvae, the virus will invade the midgut epithelium and migrate into other tissues. The abdomen becomes turgid and glassy and internal turgor may increase to cause prolapse of the rectum. The virus also infects the adult beetle with initial infection of the midgut epithelial cells causing a proliferation of cells, swelling of the gut and release of infected nuclei into the gut lumen. Infected adults will defecate

large amounts of virus during the early stages of infection and contaminate their surrounding habitat. Infection leads to cessation of feeding, reduces the fecundity of females and decreases the life span of infected beetles (Zelazny 1973a).

8.4.1 Release of the Virus for Rhinoceros Beetle Control

The first field releases of the virus were made in 1967 in Samoa. Infected rhinoceros beetle larvae were shipped from the BBA laboratory, Darmstadt, to Samoa for multiplication of the virus by feeding healthy larvae with rotting sawdust contaminated by macerates of diseased larvae (Huger 1972, 2005). Dead infected larvae were distributed and applied to breeding sites on the Samoan islands of Manono and Savaii in March and April 1967. By October 1968, infected larvae were recovered from the field and subsequently virus infection was found to be widespread throughout the Samoan islands, even on Upolu where no releases had taken place (Marschall 1970). In the site of original release on Manono, Marschall (1970) reported "the beetle had almost disappeared" and noted a conspicuous decrease in damage in areas where the virus was well established.

Following the success of the initial virus releases in Samoa, further releases were made on other rhinoceros beetle infested islands of the Pacific (Bedford 1981). *Oryctes* virus was introduced into Fiji from Samoa in 1970 and released on multiple sites by capturing healthy adult beetles, infecting them in the laboratory and

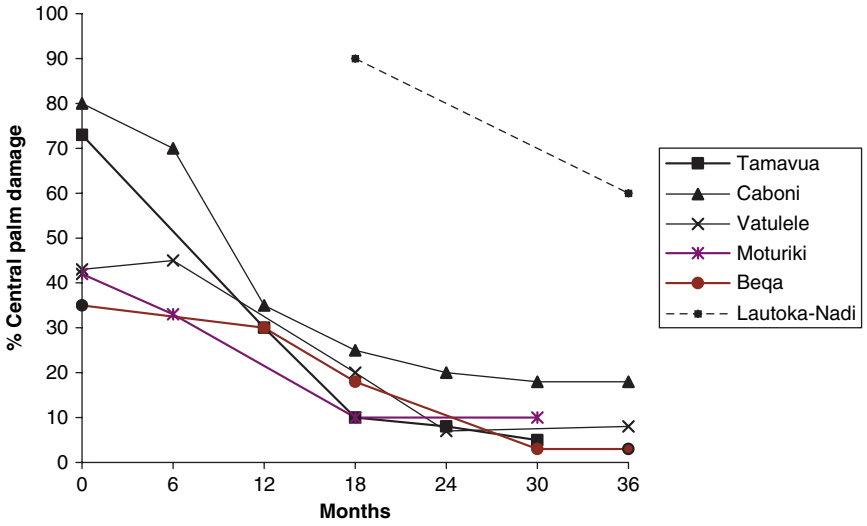

Fig. 8.2 Reduction in palm damage recorded over 36 months after release of *Oryctes* virus on five sites in the Fiji Islands from 1970–1972. No virus was released in the Lautoka area where damage remained high 18 months after the start of the programme but natural incidence of disease was recorded in the area after 36 months coinciding with a decline in visible damage (adapted from Bedford 1981)

releasing them into the field at selected sites (Bedford 1976). Between 1970 and 1974 several thousand infected beetles were released to badly infested areas and impact monitored by disease spread and estimates of damage to the palms. Several untreated sites were also monitored to provide controls for impact assessment. The results were dramatic, with 50–70% of trapped beetles infected and palm damage, which ranged from 35–85% before treatment, dropping to less that 10% on most sites within 3 years of release (Fig. 8.2). Dramatic photographic evidence of the recovery of palms post treatment is provided by Bedford (1976, 1981).

On Tongatapu, Tonga, virus was released by pouring macerate of larvae infected in Samoa onto heaps of rotting sawdust artificially infested with beetle larvae. More than 50 breeding heaps were treated on the western end of the island in 1970 and the resulting epizootic was monitored and estimated to have spread across the island at a rate of 3 km/month (Young 1974) with a peak of 40% larvae infected at the height of the epizootic. At the release site, number of palms with central crown damage dropped from 28 to 5%, 15 months from treatment.

The virus has also been released on other Pacific Islands including Tokelau (1967), Palau (1970, 1983), Wallis Island (1970–1971), American Samoa (1972) and Papua New Guinea (1978–1979) (Hajek *et al*. 2005) as well as releases within the Maldives and India. The virus has also been released for control of *Oryctes monoceros* in the Seychelles and Tanzania (Hajek *et al*. 2005).

8.4.2 Effect of Multiple Releases of Virus

Young (1974) indicated that disease incidence fell behind the "front" of the epizootic, which would be expected for the normal delayed-density-dependent activity of a pathogen following pest density reduction (Barlow 1999). In Samoa, Marschall & Ioane (1982) noted that after the spectacular decrease associated with the first release of the virus, beetles were increasing in number and palm damage was once again increasing, especially in areas where there was bush or plantation clearing and a high number of fresh breeding sites. A reinfection programme was established and monitored by trapping adults with the attractant ethyl chrysanthemumate (Maddison *et al*. 1973). Releases were made at several sites and beetle populations and disease monitored for up to 3 years with the authors concluding that damage could be reduced through implementation of this process. Increased levels of infection and reduced palm damage have been reported in a number of areas following regular release of virus including the Philippines (Alfiler 1992), Malaysia (Tuck 1996; Ramle *et al*. 2005) and India (Babjan *et al*. 1995).

8.4.3 Assessment Methods

While introduction of the virus into a new outbreak area where the rhinoceros beetle population is free of the disease is relatively simple to manage, ongoing

management through repeated introductions will be more complex and depend on some simple decision rules. The decision to re-release is generally made on the basis of need – when beetle numbers seem to be on the rise. However, a rational programme would be based on absence or low incidence of the virus from the beetle population. Diagnosis of disease and determination of disease incidence is not always simple. Several methods have been developed for diagnosis of *Oryctes* virus and Zelazny (1978) reports on visual diagnosis, bioassay and gut content smear and microscopic examination as alternative methods for assessment. He concluded that direct visual diagnosis was the easiest method of diagnosis, but was dependent on the experience of the observer. To develop a consistent method for monitoring the virus, Richards *et al*. (1999) designed a PCR primer for the virus and a monitoring system that has been tested in Malaysia and Samoa. While this method has proven effective in monitoring virus spread (Ramle *et al*. 2005), sensitivity of PCR means that care must be taken in sample collection and preparation to avoid cross contamination and overestimation of disease.

8.5 Discussion

The introduction of *Oryctes* virus into the Pacific Islands has been a major success in reducing extreme outbreak populations of rhinoceros beetle and resolving the critical problem of beetle damage to palms. The success of the virus can be attributed to its biology and mode of pathogenicity. As observed by Huger (1972) infected beetles act as "virus reservoirs" releasing large quantities of virus into the beetle habitat. Beetles aggregate at feeding and breeding sites, behaviour encouraged by the male attractant pheromone (Hallett *et al*. 1995). Not surprisingly, mature beetles caught in traps show very high levels of infection as the aggregation behaviour of the insects ensures cross contamination. Levels of infection are higher among mature beetles than neonates or larvae (Ramle *et al*. 2005) suggesting that the virus is predominantly horizontally transmitted between the adults. The virus is successful in protecting the palms by bringing about cessation of feeding and a reduction in egg laying rate by females, leading to reduced damage and population decline. The pathobiology of *Oryctes* virus also reveals its weakness as a plant protection agent and suggests that with a high density of emergent adults, the reduced level feeding will not be sufficient to prevent palm damage, especially to young establishing trees. Damage, even in the presence of virus in the population, can be observed where there is a high density of organic matter for beetle breeding such as after old palm clearing for replanting, hurricane damage or in the areas near to sawmills.

Release of virus into fresh healthy outbreak populations will remain an essential tool in rhinoceros beetle management but the question remains whether the system could be improved (Jackson *et al*. 2005). Crawford & Zelazny (1990) showed that the virus evolved rapidly after release in the Maldives and data have been collected from Malaysia (Ramle *et al*. 2005) suggesting that some of the natural isolates are only weakly virulent. Success of reintroduction programmes in Samoa

and the Philippines suggests that there may be a benefit from release of highly virulent strains even where the virus is already established. Zelazny (1973b) showed that highly virulent strains can cause cessation of feeding within a few days with a consequent reduction in damage and fecundity in the population. Virus delivery can also be improved by development of stable formulations that could be combined with attractants for a lure-and-infect strategy. A combination of new strains and new delivery systems could enhance the performance of *Oryctes* virus and provide an excellent new technology for coconut palm protection in the Pacific.

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