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CHAPTER

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Biological Control of Vertebrate Pests

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INTRODUCTION

Several species of vertebrates, especially mammals, have been successful invaders and colonizers of new territories, in particular, insular island ecosystems. Others have exhibited high environmental tolerance and adaptability after careful and repeated introductions to new locales by humans. Following establishment, several vertebrate species have become important pests. These pests harm agricultural systems by damaging agricultural lands [e.g., rabbits (*Oryctolagus cuniculus* Linnaeus) in Australia and New Zealand], by attacking crops [e.g., European starlings (*Sturnus vulgaris* Linnaeus) in the United States], and by acting as sources for communicable diseases [e.g., brushtail possums, (*Trichosurus vulpecula* Kerr), are reservoirs for bovine tuberculosis (*Mycobacterium bovis* Karlson and Lessel) in New Zealand]. Other pest vertebrates damage natural systems by threatening the continued existence of endangered flora [e.g., goats (*Capra hircus* Linnaeus) on the Galapagos Islands] and fauna [e.g., brown tree snake (*Boiga irregularis* {Merrem}) on Guam], and by adversely affecting wilderness areas by changing ecosystem functions and diversity (Vitousek *et al.*, 1996). The continued relocation of vertebrates exacerbates the ongoing problem of global homogenization of biota (Lodge, 1993).

Movement of particular vertebrates into areas where they had not previously existed has, in some instances, occurred naturally without human intervention [e.g., the passerine bird (*Zosterops lateralis* {Latham}) arrived in New Zealand unassisted from Australia]. The vast majority of vertebrate translocations have been human assisted. Accidental introduction has occurred as a consequence of human transportation [e.g., brown tree snakes, mice (*Mus musculus* Linnaeus), and rats such as *Rattus rattus* Linnaeus and *R. norvegicus* Berkenhout)]. Some releases have been intentional (but illegal) to serve self-centered private interests [e.g., monk parakeets (*Myiopsitta monachus* {Boddaert}) in New York, Florida, and Texas]. Other species have been legitimately introduced to procure public benefit by providing: (1) new agricultural products [e.g., European wild boars (*Sus scrofa* Linnaeus); sheep (*Ovis aries* Linnaeus); cows (*Bos taurus* Linnaeus), goats and rabbits for meat, and brushtail possums for fur in New Zealand], (2) recreation [e.g., red deer (*Cervus elaphus* Linnaeus), fallow deer (*Dama dama* Linnaeus), and trout (*Salmo trutta* Linnaeus and *S. gairdneri* Richardson) in New Zealand]; (3) companionship [e.g., cats (*Felis domesticus* Linnaeus) and dogs (*Canis familiaris* Linnaeus)]; or (4) biological control agents [e.g., the European fox (*Vulpes vulpes* Linnaeus), stoats (*Mustela erminea* Linnaeus), weasels (*M. nivalis* Linnaeus), and ferrets (*M. putorius furo* Linnaeus) for the control of rabbits in Australia or New Zealand; cane toads (*Bufo marinus* Linnaeus) for control of gray-backed cane beetles (*Dermolepida albohirtum* {Waterhouse}) in sugarcane plantations in Australia; and the small Indian mongoose (*Herpestes javanicus* {Saint-Hilaire}, {= *auropunctatus* (Hodgson)}) for control of rats in Hawaii].

Chemical and cultural control of vertebrate pests is expensive and nonsustainable, and at best provides a temporary local solution to problems (Hone, 1994; Williams & Moore, 1995). Biological control of vertebrates, a potentially less expensive and self-sustaining method of population suppression, has focused primarily on mammalian pests. Predators, parasites, and pathogens specific to mammals with two notable exceptions (the myxoma and calici viruses that infect rabbits) have failed to provide satisfac-

tory control (Shelford, 1942; Howard, 1967; Davis *et al.*, 1976; Wood, 1985; Smith & Remington, 1996; Whisson, 1998). Historical records indicate that the majority of attempts at vertebrate biological control have been *ad hoc* efforts and not the product of careful studies designed to elucidate factors and conditions likely to affect the impact of natural enemy introductions on pest populations. Furthermore, failure of biological control of vertebrates by predatory vertebrates has compounded problems associated with exotic vertebrates because control attempts result in addition of new species that cause biological and conservation problems.

The level of control achieved by natural enemies is dependent on ratios of natality to mortality of control agents and their host species (Davis *et al.*, 1976). For vertebrates these ratios are affected by many factors: advanced learning; social, territorial, and breeding behaviors; chemical, physical, and immunological defenses; temporal and spatial escape strategies; and genetic selection in both natural enemy and host populations for persistent coexistence. These complex interrelated factors, coupled with opportunistic feeding habits, have made vertebrate pests difficult targets for biological control with natural enemies.

Advances in understanding of mammalian fertilization biology have provided molecular biologists with necessary information to develop and investigate the concept of immunocontraception for vertebrate pest control. Immunocontraception utilizes genetically modified pathogens that express surface proteins from the target pest's egg or sperm to induce an immune response in the host. Antibodies then attack gametes in the host's reproductive tract causing sterilization (Tyndale-Biscoe, 1994b, 1995). Computer models indicate that immunocontraception may provide long-term control of vertebrate pests because genetically modified pathogens reduce net reproductive rates without killing hosts (Barlow, 1994, 1997).

In this chapter, I discuss attributes that have aided vertebrate establishment; damage resulting from colonization and uncontrolled population growth; biological control of mammalian pest species with predators, parasites, and pathogens; and future directions that biological control research for vertebrates is taking with genetically engineered microorganisms.

FACTORS THAT FACILITATE VERTEBRATE ESTABLISHMENT

Community Characteristics

Communities vary in their ability to accommodate the establishment and proliferation of new species (Primack, 1995). Elton (1958) suggested that species-poor communities (e.g., islands) or highly disturbed habitats are more permissive to successful introductions of new species. Elton's (1958) predictions have been substantiated in part by paleobiological reconstructions of invasions between newly joined communities (Vermeiji, 1991) and by mathematical modeling describing multi-species interactions in communities (MacArthur, 1955; Case, 1991). Isolated oceanic islands (e.g., New Zealand), and insular continents and habitats (e.g., Australia and lakes) often have a low diversity of native species. Such environments have typically experienced little immigration and are susceptible to invasion by vertebrates (Brown, 1989). Stable, speciose communities with high levels of interspecific competition appear to resist invasion by new species and are sources of successful colonists into less speciose or disturbed communities (MacArthur, 1955; Brown, 1989; Lodge 1993). This phenomenon has produced asymmetrical patterns of colonization, with successful vertebrate invaders usually being native to continents or extensive nonisolated habitats within continents with more diverse biotas (Brown, 1989; Vermeiji, 1991).

Continental herbivores and predators have been very successful in establishing self-sustaining populations in insular habitats, in part because such habitats often lack large generalist vertebrates and essentially have just two trophic levels, producers and decomposers (if specialist herbivores and their predators are excluded) (Vitousek, 1990). Niches equivalent to those on the mainland are largely unoccupied (Brown, 1989). Insular ecosystems therefore often appear to readily accommodate generalist herbivores and predators, perhaps because of low levels of competition for resources that are often inadequately defended chemically or physically (Vitousek, 1990; Vermeij, 1991; Bowen & van Vuren, 1997). Organisms from insular habitats that have not coevolved closely with predators or herbivores lack life history features that deter attack or permit survival despite high mortality from predation or herbivory (Bowen & van Vuren, 1997). Lack of such biological attributes may increase the competitive advantage of exotics (Case, 1996; Coblentz, 1978; Vitousek, 1990).

Introduced vertebrates can also be extremely disruptive in continental regions when habitat disturbance by urbanization or agriculture occurs. European wild boars have detrimental effects on gray beech forests in the Great Smoky Mountains in the southeast United States. Habitat disturbance—through pig rooting, trampling, and browsing—and human removal of predators (pumas and wolves) aided pig establishment and spread in this area (Bratton, 1975; Singer *et al.*, 1984; Vitousek, 1990).

Species Characteristics

Although there are well-recognized exceptions to general rules that characterize successful vertebrate colonists (Ehrlick, 1989; Williamson & Fitter, 1996b), species that establish self-sustaining populations outside their native

range typically exhibit some of the following general characters: (1) short generation times, (2) high dispersal rates, (3) high tolerance for varying geographic and climatic conditions, (4) polyphagy, (5) low attack rates from upper trophic level organisms, and (6) human commensalism (Ehrlich, 1986, 1989; Lodge, 1993; Williamson & Fitter, 1996a). The assumption of a high intrinsic rate of increase is generally unnecessary for establishment (Ehrlich, 1986; Lodge, 1993), although it is important for the establishment of some exotic bird species (Veltman *et al.,* 1996). The spread of the European rabbit in Australia and New Zealand, countries that both historically lacked significant eutherian mammal fauna, illustrates some of the preceding points.

The European rabbit originated in Spain and Portugal (Corbet, 1994) and spread through most of Europe over 3000 years ago following deforestation by humans for agriculture and overgrazing by livestock. Animals reared in captivity were carried through Europe in advance of naturally spreading populations (Flux, 1994). The spread of the European rabbit throughout Australia following its introduction in 1788 varied from 15 km/year to approximately 300 km/year. Rate of spread was fastest across dry savannas where conditions were most similar to Mediterranean climates and slowest through woodlands (Myers *et al.,* 1994).

In Australia, habitat alteration by humans such as conversion of land to pasture, overgrazing of rangelands, and predator eradication [e.g., dingos (*Canis familiaris dingo* Meyer)] aided rabbit survivorship and spread. Rabbits are polyphagous and feed on grasses and browse shrubs. During severe food and water shortages, bark, fallen leaves, seed pods, tree roots, and termites are consumed (Myers *et al.,* 1994).

Rabbits are highly fecund and exhibit rapid population growth under good conditions. Individual female rabbits 10 to 12 months of age can produce litters averaging 5 to 6 young, and with abundant food 23 to 48 offspring per year are produced (Gibb & Williams, 1994). Behavioral adaptability, sociality, and territoriality, in addition to use of elaborate underground warrens, have also aided rabbit proliferation in marginal habitats (Myers *et al.,* 1994).

Relocation of Vertebrates and Human Assistance in Establishment

Some of the first introductions of exotic vertebrates were those commensal with humans as they colonized new areas. Among well-documented early human introductions are dingoes in Australia (Brown, 1989) and Polynesian rats [*Rattus exulans* (Peale)] on Pacific islands (Roberts *et al.,* 1992). As early Europeans explored the planet, other commensal species such as *R. rattus, R. norvegicus,* and *Mus musculus* expanded their geographic range without deliberate human assistance and are now cosmopolitan in distribution (Brown, 1989). Not all vertebrate pests cohabitat with or are associated with human disturbance of the environment. Red deer and feral cats, for example, inhabit much of New Zealand's pristine habitats with no human management.

Most vertebrate translocations fail even with human assistance. Failure of exotics to establish may depend on life history parameters, responses to abiotic factors, inability to outcompete native species for resources or enemy free space, or chance (Cornell & Hawkins, 1993). Deliberate releases of exotic birds have establishment rates of around 20 to 35% (Veltman *et al.,* 1996). Establishment estimates for intentional vertebrate releases are biased because successes are more often recorded than failures (Ehrlich, 1989; Veltman *et al.,* 1996).

The amount of effort directed toward introduction is an important variable affecting successful colonization by vertebrates, and establishment rates increase with high levels of management and numbers of individuals released (Ehrlick, 1989; Griffith *et al.,* 1989; Williamson & Fitter, 1996b; Veltman *et al.,* 1996). In contrast, organisms that are casually introduced into new areas have much lower probabilities of establishing and proliferating. This phenomenon is expressed in the tens rule, a statistical characterization of probability outcomes for different levels of invasion success (Williamson & Fitter, 1996a, 1996b). For a variety of plants and animals, a general rule holds that 1 in 10 species imported (i.e., brought into new areas intentionally or accidentally) appear in the wild, 1 in 10 of those now found in the wild become established, and that 1 in 10 of those established with self-sustaining populations become pests (Williamson & Fitter, 1996b).

VERTEBRATE PEST MANAGEMENT

Following establishment, proliferation, and rise to pest status, control of exotic vertebrates is often prompted by economic, environmental, or conservation concerns. Several control strategies may be pursued, the most common being chemical control (e.g., poisoning) and cultural control (e.g., trapping, fencing, and shooting). The least used option has been biological control. Chemical and cultural control of vertebrate pests has been covered by Hone (1994) and Williams and Moore (1995).

Biological control is the intentional use of populations of upper trophic level organisms (e.g., predators, parasites, and pathogens) commonly referred to as natural enemies to suppress populations of pests to lower densities than would occur in the absence of natural enemies (DeBach, 1964; Van Driesche & Bellows, 1996). Biological control programs for vertebrates have employed all three classes of

natural enemies: predators, parasites, and pathogens. In contrast to weeds and pestiferous arthropods, however, biological control as a population suppression tactic for management of vertebrate pests has historically received much less attention. Predators of vertebrates in the few instances they have been used have not been particularly successful. Some vertebrate predator introductions also had severe impacts on nontarget organisms. Consequently, early unpromising results discouraged intensive development of this technology (Howard, 1967; Davis *et al.*, 1976; Wood, 1985).

There is a need for increased effort using biological control agents against vertebrates, especially where resistance to toxins has developed, or behavior and terrain makes chemical and cultural control difficult and expensive (Wood, 1985; Bloomer & Bester, 1992). Biological control should be fostered internationally because many countries experience similar problems (e.g., rabbits are agricultural pests in Argentina, Australia, Chile, Europe, and New Zealand; rats, cats, and dogs attack endangered faunas on many oceanic islands; feral pigs and goats in New Zealand, Australia, and the United States degrade habitat and threaten endangered flora).

Biological control can be aided by the establishment of institutions to help coordinate regional and international research activities. For example, in 1992, the Australian federal government supported the creation of the Cooperative Research Center for Biological Control of Vertebrate Pest Populations (also known as the Vertebrate Biocontrol Center), an unincorporated collaborative venture between state and federal organizations with international cooperators (Anonymous, 1997a). The principle research goal of this institution is population suppression of noxious vertebrate species by regulating reproductive rates (Tyndale-Biscoe, 1994a).

Predators as Biological Control Agents

A fundamental issue that has important implications for biological control is understanding the regulatory effects predators have on prey populations. Determining impact of introduced predators on pest and nontarget populations is becoming increasingly more important as public awareness of potential nontarget impacts increases and the impact of past introductions on nontarget organisms has become clearer (Howarth, 1983; Van Driesche & Hoddle, 1997).

Introductions of vertebrate predators as biological control agents against vertebrates have in some instances had disastrous impacts on nontarget wildlife, especially insular communities that have lacked an evolutionary history with generalist predators (Case, 1996). An example is the impact the small Indian mongoose on native rail populations in Hawaii following its release in the 1880s for rat control (Loope *et al.*, 1988). The mongoose has little demonstrable effect on rats (Cagne, 1988) and mongoose populations are now poisoned to protect native birds (Loope *et al.*, 1988). Exotic predators may enhance the success of introduced pest species by moderating the competitive impact of natives on introduced pests should predators reduce densities of native species (Case, 1996).

On the other hand, there is very good evidence that under certain circumstances introduced exotic predators can regulate target vertebrate pests. In such cases, predator efficacy may be affected by ecosystem complexity; by influence of such extrinsic factors as weather, disease, or human intervention on pest population growth; and by the availability of alternative food sources to sustain predators when pest populations are low.

In simple ecosystems such as islands, establishment of reproducing predator populations can result in the extinction of target pests. In the absence of alternative prey, releases of cats for rabbit control on Berlinger Island near Portugal by a lighthouse keeper resulted in the eradication of the rabbits, and subsequently cats died from starvation (Elton, 1927). In complex communities, alternate prey may be taken when primary prey populations are low for prolonged periods, as is the case for some microtine rodent species in Europe. European rodent populations exhibit fluctuations in size as a response to variation in food availability. These fluctuations are also influenced by predation. Population cycles are not observed in Southern Hemisphere countries where European pests and predators have been transferred (Korpimäki & Krebs, 1996; Sinclair, 1996).

Rabbit populations in Australia and New Zealand are maintained at low levels by introduced predators, but regulation only occurs after pest numbers have been reduced by other means. Poisoning programs in New Zealand in the 1950s and 1960s substantially reduced rabbit densities and populations were maintained at low levels by introduced predators, in particular, ferrets and cats (Newsome, 1990). Similarly in Australia, European foxes and cats maintain rabbit populations at low densities following population crashes caused by prolonged hot summers that reduce forage and browse (Newsome *et al.*, 1989; Newsome, 1990). Mouse populations are regulated in a similar fashion by predators (raptors and foxes) in Australia (Sinclair *et al.*, 1990). Introduction of two foxes *Dusicyon culpaeus* (Molina) and *D. griseus* (Gray) native to mainland Chile onto the Chilean side of Tierra del Fuego Island regulated rabbit populations after rabbit densities were substantially reduced by the myxoma virus (Jaksic & Yàñez, 1983).

The suppressive action of predators on rabbits in Australia has been demonstrated through predator removal experiments [referred to as perturbation experiments by Sinclair, (1996)] in which European foxes and cats were shot from four-wheel drive vehicles at night. Removal of predators

resulted in rapid rabbit population growth compared with rabbit densities in control plots in which predators were not removed (Newsome *et al.*, 1989).

The "predator pit" first conceptualized by May (1977) describes rabbit regulation in Australia and New Zealand by generalist predators. The model suggests that once prey populations fall below certain densities (i.e., because of culling or disease) predators can prevent recovery to higher levels. Generalist predators achieve this by maintaining relatively high numbers by attacking alternate prey species, and low but persistent levels of predation on species in the pit prevents pest populations from outbreaking. For the rabbit–fox system in Australia, a predator pit operates at densities of 8 to 15 rabbits per kilometer of linear transect. Below these densities foxes utilize alternate food sources (e.g., native animals) and above this critical density rabbit populations escape regulation by predators (Newsome, 1990). In New Zealand, rabbits are contained in the pit by cats and ferrets when densities are 0.4 rabbits per hectare (Barlow & Wratten, 1996). The effect of predators on long-term population dynamics of alternate prey species is generally difficult to disentangle from confounding effects of habitat degradation and competition for food and breeding sites from other introduced species (Pech *et al.*, 1995).

Foxes regulate mice and rabbits through positive density dependence at low prey densities. Increasing pest densities during outbreaks results in inverse density-dependent predation and type III functional responses (Sinclair *et al.*, 1990; Pech *et al.*, 1992). Rabbit and mouse populations escape predator regulation when favorable weather provides good breeding conditions, or when predators are controlled by shooting or poisoning (Newsome, 1990; Sinclair *et al.*, 1990; Pech *et al.*, 1992).

Predator control may be necessary for lifestock protection or for conservation of endangered wildlife and when implemented resurgence of pest populations occurs (Newsome *et al.*, 1989; Newsome, 1990). Predator numbers may increase when primary prey (e.g., rabbits) are abundant. Predation by abundant predators on secondary prey (e.g., native animals) result, leading to declines in secondary prey density. Under such circumstances native prey species may only persist in refugia or in areas with artificially reduced predator pressure (Pech *et al.*, 1995). Alternatively, declining densities of a primary pest prey species (either through management or disease) can intensify predator attacks on nontarget organisms. Therefore, when conservation of endangered natives is a concern, culling of predators may be undertaken either concurrently with the decline in prey density or in anticipation of such a decline (Grant Norbury, personal communication, 1997). In such situations an integrated approach to managing vertebrate pests and their predators is necessary (Newsome, 1990).

Predator efficacy can be enhanced either through habitat modification or resource provisioning. Cats can maintain rat and mouse populations around farm buildings below environmental carrying capacity as long as they are provisioned with additional food (e.g., milk). Dietary supplementation prevents rodent extermination and the subsequent extinction of cats. Sustaining a cat population prevents uncontrolled invasions by rodents and low pest densities are maintained (Elton, 1953). Provision of nesting boxes for barn owls (*Tyto alba javanica*) reduces crop damage by rats in Malaysian oil palm plantations; rodenticide use declined, and in some instances use was eliminated (Wahid *et al.*, 1996).

Changes in management practices can improve predator efficacy. Rodent control by *Tyto alba* (Scopoli) in *Pinus radiata* Don plantations in Chile was enhanced by clearing 4-m wide strips between trees (for owls to maneuver in while in flight) and construction of perches in forests (for resting and surveillance). Barn owl numbers and predation rates on rodents increased following habitat modification (Muñoz & Murúa, 1990). Under increased predation pressure, rodents will modify foraging behaviors by reducing activity when owls are flying or making hunger calls (Abramsky *et al.*, 1996). Learning in this manner produces behavioral adaptations because of strong selection pressures to minimize predation risks on pest populations (Davis *et al.*, 1976).

Parasites as Biological Control Agents

Parasites or macroparasites (e.g., helminths, lice, ticks, fleas, and other metazoans) do not typically kill their hosts as a prerequisite for successful development as insect parasitoids do. They tend to be enzootic (i.e., remain at fairly constant levels through time) and usually must pass through a free-living stage to complete an entire life cycle (Anderson, 1979; McCallum, 1994). The potential of parasites to regulate vertebrate host populations was first proposed as early as 1911 (Lack, 1954) and later was demonstrated theoretically with Lotka–Voltera models in which parasites increased host mortality rates (Anderson & May, 1978; May & Anderson, 1978; May, 1980).

Parasites act in a positive density-dependent manner by adversely affecting host survival or reproduction (Anderson & May, 1978; Dobson & Hudson, 1986; Scott & Dobson, 1989). Host parasite load also affects the ability of individual parasites to grow, reproduce, and survive in definitive hosts; and the severity of density dependence on host and parasite fitness is affected by patterns of parasite distribution in host populations (Scott & Lewis, 1987). Helminths, for example, tend to be aggregated within host populations so that few hosts are heavily burdened while most are lightly infected (Scott & Lewis, 1987). Density-dependent

constraints on parasite survival and reproduction occur in the few heavily infected hosts, and under such conditions, helminth population stability is enhanced (Anderson & May, 1978; May & Anderson, 1978). Furthermore, parasites with low-to-moderate pathogenicity exert stronger regulatory actions on populations than highly pathogenic species that cause their own extinction by killing hosts before transmission (Anderson, 1979).

Parasite regulation of vertebrate populations has been observed under field conditions. Botfly (*Cuterebra grisea* Coquillett) parasitism of voles [*Microtus townsendii* (Bachman)] in Vancouver Canada, is inversely density dependent; and botfly infestation significantly reduces vole survival, reproduction, and development (Boonstra *et al.*, 1980). The parasitic helminth *Trichostrongylus tenuis* (Cobbold) is the primary agent responsible for long-term population cycles in red grouse [*Lagopus lagopus scoticus* (Latham)] inhabiting Scottish heathlands (Dobson & Hudson, 1994). The regulatory effect of *T. tenuis* has been demonstrated by reducing parasite infestations with helminthicides in experimental birds. Treated grouse showed increased overwintering survival, clutch sizes, and hatching rates when compared with untreated birds (Dobson & Hudson, 1994). In the laboratory, introduction of the nematode *Heligmosomoides polygyrus* Dujardin reduced mice densities by 94% in comparison with control populations. Reduction of nematode transmission rates and elimination of parasites with helminthicides allowed infested mouse populations to increase (Scott, 1987). Although host and parasite densities in this study were higher than those found in nature, the data showed that introduction of a parasite regulated host population abundance. The potential effectiveness of nematodes as biological control agents in field situations has been evaluated for control of the house mouse, an introduced pest in Australia (Singleton & McCallum, 1990; Spratt, 1990).

Mouse (*Mus domesticus*) populations erupt every 7 to 9 years in cereal-growing regions of southeastern Australia (Singleton & McCallum, 1990; McCallum, 1993) and economic losses to mouse plagues exceed $50 million (Australian) (Beckman, 1988; Singleton, 1989). Outbreaks are associated with high autumn rainfalls following prolonged periods of drought that extend the growing season for grasses that set seeds. This high-quality food source increases high mouse survivorship and breeding throughout winter. Population crashes occur when food supplies are exhausted (Singleton, 1989). Saunders and Giles (1977) suggested that droughts are necessary to remove the regulating effects of natural enemies, and this removal combined with favorable weather conditions permitted mouse numbers to increase rapidly.

Capillaria hepatica (Bancroft), a parasitic nematode that infests mice, is naturally occurring and widely distributed in pestiferous rodents in coastal areas of Australia. It is, however, absent in mouse populations in cereal-growing areas (Singleton *et al.*, 1991). This nematode is unique because it is the only known helminth with a direct life cycle that requires host death for transmission. Female nematodes deposit eggs in the host's liver; and these eggs are liberated by predation, cannibalism, or necrophagy with subsequent digestion of infected liver. Unembryonated nematode eggs voided after ingestion undergo embryonation to become infective and are probably consumed when mice preen their fur and feet (Singleton *et al.*, 1991) (Fig. 1). Ground beetles (Carabidae) may vector *C. hepatica* eggs after they have been eaten (Mobedi & Arfaa, 1971). First-stage larvae emerge from ingested embryonated eggs and move into the liver through the hepatic portal system (Wright, 1961). Nematode infestation significantly reduces natality and numbers of young mice weaned by infected females (McCallum & Singleton, 1989; Singleton & McCallum, 1990; Singleton & Spratt, 1986; Spratt & Singleton, 1986). *Capillaria hepatica* is associated with introduced rat and mouse species in urban areas, and naturally occurring infections in native Australian mammals are rare probably because of the susceptibility of nematode eggs to ultraviolet radiation and desiccation (Spratt & Singleton, 1986; Singleton *et al.*, 1991). Native Australian mice and marsupials are susceptible to experimental infection in laboratories (Spratt & Singleton, 1986).

Rats (*R. norvegicus* and *R. rattus*) are major reservoirs for *C. hepatica* in urban areas and infestation rates range from 40 to 80% (Childs *et al.*, 1988; Singleton *et al.*, 1991). Infestation levels are lower in sympatric mice populations (0 to 40%, Singleton *et al.*, 1991). Low rat numbers in cereal-growing regions of Australia may be a factor contributing to the nonpersistence of *C. hepatica* in these areas (Singleton *et al.*, 1991). Infestation of nonrodent mammals by *C. hepatica* is rare (Singleton *et al.*, 1991) but has occurred in rabbits (Gevrey & Chirol, 1978), dogs (LeBlanc & Fagin, 1983), horses (Munroe, 1984), and humans (Pannenbecker *et al.*, 1990). Human infections can be treated successfully (Pereira & Franca, 1983).

Exploratory models investigating the impact of *C. hepatica* on mouse populations indicated that the requirement of host death for parasite transmission is strongly destabilizing. In the absence of resource limitation mouse densities increase similarly to disease-free populations before parasites have an impact and infected populations decline in density (McCallum & Singleton, 1989). Slow regulation of mouse populations occurs because of the need for host death for transmission. Consequently, the nematode's life cycle operates on the same time scale as that of its host instead of being orders of magnitude faster, as is the case with other parasites that do not require host death for transmission (McCallum & Singleton, 1989; Singleton & McCallum, 1990).

The destabilizing influence of *C. hepatica* on mouse

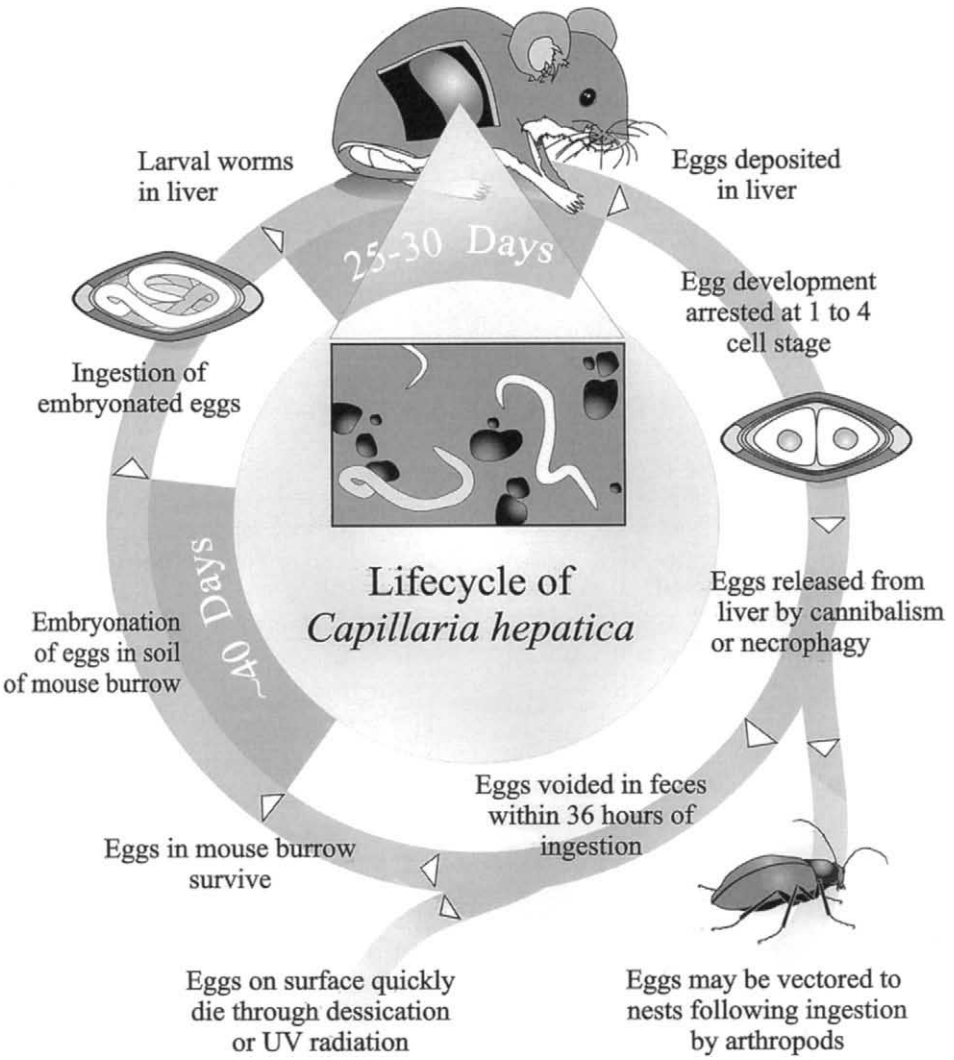

FIGURE 1 Life cycle of *Capillaria hepatica* (Nematoda) in the house mouse [*Mus musculus* (Rodentia: Muridae)] (artwork prepared by Vincent D'Amico, III). [After Singleton, G. R., & McCallum, H. I. (1990). *Parasitology Today, 6,* 190–193.]

populations may contribute to localized host and parasite extinctions. These extinctions, coupled with very low mouse densities in nonoutbreak years, result in population bottlenecks and may explain why nematodes do not persist in regions where mouse outbreaks occur. Soil type, temperature, and moisture content do not affect nematode egg survival and embryonation under favorable conditions in outbreak regions (Spratt & Singleton, 1987). Outbreak intensity can theoretically be reduced by *C. hepatica* if populations are inoculated early, preferably 1 year before an outbreak is expected (McCallum, 1993). Releases of high doses of nematode eggs in the summer or autumn when mouse densities are sufficient to enable high levels of transmission may offer the best chance for successful control (McCallum & Singleton, 1989; McCallum, 1993).

Field experiments in enclosures and with increasing populations of free-ranging mice have failed to demonstrate long-term regulation on mouse population growth with periodic inoculative releases of *C. hepatica* eggs. Unexpected declines in control populations (i.e., populations not treated with nematodes) have to some degree masked the effect of *C. hepatica* on mice populations (Barker *et al.,* 1991; Singleton *et al.,* 1995; Singleton & Chambers, 1996). Transmission of *C. hepatica* in treated populations is not density dependent and can occur at low levels for 12 to 18 months. Transmission rates show seasonal trends influenced by soil temperatures and increasing aridity (Barker *et al.,* 1991; Singleton & Chambers, 1996). Improved understanding of the influence of factors (such as temperature and rainfall on nematode persistence, survival, and transmission in field

situations) and timing of releases of parasite eggs may improve releases of *C. hepatica* for control of mouse outbreaks (Singleton *et al.,* 1995; Singleton & Chambers, 1996).

Vertebrate species that successfully colonize new habitats have reduced parasite loads in comparison with mother populations from which they originated (Dobson & May, 1986). Lower infestation levels probably occur because individuals that make up small founding populations either were uninfected or had only a limited subset of the total potential parasite species found in the area of origin, or intermediate hosts required for parasite persistence were absent in the new range. Sparrows and starlings, both successful colonizing species from Europe, have two to three times fewer parasites in North America compared with populations from which they originated. Populations established outside of Europe may have benefited from reduced parasite burdens, although there are no quantitative data to indicate that this aided establishment and proliferation (Dobson & May, 1986). Investigating the role of parasites on population dynamics of rabbits in Europe with the view for possible introduction into countries where rabbits are pests is also warranted (Boag, 1989).

Introduced mammals such as rats, goats, and cats on oceanic islands exhibit depauperate parasite faunas (Dobson, 1988). Fewer parasites coupled with presumed low genetic diversity of small founding populations, and reduced selection pressures for parasite resistance may make these pest vertebrates vulnerable to introduced host-specific parasites. The ideal parasite introduced into a high-density pest population that originated from a small founding population should have low-to-intermediate pathogenicity, because such parasites establish and maintain themselves in populations at lower densities than more pathogenic species do (Anderson & May, 1978; May & Anderson, 1978). Macroparasites that reduce both host longevity and fecundity may have the potential to cause sustained reductions of host population densities (Dobson, 1988). Low genetic variability among target populations should theoretically enable introduced parasites to become more evenly distributed among hosts, and reduction in parasite aggregation would increase natural-enemy efficacy (Dobson & Hudson, 1986).

The possibility of reassociating parasites with vertebrate pests is not limited to mammals and birds. Host-specific parasites may have the potential to reduce reproduction and longevity of pest reptile (Dobson, 1988) and amphibian species (Freeland, 1985). The brown tree snake is the proximate cause of 12 native bird extinctions on Guam following its accidental introduction after World War II on military equipment (Pimm, 1987; Savidge, 1987; Jaffe, 1994; Rodda *et al.,* 1997). The snake also has caused declines of native reptile and small mammal populations, and enters houses and attacks sleeping human infants (Rodda *et al.,* 1997). Additionally, the brown tree snake has caused economic losses by adversely affecting domestic animals (e.g., chickens and pets), and high densities of snakes on power lines regularly cause short circuits that interrupt electrical supplies and necessitate repairs. Control of the brown tree snake has been attempted through trapping, but the snake's extreme preference for live bait over artificial lures has made this approach impractical (Rodda *et al.,* 1997).

The brown tree snake—native to eastern Indonesia, the Solomon islands, New Guinea, and northeastern Australia—belongs to the family Colubridae. It is the only member of this family on Guam. There is one native species of snake on Guam, the blind snake, *Rhamphotyphlopys braminus* (Daudin), which belongs to the family Typhlopidae and is the only snake occurring on many islands in the central Pacific region (T. Fritts, personal communication, 1998). The brown tree snake has extended its range and is now established on the previously snake-free island of Saipan, and this snake has been intercepted in Hawaii; Corpus Christi, Texas, and Spain (Rodda *et al.,* 1997). Given the propensity for the brown tree snake to be dispersed to new habitats within cargo loads on planes and ships, the major social, economic, and ecological problems that are caused on islands after colonization, in addition to its distant taxonomic relationship to snakes common to Pacific islands, make the brown tree snake an excellent target for biological control.

The taxonomic relationship between colubrids and typhlopids may simplify the task and reduce the cost of finding natural enemies unique to the brown tree snake. Parasites or pathogens that are host specific just to the family (i.e., Colubridae) or genus (i.e., *Boiga*) level may be safe to nontarget snakes (e.g., typhlopids) because these organisms have not evolved the ability to cause disease in distantly related hosts.

Extreme caution should be exercised when implementing a biological control program against vertebrate pests with parasites. Parasites and pathogens can pose major threats to populations of endangered animals (McCallum, 1994; McCallum & Dobson, 1995). The susceptibility of nontarget organisms, especially endemic species, to infection by candidate biological control agents should be investigated thoroughly prior to parasite releases. Reassociating parasites that preferentially infect a competitively dominant pest species may increase species diversity of invaded communities by reducing the pest's prevalence. In this instance, the natural enemy would assume the position of a keystone parasite (Marcogliese & Cone, 1997).

Pathogens as Biological Control Agents

Pathogens or microparasites include viruses, bacteria, and protozoans. Pathogens tend to be unicellular and exhibit epizootic (i.e., boom or bust) life cycles due to rapid proliferation in hosts (Anderson, 1979; McCallum, 1994).

The potential of pathogens to regulate vertebrate population densities by reducing the longevity and fecundity of infected hosts has been demonstrated theoretically with mathematical models and by perturbation experiments using vaccines (Smith, 1994). As with macroparasites, models indicate that microparasites of intermediate pathogenicity are more effective biological control agents (Anderson, 1982). Highly virulent pathogens kill themselves by destroying hosts before they can be transmitted and avirulent strains are not transmitted because they are removed by the immune system. The immune system is theorized as being responsible for maintaining the intermediate virulence of vertebrate microparasites (Anita *et al.,* 1994). Pathogens that are readily transmitted (i.e., microparasites spread by water, air, and vectors) or have high-density host populations are more contagious than those with low transmission rates (i.e., spread is by host-to-host contact) or low host densities (Ebert & Herre, 1996).

New associations between pathogens and novel hosts are generally not more harmful than those that have evolved closely with the host. Experimental evidence indicates that novel disease-causing organisms are on average less harmful, less infectious, and less fit than the same parasite strain infecting the host it is adapted to (Ebert & Herre, 1996). Also, a microparasite's ability to infect and exploit novel hosts decreases with increasing geographic and presumably genetic distance from the host to which the pathogen is adapted (Ebert, 1994). Exceptions do occur, however, and pathogens can have devastating impacts on hosts that have no evolutionary history with the disease organism. An example is the myxoma virus, the causative agent of myxomatosis in European rabbits. The use of this natural enemy against rabbits in Australia and Europe has been the most thorough biological control program against a vertebrate pest.

The myxoma virus is a member of the genus *Leporipoxvirus* (Poxviridae) and originated from South America where it was first recognized as an emerging disease of European rabbits in laboratories in Montevideo, Uruguay, in 1896. Infected laboratory rabbits died of a fatal febrile disease that caused tumors on the head and ears. The tumors resembled myxomas (a benign tumor composed of connective tissue and mucous elements) and the disease was subsequently named infectious myxomatosis of rabbits (Fenner & Marshall, 1957; Fenner & Ratcliffe, 1965; Fenner, 1994).

The indigenous host for myxoma virus in South America is the forest rabbit [*Sylvilagus brasiliensis* (Linnaeus)]. Unlike its effect on European rabbits, myxoma inoculum injected into forest rabbits caused benign fibromas at the site of inoculation that persisted for many months, although death did not occur. Mosquitoes were implicated in vectoring the disease from forest rabbits to European rabbits being bred in South American rabbitries. Another leporipoxvirus has been isolated in California from the brush rabbit, *Sylvilagus bachmani* (Waterhouse), and is closely related to the myxoma virus (Fenner & Marshall, 1957; Fenner & Ratcliffe, 1965; Fenner, 1994; Fenner & Ross, 1994; Ross & Tittensor, 1986).

Myxoma virus has been used in Australia, Europe, Chile, and Argentina for biological control of European rabbits. The virus was first imported into Australia from Brazil in 1919 and 1926 but was not released (Fenner & Ratcliffe, 1965). Work by Australians with the virus began again in the United Kingdom in 1934 and continued with caged rabbits on Wardang Island off the south coast of Australia. The virus was successfully established on mainland Australia in 1950 (Fenner, 1994) and within 2 years it had established itself over most of the rabbit's range (Fenner & Ratcliffe, 1965). The virus initially had a major impact on the estimated 600 million rabbits and on the damage they caused, reducing population density by 75 to 95%. Efficacy was dependent on climate and rabbit population susceptibility. Populations have subsequently increased and stabilized at around 300 million because of myxomatosis.

Damage attributable to rabbits still amounts to $600 million (Australian) annually, including both lost agricultural production and cost of control applications (Robinson *et al.,* 1997). In addition to agricultural losses, rabbits severely affect native flora by eating foliage and inducing wind and water erosion of soils by overgrazing. Native fauna are also affected as rabbits out-compete indigenous herbivores and dense rabbit populations sustain exotic predator populations that feed on native animals (Gibb & Williams, 1994; Myers *et al.,* 1994; Robinson *et al.,* 1997).

Within a few years of the initial panzootic, field isolates of the virus showed less virulence when compared with the original strain that had been released. The original strain killed >99% of laboratory rabbits on average 10.8 days after infection, while circulating strains caused 90% mortality after 21.5 days. Genetic resistance in rabbits was also detected (Fenner & Marshall, 1957; Fenner & Ratcliffe, 1965). Dual natural selection had occurred, the virus had attenuated, and rabbits had increased in resistance to the disease.

Mosquitoes have been responsible for vectoring myxoma virus in Australia. The European rabbit flea, *Spilopsyllus cuniculi* (Dale), an important vector in Europe, was introduced into Australia in 1968 and increased the geographic distribution of the disease. This flea did not persist in areas with rainfall <200 mm. The xeric adapted Spanish rabbit flea, *Xenopsylla cunicularis* Smit, was introduced in 1993 and active redistribution is still ongoing (Fenner & Ross, 1994).

New Zealand also has inordinate numbers of rabbits, and attempts to establish the myxoma virus from 1951 to 1953 failed because of inclement weather and a paucity of suit-

able arthropod vectors. Further attempts at establishment were not undertaken because poisoning programs had reduced rabbits to very low numbers, additional control expenditure was unjustifiable, and the New Zealand public was not in favor of using lethal myxoma virus for rabbit control on humanitarian grounds (Gibb & Williams, 1994).

Until the 1980s, myxomatosis was the only disease known to severely affect rabbit numbers. A second highly contagious viral disease emerged in the mid-1980s and was accidentally introduced onto mainland Australia (O'Brien, 1991). It is the first pathogenic natural enemy to have established in New Zealand for biological control of rabbits. Rabbit calicivirus disease (RCD) [also known as rabbit hemorrhagic disease virus (RHDV)] emerged as a fatal disease in 1984 in Angora rabbits exported from East Germany to Jiangsu Province of China (Liu *et al.,* 1984). In 1986, the disease appeared in Italy where 38 million rabbits were estimated to have died. The disease spread rapidly through rabbit populations in Europe reaching the United Kingdom in 1992 (Chasey, 1994). The probable mechanism for dispersal in continental Europe was the movement of live rabbits and rabbit products. Transmission of RCD from France into coastal areas of southeast England is thought to have occurred by wind-borne aerosols containing virus, birds, and transchannel ferry traffic (Chasey, 1994). Outbreaks of RCD occurred in Mexico in 1988 and 1989 (Gregg *et al.,* 1991) and in Réunion Island in the Indian Ocean in 1989. Movement of RCD to these areas probably occurred with imports of frozen rabbit carcasses from China because the virus can survive freezing to temperatures of −20°C (Chasey, 1994).

The RCD virus belongs to the Caliciviridae and consists of a positive sense, single-stranded RNA genome, enclosed by a sculptured capsid composed of multiple copies of a single major protein of 60 kDa, and is 30 to 40 nm in diameter (Ohlinger *et al.,* 1990; Parra & Prieto, 1990). Disease symptoms are characterized by high morbidity and mortality in rabbits over 8 weeks of age. Younger rabbits often survive infection and may develop antibodies to RCD virus (Nagesha *et al.,* 1995). Clinically, RCD symptoms are expressed after an incubation period of 24 to 48 h in which a febrile response and increasing lethargy are observed. Infected rabbits typically die within 12 to 72 h postinfection and 90% mortality is observed after 5 days. Necropsies show a pale swollen friable liver, enlarged spleen, and clots in blood vessels. Death is ascribed to acute necrotizing hepatitis and possible hemorrhaging (Fuchs & Weissenböck, 1992; Studdert, 1994). However, necropsies close to the time of death show an absence of hemorrhaging and inclusion of hemorrhagic in the name of this rabbit disease that indicates the cause of death is misleading (Studdert, 1994).

A different viral disease is responsible for European brown hare syndrome (EBHS) which causes severe hepatic necrosis in hares (*Lepus europaeus* Pallas and *L. timidus* Linnaeus). The disease was first recorded in Sweden in 1980, and spread through continental Europe and reached the United Kingdom in 1990 (Fuller *et al.,* 1993). In Sweden, losses of hares to EBHS occurred 10 years prior to sympatric rabbit populations developing RCD. Similar observations were made in the United Kingdom where hares began dying from EBHS 2 years before RCD was observed in rabbit populations (Fuller *et al.,* 1993). Electron microscopy, nucleotide sequencing, and experimental cross-transmission studies have indicated that RCD virus and EBHS virus are closely related (Le Gall *et al.,* 1996) but distinct members of the Caliciviridae (Chasey *et al.,* 1992; Nowotny *et al.,* 1997). Disease symptoms are generally similar for rabbits and hares but show distinguishing characteristics in necrosis of liver lobules and clotting of blood vessels (Fuchs & Weissenböck, 1992). Serological studies on rabbit sera collected in 1961 from Czechoslovakia and Austria indicate that RCD virus probably evolved from an apathogenic strain endemic to Europe from at least this time (Nowotny *et al.,* 1997).

Studdert (1994) speculates that the causative agent of RCD probably existed in Europe as a quasi species, a collection of indifferent mutants with a variety of accumulated nucleotide changes. In this scenario, mutants occupied a specific ecological niche until one strain better adapted to prevailing conditions became the dominant member of the population. Adaptation may have occurred because mutations caused increased virulence in an avirulent rabbit virus or increased the host range of hare-infecting viruses by allowing mutant strains to bind more efficiently to surface receptors on rabbit hepatocytes. Given Studdert's (1994) speculative scenario, RCD virus may be a highly evolvable organism.

European rabbits appear to be the only animals susceptible to infection by RCD virus, and vaccines have been developed to protect domestic animals (Boga *et al.,* 1997). Other rabbits including cottontail rabbits (*Sylvilagus* spp.), black-tailed jack rabbits (*Lepus californicus* Gray), volcano rabbits [*Romerolagus diazi* (Ferrari-Pérez)] (Gregg *et al.,* 1991), and hares (Gould *et al.,* 1997) are not affected. The limited host range of RCD virus makes it an obvious candidate for use in a biological control program against European rabbits in New Zealand and Australia. A joint biological control program between these two countries using RCD virus was initiated in 1989 and a strain of virus from the Czechoslovakia Republic was imported into Australian quarantine facilities in 1991 to test effects on nontarget species (Robinson & Westbury, 1996).

Host-specificity testing of 28 nontarget species in Australia for susceptibility to RCD virus further verified the limited host range of this natural enemy. Test subjects in-

cluded domestic lifestock (horses, cattle, sheep, deer, goats, pigs, cats, dogs, and fowls), noxious exotic vertebrates (foxes, hares, ferrets, rats, and mice), native mammals (eight species), birds (five species), and reptiles (one species). There was no evidence for viral replication, clinical signs, or lessions in any organisms tested (Gould *et al.*, 1997). Artificial inoculation of RCD virus in North Island brown kiwis (*Apteryx australis mantelli* Bartlett) and lesser short-tailed bats (*Mystacina tuberculata* Gray), both native to New Zealand, also failed to produce disease symptoms (Buddle *et al.* 1997).

The apparent host specificity of RCD virus to rabbits, rapidity of action, and the capacity to infect rabbits from other rabbits, [through feed and feces, or from a contaminated environment (O'Brien, 1991)] prompted further evaluation of this biological control agent under field quarantine conditions in Australia. Studies monitoring the effects of RCD virus on rabbit populations were initiated on Wardang Island near Adelaide off the south coast of Australia in 1995 (Rudzki, 1995; Robinson & Westbury, 1996). In September 1995, RCD breached quarantine and appeared on mainland Australia, possibly carried there by calliphorid flies and onshore winds (Cooke, 1996; Lawson, 1995). Attempts at containment failed (Seife, 1996). Within 2 months of the initial discovery of RCD virus on the mainland, an estimated 5 million rabbits were killed in South Australia. In dry areas, 80 to 95% of infected populations died (Anderson, 1995) with dead rabbits averaging 15 per hectare. Elsewhere, fatality rates were closer to 65% (Anonymous, 1997b). In the period from October to November 1995, an estimated total of 30 million rabbits died from RCD in South Australia and the majority of surviving rabbits were less than 6 weeks of age (Cooke, 1996). The development of resistance in young rabbits may have profound effects on the long-term population dynamics on the rabbit–RCD virus system. Ten arthropod vectors of RCD virus have been identified and include flies, mosquitoes, and rabbit fleas (Anonymous, 1997b).

Rates of spread of RCD are greatest in spring and autumn at 10 to 18 km a day and are correlated with peaks of insect activity. Dispersal of the disease probably has been assisted by humans moving contaminated material to new areas (Cooke, 1996). Increased attacks on native fauna by exotic predators such as foxes because of declines in rabbit numbers do not appear to have occurred because predator populations have declined with rabbit numbers (Anonymous, 1997b). The virus is now endemic in Australia and will probably be officially declared as a biological control agent under the Biological Control Acts of the Commonwealth and States (Robinson & Westbury, 1996).

RCD virus was smuggled into the South Island of New Zealand by high country farmers in August 1997 and illegally disseminated by feeding rabbits carrots and oats saturated with contaminated liquefied rabbit livers. A network of cooperators spread the virus over large areas of the South Island and its subsequent spread (human assisted through the movement of carcasses, baiting, and insect vectors) made containment and eradication of the disease impossible. Such actions by farmers clearly violated New Zealand's Biosecurity Act, which was enacted in part to protect agriculture from unwanted introductions of pests. The New Zealand government has sanctioned controlled virus releases into new areas. The short-term impact of RCD on New Zealand rabbit populations has resulted in 47 to 66% mortality in central Otago and large-scale field studies are planned (G. Norbury, personal communication, 1997).

Cats on oceanic islands have been subjected to biological control with pathogens. Six cats were introduced onto Marion Island in the Indian Ocean in 1949 (Howell, 1984); by 1977, numbers were in excess of 3000 and were increasing an average of 23% per year (van Rensburg *et al.*, 1987). Populations were sustained by consuming approximately 450,000 seabirds yearly and cats were probably responsible for the local extinction of the common diving petrel *Pelecanoides urinatrix* (Gmelin) (Bloomer & Bester, 1992). Surveys of cats on Marion Island revealed the presence of feline herpes virus and feline corona virus, but the highly contagious feline parvo virus was absent in the population (Howell, 1984).

Initiation of a biological control program with feline parvo virus, the causative agent of feline panleucopenia, began in 1977 with the release of 93 artificially inoculated feral cats collected from the island (Howell, 1984). The disease reduced cat numbers by 82% after 5 years by reducing fecundity and increasing mortality of juvenile cats (van Rensburg *et al.*, 1987). Virions found in high concentrations in feces, urine, saliva, and vomit were transmitted through direct contact between cats or contact with contaminated objects (Howell, 1984). Annual declines of cat numbers were 29% from 1977 to 1982. This rate decreased to 8% per year from 1981 to 1983 and was accompanied by lower titers of virus in serum samples collected from feral cats, indicating that viral efficacy was decreasing (van Rensburg *et al.*, 1987). At reduced densities, hunting and trapping became viable and have been incorporated into an ongoing eradication program that may be assisted by the use of trained dogs (Bloomer & Bester, 1992).

Sexually transmitted diseases have adverse effects on domestic and wild vertebrates by reducing survival, conception rates, and numbers of offspring born and successfully weaned (Smith & Dobson, 1992). Rabbits are susceptible to infections of venereal spirochetosis (*Treponema cuniculi*), which causes sterility (Smith & Dobson, 1992). Goats can develop trichomoniasis, a sexually transmitted disease caused by the flagellated protozoan *Trichomonas foetus* (Reidmuller). This pathogen has been suggested as a bio-

logical control agent for goat populations on oceanic islands that lack this microparasite (Dobson, 1988).

NEW AVENUES FOR BIOLOGICAL CONTROL OF VERTEBRATES

Sexual transmission of diseases may further guarantee host specificity in biological control programs. It also enhances the ability of parasites and pathogens to persist in low-density populations or solitary species (e.g., predators). The rate of spread of sexually transmitted organisms is tightly correlated with mean and variance of the numbers of sexual partners per host because of the need for host-to-host contact (horizontal transmission) for transmission. Host population density is not important with respect to persistence or rate of spread of sexually transmitted diseases. This property, coupled with asymptomatic carrier states, long infectious periods, or vertical transmission (infective propagules are passed from mother to offspring), greatly enhances the ability of pathogens to persist in low-density host populations (Smith & Dobson, 1992).

Because sexually transmitted organisms can persist in low-density populations or populations of declining density, the potential of genetically engineering sexually transmitted viruses to sterilize infected hosts is being investigated (Barlow, 1994). Viruses that have antigens from the host sperm, or the zona pellucida around host eggs engineered into the genome provoke an immune system response that renders the recipient sterile. Immunocontraception (also referred to as immunosterilization) as a means to control noxious vertebrates is being actively pursued by Australia and New Zealand (McCallum, 1996). An alternative approach to immunocontraception is to use genetically modified microparasites to prevent lactation in females so that juveniles are not successfully weaned or to interfere with hormonal control of reproduction (Cowan, 1996; Jolly, 1993; Rodger, 1997).

Immunocontraception for Control of Vertebrate Pests

Many species that become pests are distinguished from nonpestiferous species by their higher intrinsic rates of increase (r_m). Pest vertebrates have high r_m values characterized by large litters, and by maturing sexually at young ages. Agents that reduce reproductive rates may be more effective for control than mortality-inducing biological control agents are because resistance development should take longer to occur and population recovery would be slower (Tyndale-Biscoe, 1994b). Resistance development may be further delayed by combined use of multiple agents that affect fertility in different ways [e.g., using agents that cause sterilization, alter levels of reproductive hormones, or affect lactation (Cowan & Tyndale-Biscoe, 1997)]. In sexually reproducing vertebrates, proteins associated with male and female gametes are potentially foreign antigens in the opposite sex. Exposure to reproductive antigens occurs when females receive sperm and accessory fluids from males during copulation. As a general rule, females do not develop antibodies to these antigens because physiological and immunological mechanisms have evolved to prevent this (Robinson & Holland, 1995). Inoculation of sperm into females of the same species either subcutaneously or intramuscularly produces high sperm antigen antibody titers in recipients. In most cases, this causes either permanent or temporary infertility in females. Such results indicate that sperm antigens in the reproductive tract are tolerated and that exposure to these antigens by different routes overcomes protective mechanisms, with infertility resulting (Robinson & Holland, 1995).

Sperm antibodies in females that can arise from either systemic or local immune responses are found in cervical mucus, genital fluids (e.g., endometrial, tubal, and follicular fluids), and blood. Antibodies bind to sperm, often in specific locations such as the head, midpiece, tail shaft, or tail tip. Once bound to sperm, antibodies cause agglutination (e.g., irreversible binding to cervical mucus that normally aids sperm transport) or immobilization of sperm. Antibodies may also interfere with acrosome reactions preventing ovum penetration and fertilization, or they block the binding of sperm to the zona pellucida (Shulman, 1996).

The zona pellucida (zona) that surrounds growing oocytes and ovulated eggs is antigenic and available to circulating antibodies during oocyte growth and ovulation. Nonreproductive tract inoculation of females with zona preparations leads to infertility (Millar *et al.,* 1989). Antibodies produced in response to administered zona antigens bind to the zona and prevent sperm penetration (Millar *et al.,* 1989). Zona glycoproteins are highly conserved among mammals, for example, nonspecific pig zona preparations cause infertility in humans, primates, dogs, rabbits, horses, and deer (Robinson & Holland, 1995). A major objective in immunocontraception research is isolation of species-specific zona glycoproteins that do not cause sterility induced by immune response in species from which zona preparations were not derived. Low variability among zona glycoproteins may limit the number of species-specific zona preparations for immunocontraception (Millar *et al.,* 1989).

Immunocontraception for wildlife population control has been successfully implemented for horses (*Equus caballus* Linnaeus) (Kirkpatrick *et al.,* 1992, 1997). Free-ranging feral mares inoculated by dart gun with porcine zona pellucida showed depressed urinary estrogen concentrations and failure to ovulate. Zona booster inoculations given 2 years after initial inoculations prevented conception in

treated horses for a third year compared with control populations that were not vaccinated. Contraceptive effects were reversible after 4 years of consecutive treatment, but prolonged treatment (5 to 7 years) with zona preparations caused irreversible ovarian dysfunction and fertility loss (Kirkpatrick *et al.,* 1992, 1997). Similar results have been achieved with porcine zona pellucida inoculations in whitetail deer [*Odocoileus virginianus* (Zimmerman)] (Kirkpatrick *et al.,* 1997).

Gametic antigens that induce immune response can be administered by baits that are ingested by target organisms or can be inoculated directly into hosts with darts or bullets (Tyndale-Biscoe, 1994b). Injection of foxes with sperm antigens reduces fertility from 75 to 35%. Baits are considered the favorable method for delivering antigens to foxes in Australia. Potential baits include dried meats that contain microencapsulated antigens. Use of recombinant bacterial vectors (e.g., *Salmonella typhimurium*) also are being considered. An orally administered agent needs to reach the lower gastrointestinal tract to stimulate a response in the common mucosal immune system in the gut-associated lymphoid tissue. This in turn induces mucosal immunity in the reproductive tract of female foxes and causes sterilization (Bradley *et al.,* 1997).

At present, an effective bait specific to foxes that is environmentally stable and easy to manufacture has not been developed. Nontarget impact is a concern because most antigens exhibit some specificity to the family level only. Effective vaccines for rabies have been delivered as oral baits to foxes in Europe, demonstrating the baiting technique is an effective dissemination method (Bradley *et al.,* 1997). Models indicate density-independent factors such as drought and rain (which affect pasture growth and rabbit numbers) strongly influence the effectiveness of bait-delivered fertility control in reducing fox abundance (Pech *et al.,* 1997).

An alternative proposal to deliver antigens orally is to develop transgenic plants to produce and deliver gametic antigens in palatable form to herbivorous pests. Plants could be sown over target areas and allowed to become self-propagating vaccines. Transgenic seeds, fruits, or leaves (e.g., transgenic carrots or maize) could be harvested and used as oral baits delivered to specific sites such as fenced watering points that allow pest animals access while excluding lifestock (Smith *et al.,* 1997).

Baiting is an expensive form of control that requires monitoring of dosage and uptake rates and multiple area-wide applications. Problems of hormonal modification of behavior and delayed population control are additional drawbacks. One advantage is that baits can be used to treat localized populations that are problematic. Similar shortcomings exist with antigen inoculations by projectiles where cost estimates are significant. To control the estimated 300,000 wild horses in Australia with dart-delivered porcine zona pellucida would cost $20 (Australian) per horse per year compared with 50 cents for permanent control with a bullet (Tyndale-Biscoe, 1991). Lethal methods of control provide immediate impact on pest populations and reduce pest status rapidly, with control being quickly observable. In contrast, fertility impairment is not immediate, population responses are delayed, and large proportions of populations need to be sterilized for this technique to be effective. Large-scale distribution of gametic antigens might be possible through releases of host-specific microparasites expressing species-specific antigen genes (Tyndale-Biscoe, 1994a, 1994b). Self-spreading and replicating parasitic vectors that have been genetically engineered and that may require periodic reinoculation into populations are analogous to augmentative biological control programs with traditional natural enemies (e.g., parasitoids, predators, or pathogens) released periodically for the control of pest arthropods.

Host-specific viruses carrying foreign DNA could be cheap and effective biological control agents that have the potential to disseminate widely by sexual transmission, contagion, or arthropod vectors. The selected micro- or macroparasite must be able to carry foreign DNA coding for gametic antigens, as well as promoters to express foreign genes and cytokines to enhance effectiveness (Tyndale-Biscoe, 1994b). Such agents must be able to reduce growth rates of infected populations and to maintain reproductive rates at lower levels (Caughley *et al.,* 1992), and should not interfere with sexual behavior or social organization (Caughley *et al.,* 1992; Robinson & Holland, 1995; Tyndale-Bisoce, 1994b). With some pests such as rabbits and foxes, dominant members of populations make the main contribution to reproduction and inhibit breeding by subordinate members by occupying prime territories.

Ideally, a sterilizing agent should not change social hierarchies by allowing individuals with lower social status to successfully rear more offspring because this will cause pest populations to increase substantially (Caughley *et al.,* 1992). Genetically engineered agents should sterilize females because models predict greater population suppression with infertile females than with sterilized males (Barlow, 1994; Caughley *et al.,* 1992). In the absence of arthropod vectors, sexually transmitted diseases engineered to cause sterilization are superior to nonsexually transmitted ones because multiple matings with sterilized females increases contact rates and the competitive ability of the engineered agent with nonsterilizing strains. The impact of immunocontraception is further enhanced if the sterilizing agent causes limited host mortality and there is low naturally occurring immunity to sexually transmitted diseases (Barlow, 1997).

Sexually transmitted herpes-type viruses are being proposed as vectoring agents to induce sterilization in brushtail possums in New Zealand (Barlow, 1994; Barlow, 1997).

The recently identified borna disease virus that causes wobbly possum disease in New Zealand may be a suitable alternative to a herpes virus (Atkinson, 1997).

The myxoma virus and murine cytomegalovirus are being investigated as gamete antigen delivery agents for rabbits and mice, respectively, in Australia (McCallum, 1996; Tyndale-Bisoce, 1994b; Shellam, 1994). Four potential insertion sites for genes coding for gametic antigens have been identified in myxoma virus and recombinants have been constructed to express two *Esherichia coli* (Escherich) enzymes and influenza virus hemagglutinin genes. The ability of a novel myxoma virus to compete and spread among existing myxoma strains in field situations has been demonstrated by monitoring the spread of virus containing identifiable gene deletions (Robinson *et al.*, 1997). The myxoma virus that can express foreign genes may operate as a vector for gametic proteins (Robinson *et al.*, 1997). Work is continuing on isolating and inserting rabbit gamete antigen genes into the myxoma virus genome (Robinson *et al.*, 1997).

The responses of experimental rabbit and fox populations in Australia to imposed sterility by surgical ligation of fallopian tubes in females have been studied in an attempt to simulate the effects of virally mediated immunocontraception after recombinant virus establishment in wild populations. This technique prevents conception among predetermined proportions of females in populations without interfering with hormones or reproductive behavior (Williams & Twigg, 1996). The dynamics of 12 rabbit populations enclosed by rabbit-proof fencing that exhibited 0, 40, 60, or 80% sterilization of females were studied in each of two locations in western and eastern Australia where climate patterns differed. Females born into treatment populations were trapped and sterilized to maintain the same overall sterility levels (Williams & Twigg, 1996). Juvenile rabbits born into populations with sterilized females exhibited greater survivorship because of lowered competition for resources. This greater survival compensated for decreased fertility, but recruitment rates were ultimately constrained by environmental factors (e.g., depletion of vegetation). In populations with 80% sterility, reduced juvenile mortality did not compensate fully for lowered reproduction, smaller numbers of rabbits were recruited into these populations, and numbers subsequently declined. These results indicate that levels of sterilization with a genetically altered micorparasite have to reach at least 80% to achieve reductions in population density (Williams & Twigg, 1996).

Surgical sterilization does not affect reproductive behavior in treated populations. Sterile dominant female rabbits maintain hierarchical dominance, increased body weight over control females, continued to defend prime territory, and engaged in normal reproductive behavior including breeding burrow construction (Tyndale-Bisoce, 1994b). Birth rates of sexually mature females were in direct proportion to the level of fertility in experimental populations, indicating that fertile females did not respond to female infertility or decreased densities of young by producing larger litters (Williams & Twigg, 1996).

Sterilized females tended to live longer than unsterilized females. This increased longevity suggests that sterile females may proportionately increase as treated populations reach an equilibrium density. Obviously, larger proportions of sterile females reduce population productivity and the numbers of fertile females that a sterilizing microparasite would have to infect and sterilize. Higher proportions of sterile females may reduce numbers of infective individuals harboring sterilizing microparasites and numbers of vectors (e.g., fleas that would spread an engineered myxoma virus), and may contribute to decline of transmission rates. These interactions need to be clarified and mathematical models may be of use here (Williams & Twigg, 1996).

Engineered microparasites that sterilize pest animals offer the possibility of humane control without killing or causing animals to suffer the effects of debilitating disease. As a form of biological control, immunocontraception may also reduce the need for broadcast distribution of toxins for pest suppression, thereby reducing environmental contamination and nontarget mortality. This is of special concern when pests inhabit suburbs, urban parks, government and state campuses, nature reserves, military bases, or other areas where lethal controls may no longer be legal or safe (Kirkpatrick *et al.*, 1997; Williams, 1997). The concept of virally mediated immunocontraception has generated considerable debate on legal and ethical issues concerning releases of engineered microorgansims into the environment. Once contagious recombinant agents that cause permanent sterilization in animals are released into the environment they cannot be recalled (Tyndale-Bisoce, 1995).

Several potential risks are recognized. First, engineered viruses that are host specific and contain species-specific antigens could mutate and infect and sterilize nontarget species after release (Anderson, 1997). Under such conditions it may be difficult if not impossible to contain and eradicate a mutant virus from an infected animal population that is abundant, secretive, and free ranging. Second, sterilizing viruses either might cross international boundaries accidentally or be maliciously moved to sterilize desirable organisms in new areas (Tyndale-Bisoce, 1994b). For example, viruses engineered with little host specificity to sterilize widely dispersed marsupial pests in New Zealand may enter Australia and infect endangered wildlife (Rodger, 1997; McCallum, 1996); engineered myxoma viruses may spread from Australia into the Americas and sterilize native rabbit species (Tyndale-Biscoe, 1995).

Third, dart-delivered contraceptives used for wildlife control in the past have had adverse effects on individuals within target populations. Changes in morphology of repro-

ductive organs, secondary sexual characteristics, and behavior have been observed. Viruses that induce sterility could alter genetic profiles of target populations because infectious agents may act as a new reproductive disease and individuals may exhibit differential susceptibility (Nettles, 1997). Fourth, public concerns over the use of viruses and genetic engineering indicate substantial apprehension about the use of sterilizing viruses for pest management, these fears that need to be fully alleviated may delay or prevent field trials and widespread application (Lovett, 1997).

Despite potential drawbacks, immunocontraception is a potentially cost-effective method for reducing pest impact on endangered native species (Sinclair, 1997) and on agricultural yields, and is an additional tool for sustainable pest management (Williams, 1997). A sterilizing agent that does not cause painful disease symptoms is an ethically acceptable form of pest control that is justifiable from animal rights perspectives, because it does not cause the suffering typical of current lethal methods (e.g., trapping, shooting, poisoning, and introduced disease) (Oogjes, 1997; Singer, 1997). Under certain circumstances, the use of vectors to disseminate genetically engineered viruses is warranted (McCallum, 1996). Experience with the myxoma virus in Australia indicates that it has not been deliberately or accidentally spread to any other country since its introduction in the 1950s because of either the lack of suitable arthropod vectors or the inability of the virus to establish where different strains are already present. This history may indicate possible difficulty for unintentional establishment of genetically engineered microparasites in new areas, and establishment of engineered myxoma viruses may be possible only with carefully timed and repeated releases into rabbit populations (Tyndale-Biscoe, 1995).

However, such safeguards may be moot if a highly competitive sterilizing strain is engineered and released. Quarantine legislation designed to prevent accidental or intentional but illegal importation of unwanted organisms would be exercised by countries under current international obligations and should impede establishment in new countries if rigorously enforced. However, current legal safeguards may be insufficient. New Zealand's experience with RCD indicates it is possible for lay people to illegally import and establish reproducing populations of exotic pathogens. In Australia, RCD breached a carefully planned quarantine on an offshore island. Unintended establishment and proliferation of engineered viruses may be contained if outbreaks are recognized early, and if proportions of susceptible individuals are removed rapidly from the population either by culling or by immunizing against the pathogen (Tyndale-Biscoe, 1995). This has never been tried with wild animal populations. The containment of contagious pathogens, such as foot and mouth disease in livestock, indicates such an approach may be possible. Highly attenuated forms of myxoma virus are used to protect wild and domestic rabbits in France and the United States, indicating the availability of such technology for this virus at least (Fenner & Ross, 1994; Tyndale-Biscoe, 1995). Limited field trails with sterilizing microorgansisms are unlikely before 2005 (Anderson, 1997).

REGULATING VERTEBRATE INTRODUCTIONS

There is abundant evidence that introduced exotic vertebrates that establish feral reproducing populations have disastrous consequences for agriculture and preservation programs for native plants and animals. Sources of current vertebrate introductions include sellers and buyers of exotic pets; acclimatization societies that import, establish, and relocate game animals and whose constituents include hunters and fishermen; and farmers and ranchers who import and experiment with novel lifestock (e.g., fitch farming). Exotic vertebrates have in some instances great economic importance (as with lifestock and game animals), they also enjoy public popularity because of interest in hunting, fishing, eating, or viewing large and unusual animals in familiar environments. The negative ecological aspects of introduced vertebrates may be poorly understood by the public at large. Such limited understanding may hinder control efforts and prevention of importation (Bland & Temple, 1993).

Legislation has been passed in the United States to minimize risks of importing new and relocating existing vertebrate species. The Lacey Act passed in 1900 and ammended in 1981 was enacted to protect certain animals and endangered habitats, and to prevent introduction of noxious pests. Under the act, violation of the law can result in fines and imprisonment [see 18 USC §42; Importation or shipment of injurious mammals, birds, fish (including mollusks and crustacea), amphibia, and reptiles; permits, specimens for museums; regulations—for more details]. Similar legislation has been developed in New Zealand. The Biosecurity (1993) and Hazardous Substances and New Organisms (HSNO) (1996) Acts were devised to protect the environment by preventing or managing the adverse effects of hazardous substances and exotic organisms.

Campbell (1993) points out that existing laws have many loopholes and are not effective when applied, indicating a need to improve existing regulations and to develop new laws to curtail unwanted entry by alien vertebrates. One proposal is to require importers of exotic organisms to develop "clean lists" and to prove that organisms are not potentially invasive and disrupting to native ecosystems (Campbell, 1993). Legislative approaches limiting imports and exports of organisms may encounter complaints under the General Agreement of Tariffs and Trade (GATT) that stricter quarantine measures are an unacceptable imposition of one country's environmental standards on others (Camp-

bell, 1993). There is an obvious need for greater cooperation among interest groups, scientists, and legislators to devise solutions to problems associated with continuing introductions of exotic species and to provide direction for future action.

BIOLOGICAL CONTROL OF EXOTIC PESTS AS AN EVOLUTIONARILY STABLE CONTROL STRATEGY

Development of resistance (behavioral or physiological) to pesticides (e.g., rodenticides) by vertebrates, and the need for repeated or multiple simultaneous control strategies (e.g., poisoning combined with trapping and hunting) indicate that control of vertebrates is an ongoing endeavor that attempts to reduce agricultural damage and losses (Greaves, 1994) or to protect wilderness areas (Cowan, 1992; Morgan *et al.,* 1986; Payton *et al.,* 1997) from pest damage. Biological control has several advantages over chemical and cultural control practices (Van Driesche & Bellows, 1996): (1) it is relatively cheap and biological control programs are often quicker to implement than to develop and to register new pesticides; (2) use of carefully screened natural enemies increases selectivity of attack toward target pests; (3) natural enemies in many instances are self-perpetuating and self-distributing; and (4) development of resistance to natural enemies is extremely rare.

One documented case of pests developing resistance to natural enemies is the development of resistance to myxomatosis by rabbits and corresponding attenuation of highly virulent strains of the myxoma virus to strains of intermediate virulence (Fenner & Ross, 1994). The myxoma virus–rabbit system in Australia and Europe is dynamic with increasing rabbit resistance selecting for more virulent strains of virus. This suggests that for the short-term, at least, the system is coupled in an antagonistic coevolutionary arms race (Dwyer *et al.,* 1990).

Flexible natural-enemy behavior patterns and physiology have the potential to weaken evolutionary responses that can cause pest resistance to introduced control agents (Holt & Hochberg, 1997; Jervis, 1997). In comparison, pesticides and cultural controls tend to target a fixed physiological or behavioral function or pattern, and the resulting selection regime is constant allowing pests either to increase tolerance to poisons or to learn and develop avoidance behaviors (e.g., bait and trap shyness).

Spatial heterogeneity of natural-enemy attack limits selection pressure on hosts by natural enemies, thus reducing the rate of resistance development by pests compared with uniformly applied selection pressures such as pesticides. Pests that escape attack move into enemy-free areas and continue breeding; thus, the rate of coevolution is reduced by susceptible pests in transient refuges (Jervis, 1997). At the metapopulation level, natural enemies may be ineffective selection agents because of widespread extinction and establishment of pest subpopulations that maintain pest susceptibility. Additionally, resistance development may involve costs leading to a corresponding decrease in fitness. For example, increased tolerance to attack may reduce the pest's reproductive capacity and ability to compete for resources, or may increase susceptibility to other mortality agents (Holt & Hochberg, 1997).

There are opportunities to enhance biological control programs against vertebrate pests that cause social, agricultural, and conservation problems. In many instances, biological control offers the best chances for long-term control, particularly in isolated areas with rugged terrain, in suburban areas with high-density human populations, or in places where pests are nocturnal or secretive. Biological control will not totally alleviate vertebrate pest problems. It may, however, reduce the vigor of pest populations, thereby reducing damage, minimizing nuisance value, or allowing native species to compete more effectively for food and breeding sites. Programs could be initiated to simply reassociate host-specific micro- and macroparasites with pest populations that have depauperate natural-enemy faunas (Dobson & May, 1986), and there is no shortage of targets as small founding populations of vertebrates continue to invade and proliferate in new habitats. Genetically engineered natural enemies are additional tools to aid biological control efforts. Research with agents that cause immunocontraception will likely diversify as advances in molecular biology continue, and routes alternative to sterilization may be taken. This area of vertebrate biological control will be tested more thoroughly once small-scale and long-term field trials begin with sterilizing microorganisms.

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