26 Parasites and pest management[1]

Herwig Leirs and Grant R. Singleton

1 Introductory remarks: Small mammals, parasites and pest control

A number of small mammals are considered pests because they cause damage to humans either directly (e.g., rat bites, vampire bat blood meals) or more often indirectly by transmitting pathogens to humans and livestock, causing losses in field crops or stored harvest produce and processed food, or damaging infrastructure and natural and cultural assets that are deemed valuable by humans. Rodents, the most abundant group of small mammals, with some species thriving very well in the human environment, are common among these pests. Fruit bats can cause considerable damage in tropical fruit orchards, whereas insectivorous bats act as reservoirs for several viruses. The European rabbit is the major vertebrate pest in Australia and high on the list in New Zealand as well, only surpassed by the possum. Some small mammals are usually not considered pests but this may depend on local conditions, whereas some others are a nuisance or carry pathogens.

Pest management, micromammals and macroparasites are linked in various ways. First, in many cases, it is simply its role as a host for zoonotic macroparasites that gives a mammal species pest status. The ultimate goal of pest population management is then to control the macroparasite rather than the host per se. Second, there have also been attempts to use macroparasites as a form of biological control of the host species. Third, pest population management usually affects the density or population turnover of the small mammal species, and this may have significant interacting effects on the parasite dynamics. Fourth, changes in landscape use by humans may lead to changes in rodent population densities and consequent

increase of risk in zoonoses. Also, landscape changes may simplify habitats and lead to zoonoses though the accidental ingestion of invertebrates that are the intermediate hosts of many macroparasites of small mammal species that previously were not considered a pest by humans (e.g., Spratt 2005). Fifth, invasive mammal species in some instances have led to an extension of the host spectrum of macroparasites. Such events have led to conservation (e.g., black-footed ferret) and human health concerns (e.g., sylvatic plague in USA).

In this chapter, we will present these different relationships, consider their biological background, and discuss how they affect pest management approaches. We start with a brief introduction of the pest problems and management strategies.

2 Parasites and the pest status of small mammals

2.1 Parasites make small mammals a pest

A number of small mammals are considered pests, exactly because they are carrying parasites that can infect human beings or livestock. *Rattus norvegicus* is a notorious pest in and around agricultural buildings because of the damage it can cause to stored food and infrastructure. On slaughter pig farms it has an especially high pest status because it can carry *Trichinella spiralis* nematodes (Kapel 2000; Meerburg et al. 2004). When pigs consume infected rats that they find on the farm, they may become infected themselves, which constitutes a health risk for meat consumers and may cause a huge economical problem for the farm owner or even a whole country's pork meat export. *Arvicola terrestris* is considered a pest in some parts of Europe in grasslands or orchards, but is also of concern since it is an intermediate host for *Echinococcus multilocularis* (Saucy 1994; Schmitt et al. 1997). The table at the end of this chapter (see Appendix) provides an overview of the most important macroparasites that are pathogenic for humans or livestock and for which small mammals are reservoirs. In addition, small mammals are used as reservoirs by numerous microparasites. The diversity of these microparasites is huge, including rabies-like lyssa-viruses, SARS-like coronaviruses and henipaviruses in bats (Hoar et al. 1998; Fooks et al. 2003; Mackenzie and Field 2004; Li et al. 2005); Ebola and Marburg filoviruses in hitherto unknown small mammals (bats seem to be likely candidates, Leroy et al. 2005); hantaviruses (McCaughey and Hart 2000), bacteria (e.g., causing leptospirosis, rat-bite

fever, plague), protozoans (e.g., *Toxoplasma gondii*) in rodents. A full treatment of all of these microparasitic zoonoses falls beyond the scope of this book, but we will nevertheless return to some of them since they are carried by arthropod vectors that feed on small mammals as well as on humans or livestock. Bubonic plague, caused by the bacterium *Yersinia pestis*, is basically a flea-vectored infection in rodents, but humans can also become infected through bites from infected rodent fleas (Gage and Kosoy 2005). The discovery of the bacterium-rat-flea relation in the late 19th century became one of the most important driving forces behind organised and enforced rodent control in urban settings and ship transport throughout the world. Even today, plague is still epithetical for any rodent borne disease. Moreover, fleas, ticks and mites also transmit between small mammals and humans a variety of other microorganisms that may cause serious disease such as the bacterium *Borrelia burgdorferi* (Lyme disease), several rickettsias (causing typhus fevers, spotted fevers, scrub fever, ehrlichiosis) or viruses (e.g., several tick-borne encephalites) (Gratz 1988; Spratt 2005).

2.2 Parasite release makes small mammals a pest

In theory, parasites can limit populations of small mammals to a certain degree (see also below and other chapters in this book). Populations of invasive species usually have only part of their native parasite (or predator) fauna, and this sometimes results in the host population reaching higher abundance or biomass. Thus, this release from the normal parasite pressure may lead such populations to become pests, while in their native area they do not have such characteristics. This has been well documented for a number of invasive plants or invertebrates (Torchin et al. 2003; Torchin and Mitchell 2004). In small mammals, invasive populations, especially on islands, are known to have a poor parasite fauna in terms of both abundance and diversity (Göuy de Bellocq et al. 2002; Milazzo et al. 2003; Pisanu et al. 2001). However, to the best of our knowledge, no examples of this being the main reason for the development of pest populations of mammals that elsewhere are harmless, are reported in the literature. Nevertheless, it is worth to consider always whether the parasite release contributes to the pest status of such populations. In fact, it has been suggested several times that neutralising the parasite release would help significantly to control invasive species.

2.3 Invasive species and landscape changes – increasing the management complexity

The introduction of new mammal species into an ecosystem either through successful dispersal and colonization (e.g., the global spread of commensal rodents such as *Rattus norvegicus, Rattus rattus, Mus musculus*) or through intentional releases (e.g., *Oryctolagus cuniculus* and *Vulpes vulpes* in Australia) has led to the range expansion of macro- and micro-parasites. A classic example is the establishment of sylvatic plague in small mammal assemblages in North America in 1908 following the plague pandemic at the turn of the 20th century. The geographic distribution of the disease has been fairly stable over the past 50 years. However, a recent study confirmed that it is distributed more widely than previously thought (Cully et al. 2000). Sylvatic plague in North America causes epizootics in prairie dog populations that lead to extirpation of colonies and is the only disease known to cause high mortality in this species (Barnes 1993). Plague also has important conservation impacts because the prairie dog is an important food resource for the highly endangered black-footed ferret (see Christe et al., this volume, for discussion of conservation impacts of macroparasites).

In Australia, native rodent species have acquired an impressive array of macroparasites from the introduced rodents (e.g., *Angiostrongylus cantonensis, Trichuris muris, Syphacia muris, Hepatojarakus pycnofasciatus, Heterakis spumosa, Mastophorus muris, Nippostrongylus brasiliensis, Strongyloides ratti, Calodium hepaticum, Eucoleus gastricus, Hymenolepis diminuta, Raillietina celebensis, Moniliformis moniliformis)* and domestic animals (e.g., *Fasciola hepatica, Spirometra erinacei* – larval stage, *Taenia taeniaeformis* – larval stage, *Linguatula serrata* – larval stage) (D.M. Spratt, personal communication). However, there is little evidence of native rodent helminths (which represent a rather rich fauna) going in the opposite direction into the introduced *R. rattus, R. norvegicus* and *M. musculus* (except for *Angiostrongylus mackerrasae*).

Landscape changes can have major consequences on the distribution and abundance of rodents and their macroparasites. In the USA, forest destruction and fragmentation reduces mammalian species diversity, with some species such as *Peromyscus leucopus* adapting well to these landscape changes. For example, they show higher population densities in small forest fragments. These population increases of an important host species for Lyme disease, have led to a dramatic increase in the density of infected tick nymphs in small (<2 ha) forest patches (Allan et al. 2003). Therefore, human induced habitat fragmentation can increase the risk of humans contracting a tick-borne disease.

An emerging issue is human-induced changes in the landscape as a result of global warming. The changes in distribution of the reservoirs or intermediate hosts of parasites, may lead to the range extension of zoonotic diseases and an increased prevalence in epizootics. In the past few decades, the expansions of ranges of rodent-borne diseases such as trypanosomiasis, Lyme disease, tick-borne encephalitis and plague have been reported (Lindgren et al. 2000; Harvell et al. 2002). Although the evidence is still being collated, if these trends continue, then there will be important management implications for the small mammal wildlife hosts of these diseases.

3 Ecologically-based rodent management

Pest management in general and small mammal management in particular have for a long time been understood to be equal to the killing of individual animals belonging to the pest species and, if possible, to the extermination of the entire pest populations. This turned out to be generally an unsustainable and, in the medium-to-long term, ineffective approach, especially in open settings such as natural, agricultural or urban environments where the entire extermination is not feasible. In the agricultural insect pest control, this led to the development of Integrated Pest Management (IPM) strategies that attempted to combine a number of different control methods. Rodent management took a longer time to change but, approximately 10 years ago, the concept of the Ecologically-Based Rodent Management (EBRM) approach has been developed (Singleton 1997; Singleton et al. 1999). This approach starts from a thorough understanding of the pest rodents' biology (ecology, physiology, taxonomy) and the damage they cause, and tries to identify ecological factors to which this damage may be most sensitive. Obviously, in many cases, albeit not always, these factors are to be related to rodent abundance, and EBRM will then investigate how to best affect rodent abundance at the moment when it matters to damage. For many species, this will best be done by increasing mortality (e.g., with rodenticides). However, in other species or under other circumstances it may be more effective to affect reproduction or dispersal. For example, population ecological models suggest that the dynamics of *Phyllotis darwini* in Chili is more sensitive to changes in survival, while *Mastomys natalensis* in Tanzania is, at least in some seasons, more affected by changes in reproduction (Lima et al. 2003). A fine example of EBRM is the Trap Barrier System (TBS) used for the control of *Rattus argentiventer* in Indonesia, where the large scale foraging movements are put to use by

luring rats into traps around an early planted crop. This reduces rodent densities over a much larger area than just that single field (Singleton and Sudarmaji 1998). Still, even the TBS is just one from the package of management options developed for *R. argentiventer* based on a solid understanding of its ecology (Singleton et al. 2005).

EBRM does not *a priori* prefer or exclude any technique but it does take into account the more indirect effects on the environment, particularly the whole ecological community in which the rodent population lives. When parasites are involved in some way, such community aspects are important *a fortiori* and an ecologically-based approach is especially appropriate. This requires a very sound understanding of the pest animals' as well as the parasites' population ecology, and this information unfortunately is often still lacking.

4 Parasite control through pest control

4.1 Why control parasite reservoirs?

There are basically two reasons why small mammal population management has its place in the struggle to reduce the burden of parasitic disease (in humans or any other domesticated or wild species that humans would like to protect). The first argument is the intuitive assumption that there is a positive relation between the abundance of reservoirs and the force of infection to humans, i.e. the risk for humans of becoming infected is higher when there are more individuals of the reservoir species, hence reservoir numbers should be lowered. A second argument comes from the theory on the ecology of infections, where it is derived that there is a threshold of host density below which an infection cannot persist. Again, this argues for a reduction in reservoir population numbers. Both arguments are less straightforward than often thought, so they need more consideration.

4.2 Force of infection to humans

The probability that an individual human becomes infected with a parasite is dependent on the probability that he or she comes in contact with an infective stage of the parasite, and that probability is again proportional to the availability of infective stages of the parasite in the environment (and, of course, the human's behaviour). The relationships between the avail-

ability of infective stages and the abundance of small mammal reservoir, however, are not necessarily straightforward. First, it will depend on whether the parasite is transmitted from one host to another via direct contact, a free-living infective stage, an intermediate host or a vector. When intermediate hosts and vectors are involved, their abundance (or even relative abundance in relation to humans) may contribute more to the force of infection than the abundance of the reservoir species. However, even in the case of a directly transmitted infection, the link between reservoir abundance and risk to humans can take different forms. Davis et al. (2005) explored this relation, starting from a simple model for the force of infection to humans λ, $\lambda = \beta N p$, where β is the transmission coefficient (the number of contacts per time a human has with the reservoir, times the probability that contact with an infected reservoir results in transmission of the infection), N is the population size of the reservoir and p is the prevalence of the infection in the reservoir population. At first glance, this relation is simple, but, since prevalence can be related to host abundance, the outcome is more complex (Fig. 1). If prevalence increases with host density, as in most infections with direct horizontal transmission (Grenfell and Dobson 1995; Mills et al. 1999; Hudson et al. 2001), then the force of infection to humans increases proportionally to N^2 (Fig. 1a). Thus, if the reservoir abundance is rising twofold, then the risk to humans rises fourfold. In a number of other infections, like those with frequency-dependent sexual transmission, there is no overt relationship between host abundance and prevalence. In this scenario, the force of infection to humans increases linearly with increasing abundance (Fig. 1b). In both cases, there is a positive relationship between the reservoir abundance and the risk for humans. Therefore, reducing the number of reservoir individuals should decrease the risk for humans.

If there is a threshold host abundance, below which the infection does not persist in the reservoir (e.g., Davis et al. 2004, see also below), then obviously there is also a non-linearity in the relation between abundance and risk to humans (Fig. 1c). In such cases, there is a host population density below which further reduction of this density does not have any effect on the risk to humans. Consequently, controlling the small mammal host below this threshold does not have direct beneficial effects but only makes sense if it contributes to keeping the host abundance low enough.

The last and, at first glance, somewhat counterintuitive scenario is that there is a negative relationship of some sort between population density and prevalence (Fig. 1d). The most obvious example, as also reviewed by Davis et al. (2005) is the "juvenile dilution effect". This effect is due to the recruitment of numerous uninfected young animals that increase the population abundance much faster than they can become infected.
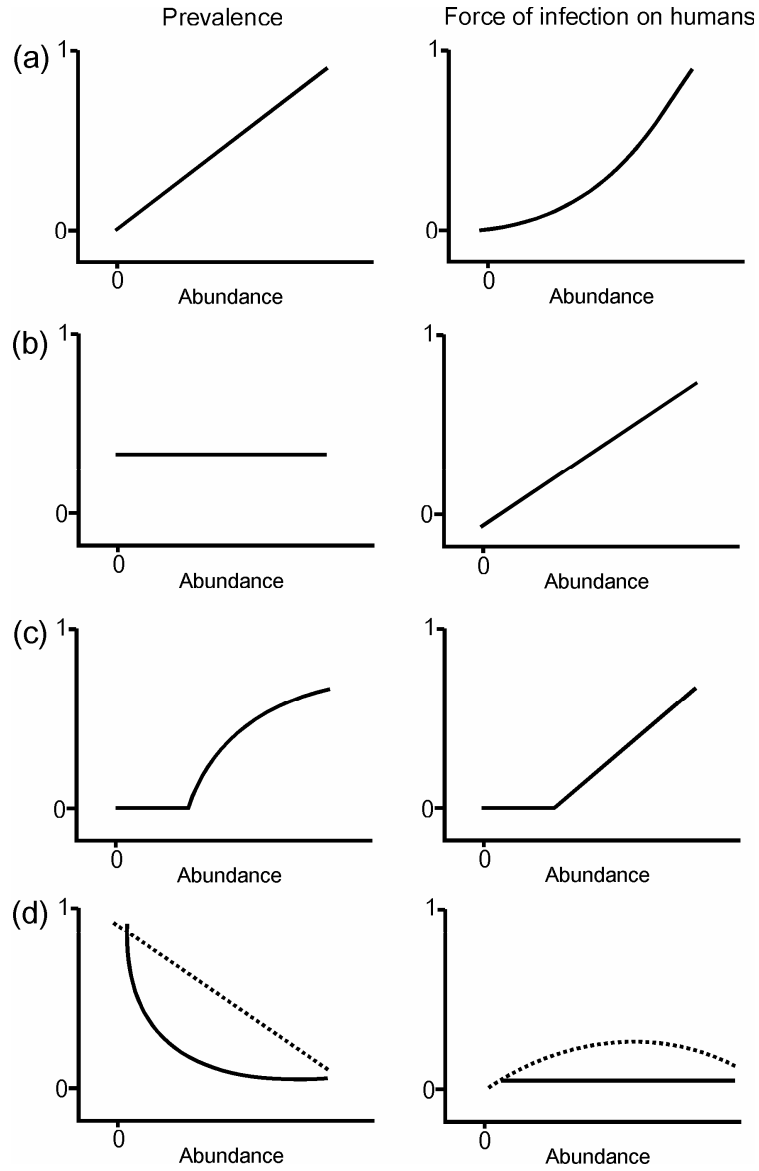
Fig. 1. Relationships between abundance and the force of infection on humans, depending on the relationship between host abundance and prevalence of disease in the host population; (a) a positive relationship, (b) no relationship, (c) a threshold relationship and (d) a negative relationship (modified after Davis et al. 2005)

There is a temporal scaling issue involved here, because while the juvenile dilution effect may work at a seasonal scale, there can still be a positive relationship between abundance and prevalence at a multiannual scale. Nevertheless, the juvenile dilution effect has its importance for population management, because it means that, within a year, an increase of the reservoir population (with uninfected juveniles) is not a reason to immediately start a control action. In fact, depending on the form of the negative relation between abundance and prevalence (e.g., the speed with which uninfected juveniles are recruited), intermediate host densities may even constitute a higher risk to humans than higher or lower densities (Fig. 1d). Similar effects can be obtained when arthropod vectors are involved in transmission of infection. The basic reproductive rate R_0, and from there p, is then proportional not to the host abundance N_h but rather to N_v/N_h, i.e. the number of available vectors per host individual. Reducing N_h may then have adverse effects on the risk to humans. That risk is further increased when such pest control action would result in the vectors looking for alternative hosts and ending up on humans. A classical example is the control of bubonic plague where a rodent control action without simultaneous action against fleas may force the latter to feed on humans, which would then effectively increase the risk to humans of becoming infected (Gratz 1999).

In conclusion, while it generally will be an appropriate disease control strategy to target small mammal species that are reservoirs of parasites, it is necessary to evaluate the relation between reservoir abundance and force of infection to humans. This requires a good understanding of the infection's ecology, but also of the reservoir host's population ecology.

4.3 Threshold population densities and herd immunity

Theories of infection ecology predict that there is a host population density below which the infection cannot persist (see references in Rosa et al., this volume). Intuitively, this is understood as a threshold density below which, on average, the chances for every infectious individual to meet an uninfected individual (to pass the infection to) are too small. More formally, it is the host density of a naive population below which the infection's basic reproductive rate R_0 becomes less than 1. There are a variety of ways to formulate R_0, but many include the number of sensitive hosts in the population. From there, the invasion threshold density S_T can be calculated. Obviously, R_0 and, thus, also S_T depend on the properties of the host (e.g., the contact rate between hosts, or between hosts and free-living parasite stages or intermediate hosts or vectors) as well as on the properties of the parasite (length of the period during which a parasite stage remains infectious, rate

of success for a parasite to colonise a host given that a host comes into contact with an infectious stage). The concept of the invasion threshold densities has a very strong theoretical support, and has been linked to observed patterns in human childhood diseases (reviewed in Hastings 1997), but observational data to support it in wildlife are very scarce (Begon et al. 2003; Lloyd-Smith et al. 2005). Study of measles epidemics in humans led to the recognition of the "critical community size", which is the size of a community that produces enough susceptible individuals to maintain transmission (Bartlett 1960). For example, there are longitudinal data suggesting that threshold numbers of susceptible individuals are needed for reinvasion of phocine distemper virus in harbour seals (Swinton et al. 1998) and corona- and parvoviruses in lions (Packer et al. 1999). A clear host density threshold was observed for infection with plague bacteria *Yersinia pestis* in *Rhombomys opimus* in Kazakhstan (Davis et al. 2004).

The existence of a threshold density opens several possibilities for disease control. In short-lived small mammals, the critical community size is probably close to the actual invasion threshold. If one manages to keep a small mammal host density below the threshold value, then an existing infection will fade out, and, even when it is reintroduced, it will not be able to establish. The effect is, thus, much more profound than a reduction of the force of infection as discussed above. The effect also can be much more sustainable, because if it is technically feasible to prevent reintroduction of the pathogen, the small mammal population can even be allowed to rise again to the original abundance level, so that fewer side-effects on the natural community are expected and no continued application of environmentally or ethically undesirable control techniques (pesticides, trapping) is needed.

The existence of a threshold can also be used as a management tool for the planning of control actions. As long as the densities remain below the threshold, no control is needed, whereas if the densities are above the threshold, authorities should be alert. In the case of plague in gerbils in Kazakhstan, there was a time lag between reaching the threshold and records of the infection (Davis et al. 2004). This allows the development of the early warning systems that predict future epizootics among gerbils and, from there, the risk to humans. It should be pointed out, however, that the threshold is not an absolute boundary for the presence of an infection. Firstly, high enough densities may be a necessary but not sufficient condition for the infection to spread, and other factors (e.g., climate) may play a role above the threshold. In addition, there is the stochastic aspect of whether a parasite arrives in a population where it is absent. If vectors or intermediate hosts are involved in the transmission of a parasite, then there may be several thresholds for each component that need to be reached, or

thresholds for relative vector/host proportions. Secondly, densities below the threshold mean that the establishment of the infection is impossible, which is not equivalent to saying that the infection never occurs. Infection can still arrive in such low-density populations and even be transmitted among individuals for some time before it fades out.

As mentioned above, it is the density of *susceptible* individuals, not the whole population, that is relevant in the threshold theory. If an infection elicits in a host an immune response with lasting effects, then animals that have been infected before are no longer susceptible to a new infection with the same pathogen. Vaccination allows a reduction of the number of susceptibles without a reduction of the total abundance of the host population. This is crucial in the concept of herd immunity, where a whole population is protected against establishment of a parasite by the immunisation of just a proportion of the population (Begon et al. 1996). The concept also applies to wildlife and should be considered in cases where reduction of abundance is undesirable or simply not feasible. A splendid example is the eradication of rabies from a large area of Western Europe. Foxes, the main wildlife host of this infection, have been hunted and poisoned for many decades in an attempt to eradicate rabies or, at least, reduce the force of infection to humans (Aubert 1999). This proved ineffective, mainly due to the high reproductive capacity of the foxes and their dispersal behaviour through which vacated places were quickly recolonised by the immigrant foxes. In fact, the increased dispersal movements contributed to a better spread of the infection. In the 1980's, a new approach was instigated. Fox populations were immunised with oral rabies vaccines hidden in baits that were distributed over large areas (Pastoret and Brochier 1998). Currently, rabies has disappeared from most of Western Europe despite increasing fox populations and the termination of vaccination campaigns (Selhorst et al. 2005). Of course, the fox population is now susceptible which means that much attention must be paid to avoiding reinvasion of rabies, either along the borders through natural dispersal of foxes or through human transport of infected dogs (Bugnon et al. 2004; Thulke et al. 2000).

A wildlife vaccination approach as described above could be promising in several other cases where it is not possible or desirable to reduce host population densities to a low enough level. It could also be considered as an alternative for vaccination of humans in cases where the latter is commercially not viable for the vaccine industry. Indeed, the development of the vaccine may not be so difficult or costly in itself, but tests of efficacy and safety in humans may be too costly or ethically difficult and, especially for diseases that occur in poor countries, investments in vaccine development may not pay off enough for commercial companies. Requirements for a vaccine for wildlife are much more relaxed, but there are a

number of other issues that need to be considered. Of course, a good quality vaccine must be available, but an equally large problem is to find adequate delivery methods for it. An oral bait with vaccine included is the most obvious approach, but one could also consider other alternatives such as a self-disseminating agent, like a genetically modified virus that carries similar antigens as the targeted parasite. The latter approach is not without ethical and environmental controversies. The needed efficiency of bait delivery is linked to the transmission properties of the pathogen in the host population: more "infectious" pathogens have a lower S_T and, thus, a higher proportion of the population will need to be vaccinated in order to achieve the herd immunity. Also, the ecology of the targeted wildlife host species must be taken into account: absolute numbers of hosts to be vaccinated, host dispersal patterns and population turnover rates are all important. Finally, one should not overlook possible resistance from human society where it may not be easily accepted that vaccination programs are targeting wild animals rather than humans or where people traditionally kill the host, rather than to protect it against infection.

4.4 If pest control equals harvesting…

If populations of pest species show strong compensatory mechanisms to cope with the increased mortality, then the classical lethal pest control strategies will not lead to a reduction in the population size. They will result, however, in an increased turn-over in the population with the recruitment of new individuals. When the recruitment is by reproduction, then the young animals entering the population will be susceptible to the infections of concern. This process will actually increase the number of susceptible individuals in the population, even when the total population size remains constant. The larger number of susceptibles leads to more intensive transmission, and since animals are constantly removed from the population, they do not have the chance to acquire a recovered immune status. If recruitment is mainly by dispersal, then the attracted individuals can be susceptible as well, but they could also be infectious. Thus, if the pest control acts as an unintended form of sustained harvesting, a parasite's prevalence may actually increase rather than decrease.

5 Pest population management using parasites

5.1 The basis for biological control

Parasites have a negative effect on host fitness and, therefore, they have long been considered as a potential pest control method (see review as early as in Elton 1942). Host-parasite models predict that parasites play a regulatory role in the population dynamics of a host (Anderson 1978; Anderson and May 1978). This conclusion has also been supported empirically by a number of experimental studies on model systems for small mammal-helminth interactions, such as the nematode *Heligmosomoides polygyrus* in *Apodemus flavicollis* (see also Rosa et al., this volume). In laboratory populations of these mice, introduction of *H. polygyrus* reduced equilibrium density levels by 10% in comparison to control populations kept under the same conditions (Scott 1987). Soon after, it was shown that this effect was caused by the decreased survival of the infected mice rather than reduced natality, and occurred in laboratory as well as in wild populations (Scott 1990; Gregory 1991; Quinnell 1992).

The evidence for a regulatory role of parasites in host population dynamics is not necessarily a good basis for biological control with parasites. Regulation is the process through which high population numbers are depressed down to an equilibrium level, but also increased up to this level when they reach low values (Begon et al. 1996). Many pest species cause damage already at low densities, and the objective of pest control is then to reduce population size well below the level of the natural equilibrium. It is not trivial to reach this goal using parasites. It requires that the negative impact of the parasite on the host more than exceeds the population's intrinsic growth rate and that the infection and its impact are persistent.

Biological control with parasites and parasitoids, especially in insect pest management, is well accepted and widespread (Pimentel 2002). On the contrary, in the control of small mammals, this management approach has been clearly successful in two cases only, both of which involved microparasites (viruses). There may actually be a biological basis for this difference as can be demonstrated by the shape of density-dependence, expressed as the per capita rate of population change as a function of density. Åstrøm (1997) noted that if the shape of this curve is downward convex (i.e. a stronger density dependence at lower densities, as suggested for insects) then even when these populations are controlled to a very low level, they are likely to be stable. If this curve is downward concave, (i.e. a stronger density dependence at higher densities, as suggested for mam-

mals), then strongly depressed populations are likely to fluctuate violently, suggesting that it is simply more complicated to biologically control mammalian than insect pests.

5.2 Biological control using parasites

Spratt (1990) described eight characteristics of the ideal candidate for biological control of mammals with helminths. These are as follows:

- there needs to be strong and persistent effect on mortality and/or fecundity;
- the impact of the bio-control agent would be more pronounced if there were density-dependent effects;
- success is likely to be higher if the helminth has a direct life cycle, otherwise time lags could diminish its effect on host population dynamics;
- persistence and diffusion of the parasite requires transmission via aerosol, long environmental persistence (e.g., resistance to desiccation) or a highly mobile and common vector;
- ideally, the bio-control agent should have high host specificity;
- the parasite needs to be cheap to maintain and able to cycle in the laboratory;
- little genetic resistance should be expected to develop rapidly in the host;
- if it is an exotic helminth, then it should need to pass the quarantine regulations of the target country.

These eight characteristics provide a very useful audit of the likely chances of success for a helminth that is proposed as a candidate biological control agent. If the candidate does not meet one of these prerequisites, then it is important to be aware of the likely implications on the parasite-host interactions or of the likely societal acceptance of the bio-control agent. Singleton (1994) also reviewed the prospects of macro-parasites as biological control agents, but specifically for rodent pest species.

Earlier, Singleton and colleagues explored the possibilities of using the nematode *Calodium hepaticum* (=*Capillaria hepatica)* as a biological control method to manage populations of house mice in Australia. House mice are an introduced mammal pest in Australia and display irregular population explosions of massive magnitude (>800 mice per ha), causing huge losses to farmers (Singleton 1997). *C. hepaticum* is a liver parasite that produces eggs that remain in the liver of the host. When the host is eaten by another animal, the eggs pass through this animal's gut, are excreted with faeces, and then mature in the soil until they are picked up by a new host. Alternatively, the eggs become embryonated in the environment after

the death and subsequent decomposition of the infected host. Infection with this nematode has a significant negative impact on the survival of house mice (Spratt and Singleton 1986). Due to this lethal effect, the transmission without an intermediate host, the possibility to formulate infectious eggs into a bait and the capacity of the eggs to remain infectious in the environment made this worm a promising agent for potential biological control, and model simulations indicated that it would indeed have a regulatory effect on house mouse populations (McCallum and Singleton 1989). Experiments in field enclosures showed that the infection successfully persisted for at least 1.5 years after release in a mouse population, but they also indicated compensations due to density-dependent mortality so that there was no difference in abundance between control and experimental treatments (Barker et al. 1991). A number of impressively extensive replicated field experiments were then set up in Eastern and Southern Australia. In the first experiment, in four treatment and three control areas of 4 km^2 each, the parasite was released during the low density phase of the population dynamics; there was successful transmission of the parasite, and after four months *C. hepaticum* had a considerable impact on survival in the mouse population; however, since survival was poor also in the control sites, there was no treatment effect on abundance (Singleton et al. 1995). Also, *C. hepaticum* did not have a noticeable effect on the population once breeding had commenced (Singleton et al. 1995). In a second experiment, started during the increase phase of a mouse population outbreak, 10% of the mice were infected in four treatment sites of 16 km^2 and again compared with three control sites. After two months, prevalence had risen to 30% but then started decreasing again and the mouse population increased to outbreak densities (Singleton and Chambers 1996). The authors attributed the failure to reach effects in both experiments to low population densities or drought conditions which limited effective and persistent transmission.

Microparasites have been more successfully applied as biological control agents. Two examples that stand out are the myxoma virus and the Rabbit Hemorrhagic Disease (RHD) virus that were used in the management of *Oryctolagus cuniculus* populations in Australia. The myxoma virus was successfully released in 1950 with a mortality often around 99% in the rabbit populations where the virus established (Cooke and Fenner 2002). The success of the myxoma virus was highest in the temperate and semi-arid zones where there was good survival of the vectors of the virus – rabbit fleas and mosquitoes. Rabbit populations bounced back within 5 years as they became immune to the various strains of the virus. The myxoma virus also changed genetically with selection favouring a virus that was more persistent, but which had lower mortality rates. Neverthe-

less, the myxoma virus is still an effective biological control agent more than 50 years after it became established in field populations.

The RHD virus escaped from quarantine facilities on an island off the Australian coast in 1995 and then spread quickly through the mainland. The drastic effects on the wild rabbit populations then incited also an un-authorised release of the virus in New Zealand. The recorded reductions in the damage by rabbits and costs to control them are huge (Saunders et al. 2002). Also in the case of the RHD, the effects of the introduced infections are strongly affected by other factors, such as aridity of the environment (Cooke and Fenner 2002; Mutze et al. 2002), virulence of the RHD strains or general condition of the rabbits (Bruce et al. 2004; Bruce and Twigg 2005; Story et al. 2004), and interactions with predator effects (Reddiex et al. 2002) or myxomatosis (Mutze et al. 2002).

It is no coincidence that the use of parasites as a biological control method for small mammals has received considerable attention in Austra-lia. The targeted pests are introduced species that colonised an environ-ment where there was only a poor community of competitors, predators and parasites and that were, thus, regulated at much higher levels, or not regulated at all, in comparison to populations in the original areas. More-over, due to the phylogenetic unrelatedness of the local fauna to lago-morphs, the risks for infection of non-target species was minimal.

5.3 Parasites as bio-rodenticides

The biological control described above aspires to establish an infection in a pest population so that the population stays at a low level for an extended period and the development of chronic and acute impacts are prevented. Parasites also have been thought of as biological rodenticides, where the objective was to kill individual rodents, much as a poison would do. This is not genuine biological control since the complex ecological host-parasite interactions discussed above do not apply here. The main issue under scru-tiny here is the pathogenicity of the parasite to individual rodents, and how to infect as many rodents as possible and as quickly as possible. Transmis-sion between rodents may be a useful characteristic, but it is not a strict necessity. Nevertheless, such an approach could still have its place in ecol-ogically-based rodent management, as an alternative to "poison", as long as the bio-rodenticide is more benign environmentally and/or has a lower impact on the non-target species.

A protozoan parasite that received some attention in this respect is the apicomplexan *Sarcocystis singaporensis*. This parasite has snakes of the genus *Python* as its definitive hosts; infectious stages are excreted with

snake's faeces and when these are eaten by rodents, they develop into muscle cysts in the rodent. When the rodent is eaten by a snake, the cycle is completed (Jäkel et al. 1997). The infection causes serious and often lethal disease in rodents. In an experiment in different agricultural habitats in Thailand, laying out bait pellets with a high concentration of the infectious stage of the parasite resulted in high (58-92%) parasite-induced mortality in rodents of three species (*R. norvegicus, Rattus tiomanicus, Bandicota indica*) (Jäkel et al. 1999). Subsequent field trials in rice fields in Thailand indicated that the bio-rodenticide was as effective in controlling rodents as conventional control (Jäkel et al. 2006). It is worth pointing out that these were direct effects of the infectious bait, without involvement of the definitive hosts. A commercial product containing the parasite has been developed and has been distributed in some Southeast Asian countries under the trade name *"PRORODENT"*. There are production issues common to this and other bio-rodenticides. These include the need for very high quantities of the infectious parasite stages and the need for biological baits to have a reasonable shelf life if they are going to be available for tactical or chronic use. Also, a bio-rodenticide has to compete with the efficacy and costs of conventional chemical rodenticides. Given that these generic challenges often discourage investment in bio-rodenticides, it is encouraging that a macroparasite based bio-rodenticide is now available commercially.

Much more widespread as a bio-rodenticide are preparations of the bacterium *Salmonella enterica*. Such formulations have been produced since the first half of the 20th century and are currently still available in a number of countries (Painter et al. 2004). The most widespread formulation is "Biorat", a preparation produced in Cuba or under Cuban license and used in a number of Central American, South American and Asian countries. Reports about the efficacy of this formulation as a rodenticide are often enthusiastic, but the major concern is the health risk that it may pose to other vertebrates than the targeted rodents, including humans (Friedman et al. 1996). The producers claim that the used strain is not pathogenic to humans but there is no substantial documentation to support this. The bacteria that are isolated from Biorat are *Salmonella enterica* serotype Enteritidis phage type 6a (Painter et al. 2004). The same type of bacteria was also used in "Ratin", a Salmonella-based rodenticide that was commercially available until 1960 in Europe and that caused several outbreaks of human illness. The dissemination of salmonellosis to other species is clearly undesirable.

5.3 Parasites as vehicles for immunocontraception

The aim of controlling the reproductive potential of a mammal pest population as a means to manage its negative impacts has led to much interest in approaches to sterilize the pest species (see Bomford 1990; Gao and Short 1993; Dell'Omo and Palmery 2002 for reviews). Thus far, there has been no successful sterility management of wild populations of small mammals. One approach that has promising potential is the delivery of a reproductive protein in a form that generates a strong and persistent immune response to the protein, thereby blocking fertilisation of the animal that receives the protein. This approach is described as immunocontraception and potentially either males or females could be sterilized (see Tyndale-Biscoe 1994). Over the past decade most of the research on immunocontraception of rodents has focused on female sterility and through using either a non-infectious agent in oral baits or an infectious species-specific virus as a carrier of an infertility agent. A system that works well under laboratory conditions has been developed for viral-vectored immunocontraception (see Singleton et al. 2002 for review).

Based on the concept developed for rodents in using viruses as vectors for a sterility protein, a program of research has been developed to try to control the brush-tail possum *Trichosurus vulpecula* using the nematode *Parastrongyloides trichosuri*, as the vehicle for the delivery and subsequent spread of a sterilizing protein (Cowan et al. 2006).

6 Concluding remarks

The presence of macroparasites can affect the pest status of small mammals and the damage they cause. Pest management of small mammal populations can also affect the macroparasite populations, in a positive as well as a negative way. Despite the effects of macroparasites on small mammal fitness, there is little hope for the near future that they can be used for biological control of small mammals, except perhaps for some bio-pesticides. Small mammals and macroparasites interact in complex ways, and the implications for pest management are equally complex.

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Appendix. Macroparasites of small mammals that are relevant for humans or live-stock

Macroparasites	Micromammals		Relevance for humans or livestock
	definitive hosts	intermediate hosts	
Trematoda			
Alaria spp.	mustelids, canids	rodents as paratenic hosts of metacercariae	occasional infection in domestic carnivores; human rare paratenic host of metacercariae
Brachylaima cribbi	house mouse		infection in humans
Dicrocoelium dendriticum	hares, rabbits, large rodents		parasitosis in sheep, goats
Euryhelmis squamula	shrews, mustelids		infection in mink
Opisthorchis viverrini	viverrids, felids		rare infection of cats and man
Clonorchis sinensis	mustelids		infection in humans
Nanophyetus salmincola	mustelids, canids, felids		*N. salmincola* is a vector for ricketsial fever agents to dogs and man
Fasciola hepatica	hares, rabbits, rodents		fascioliasis in sheep, cattle, humans
Echinostoma spp.	Norway rat, mustelids		infection in humans
Isthmiophora melis	mustelids, hedgehog		infection in mink
Paragonimus spp.	mustelids, viverrids, large rodents		infection in humans
Schistosoma japonicum, *S. mattheei*, *S. mansoni*	rodents		schistosomiasis
Schistosomatium sp.	small rodents, shrews, mustelids		cercaria cause dermatitis in humans

Cestoda

Rodentolepis (= *Hymenolepis*) *microstoma*, *R. nana*, *R. diminuta*	rodents		infections in humans
Taenia pisiformis, *T. taeniaeformis*, *T. brauni*, *T. serialis*, *T. multiceps*	carnivores	lagomorphs, rodents	infection in dogs, cats, humans
Echinococcus multilocularis	canids	microtine rodents	human alveolar echinococcosis
Echinococcus vogeli	bush dogs	rodents	infection in humans
Dipylidium caninum	canids		infection in humans
Spirometra erinacei	canids, felids		infection in humans
Inermicapsifer arvicanthidis	rodents		infection in humans

Nematoda

Toxascaris leonina	canids, felids	rodents, lagomorphs	ascariasis in dogs and cats rare in humans
Toxocara canis, *T. felis*	canids, felids	rodents as paratenic hosts	infection in dogs and cats; visceral and ocular "larva migrans" in humans
Baylisascaris procyonis	racoons	rodents	infection in humans
Ancylostoma duodenale	carnivores but only rarely		human hookworm; other *Ancylostoma* spp. may cause cutaneous "larva migrans" in humans
Uncinaria spp.	canids, felids		hookworm infection in dogs
Aelurostrongylus abstrusus	felids	rodents may act as transport hosts after eating infected snails	infection in cats
Angiostrongylus cantonensis, *Angiostrongylus* spp.	rodents, shrews, mustelids		meningitis in humans, paralysis in dogs
Gnathostoma spinigerum	canids, felids, mustelids,		erratic parasite (larval) under the skin of man - "Creeping eruption" and meningitis

Brugia sp.	viverrids, pangolins, tupaias, small primates		infection in humans
Trichinella spp.	rats		trichinosis in pigs or humans
Calodium hepaticum (=*Capillaria hepatica*)	rodents		rarely, heavy infection in humans or domestic animals
Haycocknema perplexum	dasyurids?		rare but can be fatal in adult humans
Acari			
Ornithonyssus bacoti	rodents		vector for several viruses and bacteria
Allodermanyssus sanguineus	rodents		vector for several viruses and bacteria
Echinolaelaps echidnus	rodents		vector for several viruses and bacteria
Argas sp.	some species on bats		vector for several viruses and bacteria
Ixodidae (various ticks of the genera *Ixodes*, *Amblyomma*, *Dermacentor*, *Haemaphysalis*, *Hyalomma*, *Rhipicephalus*)		larvae feed on various small mammals	adults feed on domestic carnivores, cattle, humans; vectors for several viruses and bacteria
Trombicula spp.		larvae feed on various small mammals	adults feed on domestic carnivores, cattle, humans; vectors for several pathogens
Sarcoptes scabiei	various small mammals		mange
Leptotrombidium deliensec	rodents, bandicoots		scrub typhus (*Orientia tsutsugamushi*) - humans
Insecta			
Triatoma spp.	armadillos		vectors for *Trypanosoma cruzi*
Ceratophyllus (*Nosopsyllus*) spp.	rodents		vectors for *Yersinia pestis*
Xenopsylla spp.	rodents		vectors for *Yersinia pestis*, *Rickettsia typhi*
Ctenocephalides felis	felids, opossums		vector for *Rickettsia*

		felis, *Bartonella henselae*
other Siphonaptera	rodents	vectors for *Yersinia pestis*
Mosquitoes	rodents	vector for *Plasmodium* sp., filariids
Glossina sp.	rodents	vectors for *Trypanosoma* sp.