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Can changes in social behaviour help to explain house mouse plagues in Australia?

Charles J. Krebs and Dennis Chitty, Dept of Zoology, Univ. of British Columbia, Vancouver, B.C., Canada V6T 1Z4 –
Grant Singleton, CSIRO Div. of Wildlife and Ecology, P.O. Box 84, Lyneham, A.C.T. 2602, Australia – Rudy Boonstra, Div. of Life Sciences, Scarborough Campus, Univ. of Toronto, Scarborough, Ontario, Canada M1C 1A4

House mouse plagues in the grain-growing areas of eastern Australia are a graphic illustration of the failure of social mechanisms of population control that are postulated by the self-regulation hypothesis to prevent unlimited increase in numbers. Yet house mice are well known for the strength and variety of social interactions and are clearly capable of regulating their own numbers through social mortality. Most of the research on house mouse plagues has assumed that extrinsic agencies – predators, diseases, food supplies, and weather – determine when and where mouse plagues will occur. Some aspects of these plagues cannot, however, be explained that easily, among them the low phase, which may persist for 1–3 years. We focus here on the low phase of plagues and the trigger that flips a population from the low into the increase phase of a plague. Can social factors in house mouse populations explain the low phase, and is a change in social organization a necessary condition for generating a plague? Two possible models are presented to suggest predictions to be tested by further studies of social mechanisms of population limitation in feral house mice.

House mouse plagues in Australia are superficially similar to the population cycles of small native rodents in the Northern Hemisphere. Phases of increase, peak, decline, and low follow one another at perhaps more regular 3–4 year intervals in voles and lemmings than they do in house mouse plagues (e.g. Saunders and Giles 1977). But the outbreak sequence is the key to understanding these fluctuations, whether strictly periodical or not (Chitty 1960). Much of the discussion of the causes of house mouse plagues in Australia is repetitive of a much larger literature of the causes of cycles in the Northern Hemisphere, and this could mean that house mouse studies in Australia are making some of the same errors made in the Northern Hemisphere. The purpose of this paper is to suggest a new approach to this important agricultural problem by applying an idea that has helped to explain some of the peculiarities of vole and lemming cycles in the Northern Hemisphere, namely that changes in social behaviour are associated with changes in numbers (Krebs 1985). This idea suggests some new ways of approaching the mouse plague problem.

The self-regulation hypothesis postulates that population growth can be prevented by a deterioration in the quality of individuals as populations increase (Chitty 1960, 1967). It has shifted the attention of population ecologists from the *number* of animals to the *types* of animals that make up populations. In Chitty's view social interactions between different types of animals affect population dynamics. As a population increases, peaks, and declines the critical qualitative change is proposed to be in the social environment, and some form of social mortality – infanticide, direct wounding, dispersal leading to death – is a necessary component of the mechanisms that stop population growth. The self-regulation hypothesis was proposed to explain population cycles in small mammals in the Northern Hemisphere, but Chitty (1960) claimed a generality for his model and encouraged others to investigate its applicability to other organisms. Here we ask whether it can address the problem of house mouse plagues in the grain-growing regions of southeastern Australia. Under apparently favourable conditions some house mouse populations do not necessarily increase, and the problem is to discover the difference between them and populations that do grow to plague levels. House mice do indeed have forms of spacing behaviour that should enable them to regulate their own numbers through wounding, dispersal, infanticide, or other forms of social mortality (Lidicker 1976, Macintosh 1978, Singleton and Hay 1983).

Table 1. Predictions from two possible applications of the self-regulation hypothesis to the explanation of house mouse plagues in the grain-growing regions of southeastern Australia. Two models are considered based on when territoriality is favoured.

Model I	Increase stage	Peak stage	Decline stage	Low stage
Aggressiveness	low but increasing	high	high	decreasing
Territorial behaviour	weak, but increasing	strong	?	strong
Dispersal – type	presaturation	saturation	minimal	presaturation
– rate	high	low	very low	high
Infanticide	low, increasing	high	–	low
Intruder pressure	low, increasing	high	high, decreasing	low
Wounding	low	high	high	low
Social system	open	closed	closed	closed
Model II	**Increase stage**	**Peak stage**	**Decline stage**	**Low stage**
Aggressiveness	high	decreasing	low	low
Territorial behaviour	strong	declining, none	none	none
Dispersal – type	presaturation	nomadism	nomadism	nomadism
– rate	high	very high	very high	very high
Infanticide	low	high	–	moderate
Intruder pressure	low, increasing	high	high, decreasing	low
Wounding	low, increasing	lower	low	low
Social system	closed	open	open	open

House mouse plagues occur irregularly in the agricultural areas of southeastern Australia (Newsome 1969a, b, Saunders and Giles 1977, Redhead et al. 1985, Singleton and Redhead 1989, Boonstra and Redhead 1994). As such they may not seem to offer a good example of a population system to which the self-regulation hypothesis might be applied. Plagues are associated with rainfall and drought in ways that suggest control by the physical rather than the social environment (Newsome 1969a, Saunders and Giles 1977). Virtually all authors who have studied house mice in Australia explain their population trends by changes in the extrinsic factors of food, shelter, predation, and possibly disease. Redhead (1982) is a major exception. He considered the role of spacing behaviour in mouse outbreaks, and emphasized the role of dispersal and colonization in the early increase phase.

The collapse of house mouse plagues occurs very rapidly in a matter of days or weeks (Newsome 1969b, Redhead 1982, Singleton 1989), and the proximate explanation for the collapse is food shortage aggravated by disease (Smith et al. 1993). Mouse plagues represent the failure of the machinery of population regulation and in particular the social mechanisms inherent in the self-regulation hypothesis to prevent overpopulation and subsequent starvation. Given the failure of social population regulation, the limits to growth seem to be set by food supplies or disease. Much work has been done on the increase and peak phases of an outbreak in order to explain the conditions leading to the decline. We suggest that it may be profitable to focus our attention not on the collapse of a plague but on the low phase and the processes that start population growth.

In this paper we focus our discussion on three phases of a house mouse plague, and attempt to derive predictions from the self-regulation hypothesis for these phases. We ask three questions in particular: 1. What ecological changes initiate a plague? 2. What processes stop rapid population increase? 3. What keeps populations in the low phase?

The central issue we raise is whether or not individual differences in social behaviour are relevant to understanding the generation of mouse plagues.

Plague initiation

The concept of a plague-trigger is inherent in all the conventional explanations of house mouse plagues in Australia. The timing of rainfall in relation to seasonal agricultural activities has always been central to these models. Newsome (1969a) postulated that mouse plagues could develop only when there was a coincidence of good winter rains and suitable soft soil for burrowing. Redhead (1982) emphasized the importance of autumn rains before the outbreak. Singleton (1989) suggested that high autumn and winter rainfall could trigger a plague in the Victorian mallee.

All the plague-trigger hypotheses suffer from the problem of explaining away instances of rainfall events after droughts that are *not* followed by mouse plagues. For example, Saunders and Giles (1977) described 8 mouse plagues from 1900–1970 that were preceded 1–2 years by drought-breaking rains, but recognized four droughts in these years that were not followed by plagues after the drought ended. Mutze et al. (1990) used a binomial model to predict mouse plagues in South Australia and found

that rainfall could explain at best about 41% of the variation in plague occurrence. The usual ad-hoc explanation for the failure to reach plague proportions is that heavy predation or disease prevented the outbreaks from developing. In some cases breeding may stop early and the plague be averted because of reproductive failure. The role of drought as a plague trigger was also questioned by Boonstra and Redhead (1994) who showed that the timing of a mouse plague was the same in irrigated rice-growing areas as in dryland farming areas.

One alternative explanation is that house mouse populations can increase rapidly only when they are composed of "increase" phenotypes. In this model favourable food supplies and burrowing conditions are necessary for a mouse plague to start but are not sufficient. This is the same hypothesis proposed to explain the phase of low numbers in cyclic rodents (Chitty 1960).

We can test this alternative model only if we can recognize "increase" phenotypes. We defer for the moment the question of whether there may be a genetic component to these phenotypes. One mechanism for the self-regulation hypothesis postulated by Chitty (1967) and Krebs (1978) was that increase-types were non-aggressive and as the population grew these were replaced by "peak-types" that were highly aggressive. This specific mechanism was rejected by Krebs (1985) and by Chitty (1987), who suggested that increase types were aggressive in populations of *Microtus townsendii*. Alternative mechanisms have been suggested for social regulation in voles. Relatedness was postulated by Charnov and Finerty (1980) and by Lambin and Krebs (1991) as a potential mechanism affecting population growth rates in voles. An array of strictly phenotypic hypotheses have also been suggested by Boonstra (1994) to affect vole populations through maternal effects caused by stress and through age-structure shifts.

We propose here two alternative models that can be used to apply the self-regulation hypothesis to feral house mice in agricultural landscapes. These models are conceptual and specify predictions that can be tested in field populations. Model I is the original mechanism suggested for the application of these ideas to northern voles and lemmings (Table 1). In this model increasing phenotypes are docile and as crowding increases there is selection for more aggressive phenotypes. Peak populations consist of high aggressiveness animals and these persist through the decline. During the low phase docile phenotypes slowly return. This model assumes that high reproduction is associated with docility, and that low reproduction and infanticide are associated with aggressiveness. It is the version of the Chitty Hypothesis modelled by Page and Bergerud (1984). In this version of the hypothesis, decline and low phase mice are high in aggressiveness and strongly territorial, and the key feature of the plague trigger is a loss of aggressiveness and territorial defence so that the social system becomes open, and reproductive success increases.

Model II of the self-regulation hypothesis (Table 1) is the variant that is supported by Chitty (1987) for voles and is consistent with many of the observations of Newsome (1969a, b) on house mice in South Australia. This model postulates that low-phase phenotypes in house mice are non-aggressive individuals that avoid one another, perhaps through having a nomadic social organization. The plague trigger in this model is an increase in aggressiveness leading to strongly territorial behaviour. This switch from nomadic to territorial social organization also leads to territorial social groups exporting their surplus production via presaturation dispersal (sensu Lidicker 1975). These individuals in the increase phase support a stable social organization of the type originally postulated by Anderson (1961) as typical of feral house mice everywhere. The plague-trigger in this model is the change to a rigid territorial social system, and we postulate two reasons for this change. First, on a landscape level, widespread availability of suitable breeding habitat leads to population growth and an attendant increase in intruder pressure that is countered by establishing territories. Second, on a social level, aggressive phenotypes infiltrate the breeding structure and contribute to reproduction. This shift is associated with improving reproductive success in aggressive individuals that devote energy to aggressive defence of space. From an ecological perspective, it does not matter whether these phenotypes are genetically fixed or have alternative breeding strategies determined by early experience.

For this model to work, a nomadic social organization must result in zero population growth. This could result from poor adult survival associated with movements or from increased social mortality due to poor nest defence. Nomadic social organization in which individuals are not site attached (except for the time of raising a litter) is unusual in rodents but may occur in feral house mice (Krebs et al. 1995). Infanticide by adult males could be a feature of this type of social organisation in the low phase (Wolff and Cicirello 1989). We do not know why mice would adopt a nomadic social organization at a time of low population density, and it is possible that they are trying to make the best of a bad situation.

We do not yet have the data to decide whether Model I or Model II applies to wild house mice in Australia or to determine if both are irrelevant to population outbreaks. From our field experience we suspect that Model II is closer to the field situation in Australia, but we emphasize that both models need testing.

We predict that the key process of plague initiation is a social change in the breeding system of house mice. Changes in extrinsic factors such as predation, food supplies, or disease are relevant to initiating plagues only insofar as they are permissive factors. Population growth will not occur unless there is adequate food, low levels of predation and disease, and favourable weather. But given that all these extrinsic factors are favourable, a plague will not be triggered without the proper social environment. Our conceptual models integrate both extrinsic and intrinsic factors in the generation of plagues.

What stops population growth?

The self-regulation hypothesis suggests that population growth ceases because of a deterioration in the social environment. In house mouse plagues, as the density increases to high levels late in the increase phase, a smaller fraction of the population reaches sexual maturity, and reproduction is eventually curtailed. But these social processes are not sufficient to prevent excessive densities. In peak populations house mice are non-aggressive and most individuals remain sexually inactive. Those that continue to breed presumably maintain enough aggression to defend their nests against intruders. More and more of the population becomes nomadic rather than site-attached, and once breeding has stopped in the peak of the plague, nomadism becomes the rule (Krebs et al. 1995).

Population growth stops in mouse plagues because breeding ceases, and the ultimate cause of this is presumably social strife rather than resource limitations. The peak of the outbreak is usually followed directly by a rapid decline in density during the non-breeding season. The proximate cause of the decline may be a disease (Smith et al. 1993), and declines may be associated with food shortage, though this is not necessary (Redhead 1982, Singleton 1989). There is little controversy, however, over the conventional view that the collapse is due to disease, food shortage, or limited shelter either separately or in combination. Social mortality appears to play virtually no role at this stage, as there is little sign of wounding (Redhead 1982, Singleton 1989). This is consistent with the application of the self-regulation hypothesis, according to which social strife during the late increase and peak phases, in addition to its effect on reproduction, has also rendered the animals more likely to die of anything whatever. A key difference is that house mouse plagues collapse only during the non-breeding period, whereas cyclic declines of voles and lemmings occur in both the breeding and the non-breeding seasons (Krebs and Myers 1974).

What maintains the low phase?

The low density phase of vole and lemming cycles is much less studied than the other phases, and this is also true for house mice in Australia. For both of these systems it is known that even if food resources, burrows, and climatic conditions are excellent in the year following the decline, there will not be a return to the increase phase. In house mice there must be at least a 1-year delay and in most cases a 2–3 year delay (Mutze 1990, Singleton and Redhead 1990). There are three general ideas of why there might be these long periods at low density:

1. House mice at low density are locked into a predator-pit (Sinclair et al. 1990). An array of generalist bird and mammal predators can take sufficient mice at low mouse densities to keep them in check, although once an increase begins, the mice escape from predator regulation. This explanation can be tested by reducing predation on a low house mouse population.

2. House mice need two concurrent resources – food and burrows – and these do not occur at the same time during the low phase because of irregular rainfall (Newsome 1969a). Redhead (1982) tested this idea by analysing mouse plagues on irrigated rice farms, and he rejected this idea. This explanation might apply to a few areas of specific clay soils, but does not seem to apply throughout the geographic range of mouse plagues in southeastern Australia (Mutze et al. 1990).

3. House mice are regulated by social behaviour at low densities. Two possibilities are given in Table 1. Model I postulates that strict territorial behaviour coupled with emigration and poor survival of excess juveniles might hold numbers low for a prolonged period if it is coupled with an intolerance of immigrants. Model II postulates an asocial, nomadic structure during the low phase with high losses from infanticide. These hypotheses could be tested by studying the social organization of house mice during a low phase, and by measuring the fate of juveniles. Social mortality must ultimately be a large component of loss in the low phase if this third explanation is applicable.

The first two hypotheses are based on the supposition that there is something wrong with the environment during the low phase. The third hypothesis postulates that there is something wrong with the mice in a low phase, or at least something different about them (Chitty 1967). There are three mechanisms which could lead to something being wrong with mice in the low phase. Individuals could have suffered from malnutrition via their mothers (Grandmother effect; Mech et al. 1991). Alternatively, the effects of maternal stress can be transmitted from mothers to offspring (Boonstra 1994). Third, there could be alternating selection for and against aggressiveness during a plague (Chitty 1967, Krebs 1978). Selection for or against aggressiveness has not been found in laboratory populations of *Mus* (van Zegeren 1980), but Singleton and Hay (1982) have measured a high heritability of aggressiveness in feral house mice in Australia. No one knows if selection for or against aggressiveness operates during a mouse plague, and it would be useful to find out.

An experimental test can distinguish the first two hypotheses from the third. If individuals from a low phase mouse population can be moved to a favourable field environment with good food and water, adequate burrows, and no predators, they ought not to increase under the third hypothesis. This experiment could fail because of a disruption of social organization caused by setting up the experiment. As an alternative we suggest a laboratory experiment in which samples of mice from the different phases of an outbreak are brought back to a standardized laboratory colony for the analysis of reproductive potential, studies of cross-fostering and heritability (e.g. Mihok

and Boonstra 1992). Only by experiments of this type can we determine whether changes in social organization are a *cause* of population changes or only an *effect* of density changes. We propose a two step attack: (1) to determine if there are any changes in social behaviour associated with the low phase of mouse plagues, and (2) if there are, to manipulate these behavioural changes by the use of hormones and drugs to determine whether they are a necessary cause of demographic change.

Synthesis

We attempt in Table 1 to draw these observations together and to identify two models of how the characteristics of house mice from different years of a mouse plague might differ. The important point is to identify potential experiments that can be done to help us increase our understanding of house mouse plagues. It is clear from this brief survey that the self-regulation hypothesis can be most usefully applied to the low phase of house mouse outbreaks. Social mechanisms may operate during the low phase to restrict population growth. When these break down, a plague develops, and, although high density by itself causes reproduction to stop and mortality to increase, it is clear that this does not happen in a mouse plague until food is scarce (relatively rare) or a disease intervenes to reduce numbers (more common). House mice in agricultural landscapes illustrate the failure of the mechanisms of self-regulation to prevent unlimited increase in numbers. We know that these social mechanisms – territoriality, infanticide, and aggression – are present in Australian house mice from laboratory and enclosure studies (Singleton and Hay 1983). However, the breakdown of social mechanisms of population control at high numbers does not invalidate their importance at low numbers. The key focus of future demographic work must be on the change from the low phase to the increase phase, and if we can determine the plague trigger we may be able to manage plagues and prevent agricultural damage.

To determine the dynamics of house mouse populations during the low phase we need to use radio-telemetry and mark-recapture work to measure social organization and demographic processes such as emigration and immigration. For the present it does not matter whether or not there are genetic changes in house mice during a plague. It is more important to find out if social organization differs from year to year. Since the alternative model that extrinsic factors operate exclusively as a plague trigger has been only partly successful in predicting plagues (Mutze et al. 1990), it may be useful to approach the problem from the assumption that there is something different about the mice in the low phase that prevents them from increasing. The costs and benefits of territorial defence need to be measured during the low phase if we are to understand the mechanisms behind alternative forms of social organization at different population densities.

Laboratory comparisons of the reproductive fitness of mice from increasing populations should also be contrasted with those from declining or low populations. Mihok and Boonstra (1992) used this technique to show that voles (*Microtus pennsylvanicus*) from low-phase populations did not reproduce well even in ideal laboratory conditions.

There are virtually no data on the genetic architecture of feral mouse populations in the sense of the spatial distribution of relatives, yet there is increasing evidence that the relatedness of neighbours can have important consequences for recruitment and survival in birds and mammals (Lambin and Krebs 1991, Moss and Watson 1991). The plague trigger for house mice could involve a spatial organization of relatives similar to that now demonstrated in red grouse (Moss and Watson 1991).

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