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The genus Mus as a model for evolutionary studies
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One hundred years of eruptions of house mice in Australia – a natural biological curio

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The house mouse has adapted well to the cereal crops of south-eastern Australia where populations show aperiodic outbreaks over large areas. A 20-year population study has provided a wealth of information on breeding ecology, demographic changes, spatial behaviour and epidemiology. The breeding season can be as short as 4.5 months and as long as 10 months with litter size changing seasonally from high values in spring to low values in autumn. There are marked changes in litter size between years. Rates of increase of populations also vary between years. The rate of change of populations during the breeding season is independent of density effects, but if the population density is high at the commencement of breeding then the litter size is depressed throughout that breeding season. There are density-dependent effects on survival during the non-breeding season. Rates of increase of populations over spring and summer are highly correlated with accumulated rainfall from the previous winter–spring (April–October). Studies of helminths and viruses indicate that Australian mice carry only a subset of the helminths found in Europe. There have been no published studies on murine viruses in Europe. Perhaps a reduced diversity of diseases partially accounts for the ability of mice to increase rapidly to extreme population densities in cereal-growing areas of south-eastern Australia. *Biological Journal of the Linnean Society*, 2005, **84**, 617–627.

ADDITIONAL KEYWORDS: disease – mouse plagues – management – population ecology – social behaviour.

MOUSE POPULATION OUTBREAKS – 100 YEARS ON

The house mouse (*Mus domesticus* Schwarz & Schwarz) has adapted to the cereal crops of south-eastern Australia where its populations erupt in aperiodic outbreaks ('plagues') over large areas (Mutze, 1989; Singleton & Redhead, 1989). A mouse plague in Australia is typified by synchronous eruptions of populations to densities >800 mice ha^{-1} over thousands of square kilometres (Table 1) that have significant impact on farmer livelihoods (Caughley, Monamy & Heiden, 1994). These outbreaks are unique to Australia, with the exception of reports of occasional outbreaks in the north-west plateau region of China, although the species there is *Mus musculus* (Redhead, 1988). House mouse populations also erupt in native beech forests in New Zealand, but peak densities only reach around 50 mice ha^{-1} (Ruscoe, Goldsmith & Choquenot, 2001). The year 2003 was the 100th anniversary of recorded mouse plagues in Australia. It is therefore timely to review why the house mouse has been so successful in Australia since its introduction following European settlement in 1788.

A 20-year study of mouse populations at Walpeup in south-eastern Australia has provided a wealth of information on their breeding ecology, demographic changes, spatial behaviour and epidemiology. The findings from this long-term study, and associated

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Table 1. Definitions of different phases of mouse populations in Australian cropping zones based on estimates of absolute density and trap success (measured by Caughley adjusted frequency-density index, Caughley, 1977). TN = trap nights

Descriptor	Absolute density[1,2,3] (mice ha^{-1})	Trap success[4] (no. per 100 TN)	Comments
Low population	0–50	0–8	No economic losses
Outbreak	50–200	8–32	Low losses to cereal crops; high losses to vegetable crops, stored grain, townships
Minor plague	200–800	32–125	High economic losses over hundreds of km^2
Major plague	>800	>125	Severe economic losses over thousands of km^2; maximum densities around 2700 mice ha^{-1}

[1]Saunders & Robards (1983); [2]Singleton (1989); [3]Singleton *et al*. (2001b); [4]Davis *et al*. (2003a).

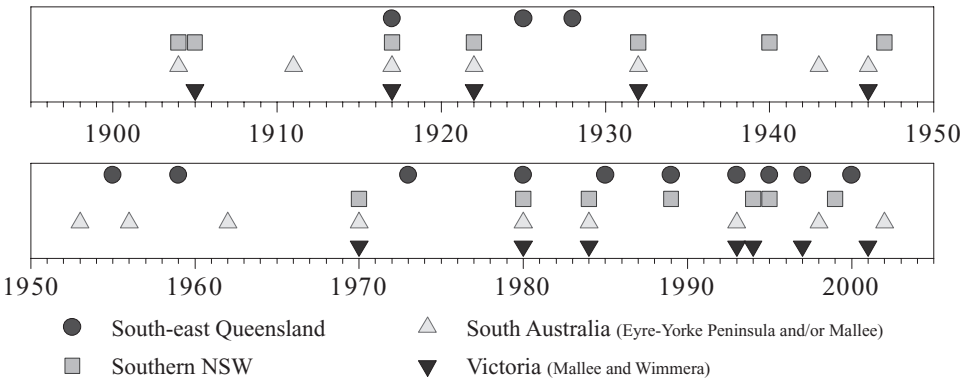

Figure 1. History of reports of mouse plagues in Australia based on a compilation of data from newspaper accounts, Government reports, published literature and our personal experiences since 1975.

short-term experimental studies, will provide the backbone of this review of the ecology of house mice in Australia.

Occurrence and distribution of mouse plagues

A 100-year chronology of mouse plagues was compiled from a detailed search of the major metropolitan (Melbourne, Victoria) and regional newspapers [western and central Victoria; southern New South Wales (NSW)] from 1890 to 1975, from annual reports of the Victorian Department of Agriculture from 1930 to 1975 (see Singleton & Redhead, 1989), from reviews by Saunders & Giles (1977) for NSW, Mutze (1989) for South Australia and Cantrill (1992) for Queensland (Fig. 1). Since 1975 mouse plagues have been recorded directly by us. There were reports of outbreaks of house mice with little detail in 1890 and 1894 (Mutze, 1989) with the first widespread mouse plague reported in NSW and South Australia in 1903/04 (Plomley, 1972; Saunders & Giles, 1977) and the first plague in Victoria occurred in the following year (Natural History Notes, *Victorian Naturalist* 1905; *Swan Hill Guardian* 1904; Singleton & Redhead, 1989). The first major mouse plague in south-east Queensland did not occur until 1917 (Plomley, 1972).

Table 2. Summary of the intervals (years) between mouse plagues in south-eastern Australia prior to and after 1980

	Mean	95% CI	Median	Range
Prior to 1980	9.9	7.7–12.1	9	3–26
After 1980	4.6	3.5–5.6	4	2–9

CI, confidence interval

The following patterns are apparent from the national dataset (Fig. 1). First, a mouse plague occurs on average about every 3.5 years (31 plagues in 100 years) in Australia with the frequency of occurrence for any particular state being about once every 7 years (12–15 plagues in 100 years). Second, there is no apparent periodicity to plagues (Table 2). Third, on only seven occasions were there concurrent outbreaks in three or more states (accounts for only 23 of 54 plague years across the states) (see also Kenney *et al*., 2003). Rainfall and food supply appear to be common factors driving population outbreaks, but major rain-

fall events are often spatially variable. In addition, other mechanisms such as soil type, cover (modifying predation effects) and agricultural systems (e.g. summer crops) that influence the development of mouse plagues can be different for each state, thereby affecting whether a minor plague develops into a major plague. Fourth, the frequency of mouse plagues has increased significantly in some states since 1980 (see Table 1; Singleton & Brown, 1999). This temporal trend is confirmed by a 10-year rolling mean of the frequency of plagues per year: this value was 0.2 in 1920, 0.3 in 1960 and 0.6 in 2000. Likely reasons for this change are increased diversity of cropping leading to different planting and harvesting times, resulting in more high-quality food available to mice for longer periods; increased cropping intensity with less land left fallow for 1 or 2 years; minimum tillage and direct seeding leading to more cover and less disturbance of mouse habitat; reduced sheep production in the cropping zone resulting in less competition for food (both unharvested grain and grass seeds along margins of crops); factors linked to climate change, particularly changes in rainfall patterns; greater awareness of mouse plagues (although this is unlikely given that plagues generally occur over large geographical areas and have received broad coverage in the media from 1903 onwards). Krebs *et al*. (2004) consider some of these factors in more detail.

Mouse plagues occur in regions where the winters are typically mild; however, they have been reported across a wide range of soil types, rainfall regimes and cropping systems (Fig. 2). In the south, the soils are often sandy-loams, winter rainfall predominates and the main crops are winter cereals with no summer cropping except in irrigated areas (which account for <1.5% of agricultural land). In the north, the soils are typically dark cracking clays, summer rainfall predominates and both winter cereals and summer crops (oilseeds, coarse grains) are grown.

One curious finding is that widespread mouse plagues rarely occur in the wheat belt of Western Australia, where about a third of the Australia cereal production occurs. There have been occasional reports of localized outbreaks of mouse populations that have caused significant economic and social problems: in the early 1960s (Western Australia Museum archives cited in Plomley, 1972), in 1975 (Chapman, 1981) and local foci in 1998 and 2000 (G. Martin & L. Twigg, pers. comm.). The recent foci are thought to be associated with the adoption of minimum tillage followed by direct seeding of lupins or canola into ungrazed wheat stubbles. The hot dry summers in this region may be

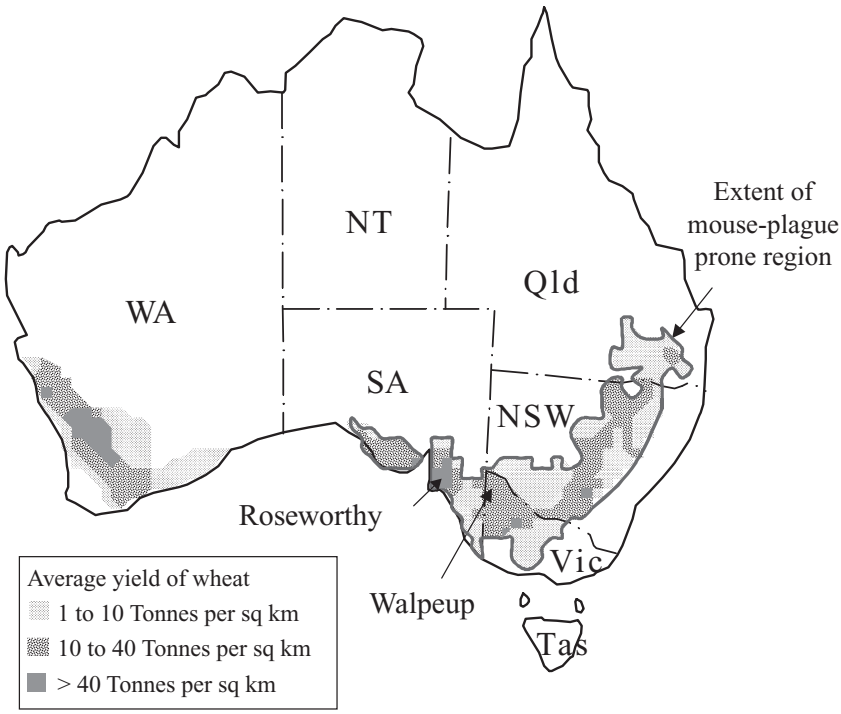

Figure 2. Distribution of the major cereal production areas in Australia, extent of the region subject to periodic mouse plagues (enclosed by the solid line) and the locations of Roseworthy, South Australia, and Walpeup, Victoria, where long-term mouse population studies have been conducted. Different levels of shading represent average yield of cereal crops (after Australian Bureau of Statistics, 2003).

an important limiting factor for mice. On an annual basis potential evapotranspiration in the Western Australian wheat belt is larger than that in semi-arid cropping regions of south-eastern Australia (1200–1300 mm in the west vs. 1000–1200 in the east) (Wang, 2001).

An outbreak of mice was reported in Tasmania for the first time in 2003 (M. Statham, pers. comm.).

POPULATION ECOLOGY

Pech *et al*. (1999) reviewed ten models (including their own model of the numerical response of mice) developed to describe the initiation of mouse plagues; these included three regional (large scale $>10^5$ km^2), five district (at the scale of local environmental variables such as soil type and land use) and two process models. Rainfall and habitat characteristics, each influencing food supply, availability of nesting sites and perhaps protection from predators, emerged as important factors that influence the population dynamics of mice (e.g. Newsome, 1969a; Redhead, 1988; Singleton, 1989; Twigg & Kay, 1994). Brown & Singleton (1999), Pech, Davis & Singleton (2003) and Stenseth *et al*. (2003) have examined further the ontogeny of outbreaks, focusing on factors that influence the rate of increase of mouse populations. These later studies have provided better estimates of the likelihood of mouse plagues and a stronger understanding of management strategies. In each case they analysed various aspects of a long-term population study of house mice at Walpeup (35°08′S, 142°01′S), Victoria, in the wheatlands of south-eastern Australia. The following sections focus on some of the key ecological findings to emerge from the set of population studies undertaken in this region. Pech *et al*. (1999) provide a good overview of population studies of mice elsewhere in Australia and the findings of these studies are referred to where appropriate.

LONG-TERM CHANGES IN POPULATION ABUNDANCE – GEOGRAPHICAL SIMILARITIES

There are very few long-term continuous population studies of house mice. However, two concurrent 20-year studies in Australia – one at Walpeup and the other at Roseworthy in South Australia (500 km to the west) (Fig. 3) – show that the respective mouse populations have had qualitatively similar trajectories. This offers hope that a generic method for forecasting mouse plagues can be produced for south-eastern Australia. Towards this end, a qualitative model based on different combinations of lagged winter and spring rainfall, and a quantitative model that also included mouse abundance in spring, were tested on the two data sets (Kenney *et al*., 2003). These approaches were based on the model developed by Pech *et al*. (1999) for predicting the trajectory of mouse populations in the Walpeup region. The Pech model is based on predicting the rate of increase of the mouse population given a starting density near the beginning of the breeding season (October) and the antecedent April–October rainfall (which is positively correlated with crop yield and is an index of food supply in spring and early summer). However, the standard Pech model was not a good predictor of the population trajectory at Roseworthy. The qualitative model provided 70% correct predictions. However, both models were poor at predicting the maximum density of the mouse population during a plague (Kenney *et al*., 2003). The comparison of the two data sets confirmed that winter–spring rainfall was the key driver for mouse

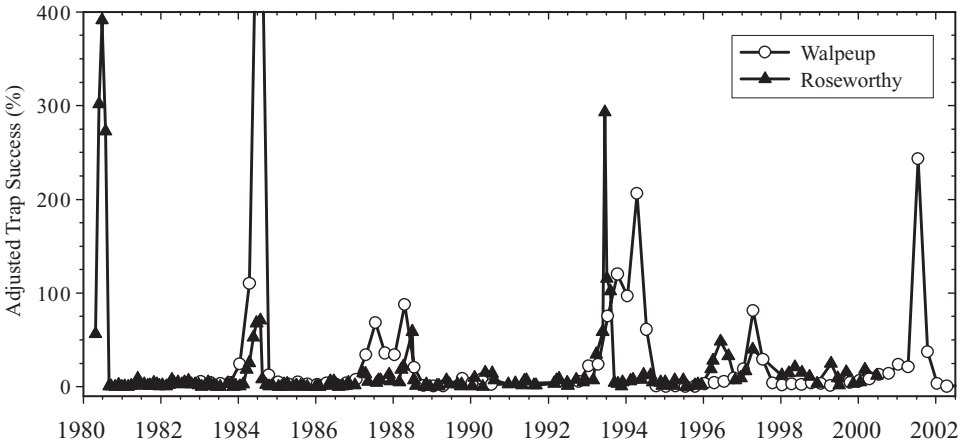

Figure 3. Comparison of mouse population abundance at two long-term study sites at Walpeup and Roseworthy in south-eastern Australia. Trap success per 100 trap nights was adjusted with a density-frequency transformation (after Caughley, 1977: 20).

plagues but the underlying mechanism was not identified.

Local differences in population dynamics

A 3-year study monitored changes in mouse population abundance every 6–10 weeks at three sites in Victoria and two in South Australia (G. R. Singleton, unpubl. data). Each site was at >100 km from its nearest neighbour, with some sites up to 300 km apart. The model developed by Pech *et al*. (1999) successfully predicted a population outbreak 5 months in advance in 2001 and low mouse densities in 2002 and 2003 for the region in the vicinity of Walpeup. However, this predictive model was less useful for the other four sites. For example, mouse densities were low at three of the five sites in 2001. Therefore, although the Walpeup and Roseworthy studies indicated similar long-term trends in mouse population trajectories at two widely separated sites, the intensive 3-year field study showed that analyses of long-term patterns need to be supplemented with local studies to understand why there are differences in population dynamics within regions.

Breeding dynamics

House mice have evolved a flexible breeding strategy. They can be seasonal or non-seasonal breeders in the field (Bronson, 1979; Pelikán, 1981). However, there have been few detailed long-term studies of the breeding biology of free-living mice (Lidicker, 1966; Berry, 1970). An 18-year study of the breeding dynamics of house mice in Australian wheatfields reported monthly mean litter sizes as high as 9.1 and as low as 4.9, with litter size changing seasonally from high values in spring to low values in autumn and winter. The length of the breeding season ranged from 20 to 45 weeks, and litter size was depressed throughout the breeding seasons that began when abundance of mice was high (Fig. 4) (Singleton *et al*., 2001b). These findings are consistent with reports from 2- to 3-year studies on seasonal breeding of mice in wheatfields in South Australia (Mutze, 1991) and irrigated cereal farms in NSW (Bomford, 1987; Singleton & Redhead, 1990).

The published studies confirm the opportunism of mice when they inhabit a variable environment that occasionally provides them with high-quality food and sufficient breeding sites. But it is not clear why mice are often able to reach their reproductive potential in Australian wheatfields but rarely in other parts of the world. Part of the explanation could be lack of competitors; it is unusual to catch any small mammal other than a mouse in Australian wheatfields. Another possible explanation is that mice in Australia are not burdened with all the diseases carried by mice in Europe.

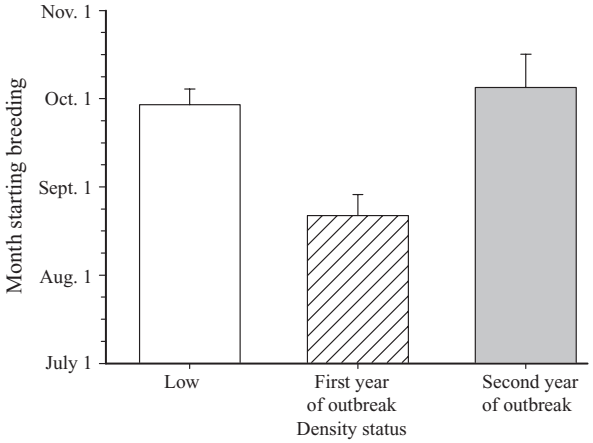

Figure 4. Month in which female mice begin to breed at Walpeup in Victoria in relation to the density status of the mouse population, 1982–2000. Breeding begins significantly earlier (in August) in moderate density years ($P < 0.01$). Sample sizes are $N = 9$ low years, $N = 4$ first years of outbreak and $N = 2$ second (plague) years. Data are given means ± 1 SE.

We will return to this point later. A reduced suite of specialist predators also may be a contributing factor. Regardless, most studies of the breeding ecology of free-living mice in Australia highlight their ability to take advantage of flushes of high-quality food: insects, grasses, dicotyledons and all the agricultural crops (see Bomford & Redhead, 1987; Tann, Singleton & Coman, 1991; although see also Ylönen *et al*., 2003). This story is repeated in New Zealand where outbreaks of mouse populations follow the seed mast years of beech forest (*Nothofagus* spp.) (Ruscoe *et al*., 2003) and the quantity of beech seed can be used to predict the growth of mouse populations (Choquenot & Ruscoe, 2000).

Survival and rates of population increase

In an effort to improve the bio-physical model of Pech *et al*. (1999), we analysed how rates of increase of mouse populations were influenced by seasonal changes in breeding performance and survival. Despite 20 years of intensive capture–mark–release studies, there is a limited dataset on seasonal differences in survival in pre-plague, plague and non-plague years because of low population densities in non-plague years, moderate to low recapture rates of mice and seasonal differences in their trappability. An analysis of the factors influencing capture probability (Davis *et al*., 2003a) and survival data from intensive capture–mark–release trapping during a period of rapid population increase from October 1983 to July 1984 (Davis, Pech & Singleton, 2003b) has enabled capture probabilities to be adjusted for seasonal

effects. These analyses revealed that the rate of increase of mouse populations (r) over the non-breeding season is negatively correlated with density, although over the breeding season r is independent of mouse density. A combined analysis of changes in survival, breeding and r in the breeding seasons has led to the development of a simulation model for the effect of fertility control on outbreaking house mouse populations. Interestingly, it appears that a moderate reduction in fertility rate of the population (one-third of all mice fail to reproduce for the entire breeding season) is sufficient to prevent mouse plagues from occurring (Davis *et al.*, 2003b). Under this level of control, there may be up to 250 mice ha^{-1} on occasions but these densities do not cause significant economic impact. This is an important result because fertility control of house mice is currently a high priority for the management of mouse plagues in Australia (for a review see Singleton *et al*., 2002). The focus has been on the development of an immunocontraceptive vaccine (Chambers, Singleton & Hinds, 1999) and one major criticism has been that fertility control would not be practicable because of the high innate reproductive potential of mice. Some earlier modelling had suggested that at least 80–90% of female mice would need to be sterilized under field conditions to prevent mouse plagues (Hone, 1999). However, the recent, more detailed analysis of seasonal population dynamics has established specifications for the use of fertility control that appear quite achievable.

BEHAVIOURAL ECOLOGY

House mice are particularly interesting behaviourally because they exhibit strong territorial behaviour in small cages and enclosure studies in the laboratory (Crowcroft & Rowe, 1963; Singleton & Hay, 1983). Territorial behaviour has the potential to limit breeding density in vertebrates (Watson & Moss, 1971), yet it seems clear that no such limitation occurs in the field when mouse plagues develop.

SPATIAL BEHAVIOUR

The spatial behaviour of mice on agricultural fields has been assessed through radio-tracking studies and from capture–mark–recapture studies. Radio-tracking showed that during the breeding season, mice were site-attached with male mice having larger home ranges than females (Krebs, Kenney & Singleton, 1995; Chambers, Singleton & Krebs, 2000). After breeding ended home ranges were significantly larger and a majority of mice became nomadic (see Pocock, Hauffe & Searle, 2005, this issue). Home ranges increased from 0.037 to 0.119 ha in a study conducted in Queensland (Krebs *et al*., 1995) and from 0.014 to 0.199 ha in a study conducted in Victoria (Chambers *et al*., 2000). This plasticity in spatial (and social) behaviour may contribute to the capacity of house mice to adjust to high population densities, which may be a prerequisite for the development of mouse plagues.

In dryland cropping areas, capture–mark–release studies indicate that undisturbed grass verges are important non-crop habitats and are used for nesting sites. Mice use these undisturbed habitats to build-up in numbers, then colonize maturing crops in late winter or early spring when cover is sufficient (Singleton, 1989; Mutze, 1991; Chambers, Singleton & van Wensveen, 1996; Ylönen *et al*., 2002). In irrigated farming systems, mice use non-crop habitats such as rice contour banks and channel banks, then invade fields once the water has been drained (Bomford, 1987; Boonstra & Redhead, 1994). Mice are present in all habitats in agricultural areas, although few actively breeding adults are found in ploughed or fallow fields or remnant woodland and so these habitats function as sink habitats (Chambers *et al*., 1996; Singleton *et al*., 2003a).

Current knowledge of the underlying social structure and spatial behaviour of mouse populations has allowed the development of appropriate management practices to reduce the impact of mice on crops. These have been tested in large replicated field studies (Singleton & Brown, 1999; Brown *et al*., 2003, 2004).

RESPONSES TO PREDATION RISK

The risk of predation is minimized by house mice at low population densities through habitat choice, with little to no feeding in open habitats. However, at high mouse abundance there is a trade-off between the need to access food in open habitats and the risk of predation. On cereal farms, ultimately mice will give up safety when there is high intraspecific competition for food in risky habitats (Ylönen *et al*., 2002). It is not clear whether increased competition for food or increased social interactions force mice into open habitats despite the associated high risk of predation. These perceived risks can generate sublethal effects of predation; mice had higher body growth rates and began to breed earlier if they had safe access to food in semi-natural grassland habitats (Arthur & Pech, 2003). Given that early breeding is a precursor to mouse plague formation, then predation risk could influence the conditions favourable to the development of a plague.

DISEASE BIOLOGY

Microparasites (viruses, bacteria and protozoans) and macro-parasites (helminths and arthropods)

have the potential to regulate populations of mammals (Anderson & May, 1978). However, there have been insufficient field studies on the distribution and impact of diseases on small mammal populations to test this potential (Begon, 2003). Moreover, the absence of some disease agents in colonizing populations of invasive species may remove an important limiting factor to the population growth of field populations (Mitchell & Power, 2003; Torchin *et al*., 2003). In Australia, there have been extensive studies of the epidemiology of a suite of micro- and macro-parasites of house mice in the search for a possible biological control agent or vehicle for transmission of an immunocontraceptive vaccine (Singleton *et al*., 1993, 2001a, 2003b; Singleton, Smith & Krebs, 2000). These epidemiological studies have generally indicated that disease does not play a major role in regulating field populations of mice. However, there is evidence that the prevalence of some of these diseases is density dependent and that they may be moderately to highly pathogenic to mice (see Table 3). Further epidemiological and manipulative studies focusing on these specific agents are warranted.

Table 3. Helminths, viruses and bacteria reported in field populations of Australian mice, their effect on field populations and whether they have a direct or indirect life cycle (modified from Singleton & Brown, 1999). The viral studies also screened for hantaan virus, mousepox (ectromelia) and Theiler's mouse encephalomyelitis virus; all mice were seronegative.

Parasite	Refs*	Density-dependent prevalence	Pathogenicity	Direct life cycle
HELMINTHS				
Nematoda				
Calodium hepaticum	1	?Yes	Moderate	Yes
Heligmosomoides polygyrus	2,3	Yes	Moderate	Yes
Syphacia obvelata	3,6	No	No	Yes
Aspiculuris tetraptera	6	No	No	Yes
Muspicea borreli	2,6	Unknown	Unknown	Yes
Heterakis spumosa	7	Unknown	Unknown	Yes
Cestoda				
Vampirolepis fraterna	3,6	No	Low	Yes
Vampirolepis straminea	2,3	?Yes	?High	No
Taenia taeniaeformis	3,6	No	No	No
Trematoda				
Brachylaima sp.	8	Unknown	Unknown	No
VIRUSES†				
Mouse hepatitis virus (MHV)	2,3	Sometimes	Moderate‡	Yes
Rota virus (EDIM)	2,3	Yes	Moderate‡	Yes
Mouse adenovirus (K87)	2,3	Yes	Moderate‡	Yes
Parvovirus–MVM	2,3	Yes	?High‡	Yes
Parvovirus–MPV		Unknown	No	Yes
Sendai	3,4	No	Unknown	Yes
Reovirus	2,3	Yes	No	Yes
Murine cytomegalovirus (MCMV)	2,3	Yes	No	Yes
Lymphocytic choriomeningitis virus (LCMV)	3,4	Unknown	Potentially	Yes
BACTERIA				
Streptobacillus moniliformis	5	No	?Moderate	Yes
Escherichia coli	9	Likely	No	Yes
OTHER				
Cryptosporidium parvum	10	Unknown	Unknown	Yes
Mycoplasma pulmonis	2,3	No	No	Yes

*1, Singleton & Chambers (1996); 2, Singleton *et al*. (2000); 3, Singleton *et al*. (1993); 4, Smith *et al*. (1993); 5, Taylor *et al*. (1994); 6, Singleton (1985); 7, Singleton & Redhead (1990); 8, Angel & Mutze (1987); 9, Gordon (1997); 10, Morgan *et al*. (1999).

†MVM, minute virus of mice; MPV, mouse parvovirus.

‡Neonates or young mice only.

Australian mouse populations carry only a subset of the helminths found in Europe (cf. Tattersall, Nowell & Smith, 1994). The main parasites missing are those that require an intermediate host for transmission. For micro-parasites, there have been no comparable studies elsewhere on the seroprevalence and epidemiology of these diseases in field populations of mice. Therefore we are unable to determine whether Australian mice have a reduced set of viruses circulating in their field populations. Further studies are required to determine whether a reduced diversity of diseases partially accounts for the ability of mice to generate plagues in Australia.

MOUSE PLAGUES: A BIOLOGICAL CURIO – WHERE NEXT?

Population ecology studies of house mice in Australia began with the pioneering studies of Newsome (1969a, b) who highlighted the importance of seasonal use of different habitats by mice, the timing of rainfall events on food supply and nesting sites, and the likely importance of social interactions in influencing dispersal patterns in the landscape. Since then the biological curio of mouse plagues and the impact they have on human livelihoods has generated an impressive set of population studies in Australia. These have provided a fascinating insight into the biology of an invasive species and its ability to achieve impressive reproductive potential and high survival in some years. Extrinsic factors such as rainfall and its effect on food supply, and changes in farm management systems that affect food supply, cover and nesting sites, all have a part to play in the generation of mouse plagues. However, models of these factors alone have been only partially successful in explaining the intra- and interannual variations in rates of increase of field populations (Kenney *et al*., 2003; Krebs *et al*., 2004). Just as important is the behavioural and physiological plasticity that mice have evolved, which is a hallmark of their success as an invasive species (Anderson, 1978; Bronson, 1984; Berry & Scriven, 2005, this issue). We have made substantial progress towards understanding the secret lives of mice and how they influence who breeds when and where, how they can rapidly switch their social organization, and how their use of the landscape can be influenced by perceived risk of predation. However, there are still many gaps in our knowledge and we offer the following as key areas of population research.

First, we need to extend our knowledge on the breeding ecology of house mice. Specifically what factors govern the length of the breeding season? What limits breeding during the hot, dry summers when typically only 20–40% of females breed? What extrinsic factors regulate the intra- and interseasonal litter size of mice and how is this controlled physiologically (current evidence suggests that the mechanism occurs prior to the development of corpora lutea and that the age at conception has an impact on litter size) (Batten & Berry, 1967; J. Jacobs unpubl. observ.). Secondly, what is the process responsible for the strong negative effect of population density on survival during the non-breeding season, and which cohorts have the best over-winter survival? Do the final or penultimate litters of the previous breeding season form the nucleus of the next breeding season? Is there interyear variation and if so what influences this variation? Is there an effect of age, determined largely by the length of the non-breeding season, on parity and an ability to have successive litters? Boonstra & Redhead (1994) highlighted this issue from their work in the mid-1980s but the answer still eludes us. Thirdly, what are the effects on survival and breeding performance of parasites that show density-dependent effects, such as *Vampirolepis straminea* and mouse parvovirus? If they have an effect only when population densities are high, what would happen if the prevalence of the respective parasites was manipulated so that it was higher at lower host densities? Fourthly, is predation an important limiting factor either directly (particularly at low densities) or through sublethal effects? There is some evidence supporting this contention but further experiments are required. Finally, the landscape ecology of mouse plagues needs more detailed analysis to understand how local factors, such as the distribution and relative proportions of source and sink habitats, influence outbreaks and to map the genetic architecture of populations during and after a plague. Many genetic studies of the population structure of house mice have been conducted in Europe (e.g. Navarro & Britton-Davidian, 1989; Berry *et al*., 1990; Berry & Scriven, 2005, this issue). The techniques for small mammal studies are now further developed (Burton, Krebs & Taylor, 2002) and could be readily applied to the ecology of house mice in south-eastern Australia.

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