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The prevalence of viral antibodies during a large population fluctuation of house mice in Australia

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SUMMARY

We studied the seroprevalence of three viruses (mouse cytomegalovirus (MCMV), minute virus of mice (MVM), and mouse parvovirus (MPV)) in house mice (*Mus domesticus*) in 1995–7. In the first year average mouse density was less than 1 mouse/ha. From November 1995 to May 1996 the population increased at an average rate of 7% per week, a doubling time of about 10 weeks. From August 1996 to May 1997 the population increased at an average rate of 10% per week, a doubling time of about 7·5 weeks. From a peak around 250 mice/ha in May 1997, the mouse population fell 19% per week to 5 mice/ha in October 1997. The seroprevalence for all three viruses varied dramatically over time. MCMV had the highest seroprevalence (61·7%), followed by MVM (8·5%) and MPV (18·4%). Time series data indicated that MCMV spread rapidly through the population of mice once trap success was greater than 14% (40–100 mice/ha). By contrast MVM and MPV seroprevalence occurred with a 2–3 month and 3–4 month time lag, respectively. The current study supports the contention that MCMV would be a good carrier for an immunocontraceptive vaccine for controlling field populations of mice.

INTRODUCTION

Populations of house mice, *Mus domesticus*, can increase to very high numbers in the grain-growing regions of eastern Australia and are an important agricultural pest [1, 2]. We have studied the demography and habitat dynamics of eruptive populations of mice [3], and the response of these populations to rainfall and food supply [4, 5] in an attempt to understand what generates these irregular outbreaks. Interest has centred also on the naturally-occurring viruses of house mice, since some of these might have potential as biological control agents [6].

There has been only one previous longitudinal study of the changes in the seroprevalence of viral antibodies in field populations of mice [7]. That 13-month study screened mice for antibodies to 13 viruses on average every 8 weeks. The two viruses of most interest to emerge from that study and a concurrent general survey of viruses of mice in Australia [8] were mouse cytomegalovirus (MCMV) and minute virus of mice (MVM; a parvovirus). MCMV is of interest because of its high seroprevalence, which in combination with its other life history parameters makes it a promising candidate to be engineered in an immunocontraception programme [9]. MVM is of interest because an epizootic of this virus occurred in association with a decline in mouse populations. In this study, the seroprevalence of a

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third virus, mouse parvovirus (MPV), is examined for the first time in wild mouse populations.

MCMV is a herpesvirus that causes persistent infection in the salivary gland of mice and latent infections associated with macrophages [9, 10]. MPV is a parvovirus that has been recently described [11, 12]. MVM and MPV are closely related, sharing high homology in the genes coding for nonstructural proteins. MVM causes an acute, self-limiting infection, whereas MPV infection persists well beyond seroconversion and is therefore also of interest.

MCMV has been extensively studied as a model of human CMV infection. Because the human virus is a frequent contaminant of transplanted organs, routes of MCMV transmission have been investigated in order to validate it as a suitable model. Spleen, salivary glands and skin have been shown to harbour MCMV DNA during acute infection [13], and infection was transferred by syngeneic and allogeneic skin grafts [14]. During latency, MCMV could be transmitted by both kidney tissue and blood transfusions [15]. Although virus could be detected in epididymal sperm, seminal vesicles and uterine sperm collected from mated female mice, productive infection of the preimplantation embryo was not demonstrated [16]. Since intranasal inoculation of MCMV causes a systemic, subclinical infection with severe interstitial pneumonitis [17], respiratory transmission may also be important in the natural history of the virus. In combination, these studies point to saliva, urine, skin, blood and respiratory secretions as sources of MCMV transmission. The virus has minimal effects on the survival or breeding of immunologically competent laboratory mice [9].

The two murine parvoviruses, MVM and MPV, infect the intestine during acute infection (about 3 weeks for MVM, as long as 6–9 weeks for MPV) [18], and MVM also replicates extensively in kidney [19]. Thus, faecal–oral transmission probably occurs with both viruses, and MVM is thought to be transmitted by urine as well. Both viruses cause subclinical infections in laboratory mice that are more than 24 h old. One allotropic variant of MVM does cause lethal infection of neonates of certain susceptible genotypes [20]. Transplacental transmission has not been demonstrated for either virus.

In this paper we report on the development of an outbreak of house mice in the Mallee region of northwestern Victoria in 1995–7 and investigate the seroprevalence of antibodies to MCMV, MVM and MPV in relation to these population changes. In particular we ask if there is any density-dependent relationship between seroprevalence of antibodies to these three viruses in this population.

METHODS

Study site

The study area was in the central mallee wheatlands of Victoria, Australia; 10 km south of Walpeup (142.02° E, 35.08° S). Annual rainfall for this region averages 340 mm and the climate is Mediterranean with hot summers. The soils are predominately sandy loams and the main crops are winter cereals with some pastures for sheep.

Capture-mark-release study

House mouse populations were live-trapped in three adjoining fields and three fence lines adjacent to these fields at monthly intervals from 8 February 1995 to 29 October 1997. Each trapping session was conducted for three consecutive nights. Longworth live-traps were used, and individual mice were ear-tagged, measured, bled, and released at the point of capture. The general methods used are described in Singleton [3].

Serology

Mice were bled from the sub-orbital venous plexus. Sera were screened for evidence of antibodies to murine cytomegalovirus (MCMV), minute virus of mice (MVM) and mouse parvovirus (MPV). Antibodies to MVM and MPV were detected by indirect immunofluorescence using sera diluted 1:10 in sterile saline [8, 21]. Antibodies to MCMV were detected by an enzyme-linked immunosorbent assay using sera diluted 1:50.

We aimed to screen a minimum of 20–25 mice each month at low population density (< 75 mice/ha) and 60 mice at higher population density (> 75 mice/ha). Where possible we tried to sample even numbers of males and females. There was no stratification across size classes. The sampling effort provides a 95% probability of detecting at least one seropositive mouse in a population with an expected prevalence of 10% [22]. If the number of mice caught was less than 25 then all of them were screened for antibodies.

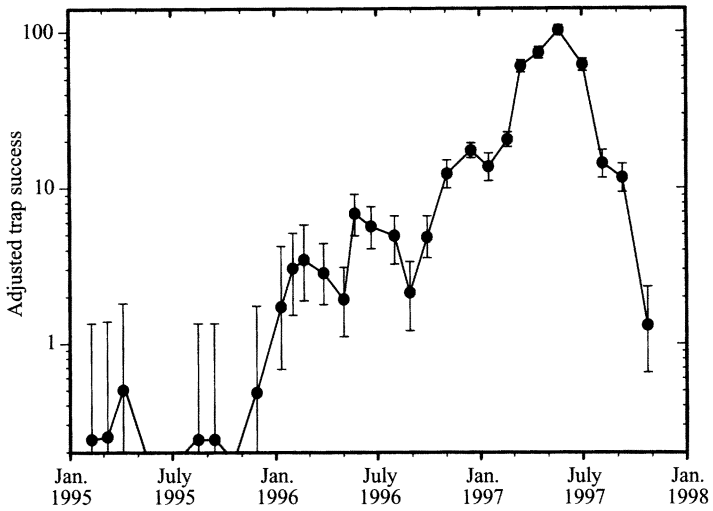

Fig. 1. Changes in house mouse density (log scale) in the Victorian mallee wheatlands during 1995–7 as measured by adjusted trap success. Binomial 95% confidence limits are given for each estimate. The population increase is over two orders of magnitude.

Population analyses

Population densities of mice were estimated in two ways. Adjusted trap success was calculated as the adjusted index of the number of mice caught per 100 trap nights as described in Caughley [23]. Petersen estimates of population density were obtained by treating days 1 and 2 of each trapping session as a combined sample and recording recaptures obtained on day 3 as the second Petersen sample [24]. In addition we estimated densities, rates of increase, and survival rates from the Jolly–Seber mark-recapture model [24].

The presence of antiviral antibodies in mouse sera (seroprevalence) was recorded as a binary response (present or absent) for each mouse. The effects of length (head-body), weight, sex and sampling time on seroprevalence were analysed by fitting generalized linear models with binomial errors and the logit link. The significance of the respective effects and their interactions were estimated using deviance values which approximate a chi-squared. The analysis did not include the first year of the study when too few mice were caught.

RESULTS

Population changes

Figure 1 shows the development of the 1997 mouse outbreak. For the first year of the study from February 1995 to February 1996 there were almost no mice caught and average densities were less than 1 mouse per ha. The population eruption began to develop during the 1995–6 breeding season and from November 1995 to May 1996 the population increased at an average rate of 7% per week, a doubling time of about 10 weeks. After a brief lull over the winter of 1996 the mouse population increased again rapidly from August 1996 to June 1997 at an average finite rate of 10% per week, a doubling time of about 7·5 weeks. From a peak around 250 mice/ha in late May 1997 the mouse population collapsed over the next 2 months, falling 19% per week, and fell to an estimated 5 mice/ha in late October 1997.

Petersen estimates of population density were closely related to the adjusted trap success ($r = 0{\cdot}97$, $n = 20$), with a peak of 101% trap success associated with a Petersen estimate of 252 per ha in late May 1997. A similar relationship was found with Jolly–Seber density estimates.

Figure 2 shows the percentage of adult females in breeding condition during this time period. Females were judged to be breeding if they were lactating or obviously pregnant. Since few mice were caught in 1995 it is not feasible to pinpoint the breeding season. During the winter of 1996 there was a brief non-breeding period in midwinter during June and July, but lactating females were recorded in late August 1996. This shortening of the winter non-breeding period is typical of developing mouse plagues [3]. By contrast in 1997 after the population collapse, breeding had stopped by late May 1997 and did not resume until October.

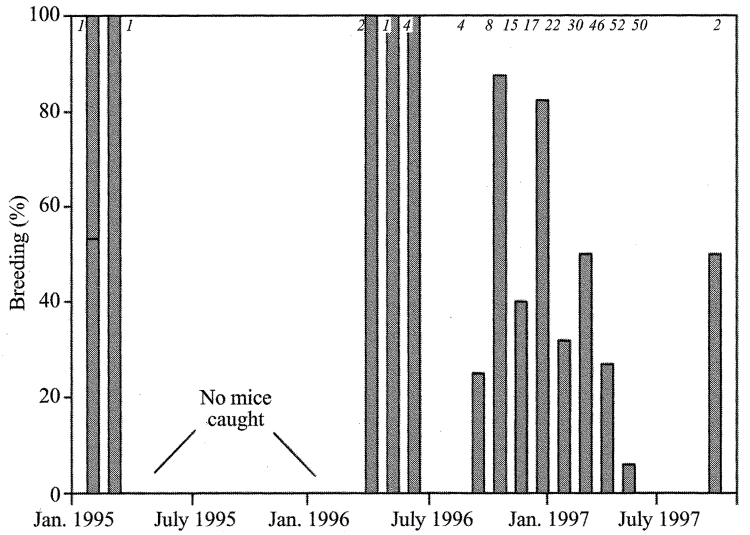

Fig. 2. Percentage of adult female mice in breeding condition. Sample sizes are shown on the graph.

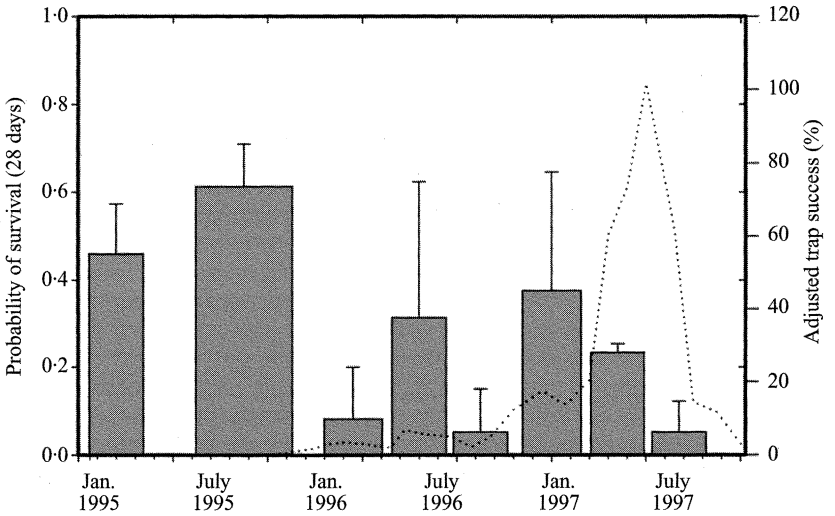

Fig. 3. Average survival probability (with 95% CI) for house mice from all habitats combined during 1995–7 in the Victorian mallee wheatlands. Changes in population abundance of mice are shown as a dotted line. Survival was estimated from the Jolly–Seber model.

Figure 3 shows the survival changes that accompanied the outbreak in numbers. In general survival was high during the period of low numbers in 1995 and then was at moderate levels during the outbreak. Two periods of low survival occurred in late winter and early spring (July–September) of 1996 and 1997. Survival was exceptionally low in 1997 when the outbreak collapsed, and only 2% of the mice survived per month during the winter of 1997.

Serology

The mouse population was seropositive for all three viruses, but their prevalence varied dramatically over time (Figs 4–6). MVM had the lowest average seroprevalence (8·5%, $n = 751$), followed by MPV at 18·4% ($n = 755$) and the highest prevalence occurred in MCMV (61·7%, $n = 731$).

Seroprevalence did not differ significantly between males and females for any of the three viruses. For MCMV and MPV there were significant weight and length (head–body) effects with larger animals having a higher seroprevalence. However, weight and length were significantly correlated ($r = 0{\cdot}84$ for males; $r = 0{\cdot}88$ for females; $r = 0{\cdot}811$ for pregnant females). The linear modelling indicated that length was the better predictor of seroprevalence. The deviance values for length for the respective viruses provided the following significance values: MCMV, $\chi^2(1) = 32{\cdot}9$, $P < 0{\cdot}001$; MPV, $\chi^2(1) = 21{\cdot}6$, $P < 0{\cdot}001$; MVM, $\chi^2(1) = 2{\cdot}8$,

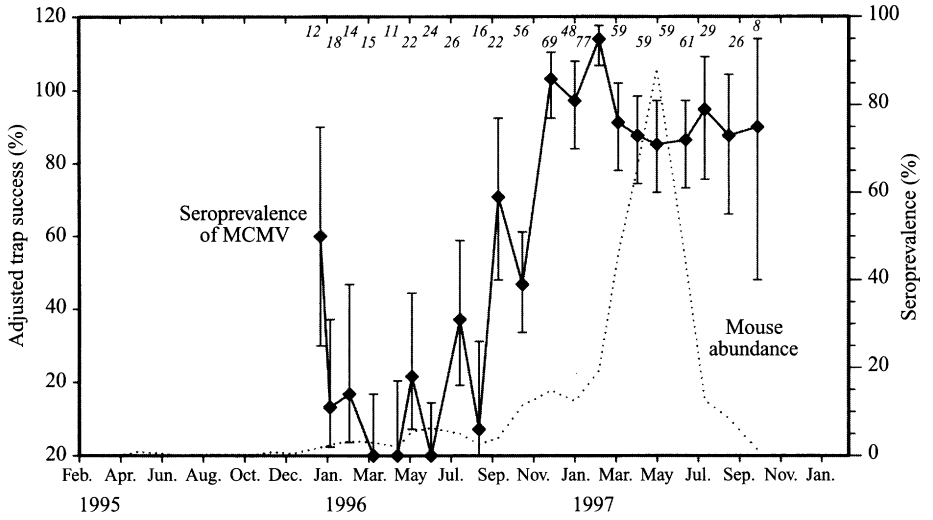

Fig. 4. Seroprevalence of murine cytomegalovirus (MCMV, ◆) in house mice from the Victorian mallee wheatlands during 1995–7. Changes in population abundance of mice are shown as a dotted line. Binomial 90% confidence limits are indicated for seroprevalence. Sample sizes for the respective sampling periods are given at the top of the graph.

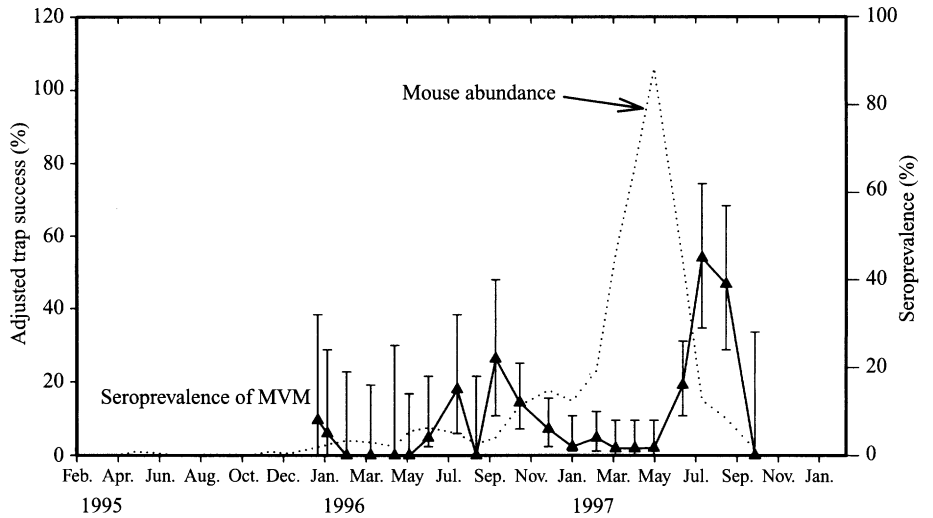

Fig. 5. Seroprevalence of minute virus of mice (MVM, ▲) in house mice from the Victorian mallee wheatlands during 1995–7. Changes in population abundance of mice are shown as a dotted line. Binomial 90% confidence limits are indicated for seroprevalence. The sample sizes for each sampling period are similar to those shown on Fig. 4.

$P > 0{\cdot}05$. Although there were significant differences with the size of mice and seroprevalence within a month, there was no apparent pattern between months; the mean length of mice each month was similar throughout the study.

The critical questions we wish to address here are whether seroprevalence is related to population density and survival rates in these mice. To determine this we used time series analysis and calculated the cross correlations first between seroprevalence and population density (estimated by adjusted trap success) and second between seroprevalence and survival rates. Table 1 presents the results of these correlations for population density. There is a broad zone of correlation between MCMV levels and population density with -4 to $+1$ months time lag. This can be seen in Figure 4 because MCMV prevalence rose rapidly to a high level around 80% once mouse numbers began to increase in October 1996, and seroprevalence remained high through the decline period.

By contrast the correlations of MVM prevalence with population density occur only with a $+2$ to $+3$ month time lag (Table 1). This is evident in Figure 5 because MVM prevalence increased dramatically as the population collapsed in July 1997.

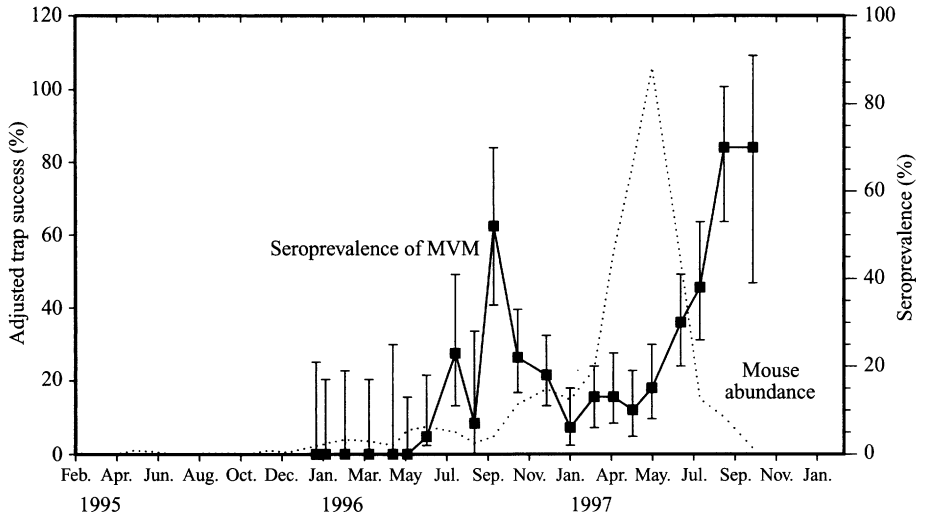

Fig. 6. Seroprevalence of mouse parvovirus (MPV, ■) in house mice from the Victorian mallee wheatlands during 1995–7. Changes in population abundance of mice are shown as a dotted line. Binomial 90% confidence limits are indicated for seroprevalence. The sample sizes for each sampling period are similar to those shown on Fig. 4.

Table 1. *Time lag correlations of seroprevalence of three viruses and house mouse population density in the Victorian mallee, 1995–7*

Time lag* (months)	Virus† MCMV	MVM	MPV
0	**0·48**	−0·08	−0·02
1	**0·49**	0·26	0·15
2	0·47	**0·69**	0·41
3	0·42	**0·68**	**0·71**
4	0·30	0·41	**0·69**
5	0·11	0·16	0·42
6	−0·01	−0·04	0·18

* Population density is correlated with seroprevalence 0–6 months later in this table.
† Bold values are statistically significant at the 5% level.

Table 2. *Time lag correlations of seroprevalence of three viruses and house mouse survival rates* in the Victorian mallee, 1995–7*

Time lag (months)	Virus MCMV	MVM	MPV
0	−0·07	−0·27	−0·17
−1	−0·11	−0·11	0·14
−2	−0·28	−0·10	−0·06
−3	−0·19	0·15	0·25
−4	−0·41	−0·03	−0·07
−5	−0·29	0·10	0·05
−6	−0·31	−0·06	−0·24

* Survival rates are correlated with seroprevalence 0–6 months previous in this table; none of these values is statistically significant at the 5% level.

The correlations of MPV prevalence with population density occur with a +3 to +4 month time lag, the longest time lag of the three viruses. This is shown in Figure 6 because MVM prevalence increased dramatically as the population collapsed in July and August 1997, and remained a high prevalence throughout the population collapse to near zero by November 1997. MPV prevalence showed as high a correlation as MVM and both of these viruses were more highly correlated to the population density changes than was MCMV (Table 1). An alternative model to explain these MVM and MPV correlations is that they are seasonal and increase in prevalence in the spring season (September to November) every year.

Table 2 presents the time-lag correlations between viral seroprevalence and survival rates. We might expect seroprevalence to be correlated with survival rates that occur earlier in the time series. Alternatively, mice could die because infection makes them more susceptible to other lethal processes leaving a residual population with high seroprevalence. None of the correlations in Table 2 is statistically significant.

DISCUSSION

This paper builds on the observations of Singleton and colleagues [7] on the associations between population changes in house mice and the seroprevalence of antibodies to viruses. Serologic tests are

not ideal for studies of epizootics because they provide a history of infection, and we do not know whether a seropositive mouse was recently infected and was shedding virus or whether the mouse had recovered from any effects of the virus infection. However, both MCMV and MPV are associated with persistent infections so it is likely that seropositive mice are also actively infected. One simplifying factor is that seroconversion for each virus generally occurs at 7–14 days [11, 21, 25, 26]. We have improved on the previous studies by sampling at about twice the frequency as Singleton and colleagues [7] and for 3 years rather than 2, so we have a better picture of the dynamics of these infections.

Our results differed significantly from those of Singleton and colleagues [7] in the seroprevalence of MCMV. In this study MCMV prevalence was correlated with population density and responded to the growth in the density of mice by increasing from less than 10% prevalence to over 80% in 3 months. Singleton and colleagues [7] found MCMV only at very high prevalence at all population densities during their one-year study. In the 1993 study the adjusted abundance index for mice was 5–10% at the beginning of the study and remained higher than 20% thereafter. Similar results were reported by Moro and colleagues [27] for an island population of mice. Over 2·5 years the seroprevalence was consistently higher than 60% (range, 45–97%), even during a 9-month period when trap success was less than 10%. Indeed, the highest estimated density of mice during the 2·5 years was about 60 mice/ha.

In the current study, the abundance index was near zero for the first 11 months of the study, and then fluctuated between 2 and 7·5% for the next 9 months (January–September 1996). There were only sufficient mice to analyse the dynamics in seroprevalence from January 1996 and in the 9 months until September the seroprevalence fluctuated markedly, ranging from 0 to 50% with high confidence limits on these estimates. The seroprevalence of MCMV increased dramatically to $> 55\%$ once the population abundance index was consistently higher than 14%. This is equivalent to approximately 40–100 mice/ha. Together, the findings of Singleton and colleagues [7] and the current study indicate that seroprevalence of MCMV is dependent on the density of mouse populations and/or changes in the social or spacing behaviour of mice resulting in higher contact rates and thence high transmission rates of MCMV. It is thought that MCMV transmission occurs via saliva and sexual secretions [13–17, 26]; both routes require close contact. Regardless of whether the dynamics of transmission is driven by changes in behaviour or population density, there appears to be a threshold level of host density above which MCMV prevalence is high, as supported by most models of host–disease interactions [28] and field studies that often show that there is not a simple correlation between rodent population density and prevalence of infection in these populations [see 29 for review]. This conclusion of a threshold density for MCMV is not supported by the study of Moro and colleagues [27] on its dynamics in an island population of mice, but this may be explained by the peculiarities associated with island populations and/or a coarser data set – they only had 11 estimates of seroprevalence over 2·5 years.

Our findings with MVM corroborate and extend the conclusion of Singleton and colleagues [7] that seropositivity to this virus increases dramatically in prevalence during the decline phase of outbreaks of mouse populations (Fig. 5). Sampling after the outbreak was over in October 1997 permitted us to confirm that the seroprevalence of MVM had also declined in the post-outbreak population. These results indicate that further studies of the epidemiology of MVM under field conditions are warranted to determine the mechanism of these epizootics in wild mouse populations.

MPV is a newly recognized virus infecting laboratory mice at moderate prevalence [10]. This is, however, the first report of infection of wild mice with this agent. In the laboratory mouse, both MVM and MPV preferentially infect the intestine and lymph nodes. MPV differs from MVM by causing a persistent rather than acute and self-limiting infection. However, MPV is cleared from the intestine sometime 4–9 weeks after infection. The site of persistence is lymphoid tissue so the potential for transmission during the persistent phase is limited. We propose, then, that the natural epizootiology of the two viruses in laboratory mice is similar, with MPV perhaps being shed for a somewhat longer time. It would be of interest to compare the pathogenesis of MPV in laboratory and wild stocks of mice since its presence in wild mouse populations suggests they were the original source of laboratory mouse infections.

As reported in the previous study, the seroprevalences of MCMV and MVM were strongly associated with the size of the animal. Larger animals have a higher probability of being seropositive. This indicates that older animals have a greater likelihood

of becoming infected with MCMV and MVM. However, the time series analysis suggests that population density is a stronger factor influencing the seroprevalence of these viruses.

In conclusion, the current study supports the contention that MCMV would be a good carrier for an immunocontraceptive agent [see 9, 30]. The time series data indicate that the virus spreads rapidly through populations of mice once the level of population abundance is greater than 10%, and that seroprevalence will remain high for at least 4 months after mouse populations have declined following an outbreak.

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