Box 1.

Summary of the main features of the current models for mouse plagues. The events leading to a plague are listed in approximate chronological order for each category of model. However, not all models include each of the steps. The locations of the regional and district models are shown in Figure 1.

(a) Regional models to predict the occurrence of plagues for:	
• *New South Wales and Victoria (NSW &V)* (Saunders and Giles 1977)	
• *South Australia (SA)* (Mutze et al. 1990)	
Sequence of events:	
1.	A low-yield crop (≈ drought) two years prior to the plague. (*SA*)
2.	A severe drought (leading to a reduced regulatory effect of disease and/or predation) followed by one or two years with good winter–spring rain. (*NSW &V*)
3.	A special sequence of rainfall events in the 12 months preceding the plague. (*SA*)
4.	High-yield crop in the summer prior to a plague in autumn. (*SA, NSW &V*)
(b) District models used to predict of the abundance of mice for:	
• *Turretfield (T)* (Newsome 1969a)	
• *Murrumbidgee Irrigation Area (MIA)* (Redhead 1982)	
• *Victorian Mallee (VM)* (Singleton 1989)	
• *Darling Downs (DD)* (Cantrill 1992)	
• *Macquarie Valley (MqV)* (Twigg and Kay 1994)	
Sequence of events:	
1.	Good autumn rain two years prior to plague to extend the breeding season into winter. *(MIA)*
2.	Mice disperse from refuges to other favourable areas during the summer breeding season one year prior to the plague. *(MIA)*
3.	High autumn–winter rainfall in the year preceding the plague. *(VM)*
4.	High abundance of female mice in a wide range of habitats in the spring prior to the plague year. *(MIA)*
5.	Correct sequence of rainfall events to provide burrowing and nesting sites in cracking soils. *(T, MqV, DD)*
6.	Favourable climatic conditions over the summer (a high-yield summer crop) in the plague year. *(T, MIA, VM, MqV, DD)*
(c) Process models for the regulation of mouse populations:	
• *Predator-regulation (P-R)* (Sinclair et al. 1990)	
• *Regulation by disease (R-D)* (McCallum and Singleton 1989; Shellam 1994)	
Conditions that prevent a plague:	
1.	Aggregation of predators in habitats with high mouse numbers (e.g. irrigated crops) due to low abundance of mice in surrounding areas. *(P-R)*
2.	Density-dependent increase in the prevalence of lethal or sterilising pathogens. *(R-D)*
Conditions that allow a plague:	
1.	Widespread favourable climatic conditions resulting in dispersed populations of prey and predators. *(P-R)*
2.	A long delay in a density-dependent increase in the prevalence of pathogens or the prevalence is independent of density. *(R-D)*

The probability of a plague in the (following) autumn was found to depend on (i) the difference between grain production in the current year and that two years earlier, (ii) the difference between the November and the October rainfall for the current year, and (iii) the autumn rainfall in the current year. The model accounts for 41% of the variation in plague occurrence. A plausible mechanism by which each of these factors could affect mouse populations has been suggested. The requirement in the *Turretfield model* (see below) for mid-summer rains was not supported even when the data set was restricted to sites with predominantly red-brown earths. However, there was a significant contribution from autumn rain in the year immediately preceding a plague which matches, to some extent, the revised model for the *Victorian Mallee* (below). In contrast to the *MIA model* (below), autumn rains from earlier years appeared to have no effect.

If projected estimates of crop yield are available, the *South Australia regional model* can be used towards the end of winter to predict the likelihood of a mouse plague in the following autumn. This may provide adequate time for preventative control measures to be implemented if, for example, farmers have access to registered in-crop rodenticides. The data requirements are the autumn (March, April and May) rainfall, the average November–October rainfall, the harvest yield from two years before and the preliminary estimate for the current year. These data provide the three variables for the model. The preliminary harvest estimates are subjective judgements made by district agronomists based on (i) autumn rainfall which determines sowing time, (ii) total (over-winter) rainfall during the growing season [Cornish et al. (1980), as reported in Mutze et al. (1990), found that total rainfall from April to October accounts for 80% of variation in crop yield], and (iii) the amount of rainfall in September and October to 'finish-off' crops. The model can be used to predict plague probabilities for each locality and a high probability is taken as an indicator that additional evidence, such as more detailed crop and rainfall records, should be examined.

The model's predictions were compared with the results of an intensive trapping program from 1980 to 1990 (Mutze 1991). Plagues were predicted for 1980, 1984 and 1985 but not in other years. The only plagues that occurred in South Australia during this period were in 1980 and 1984; the failure of mice to respond to generally favourable conditions in 1984–85 was due probably to a late break in a period of low rainfall during the winter of 1985.

District and small-scale models

Turretfield model

Newsome (1969a,b, 1970, 1971) conducted the first extensive Australian study of the dynamics of wild mouse populations at Turretfield in South Australia (Figure 1). Mice were permanent residents of small patches of favourable habitat in the landscape; reedbeds in the case of this study area. Mice colonised crops in early summer but could not over-winter there due to waterlogging of the clayey soils. According to the model, a plague of mice occurs as a direct result of an unusual sequence of events: (i) good winter rains to provide an adequate food supply through to the

following autumn and to keep the sub-soil moist over summer; (ii) a hot summer to crack the soil allowing mice access to nesting sites in the moist sub-soil; and (iii) mid-summer rain to allow mice to burrow and breed throughout summer. The data suggest it is possible for mice to increase to plague numbers in three to five months. This conclusion was supported by an experiment in which free-fed mice reached densities well in excess of those observed in plagues.

The model was later modified by Newsome and Corbett (1975) to take into account the effects of predation. In the revised version, predators can delay by one year the build-up of mouse populations generated by a pulse of favourable conditions. This is consistent with the *predation-regulation model* (below) proposed by Sinclair et al. (1990). However the suggestion by Newsome and Corbett that predators may be responsible for the failure of mouse plagues to persist under favourable conditions is contrary to the *predation-regulation model.*

Two features distinguish this model from some of the later models. Firstly, the relationship between the availability of breeding sites for mice and soil moisture over summer restricts the applicability of the results to areas of red-brown earths interspersed with patches of heavy cracking soils. Mutze et al. (1990) argued that the *Turretfield model* was relevant to only these fairly restricted parts of the South Australian wheatlands. Secondly, there is an apparent anomaly in the lead-time (the time from the first events triggering a plague to the plague itself): less than six months for the area characterised by Turretfield and up to two years elsewhere. Despite the ease for mice to burrow in sandy soils, plagues do not appear to develop immediately in areas with these soil types whenever good winter rains are followed by mid-summer rains.

The apparent ability of mice at Turretfield to reach plague densities within five months implies little prospect for long-term forecasting of outbreaks. However, the model demonstrated the role of reedbeds as the source of mice that colonise crops, and for these land systems, the strategy of selectively targeting minor refuge habitats may be effective if control was conducted routinely.

Murrumbidgee Irrigation Area (MIA) model

A 'triphasic' model of mouse plagues was developed by Redhead (1982) during an intensive four-year study in the MIA in New South Wales (Figure 1). The model is complex and includes both intrinsic (e.g. spacing behaviour) and extrinsic (e.g. food quality) factors that influence mouse population dynamics (Redhead et al. 1985; Redhead and Singleton 1988a). According to this model, the plague trigger (phase 1) is above average autumn rains two years prior to the outbreak, which extends the breeding season into winter in refuge habitats by providing high quality food. The effect of food quality was experimentally demonstrated by Bomford (1987a,b,c) and Bomford and Redhead (1987). There is high productivity of mice in the following summer (phase 2) and mice disperse from the refuges into other areas made favourable by the earlier autumn rains. At the start of the breeding season immediately prior to the outbreak (phase 3), there is an abnormally high abundance of females in a wide range

of habitat types ('induced-donor' habitats). Provided no unusual factors intervene to impede breeding, a plague will develop over summer.

Redhead (1982, 1987) used a numerical simulation model (SIMAD) to show that between-year differences in the mean litter size and the observed size of the initial population could explain the variation in the increase period for each of the three phases. However the data set for litter sizes is limited and the model does not allow for changes in litter size during a breeding season. The results of the model emphasise the importance of between-year variability in breeding performance in the MIA compared to the *Darling Downs model* (below) where the population is assumed to increase at the same rate each year.

In the *MIA model*, the plague trigger occurs in the autumn two years prior to a plague. In comparing this to the *NSW and Victoria regional model*, Redhead (1982) observed that there appeared to be a relationship between the residual mass [the accumulated difference between the long-term average and the actual monthly rainfall (Foley 1957)] increasing through winter and spring and a plague two years later. Neither this relationship nor the prior-drought hypothesis of Saunders and Giles (1977) was tested statistically, but if the relationship suggested by Redhead is true, then the importance of a prior drought is questionable. The residual mass will increase with above average rains breaking a drought (invariably in winter) or if there are simply above average winter–spring rains.

The extended build-up period (phases 1 and 2) prior to the plague year is in contrast to other models where mice can reach plague densities in one season. The difference may lie in the need for mice to colonise 'induced-donor' habitats in phase 2, which depends ultimately on the mix of crops and year-round refuge habitats in the landscape. In both the *predation-regulation model* and the *MIA model*, mice are held in a low-density state by spatial, density-dependent processes. However in the *predation-regulation model*, phases 1 and 2 were simply classed as the predator-regulated state and not necessarily as essential precursors to phase 3, the outbreak state.

In 1983–84, Boonstra and Redhead (1994) tested the hypotheses relating to phases 1 and 2 in the triphasic model. Specifically, these were that a tight social structure during the extended breeding season in phase 1 should result in high dispersal rates for mice, presumably into the 'induced-donor' habitats outside refuge areas, and that there should be a disproportionate abundance of female mice at the end of phase 2. The weather conditions prior to this study included a severe drought in 1982 and above-average rainfall in the autumn of 1983, both of which have been considered important precursors to a plague (Saunders and Giles 1977; Redhead 1982). The data on fecundity rates, dispersal rates, sex ratios and testosterone levels showed that (i) it was unlikely that social organisation had modified dispersal, (ii) the expected changes in breeding performance in phase 2 did not occur, and (iii) there was no bias towards females in the sex ratio at the end of phase 2. The conclusions were that, for irrigated rice crops, plagues could develop much faster than previously envisaged by Redhead (1982) and the <12-month time frame for the increase in mouse abundance in 1984 was similar to that suggested in other models.

However, the reasons for the lack of a subsequent plague are not clear and several hypotheses were proposed by Boonstra and Redhead (1994). The most likely explanations appear to be associated with the drought that affected the surrounding dryland farms in 1984. Non-irrigated areas may have acted as a sink for dispersing mice. Alternatively, mouse populations may have been regulated by predation, i.e. by highly mobile raptors moving away from dryer areas to concentrate around irrigated crops, as in the *predation-regulation model* proposed by Sinclair et al. (1990). During an earlier mouse plague in this area in 1979–80, Davey and Fullagar (1986) noted a large increase in the abundance of three species of mouse-eating raptors, the Australian kestrel (*Falco cenchroides*), the brown falcon (*Falco berigora*) and the black-shouldered kite (*Elanus notatus*).

The relationship between the development of mouse plagues and the timing of management actions has been examined in detail by Redhead and Singleton (1988b) and Singleton and Redhead (1989) and lead to their PICA (Predict, Inform, Control, Assess) strategy. Their analysis demonstrated that the current lack of registered in-crop rodenticides has forced a series of time delays into the ability of farmers to respond to plague warnings. The delays result from the processes necessary to obtain permission to use rodenticides. However, if suitable control techniques can be developed and be ready on demand, predictions with a time frame of less than 12 months may be sufficient for farmers to control mice effectively.

Victorian Mallee model

A long-term demographic study of mice beginning in 1983 in northwest Victoria (Figure 1) has restructured the *MIA model* for the Mallee wheatlands (Singleton 1989). In the Victorian Mallee, the development of a mouse plague may occur in the breeding season immediately following high autumn or winter rainfall, potentially more rapidly after a 'trigger' than in the MIA, but because of the different soil characteristics (Singleton 1989; Singleton and Redhead 1989) plague development may be more sensitive to the sequence of rainfall events. For example, a plague in 1988 was less severe than expected despite an increase in mouse numbers following initial favourable conditions. As in the *Turretfield* and *MIA models*, landscape heterogeneity and the role of refuge habitats appears to be important in the population dynamics of mice in the Victorian Mallee. For example, Singleton (1989) reported significant temporal differences between habitats in the increase phase of the 1984 plague.

The time taken for mice to reach plague proportions in the Victorian Mallee in 1984 was similar to that observed by Newsome (1969a) at Turretfield. Although the 1984 plague occurred 15 months after a drought, in accordance with the *NSW and Victoria regional model*, no data were collected to validate the suggestion of Saunders and Giles (1977) that predator regulation was the process responsible for this time lag. However, the prevalence of macroparasites was independent of mouse densities over a 2.5 year study prior to, and during, the plague (Singleton 1987). The data suggest that the macroparasites recorded by Singleton were unlikely to regulate mouse numbers.

Based on the *Victorian Mallee model*, a series of trapping surveys following high autumn or winter rainfall could be used to verify a prediction of conditions conducive for a mouse plague. A suitable protocol would be as follows.

- (i) Trap in the second week in September to determine (a) the start of breeding, (b) litter size [litters are larger in the build up to plagues (Singleton and Redhead 1990a)], and (c) the size of the breeding population. The data should be collected from 'donor/refuge' (e.g. fencelines) and crop habitats. If the data from September show no breeding and low populations, then there is a low probability of a plague and no further monitoring is required until the following September. Conversely, an early start to breeding, large litters and a large breeding population are conditions favouring a plague and further monitoring is required (step ii).
- (ii) Trap in November to determine the percentage of females breeding and the litter size. High numbers at this time indicate a high chance of a plague next autumn; moderate numbers may be a precursor to a plague 18 months away.
- (iii) If data from November confirm that a plague is expected in the next six months, a third trapping session may be required to detect the rapid build-up in mouse numbers over the January – February – March period.
- (iv) Moderate numbers in November (and no plague within six months) indicate that trapping the following September is important.

The application of the PICA management strategy (Singleton and Redhead 1989) is similar in the Mallee and the MIA, except that plague development may be more rapid in the former. The timing of control operations may be more constrained in the Mallee than the MIA and this may be a problem with future applications of a biocontrol agent such as *Capillaria hepatica* (see below).

Darling Downs model

The model is based on demographic data collected over 12 years from a standard set of trapping sites in the central Darling Downs in Queensland (Figure 1) (Cantrill 1992). Pooled data from all major habitat types (crops, verges, fallow etc.) show a regular annual cycle in mouse abundance with no clear separation into plague and non-plague years. There are minimal between-year differences in (i) the rate of increase of the mouse population over summer and autumn, (ii) the onset of the main breeding season, and (iii) the months of maximum population density. The most important factor that can interrupt the cycle of abundance and affect the peak density of mice in May and June is the duration of the low abundance phase (defined as less than 1% trapping success) the previous spring. In one year, a sequence of flooding rains, probably increasing nestling mortality, delayed the annual build-up in mouse numbers. A second source of between-year variation is a density-dependent decline in the over-wintering population.

Trapping data are required from fixed trapping sites for two, preferably three, periods of the year to generate predictions with the *Darling Downs model*. These are (i) in

May or June—to test the prediction from the previous year and to predict the size of the mouse population at the onset of the main breeding season in spring, (ii) in September—to determine the size of the initial spring breeding population, and (iii) in the period from October to December—to determine the starting time for the increase phase in mouse abundance. Rainfall data from spring and early summer can be used if data from the third trapping period are not available. The model is based on an established trapping protocol and its applicability to data collected elsewhere, or for a different mix of land uses, is unknown.

The model can provide forecasts 12, 8 and 5 months in advance of the peak mouse abundance in May. This translates into warning of high mouse numbers 9, 5 and 2 months in advance of the time when damage due to mice is usually reported. The model also suggests that any farming practices, such as minimum tillage, that enhance over-winter survival of mice may lead to a relatively large population at the onset of breeding the following spring. This would generate a forecast for high mouse numbers the following year with the result that farmers could be locked into annual mouse control.

Macquarie Valley model

In contrast to the 12–24 month warning-time recommended by Redhead and Singleton (1988a), Twigg and Kay (1994) suggested that decisions by farmers for managing pests would be based on relatively short-term predictions of the order of three months. They developed a series of models, based on linear multiple regression analysis, for irrigated summer crops such as soybeans, sorghum, cotton and maize, in the Macquarie valley of New South Wales (Figure 1). Soil types in the irrigation area are self-mulching clays to deep red clays that crack on drying to produce refuge sites for mice similar to those in patches of black cracking soils at the Turretfield site studied by Newsome (1969a).

Data were collected over three years that included two floods. The best models explained 68% of the variance in the index of mouse abundance and 53% of the variance in the abundance with seasonal trends removed. Significant coefficients were found for the mean daily range in temperature for each month, the mean minimum temperature, the mean maximum temperature and the total monthly rainfall in the one or two months prior to trapping. Twigg and Kay (1994) also estimated the size of the seed bank, soil moisture and soil hardness, the number of cracks in the soil and their size distribution, and indices of the structure and biomass of the vegetation. They found that the component of the seed bank from barnyard grass (*Echinochloa crus-galli*), rye grass (*Lolium rigidum*) and wild oats (*Avena fatua*) explained 70% of the variance in mouse abundance with small additional improvements contributed by indices of soil cracking and vegetation. However, the regression based on total seed bank was less satisfactory. The vegetation data provided a link between climatic variables and mouse abundance but a direct effect of rainfall and temperature on recruitment was not supported by the models. In the model for recruitment, the proportion of adult females lactating or pregnant depended on the seed bank and the distance to the closest summer crop.

The model for the abundance of mice in irrigated summer crops provides a useful, short-term predictive tool based on readily accessible climatic data. Twigg and Kay (1994) suggested that it is desirable to use routine surveys of mouse abundance to support their model's predictions, however a survey protocol suitable for use by farmers was not specified. The data from this study support the conclusions from elsewhere in Australia that the management of mice, or their food supply, in refuge habitats such as roadside verges and fencelines should help to reduce the damage caused by mice.

Simplified process models

Predation-regulation model

The model proposed by Sinclair et al. (1990) focuses on the third of the major extrinsic mechanisms — food supply, shelter, predation and disease — which may be responsible for regulating mouse populations. It is based on data collected over two years on a summer-irrigated cereal farm in the MIA (Figure 1). Although mice increased in abundance each year over the spring–summer period, years were divided into plague or non-plague categories. In non-plague years, raptors — primarily black-shouldered kites, Australian kestrels, brown falcons and brown goshawks (*Accipter fasciatus*) — and possibly mammalian predators (foxes — *Vulpes vulpes* and feral cats — *Felis catus*) could have been responsible for regulating mice at low densities despite the apparently favourable environmental conditions for mice on the farm. Also a second density-dependent factor, the effects of a pathogen thought to be *Actinobacillus moniliformis* was implicated although the data are extremely limited. In the one year when a plague occurred on the farm, mice were abundant over a wide region due to exceptionally favourable conditions over the preceding months, and the limited number of predators on the study area failed to regulate mice. Sinclair et al. (1990) suggested that the plague on the study farm was caused by a combination of wide dispersal of predators and enhanced breeding performance of mice which together allowed mice to escape predator-regulation.

The analysis of Sinclair et al. (1990) can be generalised to the following multi-state model, which is analogous to a model for rabbit plagues in semi-arid Australia (Pech et al. 1992, Pech et al. 1995, Pech and Hood 1998) and is relevant to regions where predation can be a major mortality factor for mice.

- (i) There is a plague trigger (a period of high rainfall) which provides a finite amount of food that ultimately is exhausted by mice. The time to depletion of the pulse of food is a function of the size of the trigger, i.e. the duration of the favourable conditions. A large pulse could extend over two years, a moderate pulse might last only one year, and a smaller one still should not result in an outbreak, merely a small seasonal increase in the abundance of mice. It should be possible to predict the occurrence of the pulses of food as accurately as the long-range weather forecasts.
- (ii) The trigger results in high reproduction by mice that can result in an escape from predator-regulation. If there are many predators in an area and no trigger or only a small trigger, then only a minor increase

in mice should occur. If there are few predators, then an outbreak occurs immediately following a trigger. The model predicts that if a trigger is not followed by an outbreak, then predators should have been present in large numbers.

- (iii) The trigger generates a finite amount of food in excess of that normally present. If, and only if, mice escape predator-regulation can they reach a high density state determined by the new food supply. The abundant food eventually disappears, or a drought arrives, which resets food to a low level and the system collapses back to a situation where predators can take over again.

The *predation-regulation model* is based on a very limited data set and is best viewed as an hypothesis for more extensive testing. Although the model has little to offer for predicting the likelihood of a plague, it does identify a key process that can be affected by management. No action should be taken which would deplete raptor populations, and mouse control techniques should be used to ensure that winter populations of mice stay low, i.e. within the range where regulation by predators is possible.

Pathogen-regulation model

Information on the prevalence and distribution of pathogens in wild mouse populations in south-eastern Australia is summarised in Redhead (1982), Singleton (1987), Singleton et al. (1991), Singleton et al. (1993) and Smith et al. (1993). There is some field-based evidence that endemic pathogens may be responsible for regulating mouse populations (Sinclair et al. 1990), however most effort has been directed toward a search for organisms which could be introduced into mouse populations either as conventional biological control agents (Singleton and Spratt 1990) or as vectors engineered to induce infertility in infected mice (Singleton and Redhead 1990b; Shellam 1994). Although the conditions for a candidate biocontrol agent are exacting (Spratt 1990), pathogens have considerable appeal because of their likely host-specificity, the absence of toxic residues, potential economic advantages and their possible compatibility with current management practices and land use. In addition, Australia has the advantage that local populations of *M. domesticus* lack some of the mouse-specific parasites found overseas (Singleton and Redhead 1990a).

Following a major review, Barker and Singleton (1987) concluded that the liver nematode *Capillaria hepatica* showed promise as a biological control agent for mice. Laboratory studies had established the conditions for successful transmission between mice (Spratt and Singleton 1986, 1987) and had shown that infection depressed the productivity of female mice to a level that may be sufficient to prevent plagues (Singleton and Spratt 1986). As well, surveys demonstrated that mice in plague-prone areas had no prior exposure to the parasite despite it being recorded at a range of sites in coastal south-eastern Australia (Singleton et al. 1991). McCallum and Singleton (1989) and Singleton and McCallum (1990) modelled the likely impact of *C. hepatica* on mouse population dynamics and concluded that it had the potential to significantly reduce the density of mice below infection-free levels. However, it was not clear whether time delays in the host–

pathogen cycle might negate its use for tactical release (time- and area-limited and with 'rapid' effect) (McCallum 1993). The effectiveness of *C. hepatica* as a biocontrol agent was tested in pen experiments (Barker et al. 1991) and two large-scale, replicated field experiments — the first in the Darling Downs in 1992–93 and the second in the Victorian Mallee in 1993–94 (Figure 1). The field sites were chosen to be representative of the farming systems in each region.

Three releases were conducted in the Darling Downs: (i) low density, non-breeding mouse populations in winter, (ii) low density, breeding mouse populations in summer, and (iii) high density, non-breeding mouse populations in winter (Singleton et al. 1995). Embryonated and unembryonated eggs were released on four sites using a combination of baits and direct infection of mice (oral). The treated sites were interspersed with three control sites. The parasite appeared to persist for <5 months after the first release in winter of 1992, for at least 2–4 months with low prevalence after the release in the summer of 1993, and persistence was uncertain after the winter release in 1993 because the abundance of mice declined to levels where it was not possible to trap adequate samples. Following all three releases, little or no significant difference between treated and untreated sites was detected in the age structure of mouse populations or their abundance, breeding performance or survival. An intensive trapping protocol failed to detect any transfer of the parasite beyond the experimentally infected areas. The minimal impact of *C. hepatica* was attributed to inadequate dosage rates and adverse climatic conditions leading to periods of low densities of mice, poor survival rates for mice, little fidelity in the use of burrows and loss of access by mice to eggs deposited in the cracking clay soils.

Four treated and three untreated cereal/sheep farms were used for the experiment in the Victorian Mallee (Singleton and Chambers 1996). The parasite was released in September 1993, two months prior to a period of sustained increase in the abundance of mice. About 60,000 mice, or 10% of the populations on the treated sites, were dosed with embryonated and unembryonated eggs. The results from the increase phase in mouse density (November 1993 to mid-1994) showed that *C. hepatica* persisted for approximately eight months but with little or no effect on the 28-day survival rate and the fecundity or the abundance of mice, but the treatment appeared to temporarily delay the increase in mouse density during the early part of the breeding season. The causes for the poor performance of *C. hepatica* as a biocontrol agent are uncertain but probably include low survival of eggs during hot, dry weather and delays in the life cycle of the parasite that prevent it from regulating rapidly increasing mouse populations.

A *pathogen-regulation model* based on *C. hepatica* is still in the early stages of development despite intensive efforts to collect epidemiological data in laboratory, pen and field experiments. To be implemented, the timing and conditions for release of the parasite will need to be specified, along with the information required to determine those conditions. This will determine the extent to which other control techniques might be needed to form an integrated control strategy for mice. In

addition, the model should specify when and what information is required to monitor the performance of the technique.

Although the early promise of biocontrol using *C. hepatica* has not been realised, a recent feasibility study suggests that virally-vectored immunocontraception may provide a viable alternative (Chambers et al. 1997). The fertility of laboratory mice has been impaired successfully using ectromelia virus as a model (Jackson et al. 1998) for the development of a genetically engineered immunocontraceptive strain of mouse cytomegalovirus (Shellam 1994).

Comparison of current models

There is a wealth of data, expert knowledge and experience, published and unpublished, relating to the formation of mouse plagues in Australia. The resulting models, summarised in Box 1, contain elements of the key extrinsic factors likely to affect mice: food availability, access to shelter and nest sites, predation, disease and landscape heterogeneity. In most cases food availability is estimated from climatic data although the *South Australian regional model* can include agronomic estimates of crop yields if they are available. No model includes a protocol for collecting direct estimates of the amount of food accessible to mice. The models for areas with heavy soils, *Turretfield, Darling Downs* and *Macquarie Valley*, include the effect of rainfall, or lack of rain at critical times, on the abundance of nesting and shelter sites in sub-surface cracks. In these and in other areas with lighter soils, secure access to refuge sites can depend on the frequency of disturbance caused by the prevailing system of crop rotations and periods of fallow. For this reason the recent trend to conservation farming, with emphasis on stubble retention and minimum tillage, may exacerbate problems with mice (Brown et al. 1998; Singleton and Brown 1998).

Predation and disease, two factors that are likely to be density-dependent, have been measured directly or their influence inferred as a possible explanation of mouse population dynamics. However the *predation-regulation model* (Sinclair et al. 1990) is the only example where data on predation have been analysed within the framework of predator–prey theory. Supporting evidence from Davey and Fullagar (1986), Kay et al. (1994) and Twigg and Kay (1994) reinforce the need for field experiments to determine the circumstances under which predation can regulate mouse populations. For example, Sinclair et al. (1990) hypothesised that predator-regulation may be effective only when predation pressure is concentrated on localised patches of the landscape.

Although Smith et al. (1993) and Singleton et al. (1993) identified many endemic diseases of mice in south-eastern Australia there is little evidence that any of these play a significant role in regulating mouse populations, with the possible exception of a mouse parvovirus. The effects of a disease, attributed to *Actinobacillus* by Sinclair et al. (1990), was similarly equivocal. The pathogenic agent was probably *Streptobacillus moniliformis* which has been recorded at low prevalence in fluctuating mouse populations in the Darling Downs (Taylor et al. 1994). With current technology there appears to be little scope for large-scale manipulative experiments to test for any regulatory effects of an endemic disease. The

alternative strategy of using translocated pathogenic agents, or engineered sterilising agents, offers the prospect of well-designed experimental trials.

The influence of landscape heterogeneity is inherent in all the published models and this factor is thought to interact with most, if not all, potential regulatory processes. Different habitat types provide temporal asynchrony in food supply, refugia during drought or when there is disturbance from farming activities, and may result in either concentrated or dispersed prey populations for raptors and terrestrial predators. At this stage there is very limited information on dispersal and other movements between habitats by mice (see for example, Newsome et al. 1982; Boonstra and Redhead 1994; Krebs et al. 1995b) on which to base spatially-explicit models for mouse plagues.

Krebs et al. (1995a) reviewed the case for the intrinsic regulation of mouse populations. They urged an analogy with models for the cyclic changes in populations of some rodent species in the Northern Hemisphere and proposed two variants of the Chitty hypothesis, with low, increasing, high and declining populations of mice characterised by differences in the ratio of aggressive and docile phenotypes. There are at least two problems with this view, one practical and the other philosophical. Figure 2b illustrates the difficulty of obtaining sample sizes sufficient to extract the simplest demographic parameters — abundance and rate of increase — during periods of low mouse numbers. Even if the two phenotypes, or their effects, can be distinguished in the laboratory experiments suggested by Krebs et al. (1995a), the resources required to measure aspects of the social system of low-density mouse populations in the field are unlikely to be realised. In the two hypotheses proposed by Krebs et al. (1995a), social organisation acts as a filter on the response of mice to extrinsic factors. All the suggested social differences appear plausible (docile versus aggressive behaviour, weak versus strong territorial behaviour, and open versus closed social systems) but the causal factors in population dynamics may be still extrinsic. It is probably more important to begin by testing rigorously the most parsimonious hypotheses: Boonstra and Redhead (1994) were unable to find evidence to support aspects of intrinsic regulation in the *MIA model* (Redhead 1982), and some of the extrinsic mechanisms underlying the current models appear amenable to direct experimental manipulation.

Practical application of current models

At present three models (the *South Australia regional model* and the models for the *Darling Downs* and *Macquarie Valley*) and possibly a fourth (the *NSW and Victoria regional model*) could be used unambiguously by people other than those who produced them. Most models imply that reliable forecasts can be provided only for the medium (less than 12 months) or short term (less than 6 months). This imposes some constraints on the timing of effective control options, especially those relying on biocontrol agents. All models have been developed using location-specific data and, to date, none have directly addressed the extent of the geographic range of their predictions. Some models — for example, the two regional models — should apply across state boundaries to districts

with similar soils, climate and agricultural practices. Other models are limited by attributes such as soil type or cropping regime. With the exception of the *Darling Downs model*, none of the existing models relate the density of mice, or the effect of controls, to the extent of damage caused by mice. This is an essential requirement for future models of mouse plagues.

A prerequisite for the application of a model is that the data and the methods used to generate a prediction are clearly specified. For most models the protocols for data collection have not been specified in sufficient detail. In future such protocols will need to be designed and tested for areas other than the localities where the models were developed.

One approach to improving the value of existing models is to include their predictive capability with other expert knowledge in 'decision-support systems' (Norton 1988). An example is the computer-based expert system that can provide short-, medium- and long-term predictions based on the *Darling Downs model* (Cantrill 1992). A decision support system, MOUSER, is under development for the Victorian Mallee (Brown et al. 1998). This software provides advice on how to achieve effective mouse control by modifying farming activities. The aim is to include interactive models for planning mouse-control campaigns in future editions of MOUSER. However, all the existing regional and district models focus on predicting the occurrence of plagues or the abundance of mice rather than their rates of increase. This means that none of the models are capable of assessing the impacts of control programs in an interactive way.

Modified Model for Mouse Plagues in the Victorian Mallee

Background and data

The aim of the following analysis is to construct a quantitative model, based on the conceptual model of Singleton (1989), to explain the observed rates of increase of mice over the last two decades in the Victorian Mallee. The ultimate purpose is to use quantitative models to predict when mouse plagues will occur and to assess the effectiveness of a range of control techniques, including fertility control, for managing mice in the Mallee region.

Mice were live-trapped at intervals of approximately six weeks from 1983 to 1997 in several habitats either on the Mallee Research Station or on nearby farms in northwest Victoria (35.08°S, 142.02°E). The region has a Mediterranean-type climate (Figure 2a) and is used for cropping and livestock production. Details of the trapping protocol are given in Singleton (1989). Although most trapping sessions were conducted over three nights and all mice were tagged, the number of recaptures was generally <15%. As well, the trapping grids were moved periodically to cater for the rotation of crops and pasture so that capture–mark–recapture techniques did not provide the best estimates of abundance. Instead, the number of captures, adjusted to take into account trap saturation, was used as an index of abundance. This required transforming the proportion, p, of traps catching mice per night (the frequency of captures) to the number of animals that would have been caught per trap if the traps were capable of multiple captures (an index of the density of mice). Then the adjusted

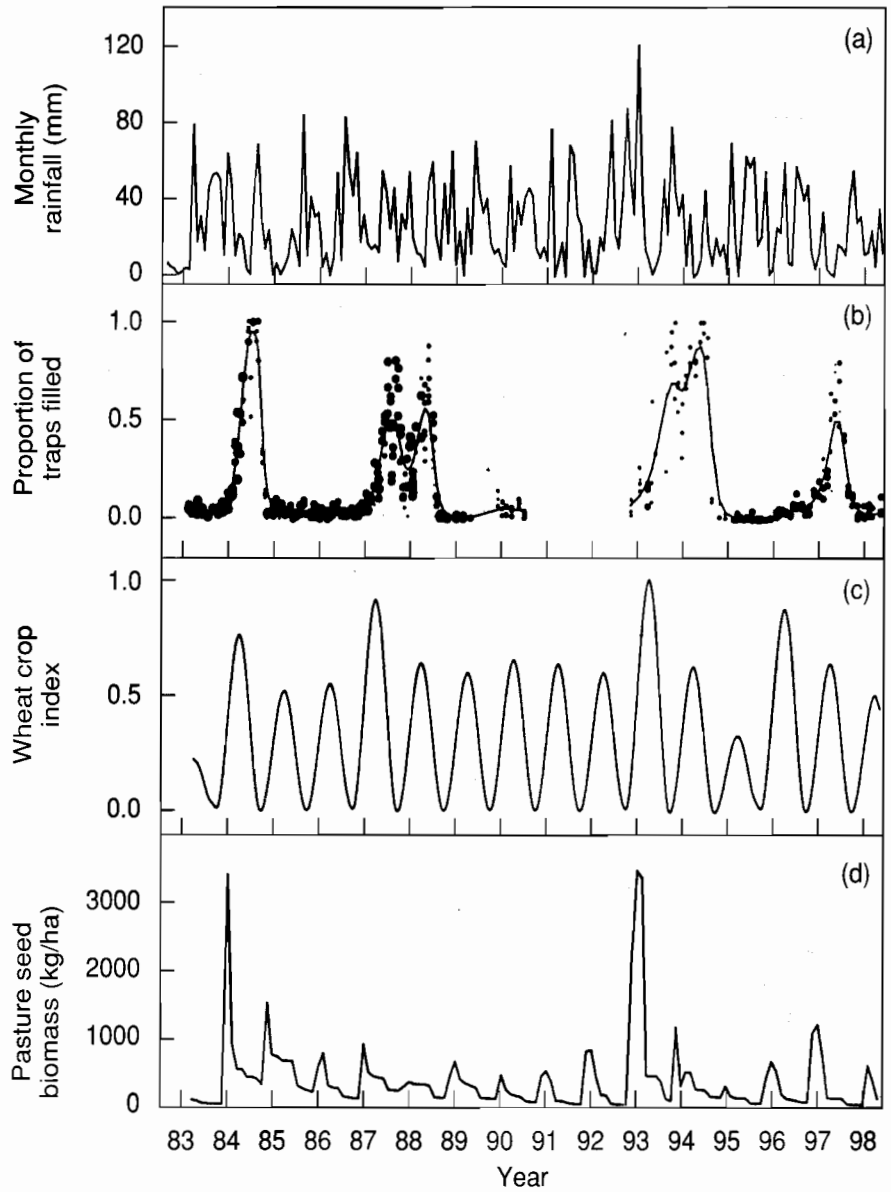

Figure 2.

(a) Monthly rainfall (mm) at the Mallee Research Station (MRS), Victoria. (b) Smoothed abundance of mice (—), indexed by the proportion of traps filled at the MRS and nearby farms. Data for 1983–85 are from Singleton (1989), for 1993–94 from Singleton and Chambers (1996) and for 1993–97 from Singleton and Brown (1998). The data are shown for each night of trapping with the size of the symbols (•) scaled according to the trapping effort (minimum = 10, maximum = 355, median = 242 trap-nights). (c) Modelled food from wheat crops (equation 1), with the mid-summer amplitude proportional to the total rainfall for the winter/spring period. (d) The estimated total seed biomass (kg/ha) from annual grasses and medic using the model GrassGro (Moore et al. 1997). Parameter settings for the GrassGro model are shown in Table 1.

density, N, is $-\ln(1-p)$ (Caughley 1977). We would expect this density to be affected proportionally by various factors and therefore the regression model would be additive on $\ln(N) = \ln[-\ln(1-p)]$.

The data for the proportion, p, are somewhat noisy (Figure 2b) and small fluctuations, particularly during periods of very low-density, are unlikely to be important in the overall population dynamics. Therefore the data were smoothed in a generalised linear modelling framework by fitting a spline in time as an explanatory variable, with the complementary log–log link function. The model was fitted in S-PLUS (1997) using a smoothing spline with 20, 30, 50 and 70 degrees of freedom (d.f.). The residual deviances (and the residual d.f.) obtained were 9067 (515), 5921 (505), 4123 (485), and 3276 (465), respectively. There was a substantial reduction in the residual deviance for a model with 30 d.f. compared to 20 d.f. which was reflected in the better tracking of the mouse plague peaks in the data. Fitting splines with more than 30 d.f. did not lead to a marked improvement in the fit to the main peaks and started to generate spurious peaks in the periods between major plagues. The inclusion of seasonal (sine and cosine) terms did not improve the model which is not surprising since mouse plagues do not occur on a regular annual cycle. The slope of the trajectory for $\ln(N)$ is the instantaneous rate of increase of the mouse population. Using S-PLUS, the estimated first derivatives were obtained from the fitted model at 40.55 day intervals ($1/9^{th}$ of a year). This approximates the mean interval between trapping sessions for mice.

During 1984 and 1985, the diet of mice was measured at a cereal farm not far (approximately 25 km) from the Mallee Research Station (Tann et al. 1991). The findings of this study showed that the main dietary components were monocotyledon seed (primarily wheat — *Triticum aestivum* and some grasses such as brome — *Bromus* spp., barley — *Hordeum leporinum*, wild oats — *Avena fatua* and ryegrass — *Lolium rigidum*) and dicotyledon seed such as medic — *Medicago* spp. and *Chenopodaceae* species. The proportion of the major component, monocotyledon seed, showed strong seasonal variation, rising from a minimum around July in mid-winter to a peak at harvest six months later. Post-harvest, the consumption of monocotyledon seed declined to a low point in the following winter. Bomford (1987a) found similar changes in the diet of mice occupying a wheatfield on an irrigated cereal farm in central New South Wales. In the Mallee, the exception to this pattern was a brief increase in wheat consumption at the time of sowing in the early winter of 1985, presumably because mice were unearthing the sown grain. No direct measurements of the availability of food items for mice were reported by Tann et al. (1991) and other data for the period from 1983 to 1997 are very limited. Consequently existing models were used to estimate the relative availability of monocotyledon and dicotyledon seed.

French and Schultz (1984) used data for 61 sites in South Australia from 1964–1975 to establish a linear relationship between wheat yield and the rainfall summed over the period from April to October, R_{A-O}. They found that 65% of the variation in wheat yield was explained by the rainfall data. Cornish et al.

(1980), using a similar model, accounted for 80% of the variation in wheat yield and Seif and Pedersen (1978) used spring rainfall to account for 86% of the variation in yield in central New South Wales. The data set used by French and Schultz (1984) included areas with climate and soils similar to the Victorian Mallee region and the validity of extrapolating from their results was tested by comparing April to October rainfall with crop yields from the Mallee Research Station for the period 1984–1997. The regression accounts for 69% of the variance in the reported values (Figure 3) indicating that, as in the French and Schultz model, R_{A-O} also provides an appropriate index of the wheat harvest in mid to late December at the Mallee Research Station. This index does not provide estimates of seed or crop biomass during the period of crop establishment and maturation. However, some measurements of spilled grain have been made immediately post-harvest and at irregular intervals for up to four months (G.R. Singleton, unpublished data). These data show a steady decline in the quantity of spilled grain post-harvest with little or none remaining on the ground by mid to late autumn. Based on this information, and the observed seasonal changes in monocotyledon seed component in the diet of mice (Tann et al. 1991; Bomford 1987a), the within-year variation in the food from wheat crops was modelled as a sinusoidal function, $[1 + \cos(2\pi(T/9)]/2$, where T is the time in ninths of a year (i.e. intervals of 40.55 days) since the first of January. Then an index of the amount of food from crops is:

$$W = \left(R_{A-O}/348\right)\left[1 + \cos\left(2\pi T/9\right)\right]/2 \qquad (1)$$

where the rainfall has been expressed as a proportion of the maximum observed value of 348 mm for the winter/spring period. The values of this index for the years 1983 to 1998 are shown in Figure 2c. A transformed version of W that helps to distinguish between average and bumper crops was used later for modelling the rate of increase of the mouse population.

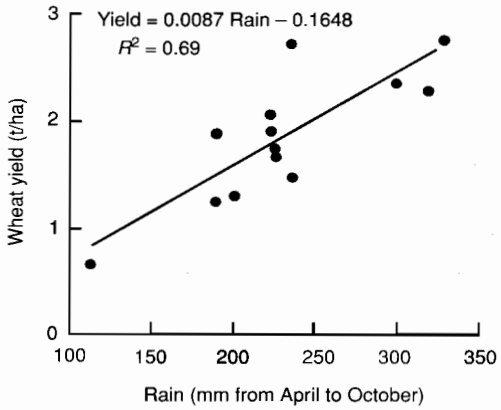

Figure 3.
Comparison of the summer wheat yields (tonnes/ha) at the Mallee Research Station with total rainfall (mm) over the preceding period from April to October. French and Schultz (1984) established a linear relationship between April to October rainfall and wheat yield using data from South Australia.

The model, GrassGro (Moore et al. 1997), was used to estimate the biomass of ripe and unripe seed, S, produced by annual grasses and medic (Figure 2d). It requires detailed information on stocking regimes, soil type and climate (Table 1). Although areas accessible to mice included grazed pastures and ungrazed grass along fencelines and roadside verges, only the estimates for grazed pasture were used. This is the predominant non-crop habitat in the Victorian Mallee and there are relatively

minor differences in the modelled total seed biomass for the two types of pasture. The seed biomass data for a grazed pasture, including medic (Paraggio) and barley grass, were estimated for the period from 1st January 1980 to 31st May 1998. The 1st January 1980 was used as a starting time to remove any distortion from initial values and runs were made over 10-year intervals to ensure that annual grass seed banks were not exhausted after droughts and to mimic pasture sowing after cropping. Each run had a two year overlap with the next.

Table 1.
Data requirements for using the GrassGro model (Moore et al. 1997) for the Mallee Research Station (MRS) at Walpeup (35.08°S, 142.02°E) in the northwest of Victoria.

Attribute	Units/description
Rainfall	daily rainfall in mm recorded at MRS
Temperature	daily maximum and minimum temperature 1965–95: MRS 1996–97: Ouyen Post Office (35.07°S, 142.32°E)[a]
Terrain	gently sloping
Soil	sandy loam, fertility rating of 0.8, soil moisture budget parameters (including thickness of each soil layer, volumetric water content of each layer, drainage and evaporation rates)
Pasture species	barley grass, medic (Parragio)
Livestock	2.0 ewes/ha managed for wool and meat production

[a] Temperature data for 1996–97 were extrapolated from records at the Ouyen meteorological station using the 1965–95 records to generate a temperature-difference profile with the MRS.

Estimates of seed biomass were then recorded for each 10-year simulation excluding the first two years to reduce the effect of 'reseeding' the pasture on the seed available to mice. Finally, seed biomass, *S*, for each 40-day time step was determined by interpolation from the nearest weekly estimates from the GrassGro model.

Modified Victorian Mallee model

Many of the existing models for predicting mouse plagues invoke a complex set of factors to explain the population dynamics of mice (see, for example, the *MIA model*). In this case, as a first step, we assessed how much of the variance in the rate of increase of mice can be explained by the most obvious factor, food availability. Then we determined whether inclusion of a density-dependent factor improved the predictive ability of the model. This additional factor is a surrogate for several processes that may modify the rate of increase when the density of mice is high. Examples include disease and changes in mating behaviour, social organisation or dispersal. The approach is similar to the generalised form of the *predation-regulation model* in that the dynamics of the mouse population are assumed to be driven primarily by annual pulses of food whose duration and magnitude is determined by the weather. However predator-regulation at low mouse densities, e.g. through the aggregation of raptors, was assumed to be less important in the croplands of the Victorian Mallee than in the localised irrigated areas studied by Sinclair et al. (1990).

An Ivlev model was used for the relationship between food availability and

the rate of increase, r, for mice. This has the general form:

$$r = -a + c\left[1 - \exp(-d.F)\right] \qquad (2)$$

where F is an index of food biomass which is seed from pasture and/or wheat in the case of mice. In equation (2), a is the maximum rate of decrease, the maximum rate of increase is $r_{max} = (c - a)$, and the demographic efficiency, d, is a measure of the ability of the mouse population to respond to scarce resources. This form for the rate of increase has been used to model the response of several species to widely fluctuating food supply in climatically-variable parts of Australia; e.g. kangaroos—*Macropus* spp. (Bayliss 1987; Caughley 1987; Cairns and Grigg 1993), feral pigs—*Sus scrofa* (Caley 1993; Choquenot and Dexter 1996) and rabbits—*Oryctolagus cuniculus* (Choquenot 1992; Pech and Hood 1998). The model applies when changes in the abundance of a population are determined primarily by the availability of food.

When food is scarce r is negative, i.e. the population declines, and when food is readily available r is positive but ultimately limited to a maximum value, r_{max}, by the species' reproductive capacity. Density-dependence was included as a linear additive factor, in a similar way to that suggested by Caughley and Krebs (1983). With the additional factor, the parameters a and $(c - a)$ in equation (2) no longer retain their demographic interpretation so the model was simplified to:

$$r = a + c.\exp(-d.F) + gN \qquad (3)$$

where g is a measure of the strength of the density-dependence.

Models were developed through an iterative process where the explanatory variables, food from wheat crops and pasture and the density of mice, were tested singly then in combination. The overall goodness of fit of each model was estimated using VENSIM® (1997) to calculate the sum of the squared errors between the observed rates of increase (from the smoothed data series) and the predictions of each model for the period 1983 to 1989. This statistic was used as a guide in model selection. A second criterion was the ability to predict the rates of increase and the trajectory of mouse abundance for the later period from 1991 to 1994. The optimal values, with 95% confidence limits, were estimated for the parameters in equations (2) and (3), although for some models the maximum rate of increase and the maximum rate of decrease were calculated directly from the smoothed trajectory of mouse abundance and used as fixed parameters. The estimates from the wheat model (equation 1, Figure 2c) do not necessarily indicate their relative availability or value for mice. Comparison of Figures 2b and 2c suggest that the high wheat yields in 1984, 1987, 1993 and 1996 correspond, with a small lag, to mouse plagues. The outbreaks appear to be a response to the difference between normal years, e.g. 1988–92, and the four high-yield years. The sensitivity of the rate of increase to this difference can be explored by transforming the amplitude of the wheat yield index to $(R_{A-O}/348)^u$, where the scaling exponent u is estimated by fitting the models to the data. Then the model of the food available from wheat crops is:

$$W = \left(R_{A-O}/348\right)^u\left[1 + \cos(2\pi T/9)\right]/2 \qquad (4)$$

If, for example $u >> 1$, then equation (4) indicates that mouse population dynamics

are sensitive to changes in high values of W, whereas $u \approx 1$ implies no transformation is necessary (Figure 4).

The results of fitting the models are summarised in Table 2 and illustrated in Figures 5 and 6. It is apparent in Figure 2b that in non-plague years there is too much variability in the field data to detect the expected seasonal variations in the mouse population, e.g. the annual spring decline in abundance (Singleton 1989). The absence of these small seasonal variations carries through to the observed rates of increase (Figures 5a–d). However, in all years, seasonal effects are quite pronounced in the models for food supply (Figures 2c,d) and it was necessary to suppress much of this within-year variability in fitting the models for the numerical response. The result is that the models including wheat, (i), (ii) and (iv) in Table 2, have scaling exponents, u, of 6.6, 9.4, and 11.3, respectively; i.e. all these models require a transformation of W similar to that shown in Figure 4b.

The corresponding numerical response functions show a rapid change from negative to positive r at low values of the wheat index, saturating at r_{max} above this threshold (Figures 6a, b and d). Even with these transformations, all of the food-only models were a poor fit to the observed r in non-plague years. Fixing the maximum rate of decrease in model (i) resulted in a reasonable fit to the post-plague declines in mouse abundance (Figure 5a) but an overall poorer fit compared to model (ii) where only the maximum rate of increase, r_{max}, was fixed. Conversely, model (ii) did not match periods with large negative rates of increase but provided a clearer distinction between plague and non-plague years (Figure 5b).

Model (iii) was based on the pasture seed biomass which showed strong seasonal variation and, apart from 1984 and 1993, was mostly in the range 0–1000 kg/ha. Since this was also the part of the fitted numerical response with a positive slope (Figure 6c), the predicted r showed a corresponding pattern that was not reflected in the observed rates of increase of mice (Figure 5c). A transformation similar to that for wheat was tried unsuccessfully with the seed biomass model.

The best fit to the observed rates of increase was obtained by combining a density-dependent effect with the wheat-based food index [model (iv) in Table 2]. This also resulted in improvements in predicting the trajectory of mouse abundance. However, the upper 95% confidence limit for the density-dependent parameter, g, could not be estimated and there was only a relatively small improvement in the sum of squared errors compared to model (ii).

The results in Figure 5d show that the model matched the high rates of increase that generated the mouse plagues in 1984, 1987 and 1993, but not the latter part of the period of high r leading to the plague in 1997. The major density-dependent contributions were during plague years (Figure 5d) and, compared to the wheat-only model (ii), produced a slightly better fit during periods of rapid decline in mouse numbers. All the models that include wheat (i, ii and iv) were optimised with high values of the scaling exponent, u.

In each case this was balanced to some extent by high values in the demographic efficiency, d, in the product dW. An independent estimate of at least one of these two parameters is required to resolve this difficulty with the Ivlev model.

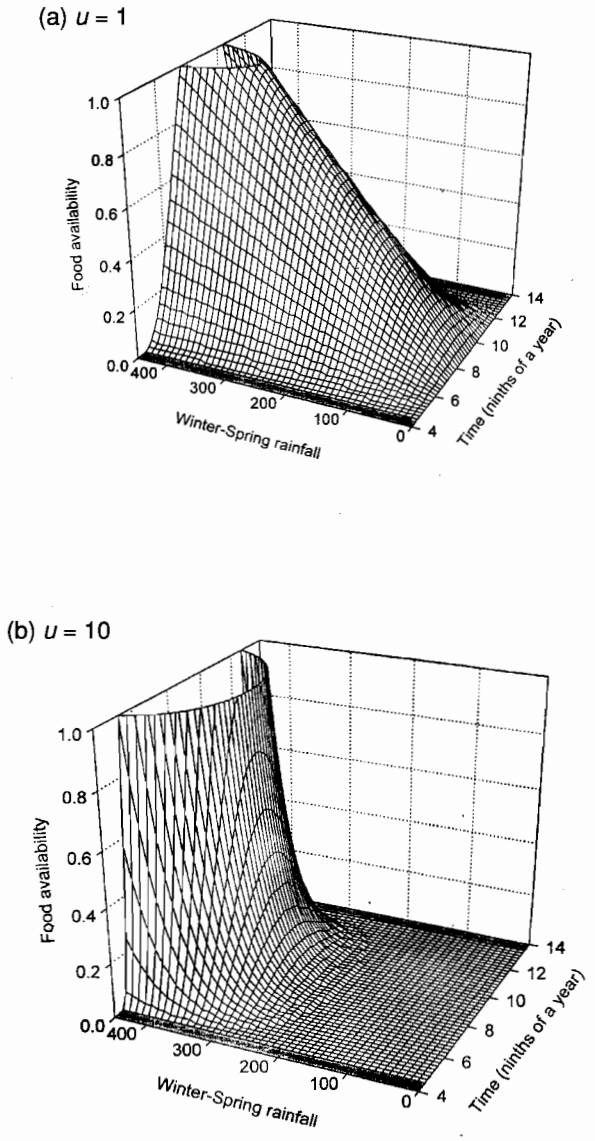

Figure 4.
The effect of the parameter, u, in equation (4) in modifying the index of food availability from wheat crops. Time, in units of $1/9^{th}$ of a year, is measured from the 1^{st} of January each year and rainfall is in mm. (a) u = 1, which is the same food index used for Figure 2c. (b) u = 10, showing the effect of $u >> 1$ in suppressing the food availability for low rainfall, <200–250 mm, and greatly exaggerating the relative availability above this threshold.

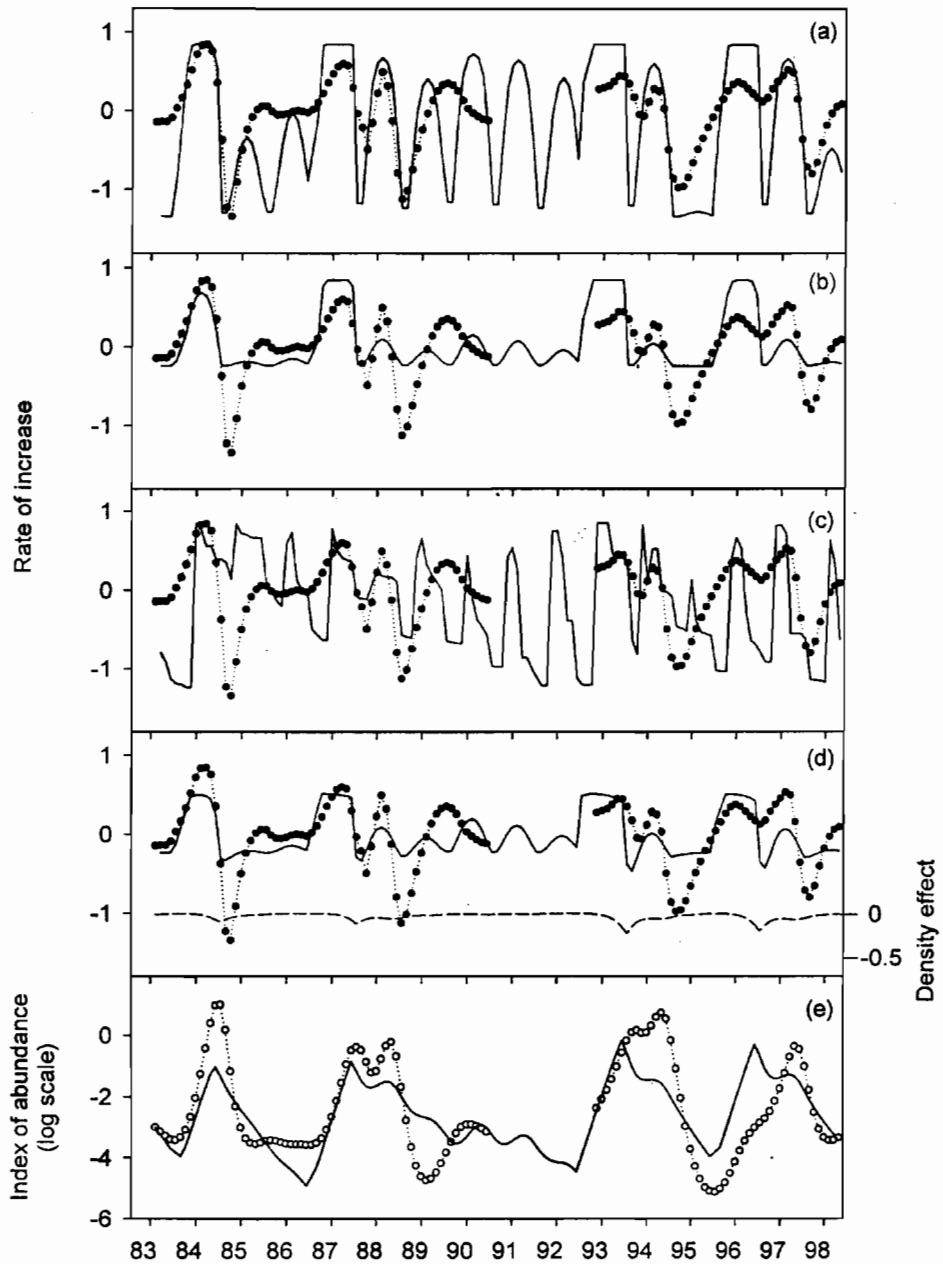

Figure 5.
(a)–(d) The observed (•) and modelled (—) rates of increase per 40 days. (e) The observed (o) and modelled (—) index of abundance of mice. For (a)–(d) the predicted trajectories correspond to the models (i)–(iv) listed in Table 2. The density-dependent contribution to *r* (- -) is shown in (d). For (e), the abundance of mice was predicted using model (iv) from Table 2.

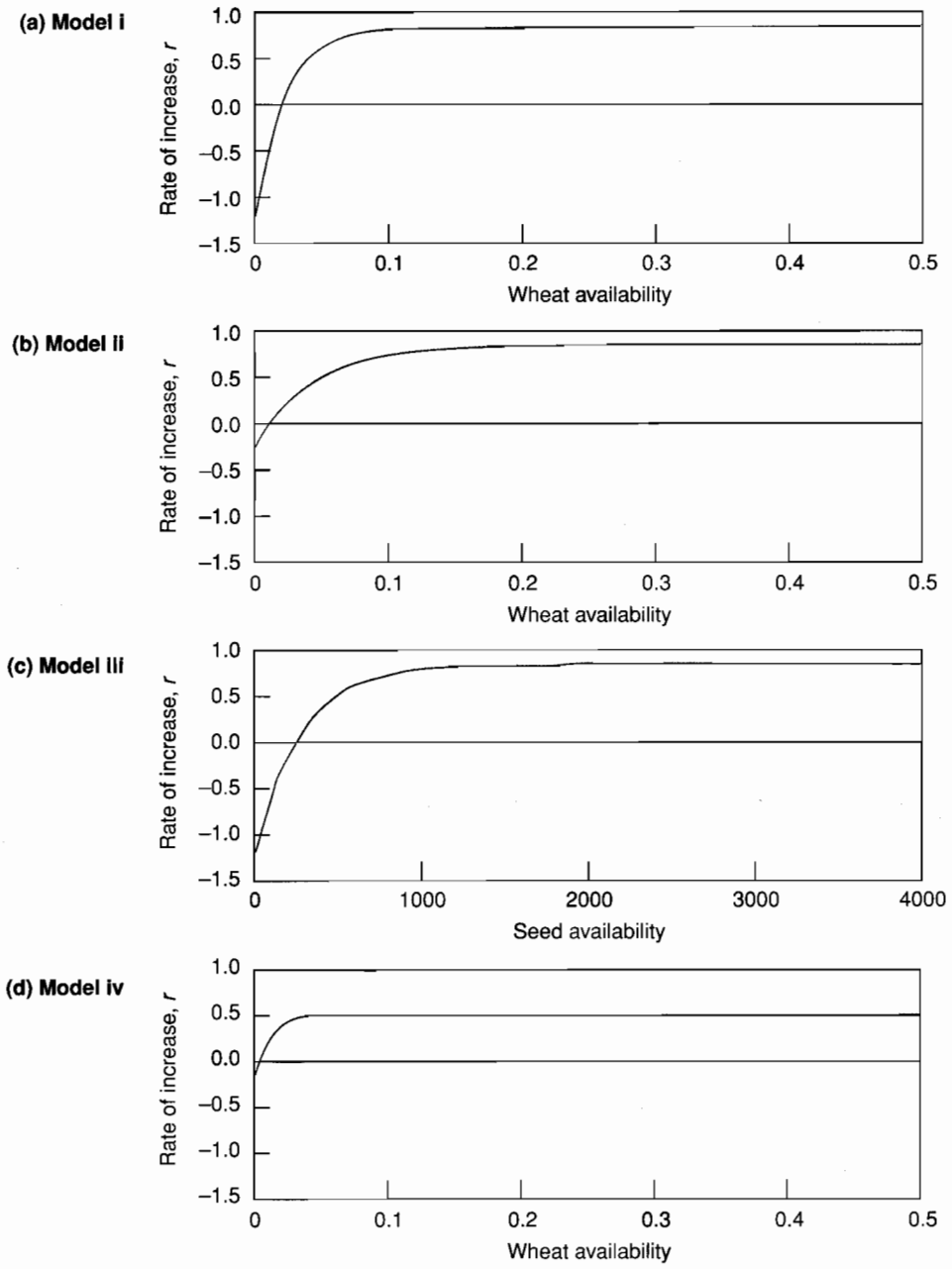

Figure 6.
The estimated numerical response functions corresponding to models (i)–(iv) in Table 2. Food is from (a) wheat crops, with r_{max} and a_W fixed at the observed values, (b) wheat crops, with only r_{max} fixed, (c) pasture seed in kg/ha, with r_{max} and a_W fixed, and (d) wheat crops with the density of mice set at 0.001 in the density-dependent term in model (iv).

Table 2.

Results from fitting models for the numerical response of mice. The food biomass, F, in equation (2) was replaced by an index of the food available from cereal crops, W, (equation 4) for models (i) and (ii), and seed biomass from grazed pasture, S, in kg/ha (model iii). The models' parameters have corresponding subscripts. In model (iv), the rate of increase is determined by equation (4) for W (replacing F in equation 3) and the index of mouse abundance, N. Parameters were optimised using VENSIM® (1997) to fit the models to the data for the rate of increase per 40 days for the period from 1983 to 1989. SSE is the sum of the squared errors in fitting each model. For the optimised values, 95% confidence limits are given in square brackets unless they could not be estimated [NE]

(i)	Food from wheat crops: $r = -a_W + c_W[1 - \exp(-d_W.W)]$, with the maximum rate of increase ($r_{max} = c_W - a_W$) and maximum rate of decrease ($-a_W$) fixed at the observed values.
	a_W = 1.34 (fixed) c_W = 2.19 (fixed) d_W = 48.0 [NE – 77.0] u = 6.6 [NE – 7.5] SSE = 34.7
(ii)	Food from wheat crops: $r = -a_W + c_W[1 - \exp(-d_W.W)]$, with the maximum rate of increase ($r_{max} = c_W - a_W$) fixed at the observed value.
	a_W = 0.24 [0.026 – 0.45] $c_W = r_{max} + a_W$ = 0.84 + a_W (fixed) d_W = 23.7 [4.8 – 61.3] u = 9.4 [7.3 – 13.6] SSE = 10.3
(iii)	Pasture seed biomass: $r = -a_S + c_S[1 - \exp(-d_S.S)]$, with the maximum rate of increase ($r_{max} = c_W - a_W$) and maximum rate of decrease ($-a_W$) fixed at the observed values.
	a_S = 1.34 (fixed) c_S = 2.19 (fixed) d_S = 0.0039 [NE] SSE = 35.5
(iv)	Wheat and density: $r = a_N + c_N \exp(-d_N.W) + gN$
	a_N = 0.22 [0.11 – 0.41] c_N = 0.74 [0.37 – 0.94] d_N = 102.5 [19.7 – NE] u = 11.3 [8.8 – 16.7] g = -0.30 [–12.6 – NE] SSE = 9.1

In addition, the estimation procedure is quite sensitive to small changes in the constants which, together with the wide confidence limits, suggests that we are searching a very flat parameter space in attempting to fit the models.

In Figure 5e, the observed densities of mice for the period for 1983 to 1997 are compared to the predicted trajectories using model (iv) in Table 2.

Some of the main demographic features are represented reasonably well. However, the results clearly demonstrate that the post-plague declines are not well modelled nor is there an obvious explanation for the 12-month difference between the predicted plague in 1996 and the observed outbreak in 1997.

Future directions for the Victorian Mallee model

The modified Victorian Mallee model represents some progress towards a quantitative model for assessing the effectiveness of mouse control operations in southern Australia. However, any assessments will be subject to the proviso that the model for the numerical response is still appropriate after a mouse population has been reduced by a control technique. Alternatively, in the case of immuno-contraception, estimates would be required of the proportional change in r resulting from an imposed level of infertility (Chambers et al. 1997, 1999).

An obvious shortcoming of the modified model is the lack of detail on seasonal changes in demographic parameters. Improvements in this area are likely in future models which will treat fecundity and survival rates separately. Examples of this approach include the density-dependent, stochastic model of Leirs et al. (1997) for the multi-mammate rat *Mastomys natalensis* in Africa and the model used by Pech et al. (1997) to assess the value of fertility control for the management of foxes in Australia. There is some evidence that the onset of breeding by mice depends on seasonal environmental triggers (e.g. Olsen 1981; Bomford 1987a,b,c; Bomford and Redhead 1987; Tann et al. 1991) and the results of model (iv) in Table 2 suggest that density-dependent factors are likely to be important in terminating plagues, presumably through decreased survival rates of mice. In addition, other factors could be included to fine-tune the model. For example, the ultimate abundance of mice in a plague can be strongly influenced by rainfall events in summer and the autumn–winter period (Singleton 1989; Boonstra and Redhead 1994).

It is likely that the strong seasonal variation in food from wheat crops and pasture will play an important role in future age-structured models for both fecundity and survival. Field data are required to validate the pasture and wheat crop models, especially the relationship between seed and crop biomass and the amount of food available to mice. In addition, manipulative experiments should be used to confirm the dependence of the numerical response on food supply. Although it may be difficult to manipulate factors like predation and disease, there may be opportunities to use future mouse control campaigns to test the importance of the density-dependence implied by the optimal model.

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5. Rodent–Ecosystem Relationships: a Review

Chris R. Dickman

Abstract

Because of their ability to use agricultural production and their role in spreading disease in humans, rodents are often viewed as having negative impacts in modified and natural ecosystems. Some species, such as the black rat, have been further implicated in the extinctions of many species of insular land birds, small mammals and invertebrates. In this review, I focus on the interactions of rodents with chemical and structural attributes of the environment, using the concept of 'ecosystem engineering' as a framework. I also discuss the direct and indirect impacts of rodents on food resources.

Many rodents alter the structure of their environment by surface tunnelling, construction of leaf or stick nests, arranging pebbles around burrow entrances, or stripping bark from trees. These activities provide living space or resource opportunities for other organisms, and represent examples of simple allogenic engineering. In more complex examples, digging, nest-building and other activities modify the environment more extensively and modulate resource flows to other organisms. Burrowing rodents such as pocket gophers, prairie dogs and mole-rats alter soil structure and microtopography, nutrient cycling and water flows over local or regional areas, and have dramatic effects on the growth and species composition of plant communities. Nest structures that divert resource flows also represent complex allogenic engineering. For example, beaver impoundments affect nutrient cycles and water flow, and consequently the species richness of aquatic invertebrates, fish and riparian vegetation at local and catchment scales. Rodents also engineer local environments biotically by dispersing seeds and the storage organs of geophytes, as well as the spores of hypogeal fungi that form mycorrhizal associations with plants. Some species probably also play a minor role as pollinators. Rodents, finally, have diverse and often pervasive effects on their food resources; there is much evidence of positive and negative effects on growth form, standing crop and the species composition and physical structure of plant communities

Rodents therefore contribute importantly to ecosystem function, and may have value as indicators of environmental change. Management of rodent pests will need to move away from the broadly destructive current approach of chemical warfare toward ecologically-based solutions that sustainably control only the target species.

Keywords

Ecosystem engineering, environment, rodent, facilitation, predation, nests, burrows

INTRODUCTION

ALTHOUGH SOME 1,800 species of modern rodents have been described (Corbet and Hill 1991), few have been well-studied and the majority remains poorly known. Not surprisingly, most knowledge has been obtained on species that impact on humans by exploiting agricultural production or by spreading diseases (Chitty and Southern 1954; Twigg 1978), or are useful in laboratory research (Barnett 1975). Different species of rodents, especially *Rattus* spp., have been implicated also in the demise of island vertebrate faunas (Atkinson 1985, 1996), and have often been subject to intensive control to achieve conservation objectives. Effective management of rodent pests remains an elusive but important goal in many parts of the world, and for different reasons. As discussed by various authors in this book, solutions may lie more with ecologically-based management than with simple one-factor approaches that have been used previously.

Despite the often negative effects of rodents in natural and modified ecosystems, many species have been shown to contribute to ecosystem function and to have value as indicators of environmental change. For example, microtine rodents are important at times in the cycling of carbon, nitrogen and other elements (Inouye et al. 1987a, Huntly 1991), while beavers cause alteration of hydrological regimes (Naiman et al. 1988). Such species have been termed 'ecosystem engineers' (Jones et al. 1994). Other species may be important as pollinators or vectors of fungal spores (Tory et al. 1997). Both microtine and sciurid rodents have been used as indicators of industrial pollution (Kostelecka-Myrcha et al. 1981; Lepage and Parker 1988), while some murids have been used to indicate the severity of impact wrought by defoliants used in chemical warfare (Sokolov et al. 1994; Evgenjeva and Fadeeva 1996). Several further species also may be sensitive barometers of climatic change (Frey 1992; Bright and Morris 1996).

The range of interactions of rodents with the environment is not well appreciated, perhaps because the interactions are diverse, often complex, or not apparent in studies carried out in small study areas or for short periods. However, such an appreciation is likely to be important for successful management of rodent pests, and essential if management is to be ecologically-based.

In the present paper, I present a selective review of rodent–ecosystem relationships, focusing on the impacts of rodents on the physical, chemical and biotic environments and the consequences of these impacts for other biota. Little attention is given to competitive relationships among rodents or to rodents as prey, because reviews of these topics are available elsewhere (Sinclair 1989; Brown and Harney 1993; Dickman and Doncaster, submitted for publication). Where possible, studies that demonstrate interactions experimentally have been emphasised, because these are most likely to identify the nature and magnitude of any interactions that occur. The concept of 'ecosystem engineering' is used to provide a framework for much of the review.

Ecosystem Engineering

The term 'ecosystem engineering' was introduced by Jones et al. (1994) and refined by Jones et al. (1997). It may be defined as follows: "Physical ecosystem engineers are organisms that directly or indirectly control the availability of resources to other organisms by causing physical state changes in biotic or abiotic materials. Physical ecosystem engineering by organisms is the physical modification, maintenance or creation of habitats. The ecological effects of engineering on other species occur because the physical state changes directly or indirectly control resources used by these other species" (Jones et al. 1997, p. 1947).

Engineers were divided into two broad groups by Jones et al. (1994, 1997). *Autogenic* engineers change the environment by their own physical structures; an example would be the shed limbs of trees that modulate microclimate and microhabitat for other organisms on the forest floor. In contrast, *allogenic* engineers change the environment by transforming living or abiotic materials from one state to another by mechanical or other means. An example would be the construction of burrows by one species that could be used by others. Rodents could be expected to be allogenic engineers.

In both their papers, Jones et al. (1994, 1997) drew a distinction between physical ecosystem engineering and other ecological processes such as pollination, dispersal, competitive and trophic interactions, including the utilisation of living or dead tissue by consumers or decomposers. In the present paper, however, I include the former two of these processes under the term 'biotic engineering'. Justification for this approach is given in Figure 1; this approach also provides consistency in this review. Trophic interactions do not fit an engineering paradigm, and the effects of rodents on plant and invertebrate prey species are discussed separately below.

Allogenic Engineering

Simple cases: changes in physical state (Figure 1a)

Rodent burrows are obvious and widespread examples of allogenic engineering. Simple burrows are made by most species at some time in their life cycle, and vary in size, orientation, depth and substrate characteristics. Among Australian desert rodents, *Pseudomys hermannsburgensis* and *Notomys alexis* dig deep, vertical burrows in summer to avoid high daily temperatures, but occupy shallow surface burrows in other seasons when temperatures, and daily variations in temperature, are less extreme (C.R. Dickman, personal observation). Other species, such as *Rattus colletti*, barely modify cracks in the soil (Madsen and Shine 1999) or, like *Rattus villosissimus*, may construct complex networks of tunnels under favourable conditions (Predavec and Dickman 1994). The burrows of many species have been described in the literature (e.g. Kemper 1981; Bronner 1992), with overviews provided by Reichman and Smith (1990), Meadows and Meadows (1991) and Hansell (1993).

The major resource created by burrows is living space for other organisms. Other rodents, lizards, snakes and many species of invertebrates make opportunistic use of burrows (Kiviat 1978; Skinner and Smithers 1990). In arid Australia, several species of

dasyurid marsupials make extensive use of abandoned rodent burrows, being unable to dig burrows themselves (Dickman and Read 1992; Dickman 1996). In one study, the burrowing activity itself, in reducing compaction of soil, was shown to have the additional effect of promoting germination of seeds of an iridaceous geophyte (Contreras and Gutiérrez 1991).

Nests provide another example of allogenic engineering. Simple constructs, such as the cup-shaped grass nests of *Micromys minutus*, may take hours or days to build and last for the duration of one breeding season (Harris and Trout 1991); more complex structures of sticks and other detritus, engineered by *Leporillus* spp. and *Neotoma* spp., often last for generations (Copley 1988). Nests are made from a variety of living and non-living materials, and are sometimes decorated with pebbles or other materials (Anstee et al. 1997) for reasons that remain unclear. As with burrows, nests provide living space for other species of vertebrates and invertebrates. Such exploitation is usually opportunistic. However, blind, wingless earwigs of the genus *Hemimerus* are found primarily in the nests of *Cricetomys gambianus*, and may be obligately associated (Knight 1984).

Two, more subtle examples of allogenic engineering may be cited. The first involves shallow scrapes created in surface soil by foraging rodents that provide sites for accumulation of seeds (McNaught 1994, see also below). The second involves bark-stripping of trees by *Sciurus* spp., *Sundasciurus* spp.and other squirrels (Medway 1983). De-barking facilitates access of fungal pathogens to vascular tissues (Abbott et al. 1977), while dead trees provide nesting, roosting and shelter sites for several species of birds, bats and other arboreal mammals (Corbet and Harris 1991; MacKinnon et al. 1996). It is likely that rodent-induced damage to plants provides opportunities for exploitation by a broad range of organisms, but few relevant studies have been carried out to confirm this (for a general discussion, see Karban and Myers 1989).

Complex cases: state changes that modulate resource flow (Figure 1b)

Continual and intensive burrowing activity by rodents may provide temporary living space for other organisms, but it also affects nutrient cycling, water flow, soil structure and microtopography. Such effects have been studied in detail in several species of fossorial and terrestrial rodents, especially North American geomyids, or pocket gophers, prairie dogs and Old World mole-rats.

The digging activities of pocket gophers (70–350 g) produce small piles of fresh surface soil that may, over extended periods, accumulate into large mounds termed mima mounds (Inouye et al. 1997). In some habitats, digging activity can cast over 15,000 kg of soil/ha/year onto the surface, and mima mounds of 25–50 m in diameter and 2 m in height may be common (Beuchner 1942; Ross et al. 1968). Some 50–100 mima mounds have been recorded per hectare in some areas, with higher densities occurring usually in disturbed prairie and agricultural landscapes (Mielke 1977). The mounds may consist entirely of topsoil, or soil with gravel and pebbles 50–60 mm diameter; in some locations the presence of soil horizons within mounds suggests a long period of stabilisation (Cox and Gakahu 1986).

Comparisons of soils from mounds and undisturbed inter-mound areas have shown differences in texture, organic content, water-holding capacity and nutrient status (Mielke 1977; Hobbs and Hobbs 1987; Inouye et al. 1987b; Huntly and Inouye 1988). These differences in turn promote heterogeneity in plant species composition and growth responses. In shortgrass prairie, the burrowing activities of *Thomomys bottae* may kill standing vegetation but provide opportunities for establishment of herbaceous perennial dicots (Martinsen et al. 1990). In serpentine grassland, mounds of *T. bottae* are invaded by different species of plants depending on prevailing rainfall conditions (Hobbs and Mooney 1991); the timing and intensity of soil disturbance may also be important (Moloney et al. 1992). Finally, in tallgrass prairie, the mounds of *Geomys bursarius* have complex effects on both vegetation and fauna. Mounds break the prairie canopy and provide recruitment sites for dicot seedlings, often increasing local plant diversity (Hartnett and Keeler 1995). Mounds also attract some herbivores such as grasshoppers, but may either attract or repel mammalian herbivores such as the meadow vole *Microtus pennsylvanicus* (Whittaker et al. 1991; cf. Klaas et al. 1998). If mounds alter local patterns of herbivory, this is likely to produce further effects on

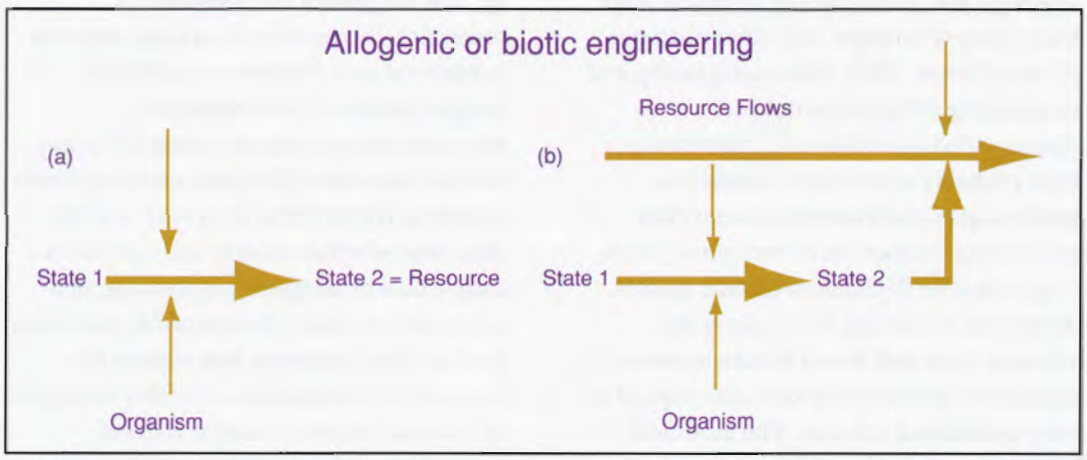

Figure 1.
Conceptual models of allogenic and biotic engineering, as applied to rodents (after Jones et al. 1994, 1997). In the simplest case, (a), living or non-living raw materials are transformed by animal activity from state 1 to state 2. The point of modulation is shown by opposing arrow heads. In allogenic engineering, state 2 is a new engineered resource such as a burrow that usually can be used immediately. In biotic engineering, state 2 is an activated but incipient resource such as a pollinated flower or dispersed seed or spore that may be structurally no different from the state 1 condition.
In the more complex case, (b), the products of state 2 modulate the flow of one or more resources to other species. Such modulation may be rapid if state 2 resources have been engineered allogenically, but slow if engineering has been biotic and is contingent on growth of plant or fungal tissue. Jones et al. (1994, 1997) discussed additional types of allogenic and autogenic engineering, but these do not appear relevant to rodents. 'Biotic engineering' is used for the first time here.

plant community structure and heterogeneity, perhaps promoting species richness over time (Klaas et al. 1998).

Like their smaller counterparts, prairie dogs (1 kg) also modulate resource flows to other species by digging. Research on the best-studied species, *Cynomys ludovicianus*, shows that colonies develop on deep, productive soils where flooding is unlikely, and range in size from tens to hundreds of hectares (Dahlsted et al. 1981; Hoogland 1994). Up to 300 burrows may occur per hectare, with soil mounds 1–2 m diameter surrounding each burrow entrance (Whicker and Detling 1988). Digging affects soil structure and compaction, increases drainage and, with grazing by prairie dogs, the cycling of nitrogen and other nutrients (Coppock et al. 1983). Although grazing and engineering effects have not been disentangled in studies of *C. ludovicianus*, both probably contribute to extensive patterning of plant communities within prairie dog colonies. In mixed-grass prairie, Coppock et al. (1983) showed that grasses decreased in biomass with colony age whereas forbs and dwarf shrubs increased; nitrogen in graminoid shoots also peaked in long-established colonies. The modified habitats produced by prairie dog excavations favour increased local abundances and diversity of open-plain birds but decreased species richness of small mammals (Agnew et al. 1986). Interestingly, colony sites also contain higher densities of soil nematodes than undisturbed areas (Ingham and Detling 1984), perhaps reflecting greater ease of establishment in loosened soil.

Burrowing and tunnelling activities by fossorial rodents such as mole-rats displace large volumes of soil and often result in the creation of surface mounds. These surficial structures resemble the mounds of pocket gophers and prairie dogs in size and composition, and have usually similar effects on nutrient status, water flow and organic content (Jarvis and Sale 1971; Cox and Gakahu 1985; Cox et al. 1987). Cox and Gakahu (1985) showed that coverage of forbs and shrubs on mima mounds of *Tachyoryctes splendens* was more than double that on inter-mound plots, whereas coverage of grass and *Acacia* trees was much reduced. These authors also noted a correlation between the activity areas of mole-rats and a fungus-gardening termite—*Odontotermes* sp., and suggested that termites preferentially use the rich organic deposits in mole-rat nest chambers to establish fungus gardens. A wide range of invertebrates has been documented using the nest mounds of the blind mole-rat *Spalax ehrenbergi* (Heth 1991). However, it is not clear here whether mound use represents a simple case of allogenic engineering, or a more complex case where mounds modulate food or other resources that sustain the invertebrate communities. Further examples of fossorial or semi-fossorial rodents modulating resource flow for other species by their burrowing activities occur within the Microtinae, Octodontidae and Heteromyidae (e.g. Chew and Whitford 1992; Contreras and Gutiérrez 1991; Gómez-Garcia et al. 1995; Borghi and Giannoni 1997). A useful review is provided by Huntly and Reichman (1994).

Nest structures that divert resource flow represent a further class of examples of complex allogenic engineering. Beaver dams are the most conspicuous examples of such

structures; similar but less extensive nests are made by muskrats *Ondatra zibethicus* and occasionally by *Myocastor coypus* (Ebenhard 1988).

Beaver dams are constructed of young and mature trees that the animals cut themselves, as well as sediments and other debris. The North American beaver, *Castor canadensis*, builds some 2–16 dams per kilometre of stream, with small dams containing 4–18 m^3 and larger dams >100 m^3 of wood (Naiman et al. 1986, 1988). The major effect of dams is to alter the stream channel by impounding water, creating patch bodies (sensu Johnston and Naiman 1987) of water, sediment, aerobic soil beneath the pond and anaerobic soil in deeper strata. The surrounding riparian zone is also affected by damming, with stream widths sometimes increased by an order of magnitude from their original condition (Naiman et al. 1988). Because of the changed hydrological regime and the additional effects of beaver herbivory, patch bodies show dramatically different fluxes of carbon, nitrogen and energy compared with unaltered streams. Impoundments usually have relatively low inputs of carbon, but high standing stocks and outputs (Naiman et al. 1986); significant fluxes arise from release of methane (Naiman et al. 1991; Yavitt et al. 1992). Impoundments have been shown further to enhance accumulation of nitrogen in sediment by 9–44 fold compared with undisturbed streams (Francis et al. 1985). The effects of impoundment on pH, dissolved oxygen, fluxes of energy, other nutrients and ions have been much studied for *C. canadensis* in many parts of its range (e.g. Wilde et al. 1950; Hodkinson 1975; Pinay and Naiman 1991; Naiman et al. 1994) and, to a lesser extent, for the related *Castor fiber* in Europe (Cirmo and Driscoll 1993; Macdonald et al. 1995).

The physical structure of beaver dams, and particularly the effects of dams on resource flows, have important consequences for aquatic and terrestrial animals and riparian vegetation. In the short term (years) impoundments may kill streamside trees and provide nest or roost sites for volant vertebrates following formation of hollows. In the longer term (decades to millenia), impoundments are likely to be colonised by wetland plants and follow successional pathways that may lead to meadows, bogs or wetlands (Figure 2). The relative roles of beaver engineering and other physical processes such as erosion, sedimentation and fire in directing particular pathways remain unclear, but likely differ between regions (Naiman et al. 1988, 1994; Johnston 1995).

Damming produces a shift from lotic (fast flowing) to more lentic (still-water) conditions, especially in higher order streams. Among aquatic invertebrates, this shift favours collector and predator species such as tubificid worms, clams and dragonflies over shredder and scraper species such as blackflies, scraping mayflies and net-spinning caddisflies (McDowell and Naiman 1986). However, lotic taxa may still be represented highly on the dam walls, perhaps because the dam acts as a net that traps drifting lotic fauna (Clifford et al. 1993). Among fishes, lotic taxa give way similarly to still-water specialists in beaver impoundments. Species richness and composition differ in dammed headwater and lower-order streams and vary also with age of the impoundment (Keast and Fox

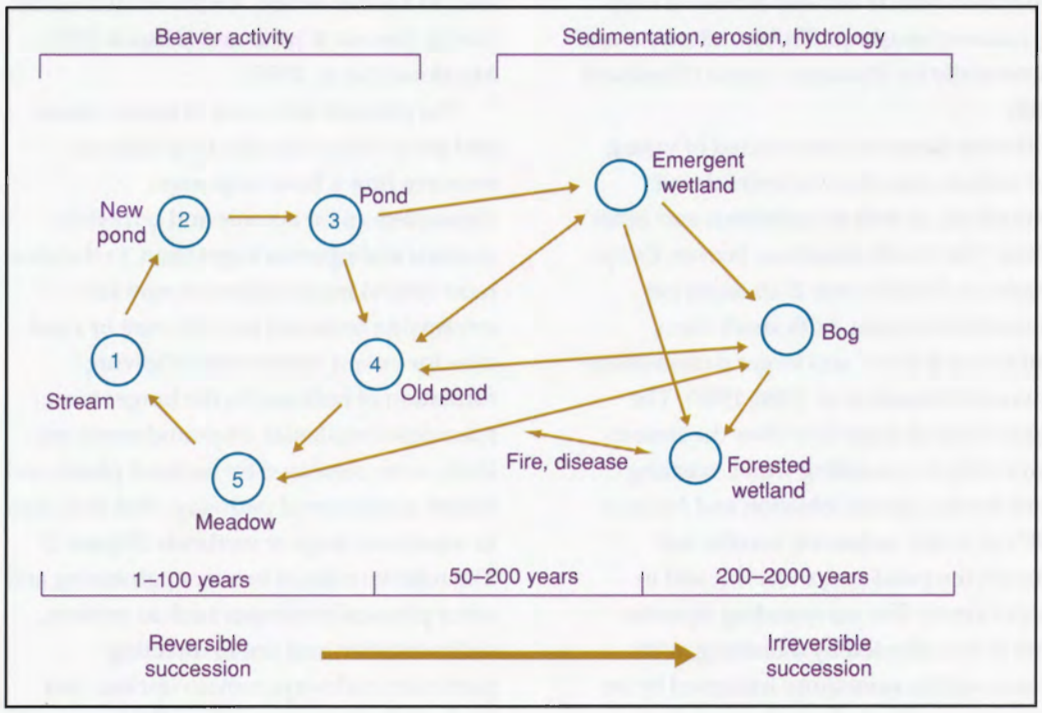

Figure 2.
Potential effects of beaver (*Castor canadensis*) on vegetation and landscape patterns, based on work by R.J. Naiman and colleagues in the boreal forests of northern Minnesota (after Naiman et al. 1988).

1990; Hägglund and Sjöberg 1999; Snodgrass and Meffe 1998).

Descriptive and experimental studies have suggested further that beaver ponds act as reproductive source populations for fish whereas adjacent streams act as sinks (Schlosser 1995). If so, beaver dams may be seen as important components of fish metapopulations at catchment or larger spatial scales.

The engineering activities of beavers may, finally, have subtle indirect effects on terrestrial invertebrates. Martinsen et al. (1998) have shown recently that resprout growth from beaver-cut cottonwood trees (*Populus fremontii* and *Populus angustifolia*) is attractive to a specialist leaf beetle, *Chrysomela confluens*. The beetles sequester phenolic glycosides from the cottonwood leaves and use them as a means of predator defense. Martinsen et al. (1998) asserted further that habitat mosaics created by beaver activity increase the diversity of arthropods and perhaps higher vertebrates as well, but provided no evidence in support of this claim.

A final class of examples of complex allogenic engineering is the surface digging activity of rodents that results in accumulation of organic material and diversion of water flow. Gutterman (1982) showed that the diggings of Indian crested porcupines, *Hystrix indica*, accumulate seeds and other organic matter, and provide

microhabitats favourable for the germination and establishment of certain species of plants. Diggings are more suitable for germination in protected than exposed habitats, apparently because they allow run-off of rainfall for longer periods (Gutterman and Herr 1981; see also Yair and Rutin 1981). Steinberger and Whitford (1983) presented similar findings from their work on the surface digging activities of desert heteromyids.

Studies of larger mammals such as brush-tailed bettongs (*Bettongia penicillata*) and grizzly bears (*Ursus arctos horribilis*) indicate that surface digging activity can dramatically decrease soil water repellency and enhance levels of mineral nitrogen (Garkaklis et al. 1998; Tardiff and Stanford 1998). Such effects might be predicted also from the digging activity of larger rodents, but do not appear yet to have been documented.

Biotic Engineering

Dispersal of seeds and spores

Although movements of seeds or spores from one place to another constitute biotic engineering as defined here, the phenomenon is ecologically more relevant after growth of the embryonic tissue has become sufficient to modulate resource flow to other organisms. Movement of seeds by rodents is well established. In some species, such as tropical squirrels, seeds are ingested and later excreted elsewhere in the animals' home ranges (Emmons 1992; MacKinnon et al. 1996). In many other species, seeds are collected and cached, or hoarded, for later consumption (Gurnell 1983; Reichman and Price 1993). Seeds often survive caching to germinate and become established (Vander Wall 1990), but the role of rodents as dispersal agents remains poorly known. In one particularly instructive recent study, Vander Wall (1997) showed that some 80% of piñon pine (*Pinus monophylla*) seeds, placed experimentally on the ground beneath trees, were gathered by rodents. Radioactively labelled seeds were mostly cached, either in scatter-hoards or larders, at distances up to 38.6 m from the source. Over a third of caches occurred beneath shrubs; these appeared to favour establishment, and served as nurse plants for young pines. Vander Wall (1997) demonstrated seed caching by four species of rodents in captivity—*Peromyscus maniculatus*, *Peromyscus truei*, *Perognathus parvus* and *Dipodomys panamintinus*—and inferred that these were the main seed dispersers in his field site too.

Fossorial rodents have also been demonstrated to move the storage organs of geophytic plants, often concentrating them within mounds or burrow systems (Galil 1967; Gómez-Garcia et al. 1995). Sprouting of storage organs at their new locations suggests that rodent-induced dispersal can be effective (Borghi and Giannoni 1997).

Dispersal of fungal spores by rodents has received relatively little attention. Many species eat the fruiting bodies of fungi (e.g. Maser et al. 1978; Claridge and May 1994; Tory et al. 1997), but it has not always been shown that ingested spores remain viable. However, spores usually remain structurally intact following passage through rodent guts, and Claridge et al. (1992) showed that spores recovered from faeces of another mammal, *Potorous tridactylus*, developed ectomycorrhizae on the roots of two species

of *Eucalyptus*. Importantly, the fungi ingested by many species of rodents are hypogeal and form mycorrhizal associations with the roots of trees and other vascular plants, thus potentially assisting plant growth. Future research should seek to clarify the extent to which rodents disperse viable spores, and also quantify their contribution to regeneration and development of forest environments (Reddell et al. 1997; Tory et al. 1997).

Pollination

Bats and primates that visit flowers for food are often effective pollinators, especially in tropical and arid habitats (Fleming and Sosa 1994). The effectiveness of rodents as pollinators, however, is less clear. Many species visit flowers and could transfer pollen that has lodged in the fur (Recher 1981). Examples include arboreal species such as dormice—*Muscardinus avellanarius* (Bright and Morris 1996), tree-rats—*Solomys* spp. (D. Fisher, pers. comm.) and desert rodents in the genus *Pseudomys* (C.R. Dickman, personal observation). Few studies have shown that rodents carry significant loads of pollen between flowers (Lumer 1980; Wiens et al. 1983; Van Tets 1997) and none has yet distinguished the relative importance of rodents as pollinators compared with other taxa (Carthew and Goldingay 1997). As Fleming and Sosa (1994) point out, the genetic effects of even the more conspicuous mammalian pollinators and frugivores on plant populations have been rarely investigated; there is much scope for new research.

Trophic Impacts of Rodents

Rodents take a very broad range of plant and animal foods, so their potential effects on prey species and communities could be pervasive. Some species of rodents specialise in taking only one or two prey taxa (e.g. the heteromyid *Liomys salvini* specialises seasonally on seeds of *Enterolobium cyclocarpum*, a Central American leguminous tree; Janzen 1981), whereas others are broadly omnivorous (e.g. many species of Australian desert rodents; Murray et al. 1999). The direct impacts of rodent predation have a long history of study, especially with respect to effects on crops and other vegetation, but indirect impacts have been recognised increasingly in recent work. This is a vast topic that can only be treated superficially here.

The best estimates of rodent impact on food resources are from agro-ecosystems in different parts of the world (e.g. Buckle and Smith 1994; Singleton and Petch 1994; other chapters in this book). In these simplified environments, rodents can reach extraordinary densities (e.g. >3,000/ha for *Mus domesticus*; Caughley et al. 1998) by eating one or a very few types of food, and cause great damage to crops. Both native and introduced species of rodents can become pests, and achieve higher densities in crop systems than in the natural environment. Very high densities may be achieved transiently by rodents in unmodified environments, often following drought-breaking rains (e.g. 1,200/ha for *R. villosissimus*; Palmer 1886), but impacts on food resources under these conditions have been little-studied (Batzli and Pitelka 1970; Noy-Meir 1988).

In natural or little modified environments, rodents may have local or broad-scale effects on vegetation. Below ground, herbivory often modifies plant community structure, reducing the standing crop but increasing local species richness (Andersen 1987; Huntly and Reichman 1994). Selective foraging on individual plant species may benefit certain life-history stages such as seeds or small bulbs by reducing intraspecific competition (Contreras and Gutiérrez 1991), but can also depress plant biomass and flower production (Reichman and Smith 1991) or even result in local plant extinction (Cantor and Whitham 1989). Above ground, rodent herbivory (including frugivory and granivory) has even more dramatic effects on vegetation. Selective foraging may again deplete favoured species in local areas, and alter trajectories of plant succession (Johnston and Naiman 1990). Generalist foraging has been shown to have pervasive effects on life form, growth, allocation of nutrients and energy stores within plants, as well as on the physical structure and species composition of plant communities (Batzli and Pitelka 1970; Brown et al. 1979; Brown and Heske 1990; Holland et al. 1992; Jefferies et al. 1994; but cf. Gibson et al. 1990). Although this topic is too broad to discuss fully here, the effects and mechanisms by which herbivores affect plant communities have been reviewed by Crawley (1983) and Huntly (1991), and the induction of plant defenses has been reviewed by Karban and Myers (1989). Short-term feedbacks and longer-term coevolution between herbivorous rodents and plants also have been discussed in detail elsewhere (Crawley 1983; Coley and Barone 1996; Pastor et al. 1997).

Perhaps because the impacts of rodents on vegetation are often obvious and economically relevant, the effects of rodents on other food groups have been seldom studied. However, limited experimental evidence suggests that high density populations of omnivorous species may deplete the local richness of epigeal invertebrates (Figure 3). On Boullanger Island, Western Australia, invertebrate species richness increased on average by 3% on plots from which *M. domesticus* had been removed, in contrast to a decrease of 18% on control plots (Figure 3a). Increases occurred primarily in beetle and spider species, which the mice ingested (C.R. Dickman, personal observation). In urban woodland in the United Kingdom, invertebrate species richness increased similarly by 83% on plots from which *Apodemus sylvaticus* had been removed, compared with only a 32% increase on control plots (Figure 3b). Increases occurred in species of beetles, spiders and snails—taxa found commonly in the diet of urban *A. sylvaticus* (C.R. Dickman, personal observation). Primarily insectivorous rodents such as grasshopper mice (*Onychomys* spp.) likely affect individual species and communities of invertebrates at times also, but evidence is lacking.

In circumstances when omnivorous rodents have been introduced to new environments, they have sometimes had dramatic effects on populations of invertebrates and small vertebrates. On Lord Howe Island, for example, an endemic phasmid, *Dryococelus australis*, disappeared following establishment of *Rattus rattus*, while numbers of two species of island snails were severely depressed (Smithers et al. 1977).