

# Of mice and men — and nematodes

Although cute enough to be kept as pets and immortalised in cartoons, mice can be a serious problem, frequently attaining the status of a destructive pest in many parts of the world. At certain times, in rural Australia (especially the cereal-growing belt in the eastern and southern parts of the country), numbers of house mice (*Mus domesticus*) increase to plague proportions. The results are often serious.

The Victorian government estimated that the 1979/80 mouse plague — mainly in the north-west of the State, but extending to parts of South Australia and New South Wales as well — caused crop losses of \$15–20 million in Victoria alone. By

extrapolation to the other States, total losses might have been \$40–50 million.

Other losses arise from damage to equipment and vehicles that mice gnaw or foul. In rural townships and small settlements, mice cause loss of stored foodstuffs

by eating them or by contaminating them with faeces and urine. (And scientists have been known to lose valuable data when mice entered an unmanned mobile laboratory — the animals chewed the accumulated computer printouts to oblivion!)

For those living on the land in stricken areas, mouse plagues can be terrifying. The animals may enter houses in swarms and chew their way through metres of wire — destroying, in the process, televisions, fridges, or stoves. They will gnaw and contaminate clothing, run into the bedrooms and over people's faces at night, and leave an unpleasant stench everywhere.

Of course, such conditions are dangerously unhygienic. More than 40 diseases can be transmitted from mice to humans. In Australia mice have not yet, as far as we know, played a part in spreading an exotic disease. However, medical researchers have implicated them in cases of food poisoning caused by *Salmonella* bacteria.

Clearly, for economic, social, and health reasons we need to be able to predict and control the outbreak of plagues. To do this we need to know a lot more about the mice themselves. Dr Trevor Redhead, in charge of the rodent control research laboratory of CSIRO's Division of Wildlife and Rangelands Research, has been studying rodent population dynamics for 10 years.

He has found that house mice are quite rare in rural Australia in the years between plagues, and indeed may often be absent from those areas they come to occupy during a plague. A series of as-yet-incompletely understood factors transforms the population from this state to plague proportions. The increase in numbers, however, is not totally unpredictable, nor is it smooth and continuous. Dr Redhead's population monitoring and close study of a plague in the Murrumbidgee Irrigation Area in 1979/80 has enabled him to devise a model of population changes, which shows that a plague comprises three phases — usually spread over 3 years. (See the diagram on page 8.)

According to this model, the original environmental trigger of the 1979/80 plague was the above-average rainfall that occurred in the autumn of 1978 — nearly 2 years before the main population increase. The good rains meant that high-quality food remained available further into autumn than usual; this, in turn, extended the breeding season.



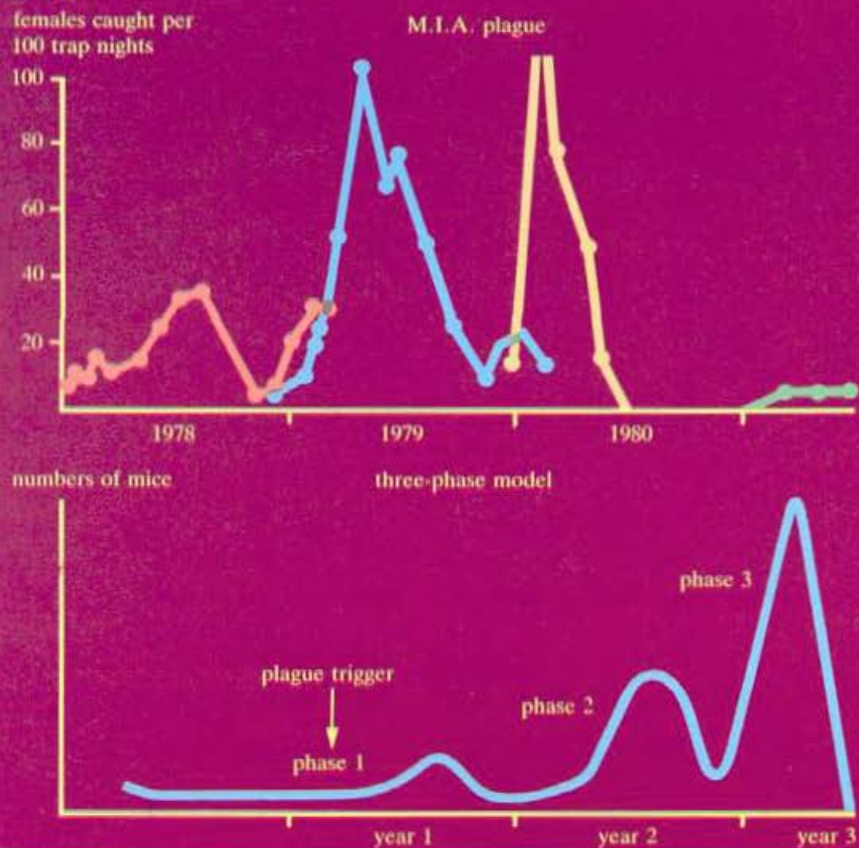
The possible start of a mouse plague.



The result can be hordes of the creatures — as shown here after 4 nights of trapping during the 1917 plague in Lascelles, Victoria.



## Build-up



Mouse catch data collected in the Murrumbidgee Irrigation Area between 1978 and 1980 (top) and Dr Redhead's model of a three-stage plague (below). Because mouse numbers peaked and declined so rapidly at the beginning of 1980, the scientists were not able to obtain a data point at the height of the plague.

Subsequently, Dr Mary Bomford of the same Division carried out an experiment to test Dr Redhead's model. She found that increasing the protein in the diet in autumn permitted a high level of breeding to continue for longer. Also, following the above-average autumn rain, the scientists found that the mice were in better condition than usual. This is assessed using a 'condition index' based on the weight and length of the animals, which had grown more rapidly than expected during the winter. (In most winters in the area studied mice do not grow at all.)

Because of this better-than-average condition, when the breeding season arrived the following summer the females produced more offspring than usual — litter size averaged 9.6, against the figure of 6.4 at the beginning of the 1977/78 breeding season. During the winter of 1979 a large number of females survived — in fact, about 3–4 times as many as at the corresponding time in 1978. This meant that the summer breeding season at the end of 1979 resulted in a huge increase in numbers —

the third and final phase of Dr Redhead's model — and the plague was upon us.

Numbers alone are not the whole problem. Mice have an exploratory instinct and, especially when crowded, tend to spread out in search of new food and new territories. It is this dispersal that causes our problems, and explains why Dr Redhead calls the house mouse a 'mammalian weed'.

The species has three important features in common with plant weeds. Firstly, it has a high reproductive potential — females as young as 6 weeks can give birth, and can then produce litters about every 4 weeks, with the average number of young per litter exceeding 9. Of course this potential is rarely realised because conditions are seldom suitable and breeding stops in winter. But the animals are quick to take advantage when conditions are good.

Secondly, mice can spread quickly, and can adapt to a wide range of different habitats.

Thirdly, they thrive in disturbed areas. Because of this last factor, many farming operations may actually provide favourable conditions for mouse plagues. Tilling may make it easier for mice to burrow, and sowing provides a ready source of edible seeds.

That is not to imply that agriculture alone is responsible. Plagues can occur in non-agricultural areas of the arid inland,

whereas they don't appear with any severity in Western Australia's wheat belt for reasons we presently don't understand. In natural bushland they are generally only found in large numbers following disturbance — for example, a fire in a eucalypt forest. They will then gradually become scarcer as the forest regenerates.

More stable populations exist in our cities and towns, particularly all along the eastern coastline and around Melbourne. The researchers have few data on the numbers present, as they have been concentrating on the more troublesome rural populations. It seems that mice rarely become apparent as plagues in the cities, although major food retailers may have problems, as may householders in the autumn. But the more benign and stable environment of the cities prevents the 'boom and bust' cycle of the animals that occurs with the fluctuating conditions of the bush.

## A biological mousetrap?

Effective control of mouse plagues will be difficult. Dr Redhead stresses that a good strategy will require several steps, which he has identified as: predict, inform, control, and assess.

By understanding the various factors, including climate, that influence mouse populations in the field, scientists seek to predict the onset of plagues with some accuracy. Monitoring the population by regular trapping will allow researchers to assess fluctuations and refine their prediction.

But knowing when a plague will occur, although this may be handy, doesn't stop it. Besides poisoning and trapping, which only give some short-term local relief, can we do anything to reduce mouse numbers?

**The spleen (top) and liver from an infected mouse. The parasites are visible as pale streaks and blobs in the liver. If present in sufficient numbers, the worms can block the movement of bile out of the liver, damaging that organ and causing death.**





Dr Grant Singleton, of the Division, who collaborates with Dr Redhead, may have an answer. With parasitologist Dr David Spratt, Dr Singleton has been studying the range of parasites that infest mice in Australia, with a view to finding one that could be used to control mouse numbers. Following the results of this parasite survey, the scientists are now concentrating on a nematode, or roundworm, called *Capillaria hepatica*, which lives as a parasite in the livers of rodents.

Central to any parasite's life is the transfer from one host to another. This may involve intermediate hosts, the production of large numbers of infective stages, or altering the behaviour of a host (a cold virus makes you sneeze, thus spreading virus particles out to infect other hosts). *C. hepatica* seems rather defective in this regard, making it a most unusual parasite.

To become infected, a mouse has to take in the worm's eggs when they are at the correct stage. Such an infective egg is said to be embryonated, which simply means that a miniature worm has developed within it and is ready to come out and grow to maturity. Once the egg is inside a mouse, this in fact happens and the adult males and females congregate in the liver, which often enlarges. Following mating there, the female deposits eggs in the liver — and for the time being the life cycle stops. None of the usual business of eggs passing out with the host faeces — so common with many parasites — occurs.

The eggs do not develop any further until they are released from the liver. For this to happen, the mouse must die, either from liver damage caused by the infection if the parasites were present in large enough numbers, or from another cause. Now the parasite has its big chance; although the host's immune response might already have killed the adult worms, viable eggs remain in the liver.

At this stage, the continuance of the parasite depends on some of the less salubrious habits of mice, to which the nematode is evidently adapted. The 'cute' little rodents are fairly partial to eating one another's dead bodies — a habit called necrophagy. This releases the eggs from the liver, and the story would be very simple if the ingested eggs then infected the greedy cannibal. Instead, they pass out with the faeces, still not in an infective state.

Mice have another feature of which the canny parasite takes advantage — the fact that they live in burrows. The egg-containing faeces of the necrophagous mouse remain in the burrow and — under the correct conditions of temperature, oxygen

levels, and humidity — the cells within each egg develop into an embryo. Now the eggs are ready to infect; and they can remain so, Dr Singleton and Dr Spratt have discovered, for 6–8 weeks at 30°C and for longer at lower temperatures. To get into another host the eggs must be eaten. Living in a burrow with droppings aplenty, the mice soon find their fur becomes soiled and, as they lick themselves clean, finally take in viable eggs; and the cycle is complete.

Once the scientists had elucidated its life cycle, they had to find out whether the nematode would actually be effective as a biological control agent. Could it reduce the number of mice?

From the start, one thing was clear: the survey of literally thousands of mice from different areas, in which Dr Singleton and Dr Spratt had identified the species of naturally occurring parasites and assessed their relative abundance, showed that *Capillaria hepatica* existed almost exclusively in those mouse populations near the coast and in the cities. It was not prevalent in the mice of the cereal-growing regions where the plagues most often occur.

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Since it already existed in coastal populations yet had not wiped out the mice there, it would certainly not be effective in completely removing mice from a large area. As an effective parasite, it was not stupid enough to kill off all of its hosts. *C. hepatica* would never work in the same way as myxomatosis, with its high kill rate.

However, the worm is unusual compared with other nematode parasites of mammals because death of individual hosts is not a calamity, but rather a necessity for the continuance of its life cycle: it depends on host death for its eggs to be released. And those eggs will have a good future provided other mice are around. So killing some hosts is advantageous to this parasite, but it is not in its interest to wipe out a population.

The scientists suspected that, at the infection levels that could be realistically achieved in the field, the outright kill rates would be relatively low.

But the important point is that the parasite would probably reduce the litter

size and the survival of the young — that is, the net 'productivity' of the mice.

In a laboratory experiment they tested this by infecting 38 female mice with 50 embryonated eggs of *C. hepatica*. They then left the females to mate with uninfected males, and compared the litter sizes and survival rate of the young with those in an uninfected control group of 36 females.

The results in fact showed no significant differences in litter size, although slightly more young died at or immediately after birth in the infected group. But among this group mice did die during the course of the experiment — some even before producing a litter, others between first and second litters. Also, the time interval between litters was greater — being 38 days between first and second litters for them, as against 31 days for the uninfected group. At weaning, young from infected females weighed significantly less.

By the end of the 90-day reproductive period, the differences between the two groups had become very obvious. When everything was added up, the control group produced an average of 15 live young per female, compared with only 9.3 in the infected group. Undoubtedly then, *Capillaria hepatica* can reduce the productivity of mice.

Now this is useful, because the work of Dr Redhead suggests that unusually high average litter sizes about 15 months beforehand are important in determining the severity and timing of the plague. Reducing the productivity may allow us to keep mouse numbers below a certain critical threshold — a level at which a plague becomes inevitable. Although we don't yet know enough about the mortality of adults, it may well be that *C. hepatica* infection in the field could lead to a decrease in the number of females present at the start of the breeding season, as well as causing less productivity in those that do breed.

### **Further checks**

Before rushing into biological control, we need to know a lot more about any proposed agent. In the case of *C. hepatica*, will it affect other mammals — the non-target species? Dr Spratt and Dr Singleton have shown that the parasite can infect some native mammals in the laboratory. However, in the wild it has only been found in native rats in the rainforests of northern Queensland, and not in any marsupials.

It primarily occurs in the introduced house mouse, Norway rat, and black rat, in urban areas. The reason is that the worm



depends for its transmission on a host that may eat dead animals and on a life style using shared burrows. (The eggs cannot survive above ground as they are quickly killed by the ultraviolet component of sunlight, and by drying out.) And for the parasite to persist, the host animals must not be spread out at low densities; furthermore, the host must have a short life span and a rapid population turnover.

It seems very unlikely, therefore, that *C. hepatica* could establish itself permanently in populations of native mammals occurring in the cereal-growing areas. However, the scientists will certainly need to carry out further research in the field before putting the worm to work.

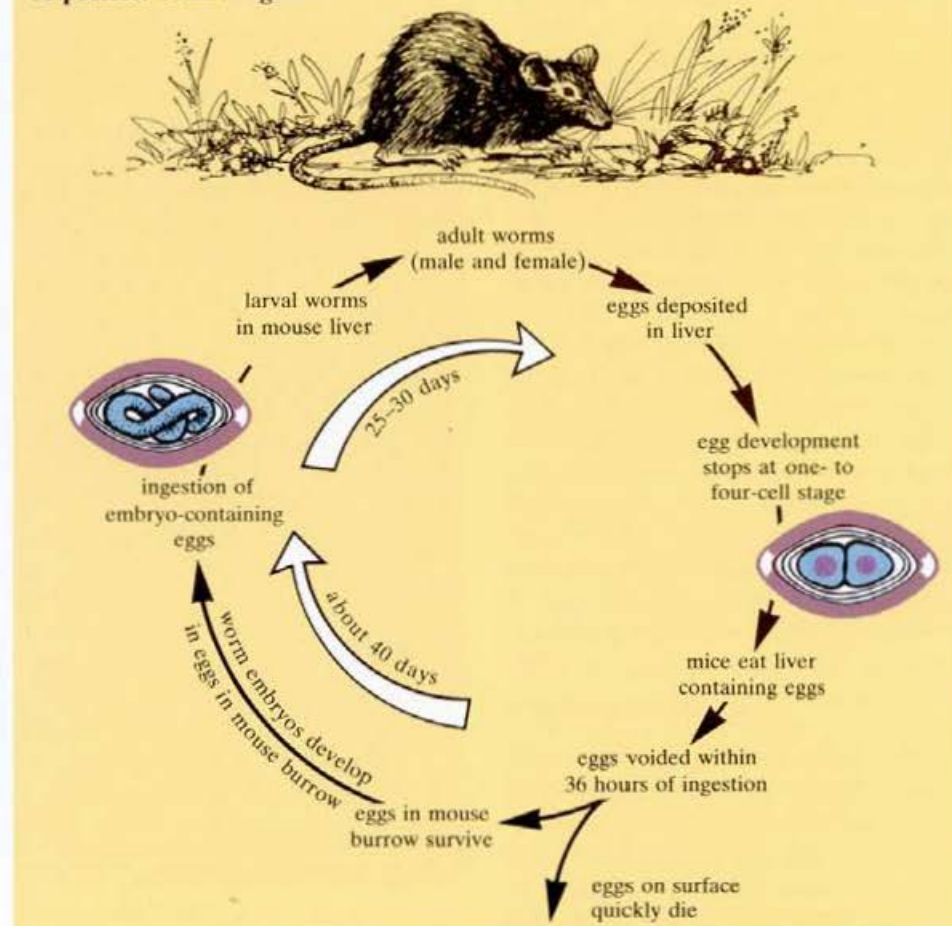
### Tactical response

What the researchers envisage is that the parasite will be used only at certain crucial times, when monitoring indicates that a plague may be in the offing. The effect, the researchers hope, will be to keep the population below the threshold at which a rapid increase becomes inevitable. But the parasite is not expected to remain in the mouse population. It seems that the severe swings in mouse numbers that occur in the Victorian cereal-growing regions make it impossible for the parasite to survive. 'Bottlenecks' arise when too few mice remain for the parasite to make a good living. So the worm would have to be re-introduced every few years, when conditions suggest that another plague may be imminent — a tactical response to any untoward developments in the mouse world.

Although this may sound awkward, it would certainly represent a better form of control than that practised at present. Currently we deal with a plague once it has already eventuated, using toxic chemicals as rodenticides. These poisons, of course,



### A possible control agent



The life cycle of the nematode parasite *Capillaria hepatica*.

are also lethal to other mammals and birds as well as posing risks to the humans who apply them. Poisoning isn't particularly effective either, as those mice that are killed can quickly be replaced by others living nearby.

A grant from the Rural Credits Development Fund (Reserve Bank) has allowed the team to employ another scientist, Mr Stephen Barker, to carry out further field work. In conjunction with Dr Singleton and Dr Spratt, he will study the effect of *Capillaria hepatica* on the productivity of mice in their natural environment, and also see whether populations of mice differ in their susceptibility to the nematode.

As a start, Dr Singleton and scientists from West Germany's Max Planck Institut are examining the genetic variability of Australian mice, with a view to assessing their ability to develop resistance to *C. hepatica*.

So we can now say with some optimism that combining the ability to predict population increases with a parasite to use in response may soon allow us to manage the mouse problem — although those remark-

ably successful ubiquitous rodents, established on earth long before us, will certainly always remain with us.

Roger Beckmann

### More about the topic

Experimental embryonation and survival of eggs of *Capillaria hepatica* (Nematoda) under mouse burrow conditions in cereal-growing soils. D.M. Spratt and G.R. Singleton. *Australian Journal of Zoology*, 1987, **35** (in press).

Studies on the life cycle, infectivity and clinical effects of *Capillaria hepatica* (Bancroft) (Nematoda) in mice, *Mus musculus*. D.M. Spratt and G.R. Singleton. *Australian Journal of Zoology*, 1986, **34**, 663-75.

The effects of *Capillaria hepatica* (Nematoda) on natality and survival to weaning in BALB/c mice. G.R. Singleton and D.M. Spratt. *Australian Journal of Zoology*, 1986, **34**, 677-81.

A field experiment to examine the effects of food quality and population density on reproduction of wild house mice. M. Bomford and T. Redhead. *Oikos*, 1987, **48**, 304-11.

Prevention of plagues of house mice in rural Australia. T.D. Redhead. In 'Rodent Pest Management', ed. I. Prakash. (CRC Press: Florida 1987, in press.)

The grain belt of south-eastern Australia.