

# Reproductive changes in fluctuating house mouse populations in southeastern Australia

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House mice (*Mus domesticus*) in the Victorian mallee region of southeastern Australia show irregular outbreaks. Changes in reproductive output that could potentially drive changes in mouse numbers were assessed from 1982 to 2000. Litter size in females is positively correlated with body size. When standardized to an average size female, litter size changes seasonally from highest in spring to lowest in autumn and winter. Litter size is depressed throughout breeding seasons that begin when the abundance of mice is high, but is similar in breeding seasons over which the abundance of mice increases rapidly or remains low. Breeding begins early and is extended on average by about five weeks during seasons when mouse abundance increases rapidly. The size at which females begin to reproduce is larger during breeding seasons that begin when mouse abundance is high. An extended breeding season that begins early in spring is necessary for the generation of a house mouse plague, but it is not in itself sufficient. Reproductive changes in outbreaks of house mice in Australia are similar but not identical to reproductive changes that accompany rodent population increases in the Northern Hemisphere. We conclude that food quality, particularly protein, is a probable mechanism driving these reproductive changes, but experimental evidence for field populations is conflicting.

**Keywords:** *Mus domesticus*; Australia; outbreaks; house mice; plagues; reproduction

## 1. INTRODUCTION

House mouse outbreaks are a particularly graphic example of the failure of population regulation. House mice increase to plague numbers at irregular intervals in the grain-growing regions of southeastern Australia and then collapse to low numbers for several years. These outbreaks are enormously costly for agriculture (Caughley *et al.* 1994; Singleton 1997) and considerable effort is being expended on developing a biological control method that will eliminate plagues (Chambers *et al.* 1999; Singleton *et al.* 1999).

Changes in reproductive input have been postulated as being a major cause behind the generation of house mouse plagues in Australia (Redhead 1982; Singleton & Redhead 1990). House mice are typical small rodents in having an enormous reproductive potential. Gestation is 19 days and postpartum oestrus allows litters to follow one another at three to four week intervals (Parkes 1926). Litter size varies from one to 13 (Laurie 1946; Rowe *et al.* 1983) and juveniles can reach sexual maturity at five to six weeks of age (Drickamer 1979; Pelikán 1981; Rowe *et al.* 1983). In spite of detailed studies on laboratory populations which have led to the development of general theories on the seasonal regulation of house mice (see Bronson & Perrigo 1987; Drickamer 1987 for reviews), we do not have a good understanding of what factors control house mouse reproduction in feral populations. In particular, while there is some evidence that food supplies in the form of ripening grain trigger the onset of breeding (Bomford 1987*a,b*) and that the provision of high-quality food can extend the breeding season (Bomford & Redhead 1987), we do not know what factors cause breeding to stop in autumn.

Plasticity in size or age at sexual maturity is an important component of reproduction that can contribute to population change. Laboratory and limited field studies of house mice indicate that they have evolved a flexible reproductive strategy; they are opportunistic rather than seasonal breeders (Bronson & Perrigo 1987). Feral populations of house mice in southern Australia can reproduce in any season of the year, but typically breed from spring to autumn (Newsome 1969; Redhead 1982; Singleton 1989; Mutze 1991; Twigg & Kay 1994). There have been no detailed long-term studies of the pattern of reproduction of field populations of mice. Mutze (1991) reported, from a six-year study of mice in southern Australia, that litter size was highest in spring and increased with maternal head-body length; however, the sample sizes in that study were low.

Population growth of mice is highly variable from year to year and our long-term goal is to find out why this is so. In this paper we use necropsy data on house mice from the mallee region of northwestern Victoria in order to evaluate the contribution of changes in reproduction to the generation of mouse plagues in the grain-growing regions of Australia. In particular we wish to test the Singleton-Redhead model (Singleton & Redhead 1990) for the generation of mouse plagues. This invokes changes in the length of the breeding season and litter size that lead to population eruptions.

## 2. METHODS

House mice were live-trapped in Longworth traps in agricultural areas of the Victorian mallee in northwestern Victoria from 1982 to 2000. Samples were obtained at approximately four-week intervals during the 1980s and at approximately six-week intervals during the 1990s, but there were periods with very low numbers (less than one mouse per hectare) in which no data on reproduction could be obtained. Trapping effort was inversely

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related to mouse density. When densities were low, we set 400–600 traps on average in each sampling period. At high density only 100–150 traps were typically set.

Trapping for breeding females coincided with a long-term capture–mark–release (CMR) study conducted at the Mallee Research Station, Walpeup, Victoria (35° 08' S, 142° 02' E) from 1982 to 1989, and at Symes Farm, 10 km south of Walpeup from 1992 to 2000. The CMR trapping was conducted over three to four consecutive nights. Traps were set in habitats representative of cereal farms in this region, with a strong emphasis on the margins of cereal crops and the crops themselves (see Singleton 1989 for details).

Abundance indices for mice were calculated using trap success per 100 trap nights and adjusted for frequency using the method of Caughley (1977, p. 20). Absolute densities of mouse populations in fields were estimated by mark–recapture using the Petersen method (Krebs 1999, p. 21).

Mice were killed immediately after capture by cervical dislocation and detailed breeding data were obtained by necropsy. In addition to body mass, head–body length and sex, we counted viable and resorbing embryos for pregnant females and examined teat size for evidence of lactation. We noted the presence of placental scars and, for some samples, we counted the placental scars for females with recent, enlarged scars. For this analysis we have relied on viable embryo counts *in utero* as estimates of litter size.

The size of females when they first bred was estimated from direct necropsy data for the 1990s data. Extensive necropsy information was lacking in the 1980s (data were collected on pregnant females only) and, therefore, the size of females when they first bred was determined from the reproduction indices of live-trapped animals. For the 1980s data, females were judged to be reproducing if they showed evidence of lactation (enlarged teats) or were judged to be pregnant by palpation. Because of these differences between the 1980s and 1990s we have separated them in the analyses and used them as replicates in order to test for the generality of the patterns observed.

The start of the breeding season marks the start of the biological year for house mice and was estimated from the time of first conception. This was determined by estimating the trimester of pregnancy (first trimester with embryos < 1.6 mm, second trimester with embryos 1.6–10.5 mm and third trimester with embryos > 10.5 mm) when breeding was first detected (most years) or from live-trapping data using a combination of breeding indices of females and the appearance of juveniles in the catch. The end of the breeding season was estimated by the timing of the last conceptions when pregnant females were captured and ranged from February to June.

All statistical analyses were carried out in NCSS 2000 (Number Crunching Statistical System, NCSS Statistical Software, Kaysville, UT, USA) using generalized linear models that adjust for our unbalanced designs. All litter sizes were analysed by generalized linear models with head–body length as the covariate. All means reported are adjusted to a standard 85-mm female mouse.

All experimental methods were approved under the National Health and Medical Research Council/Commonwealth Scientific and Industrial Research Organization/Australian Agricultural Council code of practice for care and use of animals.

### 3. RESULTS

#### (a) *Changes in abundance*

House mice in the Victorian mallee have shown three high-density years since our studies began: 1983–1984,

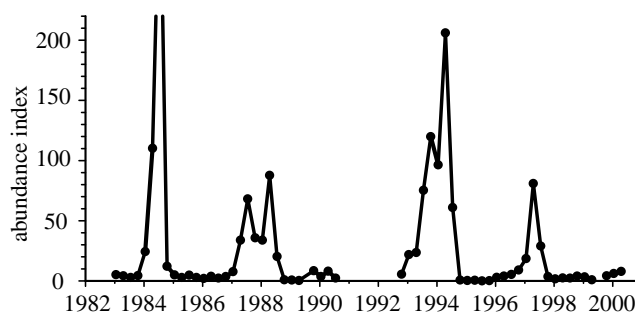


Figure 1. Abundance index for house mice from the Victorian mallee from 1982 to 2000. The abundance index is the adjusted number per 100 trap nights. Indices were averaged for each of the four seasons and each point represents one season. The abundance index exceeds 300 in autumn 1984 when the catch exceeded the number of traps.

1987–1988 and 1993–1994 (figure 1). Moderate densities were reached in 1986–1987 and 1992–1993 and these years led into high-density or plague years. In 1996–1997 mice began to increase and reached moderate numbers, but this was not translated into a mouse plague in 1997–1998. The 1983–1984 plague was unusual in that mouse numbers went from low to extremely high during just one breeding season. Low-density years were by far the most common (12 out of 18 years). Although detailed data were not available for 1990–1992, we know from infrequent sampling that mouse numbers were low throughout this period.

Two patterns can be distinguished in the development of mouse plagues. A one-year outbreak occurred in 1983–1984. In these outbreaks, mouse numbers are low the year before the outbreak and the year immediately following. In September 1983, for example, mouse abundance was four per 100 trap nights. Numbers then peaked nine months later in June 1984 when the abundance index exceeded 100 per 100 trap nights. Two-year plagues occurred in 1986/1987–1987/1988 and 1992/1993–1993/1994. In these plagues, numbers built up in year 1 and peak in year 2. For example, in September 1986 mouse abundance was three per 100 trap nights and 59 per 100 trap nights the following September in 1987. The peak occurred in the second year in May 1988 at *ca.* 90 mice per 100 trap nights.

We have divided breeding seasons into three types based on the average density of mice from spring to autumn and on the pattern of the population outbreak. Low years form the majority of breeding seasons with a mouse abundance index averaged over spring to autumn of < 10 (equivalent to densities of < 50 mice ha<sup>-1</sup>). In some low years densities are < 1 mouse ha<sup>-1</sup> and samples are difficult to obtain. There are two categories of years of high mouse abundance (density 50 to > 1000 mice ha<sup>-1</sup>). The first includes one-year outbreaks and the first year of two-year outbreaks. Four out of 18 years were in this category. The second category consists of the second year of a two-year outbreak. Only two out of 18 years were classified in this category (figure 1).

#### (b) *Litter size*

Changes in mouse abundance within a region may be driven by changes in reproduction and mortality. We

concentrate here on the reproductive component, and in this section we ask two questions.

- (i) Is litter size related to body size in Australian house mice?
- (ii) Does litter size change with population phase as predicted by the Singleton–Redhead model of outbreaks?

We analysed litter size and placental scar counts obtained from necropsy data from 1982 to 2000 for the Victorian mallee. We could detect no differences between litter size counts (viable embryos only) and placental scar counts, so we have used only litter size counts in further analyses. Figure 2 shows the relationship between litter size and head–body length for 630 pregnant females. A similar regression was determined for body weight but it has similar scatter and adds nothing new to the analysis, so we have used head–body length as a covariate in all further analyses.

Figure 2 includes all years in the analysis, and we next asked whether this regression differed between years and seasons. The 1980s data were analysed separately from the 1990s data in order to see whether the patterns were similar. Table 1 gives the sample sizes and observed and adjusted litter sizes for each season in each biological year along with their standard errors. Unfortunately, because of extremely low populations, some seasons and years have no samples of pregnant females and, because of this, it is not feasible to search for differences among the low-density years.

An analysis of covariance was calculated in order to test for equality of slopes for the regression of litter size on head–body length for all biological years and seasons and we found no differences in slopes ( $F=0.75$ , d.f. = 4,245 and  $p=0.54$ ). In order to remove the influence of body size on litter size, we used generalized linear models with head–body length as the covariate. The litter sizes reported here are adjusted to a constant size of female (85 mm in length) using the functional relationship shown in figure 2. We tested whether there was any interaction between season and density status and there was none ( $F=1.9$ , d.f. = 4,282 and  $p=0.20$ ), so we can consider their impacts on litter size as independent. Both season and density status had significant effects on adjusted litter size. Figure 3 shows the seasonal effect. There is a strong and significant drop in litter size from spring to autumn ( $F=16.9$ , d.f. = 2,282 and  $p < 0.001$ ) and this drop is consistent in every year regardless of mouse density such that summer litter size is on average 80% of spring litter size and autumn litter size is on average 71% of spring litter size. The few mice we have found breeding in the winter months continue this trend to even smaller litter sizes in winter (48% those of spring). These seasonal results are consistent in the 1980s and in the 1990s. The exception is autumn 2000, which had much higher adjusted litter sizes than average for that season (table 1).

We tested whether litter size (adjusted for head–body length) differed between the low-density years and the two types of high-density years. Figure 4 shows the impact of high density in both the 1980s and the 1990s. During both decades the second year of each of the two-year outbreaks had lower litter sizes relative to all other

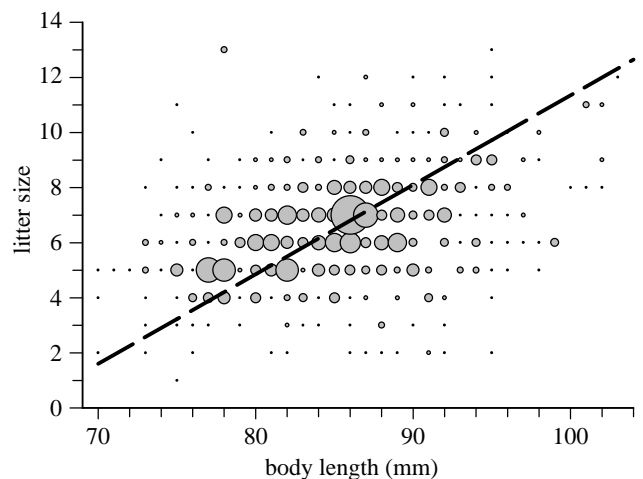


Figure 2. Regression of litter size on head–body length for 630 female house mice from the Victorian mallee from 1982 to 2000. The size of the dots is proportional to the sample size at each point. The slope of the functional regression is 0.3247, indicating that on average litter size is one embryo larger for every 3 mm increase in length ( $r=0.34$  and  $p < 0.01$ ). The regression is  $Y=0.3247X-21.128$  and the s.e. of the slope is 0.01218.

years. Since there is no significant interaction of season and density status, these high-density year effects appear to be constant over all seasons. These results imply that litter size is density dependent in house mice in the Victorian mallee. Figure 5 shows that there is a tendency towards smaller litters at high density, as litter size declines by 1.9 embryos on average for every tenfold increase in the abundance index, but that litter size is not very strongly related to population density.

There was a very low rate of post-implantation mortality in house mice from the Victorian mallee. There were only 54 dead embryos in 306 pregnant females carrying 1960 embryos, an average of 2.8% post-implantation loss. This rate changed very little from year to year over the period from 1992 to 2000.

### (c) Length of the breeding season

The onset of the breeding season ranged from August to November. The end of the breeding season ranged from February to June. In this section we examine whether the length of the breeding period changed systematically with population density.

Reproductive output could be increased dramatically if the breeding period was extended in the density build-up period. Table 2 shows the timing of the start and end of the breeding period from 1982 to 2000. There is a clear pattern that breeding starts in mid-August in years in which abundance builds quickly from a low base, but begins on average at the end of September or early October in both low years and the second year of two-year plagues (ANOVA,  $p < 0.01$ ).

The length of the breeding season shows the same pattern as the start of breeding. The breeding season is longest in years in which abundance builds quickly from a low base (35.8 weeks) and shortest in the second year of two-year plagues (23 weeks). Low years have an intermediate length of 29.6 weeks. The early start to

Table 1. Observed and adjusted litter sizes for adult house mice from the Victorian mallee wheatlands in 1982–2000.

(Adjusted litter sizes are for a standard 85-mm female. Spring is September to November, summer is December to February and autumn is March to May. The standard error is for the adjusted means.)

year and season	sample size	mean head-body length (mm)	mean observed litter size	adjusted mean litter size	litter size s.e.
1982–1983					
spring	5	83.20	6.00	6.58	0.45
summer	—	—	—	—	—
autumn	2	87.50	6.50	5.69	0.50
1983–1984					
spring	57	88.88	8.49	7.24	0.24
summer	17	86.64	7.35	6.82	0.43
autumn	5	83.80	5.20	5.59	0.81
1984–1985					
spring	30	85.33	6.90	6.79	0.33
summer	41	85.44	5.95	5.81	0.28
autumn	29	83.00	4.97	5.61	0.34
1985–1986					
spring	12	84.50	7.83	7.99	0.52
summer	1	83.00	7.00	7.65	1.81
autumn	4	84.00	5.75	6.07	0.90
1986–1987					
spring	12	84.67	9.08	9.19	0.52
summer	17	84.53	6.71	6.86	0.44
autumn	12	83.92	5.92	6.27	0.52
1987–1988					
spring	28	85.50	7.36	7.20	0.27
summer	15	87.93	5.73	4.79	0.35
autumn	—	—	—	—	—
1988–1989					
spring	5	80.60	6.60	8.02	1.36
summer	1	72.00	5.00	9.20	—
autumn	—	—	—	—	—
1989–1990					
spring	9	85.20	8.80	8.74	0.45
summer	—	—	—	—	—
autumn	—	—	—	—	—
1992–1993					
spring	—	—	—	—	—
summer	10	89.50	9.10	7.65	0.43
autumn	15	81.67	5.60	6.68	0.39
1993–1994					
spring	35	89.49	8.03	6.58	0.25
summer	100	85.70	5.65	5.42	0.15
autumn	19	85.58	4.89	4.71	0.34
1994–1995					
spring	—	—	—	—	—
summer	—	—	—	—	—
autumn	—	—	—	—	—
1995–1996					
spring	9	81.00	7.89	9.18	0.50
summer	16	84.31	6.50	6.72	0.36
autumn	6	80.67	5.50	6.90	0.61
1996–1997					
spring	30	84.23	7.90	8.15	0.36
summer	14	82.29	5.50	6.38	0.40
autumn	—	—	—	—	—
1997–1998					
spring	10	84.60	7.20	7.33	0.53
summer	18	83.11	5.89	6.50	0.46
autumn	5	83.80	5.60	5.99	0.45
1998–1999					
spring	14	82.57	8.57	9.36	0.81
summer	19	82.74	5.63	6.36	0.32
autumn	—	—	—	—	—
1999–2000					
spring	17	83.65	8.06	8.50	0.36
summer	9	85.89	7.11	6.82	0.50
autumn	22	78.77	5.23	7.24	0.37

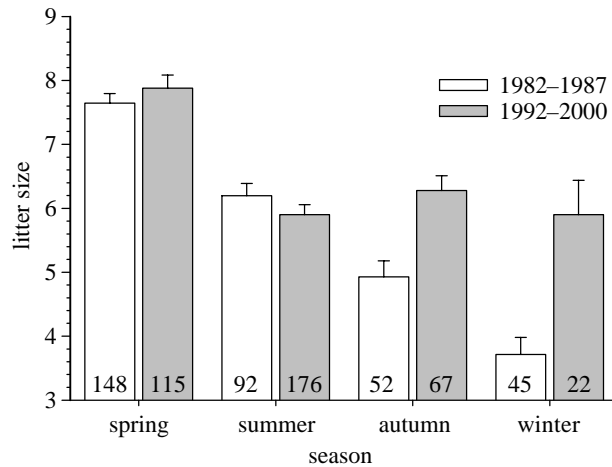


Figure 3. Change in the seasonal average litter size of a standard 85-mm female house mouse in the Victorian mallee from 1982 to 1988 (clear box) and from 1992 to 2000 (shaded box). Sample sizes are given at the bottom of the graph. Spring is September to November, summer is December to February and autumn is March to May.

breeding and the extended breeding season are important generators of population momentum. However, this increase in reproductive output does not automatically translate into a full plague either that same year (as in 1983–1984) or one year later because of extrinsic factors such as drought.

There is a slight negative correlation between density in the spring of the year and the length of the subsequent breeding season ( $r^2=0.21$ ) and there is no correlation between the length of the breeding season in year  $t$  with density in year  $t+1$  ( $r^2=0.002$ ).

#### (d) Size at reproduction

In this section we consider the variation in onset of reproduction by female mice. Estimating the size at which females begin to reproduce requires a large sample size in order to estimate median body size at maturity. We were able to contrast the second year of each of the two-year plagues with the other years in each decade. Figure 6 illustrates the proportion of adult females that were breeding at different head–body lengths during the 1990s. The high-density year of 1993–1994 had a median ( $\pm$ s.e.) length at maturity of  $79.4 \pm 0.3$  mm while other years of the 1990s had a median length at maturity of  $73.5 \pm 0.9$  mm. Breeding was clearly delayed in the high-density year of 1993–1994. In 1993–1994, necropsies were performed on 897 females of which 506 were reproductively active. In the remaining years from 1992 to 1999, 254 necropsies were carried out and 233 females were reproductively active.

The data collected during the 1980s were from live-trapping. When we divided the 1980s data into two groups, i.e. the high-density year of 1987–1988 (872 females) and all the other years during the 1980s (1385 females), we found the same pattern of change in size at the commencement of breeding. The high-density year of 1987–1988 had a median size at maturity of  $84 \pm 0.39$  mm and the low-density years had a median

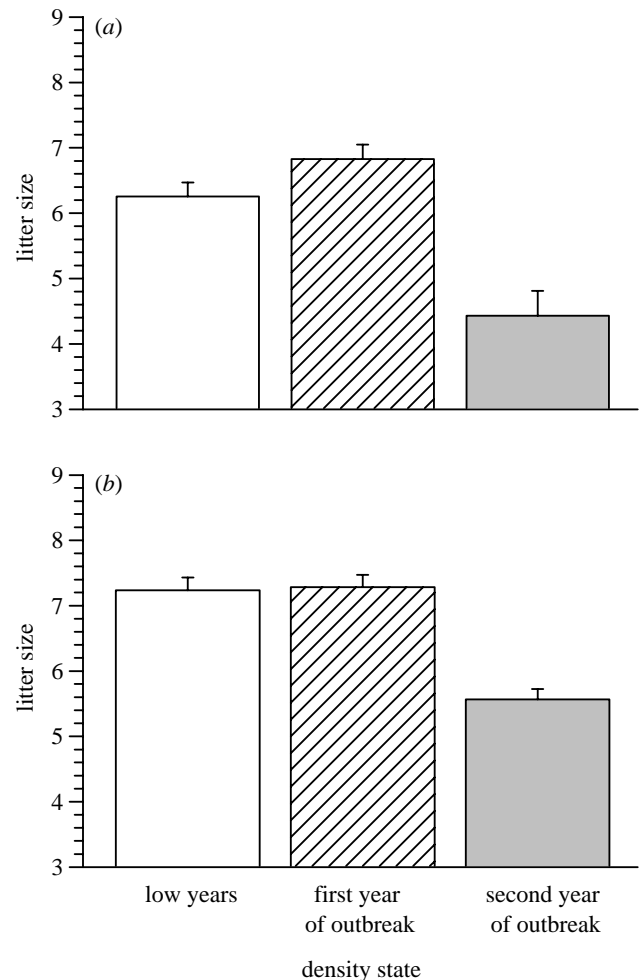


Figure 4. Change in the average litter size of a standard 85-mm female house mouse ( $\pm$ s.e.) in the Victorian mallee during low-density years, years of rapid increase and years that are the second year of two-year plagues from (a) 1982 to 1989 and from (b) 1992 to 2000. There is a strong and significant drop of ca. 25% in litter size during the plague of 1993–1994 and a drop of ca. 33% in the plague of 1987–1988.

size at maturity of  $81 \pm 0.31$  mm. The pattern for the 1980s is thus similar to that for the 1990s (figure 6).

#### 4. DISCUSSION

In southeastern Australia, house mice begin to breed earlier in spring and breed for a longer season when an outbreak is just beginning to develop. In contrast to Northern Hemisphere rodents (e.g. Krebs & Myers 1974; Norrdahl 1995), house mice showed a substantial decrease in average litter size in high-density or plague years. In some years, spring litter sizes were exceptionally large (more than nine on average) but not all of these years led to a plague the following year. We suggest that, once house mice have reached low densities, large litter sizes are not in themselves sufficient for triggering an outbreak. Two additional factors are required: an early start to the breeding season and the ability to begin reproducing at small body size (and presumably younger ages). The

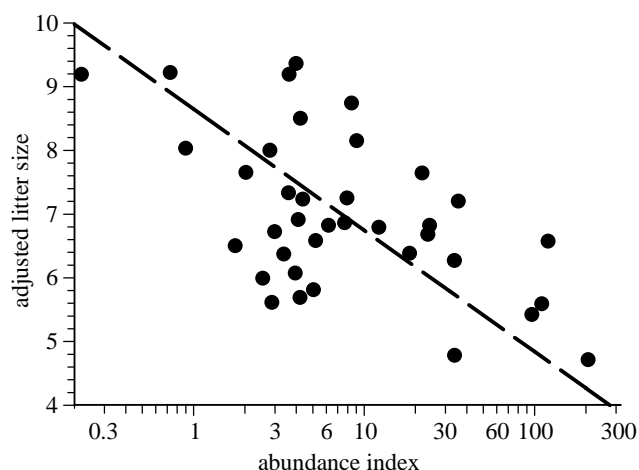


Figure 5. Relationship between litter size and population abundance in house mice from the Victorian mallee from 1992 to 2000. Data are seasonal means (spring, summer and autumn) from populations in crops. There is significant tendency for litter size to be density dependent for these populations ( $r^2 = 0.31$ ). The functional regression is shown as a dashed line (litter size =  $-1.903 \log \text{abundance} + 8.648$ ) ( $r = -0.56$  and  $n = 37$ ).

median size at commencement of breeding appears to be similar (*ca.* 73 mm) in all low-density populations, so this component of reproduction would not appear to be limiting the start of an outbreak. We are left to conclude that the primary reproductive trigger of a plague is the timing of the start of the breeding season.

Monthly variation in the proportion of female mice breeding during a season and the flexibility of mice to be seasonal or aseasonal breeders are well-documented (Bronson 1979; Pelikán 1981). The findings of the present study confirm the opportunism of mice; there was marked interannual variation for both of these important life-history traits. The seasonal trends in litter size variation also help to resolve conflicting reports from studies of one to two years duration. For example, Laurie (1946) reported no consistent changes in litter size in different populations of mice sampled monthly from four habitats (urban areas, storehouses, cold stores and corn ricks) over one year. In contrast, Batten & Berry (1967) reported a significant increase in litter size four to five months after the commencement of breeding in mouse populations living on Skokholm island. They attributed this increase to the dominance of the new season's young in the breeding population. This did not appear to be the pattern observed in our study.

Singleton & Redhead (1990) observed that, during the 1970s and 1980s, litter sizes were higher in the spring of the first year of a mouse plague (spring 1978 and spring 1983). This observation can be stated as a predictor of an incipient plague: when litter size averages above nine in spring, a plague is possible. Four years stand out in our data (table 1). High litter sizes in spring in 1983 and 1986 presaged the plagues of 1984 and 1987–1988. High litter sizes in spring 1995 should have presaged a plague in 1996–1997, but only a moderate increase in abundance occurred (figure 1). High litter sizes in spring 1998 should

have presaged a plague in 1999–2000, but this did not eventuate perhaps because mouse densities were too low following the rapid decline in population density in spring 1997.

To the authors' knowledge, this is the first paper to report on long-term patterns of seasonal changes in litter size in feral house mice. Litter size is maximal in the spring of the year and minimal in winter, as indicated by Singleton & Redhead (1990) and Mutze (1991). There are at least four hypotheses that might explain seasonal litter size effects.

- (i) Seasonal changes in litter size are a reflection of changes in parity and age structure. Younger mice that begin to breed in summer and autumn are breeding for the first time and are younger than the adults that start breeding in spring. Younger mice and primiparous mice have smaller litter sizes.
- (ii) Food supplies are maximal in spring in both quantity and quality and litter size changes track food quality seasonally. Mice of the same age and parity will have different litter sizes in different seasons because of nutritional factors.
- (iii) Juveniles survive better if born in spring and house mice have been selected for an optimal life-history strategy that maximizes reproductive effort in spring. Reduced reproductive effort in summer and autumn increases the probability of the individual surviving over the coming winter to breed in the next spring. This hypothesis assumes a cost of reproduction.
- (iv) Social cues at higher densities may lead to physiological states that reduce litter size late in the breeding season (Bronson & Perrigo 1987).

We do not have data on parity effects in feral house mice. At least some of the size effect shown in figure 2 is probably due to parity effects. Pelikán (1981) reported a positive association between the weight of mice and litter size and argued that good nutrition rather than parity effects accounted for this relationship (*cf.* Batten & Berry 1967). We note that the seasonal changes in litter size occur even after litter sizes are adjusted to a standard 85-mm female. Consequently, if age and size are closely related (we do not know whether this is true), our analysis would represent data contrary to the predictions of the first hypothesis. We need data on both parity and body size in order to evaluate this hypothesis more closely.

The third hypothesis seems unlikely in an opportunistic rodent species with a low life expectancy. We would expect house mice to operate at maximal reproductive capacity at all times rather than wait for better times in the next year. However, the questions about the costs of reproduction are important to quantify for feral house mice and we should not disregard this suggestion.

The fourth hypothesis has some support from studies of mice in corn ricks in the UK. Cover and food supply were relatively similar between ricks, yet ricks with  $< 4 \text{ mice m}^{-3}$  had litter sizes 0.5–1.1 embryos higher (mean around 5.4) than ricks with 8–16  $\text{mice m}^{-3}$  (Southwick 1955; Rowe *et al.* 1964). However, the densities of mouse populations in corn ricks were extremely high and the relatively constant resources provided by ricks may buffer seasonal effects. Interestingly, even at the

Table 2. Summary of the breeding seasons of house mice in the Victorian mallee wheatlands in 1982–2000

(The onset of breeding was estimated as three weeks prior to the time of birth of the first litters of the year. The end of the breeding season is backdated three weeks from the estimated time of birth of the last litters of the year.)

breeding season	onset of breeding	cessation of breeding	length of the breeding season (weeks)
1982–1983	first week of November	November–December and March–May	20
1983–1984	first week of August	second week of May	40
1984–1985	first week of October	first week of May	30
1985–1986	first week of September	fourth week of April	33
1986–1987	third week of August	second week of May	38
1987–1988	third week of September	third week of March	26
1988–1989	fourth week of September	third week of February	21
1989–1990	third week of September	first week of May	33
1990–1992	no data	no data	no data
1992–1993	second week of September	fourth week of March	28
1993–1994	third week of October	first week of March	20
1994–1995	third week of September	fourth week of May	36
1995–1996	first week of October	second week of May	31
1996–1997	third week of August	fourth week of February	28
1997–1998	first week of October	first week of June	35
1998–1999	third week of September	fourth week of March	27
1999–2000	third week of August	first week of July	45

lower densities in the ricks, > 10% of embryos were not viable ('resorbed') and there was no apparent density effect on the percentage of dead embryos. This compares with an embryo loss of only 2.8% in the present study and the value of 2.7% reported by Laurie (1946) for mice in the UK living in fields. Ricks may not be a good model for what happens in wheat fields. Indeed, there is evidence from a study of mice living in fields that recruitment per lactating female (as an indirect measure of litter size) was higher at densities of > 500 mice ha<sup>-1</sup> when the population was increasing than at densities < 250 mice ha<sup>-1</sup> when the population was declining (DeLong 1967). More research is required in order to examine the relationship between seasonal changes in social factors and litter size in field populations of mice.

The second hypothesis seems most likely, i.e. that mice track food supplies and adjust their litter size accordingly.

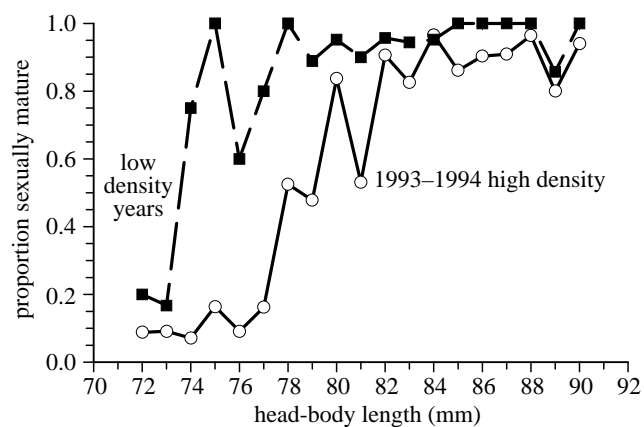


Figure 6. The proportion of house mouse females that were reproductively active (pregnant or had embryo scars) in relation to head-body length: (a) 1993–1994 versus other years in the 1990s (necropsy data) and (b) 1987–1988 versus other years in the 1980s (live-trapping data).

Bronson (1979) first suggested this hypothesis in order to explain seasonality in breeding in house mice, but the evidence in favour of this mechanism is rather mixed (Pelikán 1981 versus Batten & Berry 1967). Bomford (1987*b*) reported a significant increase in house mouse litter size in the laboratory when she supplemented diets with protein in the form of casein. However, the litter sizes of mice kept in the laboratory were all small (three to five mice) by field standards. Bomford & Redhead (1987) supplemented field populations in the autumn with high-protein seeds and increased the fraction of females breeding but did not change the average litter size. The factors that cause litter size to change seasonally as well as from year to year are not at all clear. More experimental work on field populations at the start of the breeding season is clearly needed in order to determine the factors determining litter sizes. We also need to know whether differences in the number of embryos (litter size in this paper) are positively correlated with the number of young that survive through to adulthood (see Krebs *et al.* 1994).

The delay in onset of breeding by females during high mouse density years is the second report, to our knowledge, of this phenomenon happening in field populations of mice and the first in a non-'island' population. Massey & Vandenberg (1980) monitored the breeding ecology of mice living on 'highway islands'. They collected female urine at different densities and their results strongly suggest that urinary factors present at high mouse densities may delay female puberty, thereby slowing population growth. Mouse urinary proteins have marked effects on the social interactions of female mice under laboratory conditions (Hurst *et al.* 1998) and may offer a mechanism for explaining the results reported in our study in 1993–1994. However, there have been no studies of whether specific mouse urinary proteins could influence the rate of growth of mouse populations in agricultural fields. Moreover, if mouse urinary proteins are important, it is important to know why they fail to prevent massive population eruptions in some years.

## 5. CONCLUSION

We conclude that there is dramatic year-to-year variation in the length of the breeding season, litter size and the size at which females begin to reproduce. All of these reproductive parameters help to drive population changes. In particular, mouse plagues seem to begin with large litter sizes, a small size at sexual maturity and early and long breeding seasons, so that changes in breeding season length are necessary in order to generate plague numbers of mice. However, these reproductive changes are not sufficient because not all years with extended breeding seasons are followed by plague years. We require data on the mortality schedule of feral house mice in order to complete the demographic picture.

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