Ecologically-based Management of Rodent Pests

ECOLOGICALLY-BASED MANAGEMENT OF RODENT PESTS

Edited by: **Grant R. Singleton, Lyn A. Hinds, Herwig Leirs and Zhibin Zhang**

Australian Centre for International Agricultural Research
Canberra 1999

The Australian Centre for International Agricultural Research (ACIAR) was established in June 1982 by an Act of the Australian Parliament. Its primary mandate is to help identify agricultural problems in developing countries and to commission collaborative research between Australian and developing country researchers in fields where Australia has special competence.

Where trade names are used this constitutes neither endorsement of nor discrimination against any product by the Centre.

ACIAR MONOGRAPH SERIES

This peer-reviewed series contains the results of original research supported by ACIAR, or deemed relevant to ACIAR's research objectives. The series is distributed internationally, with an emphasis on the Third World

©Australian Centre for International Agricultural Research GPO Box 1571, Canberra, ACT 2601.

Singleton, G.R., Hinds, L.A., Leirs, H. and Zhang, Z.ed. 1999. Ecologically-based management of rodent pests. ACIAR Monograph No. 59, 494p.

ISBN 1 86320 262 5
Editing and design by Arawang Communication Group, Canberra
Printed by Brown Prior Anderson, Melbourne, Australia

Contents

Author Contact Details

Berdoy, Manuel, Wildlife Conservation Research Unit, Department of Zoology, and Department of Veterinary Services, c/o University Laboratory of Physiology, University of Oxford, South Parks Road, Oxford, OX1 3PT, UK.
Email: manuel.berdoy@vet.ox.ac.uk

Boonsong, Puangtong, Agricultural Zoology Research Group, Division of Entomology and Zoology, Department of Agriculture, PO Box 9-34, Bangkok 10900, Thailand.
Email: agrizoo@bkk1.loxinfo.co.th
Fax: +662 9405396

Bounnaphol, Onechanh, Agriculture and Forestry Service, Luang Prabang, Lao PDR.
Fax: +856 71 212635

Brown, Peter R., CSIRO Wildlife and Ecology, GPO Box 284, Canberra, ACT 2601, Australia.
Email: peter.brown@dwe.csiro.au
Fax: +61 2 6242 1505

Buckle, Alan P., Zeneca Agrochemicals, Fernhurst, Haslemere, Surrey GU27 3JE, UK.
Email: Alan.Buckle@aguk.zeneca.com
Fax: +44 1428 6555668

Chambers, Lisa K., Vertebrate Biocontrol Cooperative Research Centre, CSIRO Wildlife and Ecology, GPO Box 284, Canberra, ACT 2601, Australia.
Email: lisa.chambers@dwe.csiro.au
Fax: +61 2 6242 1505

Chen, Anguo, Changsha Institute of Agriculture Modernization, Chinese Academy of Science, Changsha, Hunan 410125, P.R. China.
Fax: +86 731 4612685

Chhorn, Nel, Cambodia–IRRI–Australia Project (CIAP), PO Box 1, Phnom Penh, Cambodia.
Email: IRRI-CAMBODIA@CGIAR.ORG
Fax: +855 23 211728

Colvin, Bruce A., 16 Temple Road, Lynnfield, MA 01940, USA.
Email: bacolvin@juno.com
Fax: +1 781 334 5782

Cooper, Howard M., INSERM (Institut National pour la Santé et la Recherche Médicale), U371, Cerveau et Vision, Bron, France.
Email: cooper@albert.lyon151.inserm.fr
Fax: +33 47 2913461

Cox, Peter G., Cambodia–IRRI–Australia Project (CIAP), PO Box 1, Phnom Penh, Cambodia.
Email: Peter.Cox@bigpond.com.kh
Fax: +855 23 211728

Diarra, Wamian, CNRST (Centre National de la Recherche Scientifique et Technologique), BP 3052, Bamako, Mali.

Dickman, Chris R., Institute of Wildlife Research, School of Biological Sciences, University of Sydney, New South Wales 2006, Australia.
Email: cdickman@bio.usyd.edu.au
Fax: + 61 2 9351 4119

Douang Boupha, Bounneuang, Department of Agriculture and Extension, Vientiane, Lao PDR.
Email: j.m.schiller@cgiar.org
Fax: +856 21 414373

Duplantier, Jean-Marc, Programme RAMSE, IRD (Institut de Recherche pour le Développement, ex ORSTOM), BP 434, Antananarivo, Madagascar.
Email: duplantier@ramse.ird.mg
Fax: +261 20 22 404 51

Fan, Naichang, Department of Biology, Zhejiang Normal University, Jinhua, Zhejiang 321004, P.R. China.

Forrester, Robert I., CSIRO Mathematical and Information Sciences, GPO Box 664, Canberra, ACT 2601, Australia.
Email: bob.forrester@cmis.csiro.au
Fax: 61 2 6216 7111

Hamarit, Greangsak, Agricultural Zoology Research Group, Division of Entomology and Zoology, Department of Agriculture, PO Box 9-34, Bangkok 10900, Thailand.
Email: agrizoo@bkk1.loxinfo.co.th
Fax: +66 2 9405396

Hinds, Lyn A., CSIRO Wildlife and Ecology, GPO Box 284, Canberra, ACT 2601, Australia.
Email: lyn.hinds@dwe.csiro.au
Fax: +61 2 6242 1505

Hongnark, Sermsakdi, Agricultural Zoology Research Group, Division of Entomology and Zoology, Department of Agriculture, PO Box 9-34, Bangkok 10900, Thailand.
Email: agrizoo@bkk1.loxinfo.co.th
Fax: +66 2 9405396

Hood, Greg M., CSIRO Wildlife and Ecology, GPO Box 284, Canberra, ACT 2601, Australia.
Email: greg.hood@dwe.csiro.au
Fax: +61 2 6242 1505

Huang, Xiuqing, Guandong Institute of Plant Protection, Chinese Academy of Agricultural Science, Guangzhou, Guangdong 510640, P.R. China.
Fax: +86 20 87561757

Hung, Nguyen Manh, Institute of Agricultural Science of South Vietnam, Ho Chi Minh City, Vietnam.
Email: ias.hung@hcm.vnn.vn
Fax: +84 8 829 7650

Hung, Nguyen Quy, Institute of Agricultural Science of South Vietnam, Ho Chi Minh City, Vietnam.
Email: ias.hung@hcm.vnn.vn
Fax: +84 8 829 7650

Jackson, William B., Department of Biological Sciences, Bowling Green State University, Bowling Green, OH 43403, USA.
Fax: +1 419 372 2024

Jahn, Gary C., Cambodia–IRRI–Australia Project (CIAP), PO Box 1, Phnom Penh, Cambodia.
Email: G.JAHN@cgiar.org
Fax: +855 23 211728

Jäkel, Thomas, German Technical Cooperation (GTZ, Deutsche Gesellschaft für Technische Zusammenarbeit), 65726 Eschborn, Germany.
Email: tojack@uni-hohenheim.de

Jiang, Yongjin, Northwest Plateau Institute of Biology, Chinese Academy of Sciences, Xining 810001, P.R. China.
Fax: +86 971 6143282

Jumanta, Research Institute for Rice, West Java, Indonesia.
Email: sudarmaji@vision.net.id
Fax: +62 260 520157

Khoprasert, Yuvaluk, Agricultural Zoology Research Group, Division of Entomology and Zoology, Department of Agriculture, PO Box 9-34, Bangkok 10900, Thailand.
Email: agrizoo@bkk1.loxinfo.co.th
Fax: +66 2 9405396

Krebs, Charles, J. Department of Zoology, University of British Columbia, Vancouver, B.C. V6T 1Z4, Canada.
Email: krebs@zoology.ubc.ca
Fax: +1 604 822 2416

Lawson, Malcolm A., Pest Animal Control Cooperative Research Centre, Microbiology Department, University of Western Australia, QEII Medical Centre, Nedlands, WA 6009, Australia.
Email: mlawson@cyllene.uwa.edu.au
Fax: +61 8 9346 2912

Leirs, Herwig, Danish Pest Infestation Laboratory, Skovbrynet 14, DK-2800 Lyngby, Denmark.
Email: h.leirs@ssl.dk
Fax: +45 45 931155

Leung, Luke K.-P., CSIRO Wildlife and Ecology,
GPO Box 284, Canberra, ACT 2601, Australia.
Email: lukeleung@telstra.easymail.com.au

Macdonald, David W., Wildlife Conservation
Research Unit, Department of Zoology, University of
Oxford, South Parks Road, Oxford, OX1 3PT, UK.
Fax: +44 1865 310447

Mak, Solieng, Cambodia–IRRI–Australia Project
(CIAP), PO Box 1, Phnom Penh, Cambodia.
Email: msolieng@bigpond.com.kh
Fax: +855 23 211728

Makundi, Rhodes H., Rodent Research Unit,
Sokoine University of Agriculture, PO Box 3110,
Morogoro, Tanzania.
Email: rmakundi@suanet.ac.tz
Fax: +255 56 3748

Mathews, Fiona, Wildlife Conservation Research
Unit, Department of Zoology, University of Oxford,
South Parks Road, Oxford, OX1 3PT, UK.
Email: fmathews@ermine.ox.ac.uk
Fax: +44 1865 310447

Mills, James N., Special Pathogens Branch, Division
of Viral and Rickettsial Diseases, National Center for
Infectious Diseases, Centers for Disease Control and
Prevention (Mailstop G-14), 1600 Clifton Road,
Atlanta, Georgia 30333, USA.
Email: jum0@cdc.gov
Fax: +1 404 639 1118

Mwanjabe, Patrick S., Rodent Control Centre, PO
Box 3047, Morogoro, Tanzania.
Email: Rodent@suanet.ac.tz

Ning, Zhendong, Shanxi Institute of Plant
Protection, Chinese Academy of Agricultural
Science, Taiyuan, Shanxi 030031, P.R. China.
Fax: +86 0351 7127683

Nookarn, Piyanee, Agricultural Zoology Research
Group, Division of Entomology and Zoology,
Department of Agriculture, PO Box 9-34, Bangkok
10900, Thailand.
Email: agrizoo@bkk1.loxinfo.co.th
Fax: +66 2 405396

Oguge, Nicholas O., Department of Zoology,
Kenyatta University, PO Box 43844, Nairobi, Kenya.
Email: noguge1@excite.com *or*
noguge@africaOnline.co.ke

Pech, Roger P., CSIRO Wildlife and Ecology, GPO
Box 284, Canberra, ACT 2601, Australia.
Email: roger.pech@dwe.csiro.au
Fax: +61 2 6242 1505

Promkerd, Prasarttong, Agricultural Zoology
Research Group, Division of Entomology and
Zoology, Department of Agriculture, PO Box 9-34,
Bangkok 10900, Thailand.
Email: agrizoo@bkk1.loxinfo.co.th
Fax: +66 2 9405396

Rahmini, Research Institute for Rice, Departemen
Pertanian, Jl. Raya No. 9, Sukamandi, Subang 41256,
Indonesia.
Email: sudarmaji@vision.net.id
Fax: +62 260 520157

Rakotondravony, Daniel, Département de Biologie
Animale, Université d'Antananarivo, BP 906,
Antananarivo, Madagascar.

Salmon, Elizabeth, CSIRO Plant Industry, GPO Box
1600, Canberra, ACT 2601, Australia.
Email: Libby.Salmon@pi.csiro.au
Fax: +61 2 6246 5000

Schiller, John M., Lao–IRRI Project, PO Box 4195,
Vientiane, Lao PDR.
Email: j.m.schiller@cgiar.org
Fax: +856 21 414373

Sicard, Bruno, Laboratoire de Mammalogie, IRD-
ORSTOM (Institut de Recherche pour le
Développement, French Scientific Research Institute
for Development through Cooperation), BP 84,
Bamako, Mali.
Email: sicard@ird.ml
Fax: +223 227588

Singleton, Grant R., CSIRO Wildlife and Ecology,
GPO Box 284, Canberra, ACT 2601, Australia.
Email: grant.singleton@dwe.csiro.au
Fax: +61 2 6242 1505

Suasa-ard, Kornkaew, Agricultural Zoology Research Group, Division of Entomology and Zoology, Department of Agriculture, PO Box 9-34, Bangkok 10900, Thailand.
Email: agrizoo@bkk1.loxinfo.co.th
Fax: +66 2 9405396

Sudarmaji, Research Institute for Rice, Departemen Pertanian, Jl. Raya No. 9, Sukamandi, Subang 41256, Indonesia.
Email: sudarmaji@vision.net.id
Fax: +62 260520157

Tan, Tran Quang, National Institute for Plant Protection, Hanoi, Vietnam.
Email: tuat@hn.vnn.vn
Fax: +84 4 836 3563

van Wensveen, Monica, CSIRO Wildlife and Ecology, GPO Box 284, Canberra, ACT 2601, Australia.
Email: Monicavw@dwe.csiro.au
Fax: +61 2 6242 1505

Wan, Xinrong, Institute of Zoology, Chinese Academy of Sciences, Beijing 100080, P.R. China.
Email: xwan@public.east.cn.net
Fax: +86 10 62555689

Wang, Mengjun, Institute of Zoology, Chinese Academy of Sciences, Beijing 100080, P.R. China.
Fax: +86 10 62555689

Wang, Quanye, Northwest Plateau Institute of Biology, Chinese Academy of Sciences, Xining 810001, P.R. China.

Wei, Wanhong, Northwest Plateau Institute of Biology, Chinese Academy of Sciences, Xining 810001, P.R. China.
Fax: +86 971 6143282

Zhang, Zhibin, National Key Laboratory of Integrated Pest Management on Insects and Rodents, Institute of Zoology, Chinese Academy of Sciences, Zhongguancun Lu #19, Haidian District, Beijing 100080, People's Republic of China.
Email: zhangzb@panda.ioz.ac.cn
Fax: +86 10 62556418

Zhong, Wenqin, Institute of Zoology, Chinese Academy of Sciences, Beijing 100080, People's Republic of China.
Email: zhongwq@panda.ioz.ac.cn
Fax: +86 10 62555689

Zhou, Wenyang, Northwest Plateau Institute of Biology, Chinese Academy of Sciences, Xining 810001, P.R. China.
Fax: +86 971 6143282

Abbreviations

ACIAR	Australian Centre for International Agricultural Research
AHF	Argentine haemorrhagic fever
AZRG	Agricultural Zoology Research Group (Thailand)
CIAP	Cambodia–IRRI–Australia Project
CRS	Catholic Relief Service
CSIRO	Commonwealth Scientific and Industrial Research Organisation (Australia)
EBRM	ecologically-based rodent management
ECC	endogenous circadian clock
ECTV	ectromelia virus
EWS	early wet season (crop)
GMO	genetically modified organism
GTZ	Deutsche Gesellschaft für Technische Zusammenarbeit (German Technical Cooperation)
HFRS	haemorrhagic fever with renal syndrome
HPS	hantavirus pulmonary syndrome
IPM	integrated pest management
IRD-ORSTOM	Institut de Recherche pour le Développement; the French Scientific Research Institute for Development through Cooperation
IRRI	International Rice Research Institute
Lao PDR	Lao People's Democratic Republic
MCMV	murine cytomegalovirus
MIA	Murrumbidgee Irrigation Area (Australia)
PICA	Predict, Inform, Control, Assess (strategy)
RPM	rodent pest management
SNV	Sin Nombre virus
TBS	trap–barrier system
TBS+TC	TBS plus trap crop
TBW	total body water
VVIC	viral-vectored immunocontraception
ZP	zona pellucida (glycoproteins)

List of Species

Species name	Common Name
Acomys cahirinus	spiny mouse
Apodemus agrarius	striped field mouse
Apodemus flavicollis	yellow-necked field mouse
Apodemus sylvaticus	wood mouse; long-tailed field mouse
Arvicanthis niloticus	unstriped grass rat; Nile grass rat
Bandicota bengalensis	lesser bandicoot rat
Bandicota indica	large bandicoot rat
Bandicota savilei	
Bolomys obscurus	dark field mouse
Brachyuromys ramirohitra	
Calomys callosus	"laucha grande"
Calomys musculinus	corn mouse
Castor canadensis	North American beaver
Castor fiber	Eurasian beaver
Citellus dauricus	Daure ground squirrel
Clethrionomys glareolus	bank vole
Clethrionomys rufocanus	red backed vole
Cricetomys gambianus	African giant pouched rat
Cricetulus barabensis	striped hamster
Cricetulus longicaudatus	lesser long-tailed hamster
Cricetulus triton	rat-like hamster
Cynomys ludovicianus	plains prairie dog
Dipodomys panamintinus	Panamint kangaroo rat
Geomys bursarius	eastern American pocket gopher; plains pocket gopher
Gerbillus nigeriae	Nigerian gerbil
Hystrix cristata	crested porcupine
Hystrix indica	Indian crested porcupine
Liomys salvini	Salvin's spiny pocket mouse
Mastomys coucha	multimammate rat
Mastomys erythroleucus	multimammate rat
Mastomys huberti	multimammate rat
Mastomys natalensis	multimammate rat
Marmota himalayana	Himalayan marmot
Meriones unguiculatus	Mongolian gerbil; clawed jird
Micromys minutus	harvest mouse
Microtus brandti	Brandt's vole
Microtus californicus	California vole
Microtus fortis	oriental vole
Microtus mandarinus	brown vole
Microtus oeconomus	root vole
Microtus pennsylvanicus	meadow vole
Mus caroli	rice mouse
Mus cervicolor	ryukyu mouse
Mus domesticus	house mouse
Mus musculus	house mouse
Muscardinus avellanarius	dormouse
Myocastor coypus	coypu; nutria
Myospalax baileyi	plateau zokor
Myospalax fontanieri	Chinese zokor
Nesokia indica	short-tailed bandicoot rat
Nesomys rufus	
Notomys alexis	spinifex hopping mouse
Ochotona cansus	Gansu pika
Ochotona curzoniae	plateau pika; black-lipped pika
Ochotona daurica	Daurian pika

Species name	Common Name
Oligoryzomys longicaudatus	long-tailed pygmy rice rat
Ondatra zibethicus	muskrat
Onychomys spp.	grasshopper mice
Perognathus parvus	Great Basin pocket mouse
Peromyscus boylii	
Peromyscus maniculatus	deer mouse
Peromyscus truei	big eared cliff mouse; Pinyon mouse
Pitymys irene	
Pseudomys hermannsburgensis	sandy inland mouse
Rattus argentiventer	rice-field rat
Rattus bowersi	Bower's rat
Rattus colletti	dusky rat
Rattus exulans	Polynesian rat
Rattus flavipectus	buff breasted rat
Rattus germaini	
Rattus koratensis	Sladen's rat
Rattus losea	lesser rice-field rat
Rattus nitidus	Himalayan rat
Rattus norvegicus	Norway rat; brown rat
Rattus rattoides	Synonym for lesser rice-field rat and Turkestan rat
Rattus rattus	black rat; house rat; roof rat
Rattus rattus diardii	Malaysian house rat
Rattus tanezumi (formerly *Rattus rattus mindanensis)*	Philippine rice-field rat
Rattus tiomanicus	Malayan wood rat
Rattus villosissimus	long haired rat
Rhabdomys pumilio	
Sigmodon alstoni	Alston's cotton rat
Sigmodon hispidus	cotton rat; Hispid cotton rat
Solomys spp.	tree rats
Spalax ehrenbergi	blind mole-rat
Suncus murinus	common shrew
Tachyoryctes splendens	African mole rat
Tatera indica	
Taterillus gracilis	gerbil
Taterillus petteri	gerbil
Taterillus pygargus	gerbil
Thomomys bottae	western American pocket gopher; valley pocket gopher
Thomomys talpoides	northern pocket gopher
Thryonomys spp.	cane rat; cutting grass rat
Xerus erythropus	ground squirrel
Zygodontomys brevicauda	cane mouse

Preface

THE SEED FOR this book was sown in Morogoro, Tanzania, in 1996, following the strong ecological theme that emerged at an international workshop: Rodent Biology and Integrated Pest Management in Africa. Herwig Leirs and Grant Singleton were encouraged that the theme of ecologically-based rodent research came through strongly as the future direction for rodent management in developing countries in both Africa and Asia. The opportunity to germinate the seed arose in 1997 when Zhibin Zhang approached Grant Singleton and Lyn Hinds to co-convene an international conference on rodent biology and management. The focus would be broader than the Morogoro workshop and it was obvious that to augment the charm and appeal of Beijing in early autumn, an impressive line-up of international speakers would be required to attract participants to the conference. The Australian Centre for International Agricultural Research (ACIAR), the Chinese Academy of Sciences and the Commonwealth Scientific and Industrial Research Organisation (CSIRO) Wildlife and Ecology each pledged support for the conference. This led to a successful recruiting drive with all the speakers we approached accepting an invitation to present a paper at the First International Conference on Rodent Biology and Management held in Beijing in October 1998.

In January 1998, the editors approached ACIAR with the concept of a book on ecologically-based management that would bring together leading researchers of the basic biology of rodents and those charged with developing and implementing management strategies for rodent pests, especially in developing countries.

The book consists primarily of a selection of papers presented at the Beijing conference and comprises three sections. Section 1 sets the scene with contributions from leading small mammal biologists interested in theory and current paradigms of rodent biology and management. Section 2 covers state-of-the-art technologies of the different approaches to management of rodent pests—rodenticides, physical control, urban management and biological control. Section 3 describes regional case studies of rodent pest problems and progress with their management for a selection of developing countries in Asia and Africa.

Internationally, there have been two previous books of note on rodent pest management: one edited by Ishwar Prakash, published in 1988, the other edited by Alan Buckle and Robert Smith, published in 1994. Both provided a good mix of papers on the principles and practices of rodent pest management, and are compulsory reading for students and practitioners of rodent biology and management. Our book differs from these two books in providing a considerably stronger emphasis on (i) ecologically-based management, (ii) recent developments in innovative approaches to biological control, and (iii) the problems, progress and challenges of rodent pest management in developing countries. One important element missing in our book, and in the previous two books, is a substantial contribution on rodent management in Central and South America. We hope that this void is filled in the near future. In the interim, we hope that our book is of interest and practical value to researchers in that region of the world.

This has been a challenging project with more than half of the contributing authors not having English as their native language. We thank these authors for their perseverance in the face of obvious frustration in not being able to write in Bahasa, Cantonese, Flemish, French, Kiswahili, Lao, Mandarin, Thai, Vietnamese etc. We commend them for their responsiveness to our requests for many points of clarification and in keeping to a tight schedule.

All chapters were refereed by two people and then edited. We thank fellow authors for their contributions to the reviewing and editing process as well as David Spratt, Abigail Smith, Wang Zuwang, Lam Yuet Ming, David Freudenberger, Alison Mills, Christopher Hardy and Geoffrey Shellam. The support and enthusiasm of John Copland and Peter Lynch have ensured that the seed of an idea developed into a bountiful crop—a crop which it is hoped will be eyed despairingly by rodents in our ecologically-led quest to battle their impact on our lives.

Grant Singleton
Lyn Hinds
Herwig Leirs
Zhibin Zhang

March 1999

1. Ecologically-based Management of Rodent Pests—Re-evaluating Our Approach to an Old Problem

Grant R. Singleton, Herwig Leirs, Lyn A. Hinds and Zhibin Zhang

Abstract

Rodent pest management has gone through a period of stagnation mainly because there has been too little research effort to understand the biology, behaviour and habitat use of the species we are attempting to manage. There is a growing demand, particularly in developing countries, for rodent control strategies that either have less reliance on chemical rodenticides or can better target their use. Similar concerns exist with the control of insect and weed pests. This has led to the development of the concept of ecologically-based pest management (EBPM) which builds on the progress made with integrated pest management (IPM). We analyse this idea for rodent pests and provide examples where research on the basic biology and ecology of rodent pests has provided management strategies that are more sustainable and environmentally benign. The theme of ecologically-based rodent management (EBRM) was foremost in our minds when we invited people to contribute to this book. The other significant considerations were a focus on rodent pest management in developing countries and the importance of marrying basic and applied research on rodents. If in developing countries we can foster the importance of population ecology and an emphasis on management directed at the agro-ecosystem level, then we are confident that the next decade will see rapid advances in rodent pest management.

Keywords

Rodent management; IPM; rodent ecology; ecologically-based rodent management

INTRODUCTION

THE GENESIS of this book was a common concern on the lack of progress in rodent pest management over the past 20 years in both developing countries and elsewhere. This has occurred despite the advent in the 1970s of sophisticated chemical rodenticides and effective strategies for their use (see Buckle 1988; Buckle and Smith 1994).

We contend that rodent pest management has gone through a period of stagnation for four primary reasons. First, there has been too great an emphasis on how to develop, use, compare and market rodenticides, with particular attention on commensal rodents in industrialised countries. In developing countries, on the other hand, the lack of a critical approach to the use of rodenticides for particular species has in some instances led to an unreasonable aversion to rodenticide use. Second, the development of rodent control strategies generally has been based on short-term experiments where immediate declines in rodent numbers were seen as a success, without much consideration of long-term consequences or ecosystem effects. Third, field studies have rarely progressed beyond alpha-level, descriptive population studies (see Krebs, Chapter 2). Fourth, the recommended management protocols have been too prescriptive. They rarely take into account the particular characteristics of the pest species or of the socioeconomic constraints of the end-users of the technology.

What has been lacking is a solid understanding of the biology, behaviour and habitat use of the respective species we are attempting to manage. Armed with such knowledge we will be able to focus on disentangling the major factors that limit the growth of pest populations. This requires experimental field studies conducted at an appropriate scale and for an appropriate length of time. Recently there has been some progress in the assessment of rodent management methods using replicated, manipulative field studies based on our understanding of the ecology of the pest species (e.g. Singleton and Chambers 1996; Brown et al. 1998; White et al. 1998; Fan et al., Chapter 13), but there is still much to be done.

In the interim, there has been a marked attrition in the number of wildlife researchers working on rodent pests. Ishwar Prakash (1988) noted this trend in his introduction to the pioneering book *Rodent Pest Management*.

> It is also felt that this work ... will trigger more research effort for the benefit of mankind, ... (which) it appears has dampened during the last few years.

Unfortunately, his plea did not arrest this trend.

Since 1993 there has been encouraging evidence of an increase in the number of young wildlife researchers interested in the biology and management of rodent pests in developing countries. This has been due primarily to funding support provided by the Australian Centre for International Agricultural Research in Southeast Asia, the European Union, Belgium and Denmark in eastern Africa and ORSTOM (French Scientific Research Institute for Development through Cooperation) in Western Africa. We are pleased that some of these researchers have been able to contribute to this book.

China, through necessity, also has seen a marked increase in research effort on rodent pests. Rodent problems increased in severity in the 1980s resulting in rodent control being listed as one of the top three priorities for the national plant protection program in 1985. Since 1985, rodent control has been listed in three successive national five-year-plans (1985–1990; 1991–1995; 1996–2000). There are now approximately 100 scientists with the Chinese Academy of Sciences, Ministry of Agriculture and universities working on rodent control. Many of these are young scientists, who received their degree in biology or post-graduate qualifications in the 1990s.

In this opening chapter we will set the scene with a brief overview of the magnitude of the impact of rodent pests, the concept of ecologically-based management and the aims and structure of the book.

RODENT PESTS — STILL A PROBLEM

The quest to control the depredations of rodents, especially in agricultural systems, has been ongoing for thousands of years. Aristotle (384–322 BC) recounts

> The rate of propagation of field mice in country places, and the destruction that they cause, are beyond all telling.

Although the last 50 years in particular have provided good progress with rodent pest management, rural people in many countries still rank rodents in the top three of their most important pests. Of particular concern are the losses caused in developing countries where rodents are literally competing with humans for food.

A meeting on rodent pest management in Southeast Asia was held in early 1998 at the International Rice Research Institute (IRRI) in the Philippines. Reports of present-day rodent problems were presented for Australia, Cambodia, East Africa, Indonesia, Lao People's Democratic Republic (PDR), Malaysia, Philippines, Thailand and Vietnam; the accounts were impressive in their extent and impact. Rodent problems ranged from eruptive populations of mice in south-eastern Australia and rats in the uplands of Lao PDR, to the chronic problems that occur annually in the rice fields of most Southeast Asian countries.

There were two telling commentaries from the meeting in the Philippines, which place in context the impact of rodent pests in developing countries. One reported that although rodents were not the most important pre-harvest pest to Laotian farmers, they were the pest they felt they had the least control over. The other presented losses caused by rodents in Cambodia not in monetary terms but in how much rice could have been available for annual human consumption if not for rat depredations. If we apply this line of reasoning to Indonesia where rats cause annual pre-harvest losses of approximately 17%, then rats consume enough rice annually to feed more than 25 million Indonesians for a year. In countries such as Indonesia, rice provides 50–60% of the daily energy requirements for people.

In some cases, the 'official' national level of annual pre-harvest losses caused by rodents is not high. For example, 3–5% losses are reported in Malaysia (Singleton and Petch 1994) and 1–3% in the Philippines (Sumangil 1990; Wilma Cuaterno, April 1998, pers. comm.). However, when detailed damage assessment is conducted, the damage caused by rats generally is more severe. For example, Buckle (1994b) reported a conservative loss estimate of 7.3% in the entire Penang State of Malaysia. Also, both in the Philippines and Malaysia, the patchy nature of rodent damage often results in farmers losing more than 60% of their crop, which means that rodents are still a significant national problem (Lam 1990). In other places, rodent damage may vary widely with limited damage in most years, and the most extreme losses of more than 80% of the harvest in outbreak years (e.g. Boonaphol and Schiller 1996). In countries that live at the brink of subsistence, such figures are a constant threat to food security.

This book contains detailed accounts of the magnitude and importance of the impact of rodent pests, particularly in agricultural systems. This information in itself is important because it provides a spotlight on rodent problems that generally have a lower profile than insect, weed and disease impacts on agricultural crops. The latter group of problems has a higher profile for two reasons. One is that, in developing countries, there are many entomologists, botanists and plant pathologists who are able to identify, quantify and sell the need for research, education, extension and action in their respective fields. In comparison, there are few rodent biologists; most of these have an entomological training and there is a poor infrastructure for research on rodent pests.

The second reason is that farmers have a stronger identity with rodents than other pests. Rodents are perceived as 'intelligent' pests, which learn to counter whichever control measures farmers use. Over the centuries, farmers have learned to accept the depredations caused by rats. A common response is,

> for every eight rows of rice we sow for our family, we sow two for the rats.

Unfortunately, with the increasing human population and the shortage of food in developing countries, this level of loss can no longer be tolerated.

Clearly, rodents are still an important problem, and this is without consideration of the losses they cause post-harvest, and the role they play as reservoirs for debilitating diseases of humans and their livestock.

IPM, RODENTICIDES AND ECOLOGICALLY-BASED MANAGEMENT

Integrated pest management (IPM) is simply the integration of a range of management practices that together provide more effective management of a pest species than if they are used separately. IPM was developed with the aim of promoting methods for managing insect pests and plant diseases that were least disruptive to the ecology of agricultural systems (Smith and van den Bosch 1967).

Ecologically-based pest management

In 1996, a review of pest management of insects and weeds by the Board on Agriculture of the National Research Council (NRC) of the United States of America, highlighted that the practice of IPM has generally not been consistent with the underlying philosophy of IPM. They contend that there has been too much focus on pest scouting and precise application of pesticides. They argue that there is a need to refocus objectives from pest control to pest management and this requires greater emphasis on ecological research and a systems approach (National Research Council 1996). This extension and refocusing of the ecological aspects of IPM led the NRC to develop a concept termed 'ecologically-based pest management' (EBPM). The fundamental goals of EBPM are threefold. One is to minimise adverse effects on non-target species and the environment. The second is to develop an approach that is economic for end-users, particularly farmers, in both the developed and developing world. The third is to establish an approach that is durable.

The development of IPM for rodents has followed a similar path to IPM for insects. The primary foci have been the development of simple monitoring systems to decide whether or not to instigate a baiting campaign, and the development of effective patterns of use for particular rodenticides. Generally, the focus in rodent control has been mostly to achieve a visible increase in mortality, without appropriate attention to other demographic processes or ecological compensation mechanisms. There have been attempts to develop rodent IPM based on an understanding of the habitat use and population dynamics of rodent pests (see Wood and Liau 1984 a,b; Redhead and Singleton 1989; Whisson 1996; Brown et al. 1998; White et al. 1998) or the use of biological control (e.g. Lenton 1980; Singleton and Chambers 1996), but with the possible exception of *Rattus tiomanicus* in oil palm plantations (Wood and Liau 1984a,b), these have not been adopted successfully over a large area. The progress of rodent IPM in Southeast Asia and Australia has been reviewed by Singleton (1997).

Also, biological control needs to be viewed in the context of ecologically-based management of pests because often it is limited in its specificity and efficacy. This is supported by a review of one of the success stories of biological control, the weevil — *Cyrtobagous salviniae*, for controlling the floating fern salvinia (*Salvinia molesta*). Following its establishment in South Asia in 1939, salvinia was spread by man to Southeast Asia and Australasia. It severely disrupts the lives of people by forming dense mats a metre thick, choking slow moving waterways, rice fields and lakes (see Thomas and Room 1986 for details). Efforts to develop biological control were thwarted initially because the fern was incorrectly identified, resulting in the testing of the wrong herbivores. In 1978, salvinia was found in Brazil where it is relatively rare. Field studies identified three potential herbivores and one of these, *C. salviniae*, was released into a lake in northern Queensland and destroyed 30,000 t of salvinia within a year (Room et al. 1981).

When tested in other waterways the weevil was not a success. Subsequently, a combination of ecological and laboratory studies revealed that, if the level of nitrogen was too low in the fern, the weevil population declined. Nitrogen was added to waterways which increased the weevil population, until it eventually reached a critical density at which the damage it caused to the plant resulted in a sufficient increase in nitrogen in the plant itself for the weevil population to be self-sustaining (Room 1990). This was an unexpected result because higher levels of nitrogen generally make weed problems worse. The salvinia story highlights how taxonomic and ecological research provided a strong basis for a successful systems approach for pest management.

Ecologically-based rodent management

For rodents, an ecological basis for control was suggested many years ago (Hansson and Nilsson 1975; see also Redhead and Singleton 1988) but the implementation of those early ideas has been largely overlooked. One success was the eradication of coypu (*Myocastor coypus*), an introduced rodent pest, in Britain in the 1980s. After several decades of unsuccessful control, a new strategy was developed based on a long-term population dynamics study and biological simulations. A complete solution of the problem was obtained in less than six years through integrating knowledge about the animal's biology and behaviour with a well-organised control scheme with attractive incentives for trappers (Gosling and Baker 1989). There are other good examples in the rodent literature which illustrate the importance of ecological, taxonomic and behavioural studies for developing effective strategies for managing rodent pests. We provide some further examples later in this chapter, with more detailed case studies provided in the ensuing chapters (Macdonald et al., Chapter 3; Leung et al., Chapter 14).

The advantages of viewing biological control of rodents as part of an integrated ecologically-based approach to rodent management rather than a single panacea for control has been reviewed by Singleton and Brown (1999). For simplicity, we propose that this strategy be termed 'ecologically-based rodent management' (EBRM). The contributions by Pech et al. (Chapter 4) and Hinds et al. (Chapter 10) further portray the advantage of having a strong ecological understanding of the biology of both the rodent pest and the disease agent when developing techniques for biological control. In this instance, the focus is on developing fertility control of house mice. Without a multi-disciplinary approach, the requisite knowledge of reproductive biology, social behaviour patterns and population dynamics of the wild house mouse could not be consolidated to allow full development of a product which can then be tested for efficacy.

Rodenticide-based control strategies have a clear need for a good biological basis to build upon. Toxicity of active ingredients and bait palatability are obvious factors which have been studied under laboratory conditions for many decades (see e.g. Buckle 1994a; Johnson and Prescott 1994). Less common, but equally important, is a proper understanding of how poisons can be delivered. For example, rodenticides in Hawaiian macadamia orchards were commonly distributed by broadcasting on the ground. Recently, population and behavioural studies of the black rat, *Rattus rattus*, revealed that those rats which damage the nuts forage only in the trees. This information led to placement of bait stations in trees leading to more efficient use of rodenticides for controlling damage (Tobin et al. 1997).

In China, chemical rodenticides, mostly anticoagulants, are still the routine weapons for controlling rodents in farmland and grassland. However, such rodent control campaigns in the absence of a sound ecological knowledge of the pest species have generally only achieved short periods (6–9 months) of respite from the ravages of the rodents. In the rice fields of southern China the effects have been even shorter (Huang and Feng 1998). Indeed, many studies (Liang 1982; Liang et al. 1984; Zhang 1996; Huang and Feng 1998; Qi et al. 1998) have shown that the response of rodent populations after chemical control is non-linear. Killing some individuals may reduce the population numbers initially, but the remaining animals compensate with better survival and better breeding performance. For example, following an 88% reduction in a population of the Mongolian gerbil (*Meriones unguiculatus*), the body mass at first pregnancy was reduced from 58 g to 35–50 g (Wang et al. 1998).

In Malaysia, populations of the Malayan wood rat (*R. tiomanicus*) also showed a rapid population response after control, with a full recovery in population density occurring over 12–18 months. In this case, knowledge of the population dynamics and factors limiting population growth resulted in an effective management program of rats in oil palm plantations. Management consisted of an intensive baiting campaign followed by recurrent placement of baits every six months (see Wood and Liau 1984a).

Re-invasion is another factor resulting in populations returning quickly to pre-control densities (e.g. Guruprasad 1992). This is particularly a problem in developing countries where farmers often manage their own rodent problems on small plots of land (0.25–2 ha) at different times to their neighbours. The land use patterns on these small holdings also generally result in a patchy landscape. We therefore need ecological studies to examine the relative demographic importance of each patch and the timing and rates of movements by rats between patches (Singleton and Petch 1994). This metapopulation approach to rodent control is achieving more attention (see Smith 1994), but appropriate field studies of the spatial dynamics of rodent populations in agro-ecosystems in developing countries (e.g. Leirs et al. 1997b) are few.

Ethology in rodent pest management

The development of resistance by rodent pest species to first and second generation anticoagulants explicitly necessitated an integrated approach to rodent management, where use of one poison type was complemented or alternated with the use of other poison types, physical control methods, exclusion, or other control measures (Greaves 1994). Here again, more attention was paid to short-term, and indeed often urgently needed, quick solutions like changing to a stronger poison. Much less effort has been directed towards preventing the development of, or containing the geographical distribution of, resistance. So-called 'behavioural resistance', where rodents refuse to eat the poisonous baits, poses other challenges. In the Birmingham restaurant area, house mice were impossible to control until detailed studies revealed that they had difficulties in digesting starch and were therefore unlikely to eat grain-based baits; changing to fish baits solved the problem quickly (Humphries et al. 1996).

The Chinese zokor (*Myospalax fontanieri*) provides another practical example of the importance of understanding rodent behaviour in developing effective management. In the farmland of Northwest Loess Plateau, the zokor, which lives underground, shows a cautious response to chemical baits. Less than 70% of a zokor population can be killed by using the best possible baiting technique for this species: setting baits in their underground tunnels (Zou et al. 1998). Further improvement in this kill rate depends on a better understanding of the behavioural aspects of feeding for this species, particularly in overcoming its neophobic response to baits (Zhang and Wan 1997) or perhaps whether they show social learning of food preferences (see Galef 1994; Berdoy 1994 for reviews).

A good ecological basis to management strategies can help to provide excellent rodent damage control without interfering with rodent demography. Wood mice (*Apodemus sylvaticus*) in Germany can be lured away from sugar beet seeds during the short period after sowing when they are prone to rodent damage by providing an attractive, unpoisoned alternative food in the periphery of the fields (Pelz 1989). As all the above examples show, however, solutions are often specific and require a detailed knowledge of the biology, ecology and behaviour of the pest species. Obtaining such knowledge is a laborious yet rewarding task that will allow the development of new damage control strategies.

Further examples of the benefits of combining knowledge of the ecology and ethology of rodent species for developing better integrated control are provided by Santini (1994) for three European species of rodents in agriculture and forestry, and Buckle et al. (1997) for the Malayan wood rat in oil palm plantations.

RE-EMERGENCE OF POPULATION ECOLOGY OF RODENT PESTS

The current book builds on the strong ecological theme that emerged at an international workshop on rodent biology and integrated pest management in Africa, held in Morogoro, Tanzania, in 1996 (for published proceedings see Belgian Journal of Zoology Volume 127, Supplement). Africa is an economically poor continent and control strategies which rely primarily on rodenticides are unrealistic. This has sparked interest in a more integrated ecological approach to rodent pest management. One of the conclusions of the workshop was, however, that such strategies cannot materialise without the availability of population data from long-term studies (more than three years) (Leirs 1997). In West Africa, much information was collected by Hubert and co-workers in the 1970s (e.g. Hubert 1982), while in East Africa it is only in the past few years that long-term ecological studies have begun to provide insights into the main factors driving rodent population dynamics (Leirs et al. 1996, 1997a). Building on these insights, the focus has now switched to experimental field studies.

The workshop in Morogoro formulated recommendations, many of which are relevant to the present book (Leirs 1997a). The key recommendations are as follows:

- The taxonomy of many pest rodents must be clarified so that control actions can target the correct species.
- Life-history studies and physiological comparisons between these species are imperative.

- Experimental ecological studies, properly designed with appropriate controls, must be set up to evaluate management strategies and, in the first place, test our hypotheses (or, rather, unsubstantiated beliefs) about rodent population dynamics.
- Poisons in this framework are not considered as something to avoid, but as only one of the possible approaches which should be used more effectively and integrated with other approaches.

The development of the concept of EBPM is important, because it builds on the solid foundations developed by IPM. In effect, EBPM is refocusing IPM towards understanding the population biology of the pest and the agro-ecosystem in which it lives. From the viewpoint of a population ecologist, one wonders what all the fuss is about; EBPM is self-evident. However, when one moves into applied wildlife management, especially of rodents, then the need to sell a concept such as ecologically-based management of rodent pests becomes a reality (Singleton and Brown 1999). Unfortunately, too often there is a divide between practitioners, who are more concerned with the details of how to apply specific control technologies, and wildlife researchers who focus on understanding the theory and the context of the problem (Sinclair 1991). We have provided a mix of pure (Section 1 and parts of 2) and applied (Sections 2 and 3) rodent biology in this book in an attempt to bridge this divide.

AIMS AND STRUCTURE OF THE BOOK

This book has four broad aims:

- to raise the profile of the importance of basic research for developing effective, applied management of rodent pests;
- to argue the need for an ecologically-based approach to rodent pest management;
- to raise the profile of rodent pest management in developing countries; and
- to spark interest in prospective students in a challenging but rewarding field of endeavour.

The book begins with a section on theory and current paradigms of rodent biology and management.

This section includes contributions from leading small mammal ecologists. Krebs (Chapter 2) provides a thought-provoking paper on the different phases of small mammal ecology and concomitant shifts in research paradigms. Macdonald and coworkers (Chapter 3) present the results of a series of novel studies used to disentangle the interesting social behaviour of Norway rats. Dickman (Chapter 5) examines, at the ecosystem level, the positive role rodents play as 'ecosystem engineers' through their impact on the chemical and structural attributes of the environment. Mills in his chapter on arenaviruses and hantaviruses (Chapter 6), and Pech and his coworkers through their synthesis of models for predicting mouse plagues in Australia (Chapter 4), both provide a different perspective of the need for strongly focused population studies of rodents.

One common theme is addressed by all authors—the importance of basic research for developing effective management of rodents.

The second section covers broad methods of management—rodenticides, physical control and biological control. This section provides overviews on the state-of-the-art technologies for fertility control (Chambers et al., Chapter 10), chemical control (Buckle, Chapter 7) and the control of rodent pests in urban environments (Colvin and Jackson, Chapter 11). Reviews are provided also on physical methods of control, particularly in rice agro-ecosystems in developing countries (Singleton and coworkers, Chapter 8) and on the ecological management of Brandt's vole in the grassland of Inner Mongolia (Zhong and coworkers, Chapter 9). The common theme for this section is ecologically-based pest management.

In a conscious effort to ensure the book is relevant to developing countries, regional case studies of rodent problems and the progress with associated research are provided for Asia and Africa in Section 3. This section has contributions from selected countries edited by G.R. Singleton and Z. Zhang (Asia—contributions from Cambodia, China, Indonesia, Lao PDR, Thailand and Vietnam) and H. Leirs (Africa—contributions from Burkina Faso, Kenya, Madagascar, Mali and Tanzania). The information on the biology and management of rodent pests in developing countries, and the infrastructure for research and extension, varies considerably. In some countries, such as Cambodia and Lao PDR, the problem is only just being defined and it is still not known which species cause the major problems in the different agro-ecosystems (see contribution by Schiller et al., Chapter 18). The contributions in this section comprise a mix of biological studies aimed directly at management, and general overviews of rodent problems and how they are currently being managed in various developing countries.

In seeking contributions for this book we were heartened by the enthusiasm that it generated from researchers across the spectrum of pure and applied research. We received no 'knock backs' from contributors we targeted. Indeed, we had to limit the contributions that were on offer. What pleasantly surprised us was the strong interest by 'pure' scientists in hoping their work would not only be of heuristic value. They were keen for their findings to be accessible to researchers in developing countries because they felt their research could make a significant contribution to tackling the problem of rodent pests in these regions. So perhaps Denis Chitty is indeed correct in stating "pure and applied science differ mainly in aims, not methods". If this book acts as a catalyst for pure and applied scientists to work together towards a common aim of reducing the impact of rodent pests in agricultural ecosystems of developed and developing countries, then we will be more than satisfied with our toil.

REFERENCES

Berdoy, M. 1994. Making decisions in the wild: constraints, conflicts and communication in foraging rats. In: Galef, B.G., Mainardi, M. and Valsecchi, P., ed., Behavioural aspects of feeding: basic and applied research in mammals. Switzerland, Harwood Academic Publishers, 289–313.

Boonaphol, O. and Schiller, J.M. 1996. Rodent problems and research priorities in Laos. Paper for ACIAR Project Planning Meeting on Management of Rodent Pests in Southeast Asia, 10–12 January 1996, UPM Malaysia, 14 p.

Brown, P.R., Singleton, G.R., Dunn, S.C. and Jones, D.A. 1998. The management of house mice in agricultural landscapes using farm managment practices: an Australian perspective. In: Baker, R.O. and Crabb, A.C., ed., Proceedings of the 18th Vertebrate Pest Conference, Costa Mesa, California, USA, 2–5 March 1998. Davis, University of California, 156–159.

Buckle, A.P. 1988. Integrated management of rice rats in Indonesia. FAO (Food and Agriculture Organization of the United Nations) Plant Protection Bulletin, 36, 111–118.

Buckle, A.P. 1994a. Rodent control methods: chemical. In: Buckle, A.P. and Smith, R.H., ed., Rodent pests and their control. Wallingford, UK, CAB International, 127–160.

Buckle, A.P. 1994b. Damage assessment and damage surveys. In: Buckle, A.P. and Smith, R.H., ed., Rodent pests and their control. Wallingford, UK, CAB International, 219–248.

Buckle, A.P. and Smith, R.H. (ed.) 1994. Rodent pests and their control. Wallingford, UK, CAB International, 405p.

Buckle, A.P., Chia, T.H., Fenn, M.G.P. and Visvalingam, M. 1997. Ranging behaviour and habitat utilisation of the Malayan wood rat (*Rattus tiomanicus*) in an oil palm plantation in Johore, Malaysia. Crop Protection, 16, 467–473.

Chitty, D. 1996. Do lemmings commit suicide? Beautiful hypotheses and ugly facts. New York, Oxford University Press, 268p.

Galef, B.G. Jr 1994. Olfactory communication about foods among rats: a review of recent findings. In Galef, B.G., Mainardi, M. and Valsecchi, P., ed., Behavioural aspects of feeding: basic and applied research in mammals. Switzerland, Harwood Academic Publishers, 83–101.

Greaves, J.H. 1994. Resistance to anticoagulant rodenticides. In: Buckle, A.P. and Smith, R.H., ed., Rodent pests and their control. Wallingford, UK, CAB International, 197–217.

Gosling, L.M. and Baker, S.J. 1989. The eradication of muskrats and coypu from Britain. Biological Journal of the Linnean Society, 38, 39–51.

Guruprasad, B.K. 1992. Reinfestation of *Bandicota bengalensis* (Gray) in irrigated field habitat. In: Borrecco, J.E. and Marsh, R.E., ed., Proceedings of 15th Vertebrate Pest Conference, 3–5 March 1992, Newport Beach, California. Davis, University of California, 277–279.

Hansson, L. and Nilsson, B. (eds) 1975. Biocontrol of rodents. Ecological bulletins, No 19, Swedish Natural Science Research Council. 306p.

Huang, X.Q. and Feng, Z.Y. 1998. Ecology and management strategies for *Rattus losea*. In: Zhibin Zhang and Zuwang Wang, ed., Ecology and management strategies of rodent pests in agriculture. Beijing, China Ocean Press, 178–194 (in Chinese).

Hubert, B. 1982. Dynamique des populations de deux espèces de rongeurs du Sénégal, *Mastomys erythroleucus* et *Taterillus gracilis* (Rodentia, Muridae et Gerbillidae). I. Etude démographique. Mammalia, 46, 137–166.

Humphries, R.E., Sibly, R.M. and Meehan, A.P. 1996. The characteristics of 'behavioural resistance' and bait avoidance in house mice in the UK. Proceedings of Brighton Crop Protection Conference—Pests and Diseases, 157–164.

Johnson, R.A. and Prescott, C.V. 1994. The laboratory evaluation of rodenticides. In: Buckle, A.P. and Smith, R.H., ed., Rodent pests and their control. Wallingford, UK, CAB International, 161–179.

Lam, Y.M. 1990. Cultural control of rice field rats. In: Quick, G.R., ed., Rodents and rice. Report and proceedings of an expert panel meeting on rice rodent control, 10–14 Sept. 1990. Los Baños, Philippines, IRRI (International Rice Research Institute), 65–72.

Leirs, H. 1997. Preface. Rodent biology and integrated pest management in Africa. Proceedings of the International Workshop held in Morogoro, Tanzania, 21–25 October 1996. Belgian Journal of Zoology, 127(suppl.), 3–5.

Leirs, H., Stenseth, N.C., Nichols, J.D., Hines, J.E., Verhagen, R. and Verheyen, W.N. 1997a. Seasonality and non-linear density-dependence in the dynamics of African *Mastomys* rats. Nature, 389, 176–180.

Leirs,H., Verhagen, R., Sabuni, C.A., Mwanjabe, P. and Verheyen,W.N. 1997b. Spatial dynamics of *Mastomys natalensis* in a field–fallow mosaic in Tanzania. Belgian Journal of Zoology, 127(suppl.), 29–38.

Leirs, H., Verhagen, R., Verheyen, W., Mwanjabe, P. and Mbise, T. 1996. Forecasting rodent outbreaks in Africa: an ecological basis for *Mastomys* control in Tanzania. Journal of Applied Ecology, 33, 937–943.

Lenton, G.M. 1980. Biological control of rats by owls in oil palm and other plantations. Biotrop Special Publication No. 12, 87–94.

Liang, J.R. 1982. Population recovery of Chinese zokor (*Myospalax fontanieri*) and plateau pika (*Ochotona curzoniae*). In: Wuping Xia, ed., Alpine ecosystem. Gansu, Gansu People's Publishing Press, 93–100 (in Chinese).

Liang, J.R., Zhou, L., Wang, Z.W. and Song, R.Y. 1984. Mathematical model of population recovery of Chinese Zokor (*Myospalax fontanieri*) and plateau pika (*Ochotona curzoniae*). Acta Ecologica Sinica, 4, 1–11 (in Chinese).

National Research Council 1996. Ecologically based pest management: new solutions for a new century. Washington, D.C., National Academy Press, 144p.

Pelz, H.-J. 1989. Ecological aspects of sugar beet seeds by *Apodemus sylvaticus*. In: Putman, R.J., ed., Mammals as pests. London, Chapman and Hall, 34–48.

Prakash, I. (ed.) 1988. Rodent pest management. Boca Raton, CRC Press, 480p.

Qi, G.X., Yao, W.L., Wang, J. and Yang, B. 1998. Research on population dynamics and control strategies for rodents in cities and towns of southern China. Acta Therio-logica Sinica, 18, 226–230 (in Chinese).

Redhead, T.D. and Singleton, G.R. 1988. The PICA Strategy for the prevention of losses caused by plagues of *Mus domesticus* in rural Australia. EPPO (European Plant Protection Organization) Bulletin, 18, 237–248.

Room, P.M. 1990. Ecology of a simple plant–herbivore system: biological control of *Salvinia*. Trends in Ecology and Evolution, 5, 74–79.

Room, P.M., Harley, K.L., Forno, I.W. and Sands D.P.A. 1981. Successful biological control of the floating weed salvinia. Nature, 294, 79–80.

Santini, L. 1994. Knowledge of feeding strategies as the basis for integrated control of wild rodents in agriculture and forestry. In: Galef, B.G., Mainardi, M. and Valsecchi, P., ed., Behavioural aspects of feeding: basic and applied research in mammals. Switzerland, Harwood Academic Publishers, 357–368.

Sinclair, A.R.E. 1991. Science and the practice of wildlife management. Journal of Wildlife Management, 55, 767–773.

Singleton, G.R. 1997. Integrated management of rodents: a Southeast Asian and Australian perspective. Belgian Journal of Zoology, 127, 157–169.

Singleton, G.R. and Brown, P.R. 1999. Management of mouse plagues in Australia: integration of population ecology, bio-control and best farm practice. In: Cowan, D.P. and Feare, C.J., ed., Advances in vertebrate pest management. Fürth, Filander – Verlag, 189–203.

Singleton, G.R. and Chambers, L.K. 1996. A large scale manipulative field study of the effect of *Capillaria hepatica* on wild mouse populations in southern Australia. International Journal for Parasitology, 26, 383–98.

Singleton, G.R. and Petch, D.A. 1994. A review of the biology and management of rodent pests in Southeast Asia. Canberra, ACIAR Technical Reports No. 30, 65p.

Smith, R.H. 1994. Rodent control methods: non-chemical and non-lethal chemical. In: Buckle, A.P. and Smith, R.H., ed., Rodent pests and their control. Wallingford, UK, CAB International, 109–125.

Smith, R.F. and van den Bosch, R. 1967. Integrated control. In: Kilgore, W.W and Doutt, R.T., ed., Pest control: biological, physical, and selected chemical methods. New York, Academic Press, 295–340.

Sumangil, J.P. 1990. Control of ricefield rats in the Philippines. In: Quick, G.R., ed., Rodents and rice. Report and proceedings of an expert panel meeting on rice rodent control, 10–14 Sept. 1990. Los Baños, Philippines, IRRI (International Rice Research Institute), 35–47.

Thomas, P.A. and Room, P.M. 1986. Taxonomy and control of *Salvinia molesta*. Nature, 320, 581–584.

Tobin, M. E., Sugihara, R.T. and Koehler, A.E. 1997. Bait placement and acceptance by rats in macadamia orchards. Crop Protection, 16, 507–510.

Wang, M.J., Zhong, W.Q. and Wan, X.R. 1998. Ecology and management strategies for Mongolia gerbils (*Meriones unguiculatus*). In: Zhibin Zhang and Zuwang Wang, ed., Ecology and management strategies of rodent pests in agriculture. Beijing, China Ocean Press, 225–240 (in Chinese).

White, J., Horskins, K. and Wilson, J. 1998. The control of rodent damage in Australian macadamia orchards by manipulation of adjacent non-crop habitats. Crop Protection, 17, 353–357.

Whisson, D. 1996. The effect of two agricultural techniques on populations of the canefield rat (*Rattus sordidus*) in sugarcane crops of north Queensland. Wildlife Research, 23, 589–604.

Wood, B.J. and Liau, S.S. 1984a. A long term study of *Rattus tiomanicus* populations in an oil palm plantation in Johore, Malaysia. II. Recovery from control and economic aspects. Journal of Applied Ecology, 21, 465–472.

Wood, B.J. and Liau, S.S. 1984b, A long term study of *Rattus tiomanicus* populations in an oil palm plantation in Johore, Malaysia. III. Bionomics and natural regulation. Journal of Applied Ecology, 21, 473–495.

Zhang, Z.B. 1996. Techniques and strategies for rodent control by contraception. In: Zuwang Wang and Zhibin Zhang, ed., Theory and practice of rodent pest management. Beijing, Science Press, 367–368.

Zhang, Z.B. and Wan, Y.L. 1997. Ten years' review on rodent pest management in China. In: Nou, D.S, Lou, Z.P., Wan, Y.L. and Tang, H.Y., ed., Research on agricultural biology and sustainable development of agriculture. Beijing, Science Press, 146–151 (in Chinese).

Zou, B., Ning, Z.D., Wang, T.L. and Chang, W.Y. 1998. Ecology and management strategies of Chinese zokor (*Myospalax fontanieri*). In: Zhibin Zhang and Zuwang Wang, ed., Ecology and management strategies of rodent pests in agriculture. Beijing, China Ocean Press, 41–64 (in Chinese).

Section 1

Basic Research — the Foundation for Sound Management

Pure and applied science differ mainly in aims, not methods

Chitty 1996

2.

Current Paradigms of Rodent Population Dynamics — What Are We Missing?

Charles J. Krebs

Abstract

Rodent population studies have played a key role in developing our understanding of population dynamics. The proximal stimulus to this understanding is to alleviate problems of rodent pests in agriculture and disease transmission to humans.

Ideas about rodent population dynamics have gone through three phases. In the 1930s there were almost no quantitative data, and population control was believed to be caused by biotic agents that operated in a density-dependent manner. By the 1950s a new paradigm of social control of numbers emerged with emphasis on physiological stress and social aggression within populations. By the 1970s a synthesis of sorts had emerged suggesting that multiple factors caused population changes. Experimental manipulation of field populations in the 1960s enlarged our outlook on the complexities of rodent populations, and the emergence of modelling and rigorous statistical analyses of survival and reproduction in the 1980s and 1990s has shown again that rodents have been the *Drosophila* of population ecology. But as precision has increased over time, generality and simplicity have declined to near extinction.

What is missing and what do we need to do in the next 20 years? Experimentation is the key to understanding, and no study should be undertaken without a clear set of experimental predictions. The era of alpha-level descriptive population studies should be over. We need large-scale, extensive studies coupled with short-term experimental studies. Rodents are good candidates for studies of spatial dynamics, a strongly emerging subdiscipline in ecology. Also, rodent management should focus on the factors limiting populations and use an experimental approach. The era of pest eradication via killing alone should be over and we need to be smarter in developing our management options. The development of genetic resistance to anticoagulants and chemical poisons is a call to the ecologists of the 21st century to think more clearly about how we might outwit rodent pests. The accumulated knowledge of the physiology, behaviour, and genetics of rodents needs to be integrated into our management options. There is much to be done both to understand and to outsmart these clever mammals.

Keywords

Population regulation, population limitation, food, predation, social behaviour, rodents, pest management

INTRODUCTION

POPULATION DYNAMICS is without question the most highly developed of the subdisciplines of ecology. From abstract mathematical models to field experiments, ecologists have made progress over the last 50 years in analysing population changes in many species. In particular, rodents have been model organisms for studies of population dynamics for three reasons. First, they are conveniently short-lived so that a scientist or a postgraduate student can accomplish something within the constraints of a 3–4 year time window. Second, they are ubiquitous, occur in abundance nearly everywhere, and are relatively cheap to study, and are often of economic importance (Singleton et al., Chapter 1). Third, they do interesting things such as have population outbreaks that occur frequently enough that even politicians think that something must be done about them, at least when they are superabundant. All these features have combined to produce a very large literature on rodent population dynamics that is somewhat overwhelming to the novice. It is important therefore to step back and ask what we have accomplished with these studies, how useful it has been for pest control, and what is to be done next. This book brings together ecologists, physiologists, and ethologists with a common interest in rodent biology and thus provides an ideal time to address these larger issues for rodents.

After a historical overview I will summarise the three current paradigms of rodent population dynamics, assess their strengths and weaknesses, and suggest some paths for future growth.

WHAT ARE THE PROBLEMS?

Ecological questions are complex and one thing we have learned is to ask very specific questions about populations so that we can answer them clearly. Three major questions have formed the focus of population dynamics (Krebs 1994, p. 322; Krebs 1995):

- What stops population growth?
- What limits average abundance?
- What constrains geographical distributions?

To find out what stops population growth, we must compare a growing population to one that is not growing, and the usual approach is to look for some factors causing negative feedback in the form of density dependence. The second question is very broad and is answered by the use of the comparative approach in which a high-density population is compared with a low-density population to see what factors are associated with the observed differences in density. In both these cases an experimental approach is useful to answering the question most quickly and avoiding spurious correlations (Underwood 1997).

Most academic rodent ecologists have addressed the first question—the problem of regulation (Berryman 1986; Sinclair 1989), and this has engendered much discussion about density dependence in natural populations. Fewer ecologists have worked on the second question—limitation of numbers, and yet this is the critical question for pest management. In a simple world, the same ecological factors would limit and regulate a population, but this has never been found in the real world. Limitation often comes from habitat factors that students of regulation seldom consider, as we shall see. In a sense these two aspects of population dynamics correspond to the two statistical concepts of the mean and the variance of a set of measurements. We shall be repeating history to complain, as do many statisticians, that scientists are often preoccupied with the mean and tend to forget about the variance.

The question of what constrains geographic distributions has fallen out of favour until fairly recently when the consequences of global warming on north–south geographical distribution boundaries became a hot topic of worry. It is an important issue that I cannot deal with here, and there has been much discussion of the consequences of these biological invasions (Ehrlich 1989; Ruesink et al. 1995; Vitousek et al. 1996).

HISTORICAL OVERVIEW

Population dynamics has gone through three phases during the last 75 years. They have overlapped little in time but have phased into one another, with an abundance of outliers of the 'flat-earth' society type that bedevils ecology in general.

Phase I

The first phase began with the debate in the 1920s and 1930s about the role of biotic and abiotic factors in population regulation. The champions were A.J. Nicholson (1933) for the biotic school and a variety of opponents for the abiotic school (e.g. Thompson 1929; Uvarov 1931). The winners were the Nicholsonians with their focus on regulation via density-dependent processes, in which the main agents were predators, parasites, diseases, and food shortage. The habitat was nowhere to be seen, and weather was noise for population dynamics. Most of this early discussion was about insect populations, and rodents were not a part of the discussions. This was an age of data-free ecology, and the arguments were typically theoretical in the bad sense of this word with no experiments on natural populations available. I have referred to the Nicholsonian world-view as the density-dependent paradigm (Krebs 1995).

It is important to remember that from the start all ecologists implicitly believed that a population can be identified, that community interactions are all direct and easily definable, and that population processes are repeatable in space and in time. These are three gigantic leaps of faith that came back later to challenge simplistic models.

Phase II

The second phase of population dynamics began in the 1940s when ecologists began to realise that social processes could affect births, deaths and movements. Among the leaders of this phase were David E. Davis and John Christian in the United States and Dennis Chitty in England (Christian 1950; Chitty 1952; Davis 1987). Rodents were the key to this new phase, which built partly on the earlier recognition by some ornithologists that territoriality could regulate the breeding density of some bird species. Attention turned in this phase to studying the physiological and behavioural impacts of individuals on one another. One of the early striking experiments was done on rats in Baltimore by Davis and Christian (1956, 1958) who showed that one could reduce the population of rats in a city block by *adding* rats to the population (Figure 1), a completely counterintuitive result for the 1950s. Social strife for breeding space in rodents became a hot topic, and John Calhoun suggested crowded mice and rats as potential models for people in cities (Calhoun 1949). Much of this early work was done on house mice and rats in enclosures, and one of the dominant themes of criticism was that these enclosures were very high density, artificial environments and of little relevance to what went on in natural populations.

Social regulation of population size arose as an alternative explanation of population changes in populations that did not seem to be regulated by the conventional Nicholsonian predators, parasites, or food shortages (Chitty 1960). These studies interfaced well with emerging work in ethology and behavioural ecology, which indicated the complex social structure of many mammal populations, and the interest population geneticists began to show in the dynamics of natural populations (Ford 1975). There was, among many ecologists, considerable scepticism that social processes, in contrast to the extrinsic factors of predators, food supplies and parasites, might explain changes in numbers. A series of elegant experiments on bird populations (e.g. Watson and Moss 1970; Moss and Watson 1980) helped to convince some sceptics, and parallel work on rodents (e.g. Krebs et al. 1969; Tamarin and Krebs 1969; Gaines and Krebs 1971) strongly supported the concept of social limitation of population density.

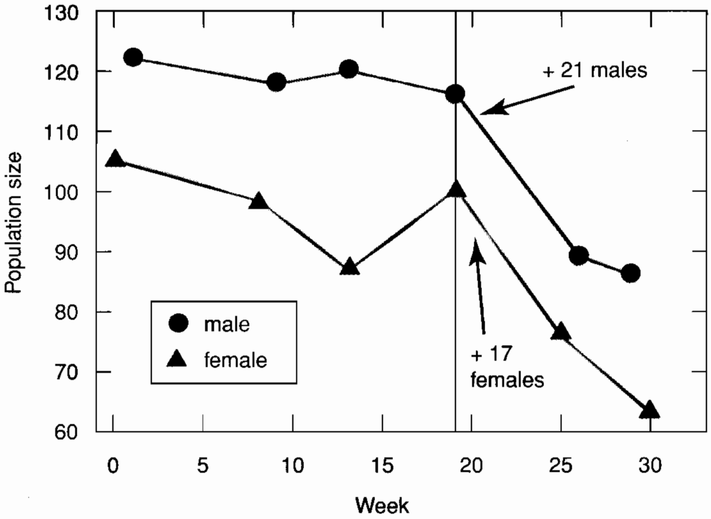


Figure 1.
Introduction experiments of Norway rats (*Rattus norvegicus*) into two city blocks in Baltimore in 1954. Adding rats to a stationary population did not increase numbers but caused them to drop (after Davis and Christian 1956).

Phase III

By 1970 nearly all the ideas about population regulation and limitation were on the table for consideration and a synthesis began by suggesting that everyone might be correct, that multiple factors could be involved in both regulation and limitation (Lidicker 1973, 1988). Two developments accompanied this phase of population studies. First, experimental testing of hypotheses in field situations became the norm in ecology. Second, mathematical models began to be applied to specific questions about rodent systems in order to explore assumptions with rigour (e.g. Stenseth 1978, 1981b). The question then became how to articulate multiple factor hypotheses within the paradigm of experimental ecology. All ecologists are happy to conclude that the world is a complex multivariate system, but almost all agree that we must abstract from this complexity to some order to make progress.

Many multiple-factor hypotheses suffer from three deficiencies. Excessive complexity is the first lethal deficiency. A good example occurs with many flow chart models of population processes. Batzli (1992), for example, lists 22 hypotheses for rodent population cycles and gives a complex flow chart to illustrate some of the interrelationships involved. Limited predictability is a second problem with multiple-factor hypotheses. It does us no service to tell managers that we cannot predict anything about their potential pest problems because the world is complex. Third, many multiple-factor hypotheses are impossible to test experimentally. Without an experimental approach rodent ecology will make little progress.

The solution to these problems is fairly straightforward. We should encourage multifactor models of limited complexity, quantitative predictability, and feasible experimental tests. Note that there are two distinct types of multi-factor models of population limitation.

Several independent factors limit average abundance

The key point in this alternative is that the several factors that affect abundance are independent in a statistical sense. In practice this means hypothetically that if you change factor A and double numbers, and change factor B and triple numbers, you expect that if you change both factor A and factor B at the same time you will change numbers by the simple multiple (2 × 3) or 6 times.

Several interacting factors limit average abundance

This is the most complex alternative hypothesis since it postulates a statistical interaction between some factors. In practice you would recognise an interactive explanation by the fact that changing factor A and factor B at the same time does not result in their joint effect being predictable. In the above example, changing factor A and factor B might change numbers much less than 6 times, or much more than 6 times. If this hypothesis applies to your rodent population, interest centres on exactly how the ecological interaction of factor A and factor B operates mechanistically.

A straw poll among rodent ecologists would probably find most of them supporting multi-factor hypotheses of regulation and limitation. If this turns out to be the most frequent model for rodents, it raises the multifactor dilemma that it is difficult to deal with more than three factors in any realistic model. There are two possible solutions to this dilemma. First, we can hope that all factors operate independently (hypothesis 1 above), so that if we have four or five significant factors for a particular herbivore, the factors do not interact. Second, we can hope that for systems with interactions only two or at most three factors show interactive effects (hypothesis 2).

The recent history of rodent population studies has been a history of reduced generality, increased precision, and decreased simplicity. Philosophers would be appalled at this, but ecologists should be happy to see us move away from superficial generality and simplicity. The touchstone of our progress must be the management of rodent pests, and we must try to answer this important question:

> how much have our ivory tower studies of rodents in the laboratory and in the field helped us to solve problems of rodent pests?

THREE CURRENT PARADIGMS

There are three current paradigms that represent the dominant focus of work today on small rodent populations.

The food paradigm

The food paradigm states that both the quantity and the quality of food supplies regulate rodent population density. Food supplies also limit the average density of populations, and outbreaks of rodents are caused by changes in their food supplies. The most important thing you need to know, under this paradigm, is what do your rodents eat and how much of it is out there in their habitat. These are themselves complex issues since diets change seasonally and may be affected by an individual's sex and age and also by changes in plant productivity from year to year and season to season. A test of the food paradigm is done most easily by supplementing food supplies artificially, although these experiments themselves can be called into question if the food given is not adequate nutritionally.

The food paradigm cannot be tested as a unit and needs to be applied to specific cases to make predictions that can be falsified. For example, the average abundance of a rodent pest might be higher where more food is available. Ecologists often pyramid hypotheses about food supplies. A recent example is the hypothesis about Lyme disease in eastern United States of America (Ostfeld 1997; Jones et al. 1998): food supplies in the form of acorns from oak trees are postulated to limit the average abundance of deer mice (*Peromyscus maniculatus*), trigger outbreaks of these mice (when acorn crops are heavy), and regulate density through starvation. Boutin (1990) concluded in his review of feeding experiments that, by adding food to terrestrial herbivore populations, one could increase density two to three-fold but not more, so that clearly for some populations food limits density over some restricted range only. Ecologists tend to despair when their favourite explanation does not apply to all species in all situations. We should be more modest in our aims. Food is clearly one of the dominant ecological factors limiting and regulating rodent populations, and the question is which populations and under exactly what conditions.

The predator paradigm

Many things eat rodents and some ecologists look to these trophic links to explain regulation and limitation of populations. The predator paradigm states that mortality caused by predation regulates rodent populations, that generalist predators limit the average density of populations below the limits that might be set by food supplies, and that outbreaks of rodents are caused by predator control activities, artificial or natural. The most important thing you need to know, under this paradigm, is who eats whom in your community. Since this can vary seasonally, and predators are often selective for sex and age groups, obtaining this information with quantitative rigour is not easy.

Paul Errington presented the most serious challenge to the predator paradigm more than 50 years ago by suggesting that predators consumed only the doomed surplus from rodent populations (Errington 1946). This question has been restated more recently as the question of whether predation mortality is *additive* or *compensatory* (e.g. Bartmann et al. 1992). Errington suggested that it was often compensatory. This question can be answered directly by removing predators or indirectly by showing what fraction of mortality is due to predation kills. There are considerable problems with inferring predation limitation from predator kills alone. If territoriality causes dispersal movements, or parasites cause debilitation, or food shortage causes poor condition, predators may be the executioners rather than the primary cause of population changes (Murray et al. 1997).

The usual argument against predation as a regulating factor has been that rodents have such a high rate of reproduction, that it is impossible for predators to kill enough of them (e.g. Chitty 1938, 1996; but see Korpimäki and Norrdahl 1998). It is certainly correct that sufficient numerical and functional responses must be present for predation to be a potential regulator of rodent populations (Hanski and Korpimäki 1995). From a practical viewpoint the key is to manipulate predator numbers. For example, to see if they could reduce crop damage by house mice in Australia, Kay et al. (1994) provided perches in agricultural crops for raptors. The important point is not to be convinced that predators are limiting or regulating numbers just because predators kill many rodents. It is convenient politically to show lots of dead rodents to our political masters, but scientifically dubious to infer from these piles of dead bodies that predators are helping to alleviate pest problems.

The social paradigm

The social structure of a rodent population can affect its ecology. The social paradigm states that social interactions between individuals can lead to changes in physiology and behaviour that reduce births, and increase deaths, and thereby regulate populations. In particular, territoriality may limit the average density of rodent populations. Outbreaks of rodents are postulated in this paradigm to be caused by changes in the social environment (e.g. Krebs et al. 1995). The social paradigm is the least popular of the three paradigms under which population ecologists operate. This is usually because ecologists assume that the social environment is primarily determined by habitat which is highly correlated with food supplies. Thus, for example, food supplies determine territory size and territory size limits population density. The problem is that other factors may influence social behaviour as well, and thus the linkage of habitat to social processes can be very loose.

Practical problems of rat and mouse control had highlighted already by the 1940s that killing of rats and mice often did not result in control, especially when the pests were at high density (Chitty 1954, p. 6; Elton 1954). Achieving controls in rat populations has typically involved intensive large-scale campaigns of killing rats either directly or by poisoning (see Singleton et al., Chapter 8). Only recently has the possibility of using other methods of control like parasites (Singleton and McCallum 1990) or immunocontraception (Caughley et al. 1992; Chambers et al. 1997) been able to be explored.

The social paradigm has highlighted the role of immigration in local population dynamics. Removal experiments on rodents and other small mammals have illustrated the difficulties of controlling rodents by increasing mortality. Figure 2 illustrates one of the first experimental field removal studies on voles. In spite of very high and continuous mortality imposed by removals, the vole population continued to maintain high density and grow via immigration. Sullivan and Sullivan (1986) obtained a similar result for snowshoe hares. After a series of laboratory and field studies it became clear to ecologists that pest species with high turnover (high reproduction, high mortality, short generation times) are most sensitive to reductions in fecundity rather than increases in mortality rates (Figure 3) (Stenseth 1981a; Lebreton and Clobert 1991).

The fence effect (Krebs et al. 1969) is one example of an experimental result that was completely unanticipated by the food or the predator paradigms (Krebs 1996). If fencing a vole population without altering the food supply or the predator fauna could produce a 3–4-fold increase in population density, what role are immigration and emigration playing in population regulation? Lidicker (1962) had raised this question long ago but few rodent workers have responded to analyse this phenomenon (Ostfeld 1994). Unfortunately if you are interested in pest control you do not wish to find a procedure that will increase rodent density! My point is that surprise results that are unexpected under the conventional wisdom can result from ignoring social processes in rodent populations.

I do not wish to argue the merits of the social paradigm here. The important point for those interested in pest control is whether or not it suggests any kinds of manipulations that could reduce pest numbers. To date the major contribution of the social paradigm to rodent pest management has been to show that dispersal and social structure can render useless simple forms of pest control via mortality (e.g. Sullivan and Sullivan 1986).

OPTIMAL POPULATION STUDIES

Given these three paradigms, what ought we to be doing in rodent population studies? We can start by asking what an ideal world of population data would look like. It would have four components.

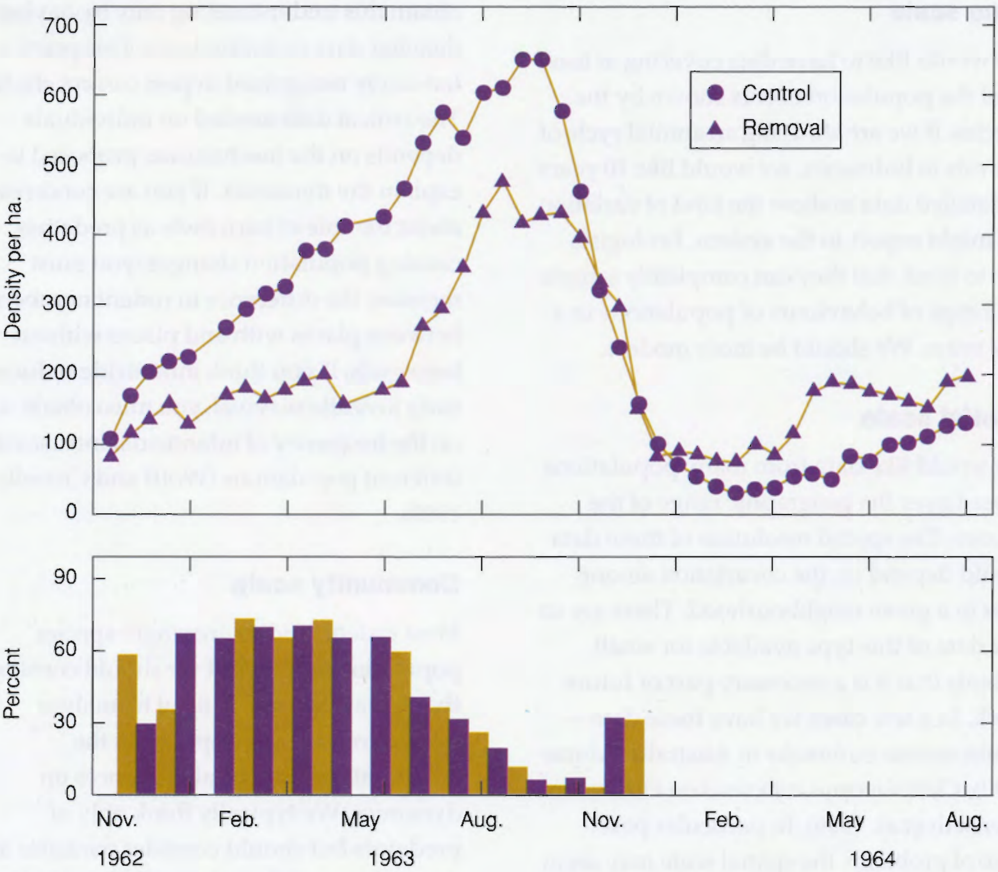


Figure 2.

A removal experiment on the California vole (*Microtus californicus*). All adult voles were removed every two weeks from the removal area of 0.8 ha. From November 1962 to July 1963 an average of 62% of the population was removed every two weeks with little impact on population growth because of immigration (after Krebs 1966).

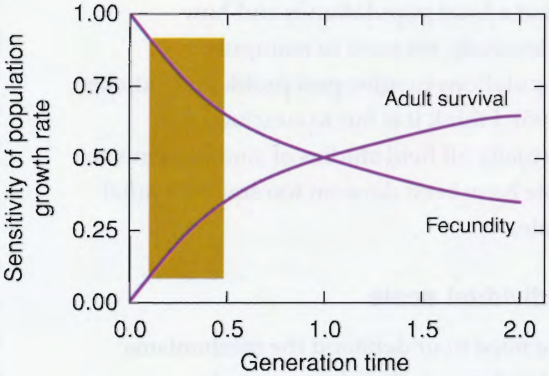


Figure 3.

Relative sensitivity of the population growth rate to survival after weaning and to fecundity for mammal populations. The shaded area is the zone occupied by many rodent pests (modified after Lebreton and Clobert 1991).

Time scale

We would like to have data covering at least 10 of the population events shown by the species. If we are studying an annual cycle of rice rats in Indonesia, we would like 10 years of detailed data to show the kind of variation we might expect in the system. Ecologists like to think that they can completely sample the range of behaviours of populations in a few years. We should be more modest.

Spatial scale

We would like data from many populations spread over the geographic range of the species. The spatial resolution of these data would depend on the covariation among sites in a given neighbourhood. There are so few data of this type available for small rodents that it is a necessary part of future work. In a few cases we have these data—house mouse outbreaks in Australia (Mutze 1990), *Clethrionomys rufocanus* on Hokkaido (Stenseth et al. 1996). In particular pest control problems the spatial scale may seem to be irrelevant, but it is not if we remember that the local spatial scale can also be critical (Stenseth 1981a). The concern about dispersal and population structure has focused attention on the need to find out what a local population is and how extensively we need to manipulate populations to solve pest problems (Lidicker 1995). I think it is fair to conclude that virtually all field studies of small rodents to date have been done on too small a spatial scale.

Individual scale

We need to understand the mechanisms behind population changes, and we can obtain this understanding only by having detailed data on individuals. This point is too rarely recognised in pest control studies. The critical data needed on individuals depends on the mechanisms proposed to explain the dynamics. If you are concerned about the role of barn owls as predators causing population changes, you must measure the difference in rodent numbers between places with and places without barn owls. If you think infanticide reduces early juvenile survival, you must obtain data on the frequency of infanticidal intrusions in different populations (Wolff and Cicirello 1989).

Community scale

Most rodent studies are single-species population studies but we should consider that it may be more fruitful to analyse interactions between species in the community as potential influences on dynamics. We typically think only of predators but should consider parasites and diseases as well (Saitoh and Takahashi 1998). In most pest rodent studies, competition for resources between species is presumed to be minimal and single-species interactions are paramount so that these community interactions can be ignored. Generalist predators are perhaps the most common factor operating on small mammals in which community interactions, including indirect effects (Menge 1997), need to be considered.

WHAT DO WE HAVE ALREADY?

Given this ideal world, we should take stock of what we have already accomplished and then move on to what we are lacking. Three strengths stand out.

- Population ecologists are fortunate in having a set of good quantitative methods for dealing with the arithmetic of population change. From the Leslie matrix to metapopulation models, there is quantitative rigour in abundance. The importance of this is not always appreciated by population ecologists, yet it is one of the great intellectual achievements of this century. We can use this arithmetic to balance the books. If we know the birth rates and death rates of a population (as well as immigration and emigration) we can compute exactly the rate of population increase or decrease. We need to use this more often to check on our estimates of these parameters (e.g. Haydon et al. 1999). For many rodent pests, control through increasing mortality is the only option available. For these cases quantitative demographic models can estimate the mortality required to reduce a population a specified amount in order to plan an optimal control program.
- Second, we have a set of good paradigms for analysing population regulation and limitation. I have outlined these above, and others can be articulated. The importance of being able to articulate clear, testable hypotheses is underappreciated in ecology (Platt 1964; Underwood 1997). Prediction, absolutely essential for scientific respectability, is almost unknown in population ecology (Peters 1991).
- Third, we have good field methods for estimating population parameters to feed into quantitative models and into statistical analysis of our experiments. Population estimation methods have been extensively improved (Pollock et al. 1990), elegant methods for analysing survival rates are available (Lebreton et al. 1993), and statistical methods for analysing reproductive changes and separating immigration from births are being developed (Nichols and Pollock 1990; Nichols et al. 1994). We have the demographic tools to understand rodent populations with a level of precision that was not available 25 years ago.

WHAT ARE WE LACKING?

I address here six problems that I think are central to future studies on rodent populations. They are not in any particular order of importance, since some are more relevant than others to particular situations.

Good methods for spatial dynamics

One of the contributions of the social paradigm to rodent population dynamics has been the stress on the importance of immigration and emigration for understanding population changes. But we still lack good methods for studying the spatial aspects of populations. Radio-telemetry has made it possible to get some data on individual movements, but we are rarely able to do it on a scale that would be sufficient to get a broad picture of landscape dynamics. We know too little about how we should structure our studies of spatial processes. Should we have many small trapping grids or a few very large grids? How large an area should we attempt to study? What fraction of movements that we can document are genetically effective (i.e. the immigrant individual survives, breeds and leaves offspring rather than dies after immigrating)? We have much to learn about just how to study spatial dynamics successfully in rodents, yet spatial processes underlie all of the problems of pest management. If we can reduce rats on one rice farm, will the neighbouring farms be affected or not? Much empirical work needs to be done on these questions. We can model pest populations as metapopulations in space but if we do not know the linkage parameters for these populations, our models will not be very useful.

Long-term experiments on limitation

There are no long term experiments on population limitation in any rodent species. If we feed a population for two years, we often get a population increase (Boutin 1990). What happens if we continue this experiment for 10 years? Is the system in equilibrium after two years so that we will learn nothing more from the longer study? There are numerous examples in ecology of short-term effects that were not sustained or even were reversed in the longer term (Norby et al. 1992; Wilsey 1996). There are also many examples from pest control in which initial encouraging results were followed by failures (DDT resistance, anticoagulants). The message is to be cautious about long-term conclusions.

Good interplay of models and field studies

Many ecologists have lamented the lack of interaction between field ecologists and modellers (e.g. Kareiva 1989). There are signs that this is finally breaking down (Stenseth and Saitoh 1998) but I think it is a failure on both sides that holds back progress. Models can help us to explore the logical consequences of assumptions we make in field experiments, and provide a quantitative estimation of the anticipated effect sizes. I think it is particularly important that rodent pest control studies incorporate both adequate controls and modelling studies as part of their overall approach.

Methods for evaluating weather-driven hypotheses

Climatic change is the wave of the future and we should be more concerned that our understanding of rodent systems will be transient and modified by weather changes. Hypotheses about weather-driven events are difficult to test. Post-hoc correlational studies are useful but inconclusive. They test more the cleverness of the statistician than the reality of the biological cause. We need to state weather hypotheses clearly so they can be tested next year, not last year, and we need to abide by the simple rules of experimental falsification when our predictions fail. Ad hoc explanations are available by the shipload for ecological systems, and we should not get in the habit of using them to bail out our failures of understanding. The exact mechanisms by which weather acts on populations need to be determined, since we need to know whether births or deaths are driving the change.

Economic and environmental analyses of pest control alternatives

This is not my area of specialty but I would like to think that we should aim in pest control work to achieve the best gain for the least cost—both environmental and economic. If we cannot achieve this, e.g. because the lowest economic cost method produces the highest environmental damage, we need to state this clearly so that the public can make an informed decision about alternatives.

Strategies for analysing the pest community of crops

In viewing rodent pests as single-species populations we overlook the broader strategy of looking at the whole community of pests of a particular crop. If the pests are truly independent, we can work on them one by one. But community interactions have ways of producing surprises via indirect effects (Holt 1987; Menge 1995), and we should be preemptive in looking for these possibilities.

CONCLUSION

The ivory tower of basic research studies on rodents has contributed little to the practical successes of rodent pest management, either short or long term. Much more insight has flowed in the opposite direction, and our understanding of rodent dynamics has been greatly improved by the practical studies of rodent pest control. What basic ecology can contribute to pest management is in the methods of study needed. The need for clear hypotheses, rigorous experimental tests based on good knowledge of natural history, a sceptical view of existing ideas, and the need to measure our successes and failures—all of these features of good science should be part and parcel of rodent management.

The major deficiencies of rodent population studies as we move into the new millennium are three. We need to apply the insights of theoretical ecology, behavioural ecology, physiology, and genetics to rodent pest problems. A promising start in this direction is immunocontraception, (Chambers et al. 1997; Chambers et al., Chapter 10). We need studies of tropical species in varied tropical environments, since much of our knowledge of rodent ecology comes from the Temperate Zone (c.f. Leirs et al. 1996). Finally, we need more studies of parasites and diseases in field populations. Conventional wisdom suggests that they are of little impact on highly fecund rodents, but their potential for biological control is largely untested (c.f. Singleton and McCallum 1990). There are many experiments waiting to be done and much promising modelling ahead with the goal of understanding population processes in rodents and at the same time alleviating the suffering caused by rodent pests around the world.

REFERENCES

Bartmann, R.M., White, G.C. and Carpenter, L.H. 1992. Compensatory mortality in a Colorado mule deer population. Wildlife Monographs, 121, 1–39.

Batzli, G.O. 1992. Dynamics of small mammal populations: a review. In: McCullough, D.R. and Barrett, R.H., ed., Wildlife 2001: populations. New York, Elsevier Applied Science, 831–850.

Berryman, A.A. 1986. Stabilization or regulation: what it all means! Oecologia, 86, 140–143.

Boutin, S. 1990. Food supplementation experiments with terrestrial vertebrates: patterns, problems, and the future. Canadian Journal of Zoology, 68, 203–220.

Calhoun, J.B. 1949. A method for self-control of population growth among mammals living in the wild. Science, 109, 333–335.

Caughley, G., Pech, R. and Grice, D. 1992. Effect of fertility control on a population's productivity. Wildlife Research, 19, 623–627.

Chambers, L., Singleton, G. and Hood, G. 1997. Immunocontraception as a potential control method of wild rodent populations. Belgian Journal of Zoology, 127, 145–156.

Chitty, D. 1938. A laboratory study of pellet formation in the short-eared owl (*Asio flammeus*). Proceedings of the Zoological Society of London Series A, 108, 267–287.

Chitty, D. 1952. Mortality among voles (*Microtus agrestis*) at Lake Vyrnwy, Montgomeryshire in 1936–9. Philosophical Transactions Royal Society of London, 236, 505–552.

Chitty, D. 1954. Control of rats and mice, vol. 1. Rats. Oxford, Clarendon Press, 305p.

Chitty, D. 1960. Population processes in the vole and their relevance to general theory. Canadian Journal of Zoology, 38, 99–113.

Chitty, D. 1996. Do lemmings commit suicide? Beautiful hypotheses and ugly facts. New York, Oxford University Press.

Christian, J.J. 1950. The adreno-pituitary system and population cycles in mammals. Journal of Mammalogy, 31, 247–259.

Davis, D.E. 1987. Early behavioral research on populations. American Zoologist, 27, 825–837.

Davis, D.E. and Christian, J.J. 1956. Changes in Norway rat populations induced by the introduction of rats. Journal of Wildlife Management, 20, 378–383.

Davis, D.E. and Christian, J.J. 1958. Population consequences of a sustained yield program for Norway rats. Ecology, 39, 217–222.

Ehrlich, P.R. 1989. Attributes of invaders and the invading processes: vertebrates. In: Drake, J.A., ed., Biological invasions: a global perspective. New York, John Wiley and Sons, 315–328.

Elton, C. 1954. Research on rodent control by the Bureau of Animal Population September 1939 to July 1947. In: Chitty, D., ed., Control of rats and mice, vol. 1. Rats. Oxford, Clarendon Press, 1–24.

Errington, P.L. 1946. Predation and vertebrate populations. Quarterly Review of Biology, 21, 144–177, 221–245.

Ford, E.B. 1975. Ecological genetics, 4th edition. London, Chapman and Hall.

Gaines, M.S. and Krebs, C.J. 1971. Genetic changes in fluctuating vole populations. Evolution, 25, 702–723.

Hanski, I. and Korpimäki, E. 1995. Microtine rodent dynamics in northern Europe: parameterized models for the predator–prey interation. Ecology, 76, 840–850.

Haydon, D.T., Gillis, E.A. and Krebs, C.J. 1999. Biases in the estimation of the demographic parameters of a snowshoe hare population. Journal of Animal Ecology, 68, 501–512.

Holt, R.D. 1987. Population dynamics and evolutionary processes: the manifold roles of habitat selection. Evolutionary Ecology, 1, 331–347.

Jones, C.G., Ostfeld, R.S., Richard, M.P., Schauber, E.M. and Wolff, J.O. 1998. Chain reactions linking acorns to gypsy moth outbreaks and Lyme disease risk. Science, 279, 1023–1026.

Kareiva, P. 1989. Renewing the dialogue between theory and experiments in population ecology. In: Roughgarden, J., May, R.M. and Levin, S.A., ed., Perspectives in ecological theory. New Jersey, Princeton University Press, 68–88.

Kay, B.J., Twigg, L.E., Korn, T.J. and Nicol, H.I. 1994. The use of artificial perches to increase predation on house mice (*Mus domesticus*) by raptors. Wildlife Research, 21, 95–106.

Korpimäki, E. and Norrdahl, K. 1998. Experimental reduction of predators reverses the crash phase of small-rodent cycles. Ecology, 79, 2448–2455.

Krebs, C.J. 1966. Demographic changes in fluctuating populations of *Microtus californicus*. Ecological Monographs, 36, 239–273.

Krebs, C.J. 1994. Ecology: the experimental analysis of distribution and abundance. New York, Harper Collins.

Krebs, C.J. 1995. Two paradigms of population regulation. Wildlife Research, 22, 1–10.

Krebs, C.J. 1996. Population cycles revisited. Journal of Mammalogy, 77, 8–24.

Krebs, C.J., Chitty, D., Singleton, G.R. and Boonstra, R. 1995. Can changes in social behaviour help to explain house mouse plagues in Australia? Oikos, 73, 429–434.

Krebs, C.J., Keller, B.L. and Tamarin, R.H. 1969. *Microtus* population biology: demographic changes in fluctuating populations of *M. ochrogaster* and *M. pennsylvanicus* in southern Indiana. Ecology, 50, 587–607.

Lebreton, J.D., Pradel, R. and Clobert, J. 1993. The statistical analysis of survival in animal populations. Trends in Ecology and Evolution, 8, 91–94.

Lebreton, J.-D. and Clobert, J. 1991. Bird population dynamics, management, and conservation: the role of mathematical modelling. In: Perrins, C.M., Lebreton, J.-D. and Hirons, G., ed., Bird population studies: relevance to conservation and management. Oxford, Oxford University Press, 105–125.

Leirs, H., Verhagen, R., Verheyen, W., Mwanjabe, P. and Mbise, T. 1996. Forecasting rodent outbreaks in Africa: an ecological basis for *Mastomys* control in Tanzania. Journal of Applied Ecology, 33, 937–943.

Lidicker, W.A.J. 1988. Solving the enigma of microtine "cycles". Journal of Mammalogy, 69, 225–235.

Lidicker, W.Z.J. 1962. Emigration as a possible mechanism permitting the regulation of population density below carrying capacity. American Naturalist, 96, 29–33.

Lidicker, W.Z.J. 1973. Regulation of numbers in an island population of the California vole, a problem in community dynamics. Ecological Monographs, 43, 271–302.

Lidicker, W.Z.J. 1995. The landscape concept: something old, something new. In: Lidicker, W.Z.J., ed., Landscape approaches in mammalian ecology and conservation. Minneapolis, University of Minnesota Press, 3–19.

Menge, B.A. 1995. Indirect effects in marine rocky intertidal interaction webs: patterns and importance. Ecological Monographs, 65, 21–74.

Menge, B.A. 1997. Detection of direct versus indirect effects: were experiments long enough? American Naturalist, 149, 801–823.

Moss, R. and Watson, A. 1980. Inherent changes in the aggressive behaviour of a fluctuating red grouse *Lagopus lagopus scoticus* population. Ardea, 68, 113–119.

Murray, D.L., Cary, J.R. and Keith, L.B. 1997. Interactive effects of sublethal nematodes and nutritional status on snowshoe hare vulnerability to predation. Journal of Animal Ecology, 66, 250–264.

Mutze, G.J. 1990. Mouse plagues in South Australian cereal-growing areas. I. Occurrence and distribution of plagues. Australian Wildlife Research, 16, 677–683.

Nichols, J.D., Hines, J.E., Pollock, K.H., Hinz, R.L. and Link, W.A. 1994. Estimating breeding proportions and testing hypotheses about costs of reproduction with capture–recapture data. Ecology, 75, 2052–2065.

Nichols, J.D. and Pollock, K.H. 1990. Estimation of recruitment from immigration versus in situ reproduction using Pollock's robust design. Ecology, 71, 21–27.

Nicholson, A.J. 1933. The balance of animal populations. Journal of Animal Ecology, 2, 132–178.

Norby, R.J., Gunderson, C.A., Wullschleger, S.D., O'Neill, E.G. and McCracken, M.K. 1992. Productivity and compensatory responses of yellow-poplar trees in elevated CO_2. Nature, 357, 322–324.

Ostfeld, R.S. 1994. The fence effect reconsidered. Oikos, 70, 340–348.

Ostfeld, R.S. 1997. The ecology of Lyme-disease risk. American Scientist, 85, 338–346.

Peters, R.H. 1991. A critique for ecology. Cambridge, Cambridge University Press.

Platt, J.R. 1964. Strong inference. Science, 146, 347–353.

Pollock, K.H., Nichols, J.D., Brownie, C. and Hines, J.E. 1990. Statistical inference for capture–recapture experiments. Wildlife Monographs, 107, 1–97.

Ruesink, J.L., Parker, I.M., Groom, M.J. and Kareiva, P.M. 1995. Reducing the risks of nonindigenous species introductions. BioScience, 45, 465–477.

Saitoh, T. and Takahashi, K. 1998. The role of vole populations in prevalence of the parasite (*Echinococcus multilocularis*) in foxes. Researches on Population Ecology, 40, 97–105.

Sinclair, A.R.E. 1989. Population regulation in animals. In: Cherrett, J.M., ed., Ecological concepts. Oxford, Blackwell Scientific, 197–241.

Singleton, G.R. and McCallum, H.I. 1990. The potential of *Capillaria hepatica* to control mouse plagues. Parasitology Today, 6, 190–193.

Stenseth, N.C. 1978. Demographic strategies in fluctuating populations of small rodents. Oecologia, 33, 149–172.

Stenseth, N.C. 1981a. How to control pest species: application of models from the theory of island biogeography in formulating pest control strategies. Journal of Applied Ecology, 18, 773–794.

Stenseth, N.C. 1981b. On Chitty's theory for fluctuating populations: the importance of genetic polymorphism in the generation of regular density cycles. Journal of Theoretical Biology, 90, 9–36.

Stenseth, N.C., Bjornstad, O.N. and Saitoh, T. 1996. A gradient from stable to cyclic populations of *Clethrionomys rufocanus* in Hokkaido, Japan. Proceedings of the Royal Society of London Series B, 263, 1117–1126.

Stenseth, N.C. and Saitoh, T. 1998. The population ecology of the vole *Clethrionomys rufocanus*: a preface. Researches on Population Ecology, 40, 1–3.

Sullivan, T.P. and Sullivan, D.S. 1986. Resiliency of snowshoe hares to population reduction. Journal of Applied Ecology, 23, 795–807.

Tamarin, R.H. and Krebs, C.J. 1969. *Microtus* population biology. II. Genetic changes at the transferrin locus in fluctuating populations of two vole species. Evolution, 23, 183–211.

Thompson, W.R. 1929. On natural control. Parasitology, 21, 269–281.

Underwood, A.J. 1997. Experiments in ecology: their logical design and interpretation using analysis of variance. Cambridge, Cambridge University Press.

Uvarov, B.P. 1931. Insects and climate. Transactions of the Entomological Society of London, 79, 1–247.

Vitousek, P.M., D'Antonio, C.M., Loope, L.L. and Westbrooks, R. 1996. Biological invasions as global environmental change. American Scientist, 84, 468–478.

Watson, A. and Moss, R. 1970. Dominance, spacing behaviour and aggression in relation to population limitation in vertebrates. In: Watson, A., ed., Animal populations in relation to their food resources. Oxford, Blackwell, 167–220.

Wilsey, B.J. 1996. Plant responses to elevated atmospheric CO_2 among terrestrial biomes. Oikos, 76, 201–206.

Wolff, J.O. and Cicirello, D.M. 1989. Field evidence for sexual selection and resource competition infanticide in white-footed mice. Animal Behaviour, 38, 637–642.

3. The Behaviour and Ecology of *Rattus norvegicus*: from Opportunism to Kamikaze Tendencies

David W. Macdonald, Fiona Mathews and Manuel Berdoy

Abstract

While rat population management is clearly possible in the absence of a knowledge of rat biology, we aim to show in this review how control is likely to prove more effective if woven into a robust framework of understanding. For example, rats have flexible population dynamics and can delay the onset of fertility in times of food shortage. Detailed observations have demonstrated the presence of stable, near-linear dominance hierarchies, where male social status tends to be age-related. We discuss how the success of poisoning strategies are crucially dependent on the foraging decisions which are made against this background of dominance hierarchies and competition for mates. Feeding patterns also can be altered in order to avoid predators. Rats are notoriously neophobic, and poor bait uptake is one of the main reasons that control strategies fail. Our review highlights the importance of social cues and *Toxoplasma gondii* infection in the modulation of neophobic responses. The potential impact of ill-planned rat control operations on the spread of zoonotic diseases is also considered. Finally, we discuss the development of behavioural and physiological resistance by rats in the face of continued poisoning pressure, and the apparent evolution of a new type of resistance which benefits the rat even in the absence of poison.

Keywords

Rattus norvegicus, rats, behaviour, society, neophobia, resistance, disease

INTRODUCTION

EXPLOSIVE DEMOGRAPHY, adaptable ecology and opportunistic behaviour are capacities that cause rats to rank high amongst those mammals that have most affected the course of human history. Today rats exact an immense toll on society worldwide, whether through the costs of prophylactic or remedial control, or through disease transmission and damage to crops and stored food. Throughout Southeast Asia, for example, pre-harvest damage caused by the rice-field rat, *Rattus argentiventer,* is reckoned to reduce crop yields by 17%, a figure which translates into the squandered rice requirements of in the order of 20 million people (Singleton 1997). Such losses may also have indirect environmental costs; lower yields forcing larger areas into production and accelerating the cultivation of wilderness with consequent threats to biodiversity. More directly, rats threaten the survival of endemic fauna on a number of islands, from the Galapagos to Guam (e.g. Amarasekare 1993; Robertson et al. 1994; Cree et al. 1995); alien species are, second only to habitat loss, the greatest contemporary force for extinction. Finally, the enormity of the threat posed to humanity by rat-borne disease is heightened in the context of our huge populations, rapid transportation and antibiotic resistance. Amongst the emerging infectious diseases, the viral haemorrhagic fevers and Lassa fevers add to the already lengthy list of blights which can be transmitted by rodents. The 200,000 cases of haemorrhagic fever with renal syndrome diagnosed annually in Asia is doubtless just the tip of an epizootiological iceberg. These threats are not confined to the developing world: the 1973 wave of deaths through hantavirus with pulmonary syndrome amongst healthy young Americans led to the discovery of hantavirus in the deer mouse, *Peromyscus maniculatus* (Childs et al. 1987). Hantavirus infection has also been discovered in Norway rats in rural Britain (Webster and Macdonald 1995a).

Amongst the diversity of problems caused by rodents, the Norway rat (*Rattus norvegicus*), ranks high amongst the miscreant species. Originally from Southeast Asia, the Norway rat's versatility rivals mankind's and our two species have, in company, spread around the globe. Trawling bibliographic indices reveals that some 24,000 technical publications refer annually to *R. norvegicus* (Berdoy and Macdonald 1991). This stunning total is, however, neither a fitting tribute to the fascination of its adaptability nor recognition of the enormity of its pest status; rather it stems largely from the utility of its domestic form as a model for studies ranging from biochemistry to experimental psychology. Publications on wild-type Norway rats largely concern toxicological studies of candidate poisons, while the behaviour and ecology of the species in the wild—which is where it actually does damage—account for scarcely a handful of those 24,000 publications annually. Indeed, as we shall show, while a little is known of the ecology of wild rats in farmscapes and a few cities in a smattering of developed countries, they are perversely unstudied where they impact the most. Most startling of all, effectively nothing is known of the biology of the sewer-dwelling rat.

Our objective in this chapter is to review the behavioural ecology of wild rats, largely within the context of our own team's findings. Our contention is that while rat control is manifestly possible in the absence of much knowledge of rat biology, it is likely to be much more effective if woven into a robust framework of understanding. This proposition rests on the oft-proven wisdom of the maxim: 'know thyn enemy'. In particular, our aim is to reveal that seemingly disjunct, and perhaps even rarefied, research topics, such as dispersal, social status, feeding patterns and disease transmission are actually inextricably linked in formulating a biological basis for rat management.

POPULATIONS, DEMOGRAPHY AND DISPERSAL

Questions about the population biology of Norway rats were at the forefront of mammalian ecology in post-war years, thanks to the pioneering work by Elton and Chitty (Chitty 1954) and Davies (1949). Considering the enormity of the rat's agronomic and public health impact, it is remarkable that the momentum of these early investigations was soon dissipated.

Numerous studies on farmland (Errington 1935; Aisenstadt 1945; Emlen et al. 1948; Zapletal 1964; Hartley and Bishop 1979; Brodie 1981; Huson and Rennison 1981; Homolka 1983) in differing temperate climates indicate that hedges and fields are generally a marginal habitat for rats, except when crops are available as food. Middleton (1954) noted that all rat infestations in hedgerows at his study site in Berkshire (United Kingdom) were either short-lived or were associated with rat colonies in corn-ricks or field-barns. He suggested that scattered field colonies were themselves ephemeral, but were probably the main reservoirs from which infestation of farm buildings occurred in the autumn and winter.

Colonists tend to be rats that are approaching, or have recently achieved, sexual maturity (Zapletal 1964). Telle (1966) found that most colonists weighed between 160–250 g, while Farhang-Azad and Southwick (1979) reported a mean weight of 190 g for 26 rats collected from newly recolonised burrows. Both concluded that emigrants are mainly young animals, but neither reported the sex of new colonists. However, Bishop and Hartley (1976) found approximately twice as many 'new' adult males as females entering their hedgerow population. Similarly, Calhoun (1962) reported that more males than females were ejected, or at least departed, from more socially stable colonies. Kendall (1984) and Leslie et al. (1952) found that the sex ratio of rats in environments where they breed tended to be biased towards females.

Rat population dynamics, and thus the success of control operations, are intimately linked with the availability of food supplies. In the simplest case, bait uptake is most likely when other sources of food are unavailable. However, we have found that food supply, and other environmental factors, also produce concomitant changes in behavioural and reproductive ecology (D.W. Macdonald and M.G.P. Fenn, unpublished data). These changes in turn may be of fundamental importance to rat population size and to control efforts. We live-trapped and radio-tracked rats in three contrasting United Kingdom farmland habitats, all of which were surrounded by winter wheat: (i) a resource-rich agricultural tip; (ii) a woodland where grain was intermittently provided for pheasants; and (iii) an adjacent, resource-poor stream bank. Several salient findings emerge. First, and unsurprisingly, more rats occurred at the farm rubbish tip, where food was most abundant, and fewest along a stream where it was most scarce (Figure 1). Second, numbers varied at each site: in general, there was a cycle in rat abundance corresponding to seasonal changes in breeding activity—pregnant females were generally captured from March until October, and peak numbers of juveniles were found between November and December (see also Davies and Hall 1951; Farhang-Azad and Southwick 1979, who report a bimodal pregnancy rate, with highs in spring and late summer). There is also evidence that reproduction ceases in cold winters (Leslie et al. 1952; Andrews et al. 1972; Lattanzio and Chapman 1980). However, in our study (D.W. Macdonald and M.G.P. Fenn, unpublished data) breeding activity was not simply a function of season but also depended on food

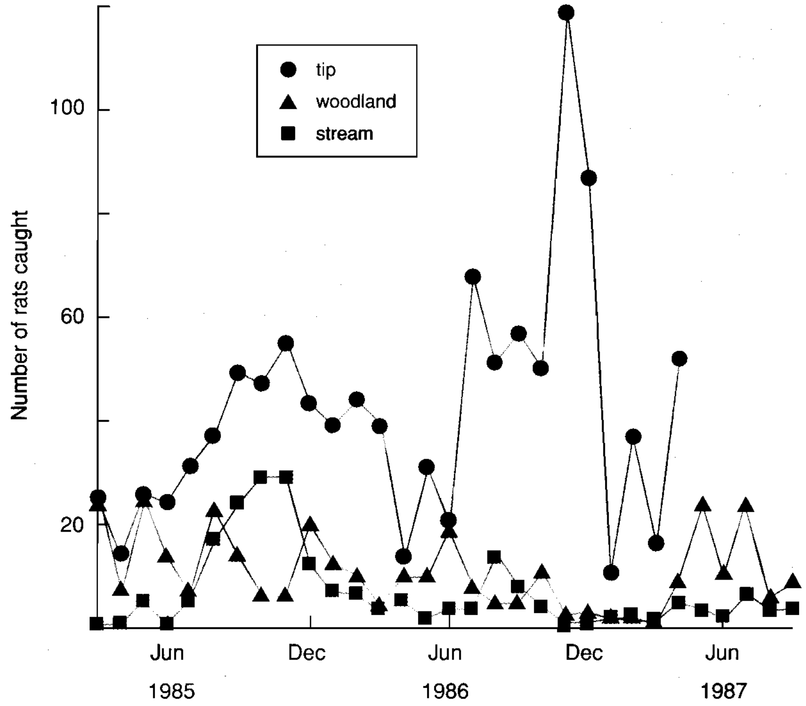


Figure 1.
Variation in rat abundance across time in three habitats with different food resources. The figure illustrates that more rats were captured in the resource-rich environment of the tip than in the woodland (moderate food availability) or stream (poor availability of resources). Cyclical fluctuations in abundance were also more marked in the resource-rich environment (D.W. Macdonald and M.G.P. Fenn, unpublished data).

availability. In the woodland site, winter breeding was stimulated by the provision of grain in January for the pheasants, with peak numbers of juvenile rats occurring in the population approximately three months later, in March and April.

Fertility in both sexes was affected by season. Using perforation as a measure of attainment of sexual maturity, the median weight of females at perforation at the rubbish-tip site was higher in the summer sample than the winter one. Fertility in males was also modulated by season and food supply, with evidence for delayed onset of sexual maturity and facultative cessation of spermatogenesis where food was scarce. For example, males achieved a greater weight before their testes became scrotal in winter than was the case in summer. Similarly, those animals living in the relatively poor environment of the stream delayed sexual maturity until they had achieved a greater weight than their counterparts in more productive habitats (Figure 2) (D.W. Macdonald and M.G.P. Fenn, unpublished data). Indeed, it was not unusual for males weighing more than 300 g to have abdominal testes [the median weight of rats with scrotal testes reported elsewhere was 136 g in rural rats (Davies 1949) and 145 g in rats living around Baltimore zoo (Farhang-Azad and Southwick 1979)]. Clearly, the control of food availability may have an important role in maintaining rat populations at manageable levels.

Movements

Farmland rats may either occupy stable home ranges or travel widely as transients. Hartley and Bishop (1979) estimated that three-quarters of rats fell in the former category and therefore were unlikely to have access to bait points positioned outside their home ranges. Within the home range, rats regularly retreat to rest sites (Orians and Pearson 1979; Galef 1988), which in arable areas tend to be located in hedge bottoms and banks (Brodie 1981).

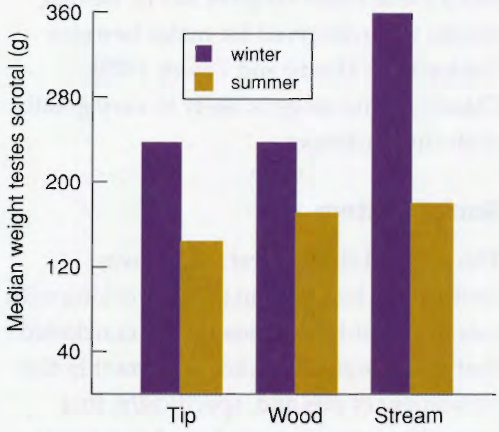


Figure 2.
Median weight at which testes became scrotal in winter and in summer at sites which differed in resource availability (tip = resource rich, wood = moderate, stream = poor). The figure illustrates that males achieved sexual maturity at lower weights in summer than in winter, and also in resource-rich environments ($p < 0.05$ for seasonal differences, $p < 0.001$ for resource differences) (D. W. Macdonald and M.G.P Fenn, unpublished data).

We radio-tracked rats in and around farms (Fenn et al. 1987; Macdonald and Fenn 1995). Males ranged widely through the fields when crop cover was available to provide protection from predators [mean linear home range = 678 m, standard deviation (SD) = 535]. However, their ranges contracted after harvest (90 m, SD = 28.2). This effect appeared not to apply to females, which generally had smaller home ranges. In addition to farming activity, the absolute availability of food also influenced home range size.

Females at the Wytham tip, where food was plentiful, had linear ranges averaging only 85 m whereas those based in the relatively poor environment of the stream had a mean home range of 428 m. Similar results were observed for males (see also Taylor 1978; Hardy and Taylor 1979). Clearly, home range is likely to vary greatly with circumstances.

Social system

The seminal study of rat society was undertaken by Calhoun (1962) working with rats in an outdoor enclosure. He concluded that social pressures were important in the movement of rats and, specifically, that subordinate males were forced to migrate into less favourable sites, leading to an unbalanced sex ratio (as low as 0.38) in good habitats. Similar reports of unbalanced sex ratios in areas suitable for breeding have also been made by others (e.g. Leslie et al. 1952). This socially-induced migration away from good sites may explain the transient infestation of fields and corn-ricks often reported during the summer. Social dominance confers feeding priority (Smith et al. 1991a), greater reproductive access and success (Gärtner et al. 1981; McClintok et al. 1982; Adams and Boice 1983, 1989) and reduces susceptibility to adverse conditions (Barnett 1955, 1958a; Christian and Davies 1964; Boice 1969).

We radio-tracked rats at our three farmland study sites (D.W. Macdonald and M.G.P. Fenn, unpublished data), and various other farm situations (Fenn et al. 1987; Fenn and Macdonald 1987). In summary, where resources were abundant and clumped, rats formed multi-male, multi-female groups which we suspect defended a clan territory. In areas with scattered or sparse resources, male movements were consistent with exclusive male ranges with access to several females. Insofar as any spatial exclusivity would affect access to baiting stations, the spatial organisation of rat populations is clearly important to poisoning operations. On one farm we found that almost all radio-tracked rats visited a particular grain mill at some stage; when poison was placed there an estimated 95% of the population was killed (Fenn et al. 1987).

The sex ratio of the populations along the stream was constant, and equal, throughout the year. In contrast, in the wood (around the pheasant hoppers) the sex ratio was significantly skewed: only approximately one fifth of rats captured between January and June were male [$\chi^2 = 5.828$, degrees of freedon (d.f.) = 1, $p = 0.016$ from January–March; $\chi^2 = 11.765$, d.f. = 1, $p = 0.0006$ from April–June]. We conclude that where groups developed around reliable food supplies, resident males excluded transient incomers, whereas these were numerous in passage through the poorly resourced stream habitat.

In order to examine further the consequences of ecological variation on social structure, we observed several colonies of wild Norway rats housed in large outdoor enclosures (c. 266 m^2). In an effort to bring the field into the laboratory, we provided rats with problems which they would encounter in the wild, including sufficient space, a diverse and multi-generational social environment and a dispersed food supply. Nocturnal observations showed that rat colonies were organised into steady, near-linear dominance hierarchies where male social status tended to be age-related (Berdoy et al. 1995a). Larger rats stood a better chance of winning contests, particularly when interacting with unfamiliar individuals, (Figure 3a). But social status within colonies became fixed: the results of earlier encounters appeared to determine the outcome of future ones, and dominant individuals thus maintained their social status long after initial body weight asymmetries with younger individuals had disappeared or even been reversed (Figure 3b). In stable groups, therefore, age is often a better predictor of social status than is size, with the alpha male being smaller than many of its subordinates (see also Calhoun 1962). Such 'settled dominance' (Berdoy et al. 1995a) may be found in a variety of other species (e.g. American bison, Rutberg 1983, but see Wolff 1988; red deer, Thouless and Guiness 1986; blue-footed booby chicks, Drummond and Osorno 1992; see also Huntingford and Turner 1987, for discussion of endocrine consequences of fighting). In rats it may explain why, despite considerable attention, the evidence that dominance is correlated to body weight has been conflicting—e.g. Barnett (1958a), Boreman and Price (1972), Nott (1988) and Smith et al. (1994) found a positive relationship whilst Baenninger (1966, 1970), Boice (1972), Price et al. (1976) and Sridhara et al. (1980) did not.

Why do large subordinates seem to accept the status quo rather than challenging smaller dominant individuals? We suspect that the costs of escalated aggression are great relative to the value of the contested resource. Whilst adult male rats were obviously in competition for food and mates, even the lowest subordinates could achieve access to feeding sites by adapting their feeding patterns accordingly (Berdoy and Macdonald 1991; Berdoy 1994). Moreover, dominant males could not monopolise access to oestrous females (Berdoy et al. 1995b).

In one colony, observations showed that whilst the alpha male enjoyed the highest number of copulations, there was no straightforward correlation between social status and mating success, with some subordinates achieving more copulations than individuals far higher in the dominance hierarchy. In another colony, the dominant male actually had less mating success than his two immediate subordinates.

This state of affairs is likely to be a consequence of the rat's mating system and may even be encouraged by the female's behaviour. Observations in the naturalistic environment of our enclosure colonies showed that mating was achieved by a type of scramble competition. Males pursued the oestrous female wherever she moved outside her burrow. Females were typically in oestrus for one night only, during which they were assiduously followed by a string of two to three males (up to seven), and mated repeatedly with several of them. Importantly, males had little time to interact with each other during the pursuit of oestrous females because they would lose a mating opportunity, and as a consequence even the most dominant males could not monopolise access to the female.

The scramble amongst males to mate with a female was so intense that it was hard to detect whether females displayed any

Figure 3. (a)
The effect of body weight asymmetry on social dominance in 132 pairs of adult male rats. There was a significantly increasing effect of weight on the chance of winning contests as the weight/age asymmetries between contestants became larger (G = 20.17; d.f. = 4; *p* = 0.0003). Asterisks show the statistical significance of weight asymmetry on the likelihood of winning a contest within each weight asymmetry class (ns = non significant, * = *p* < 0.05, ** = *p* < 0.01, * = *p* < 0.001, using binomial one-tailed test).**

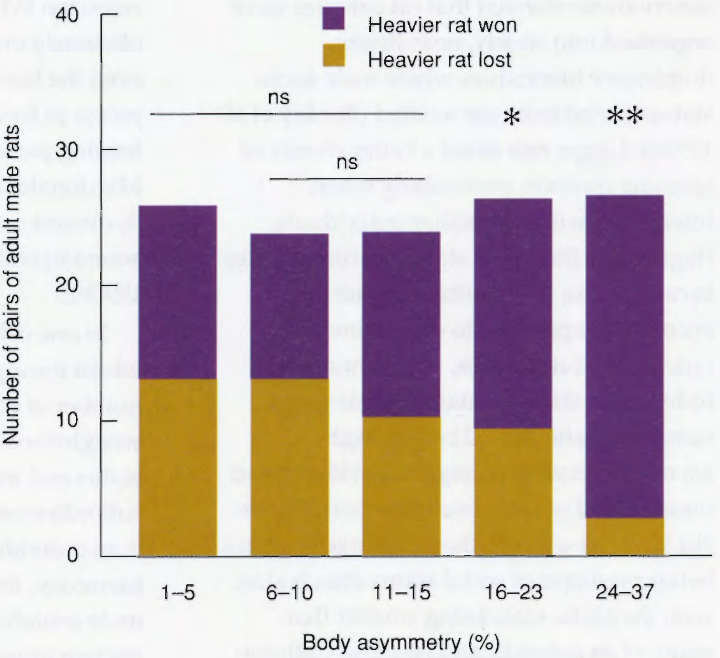
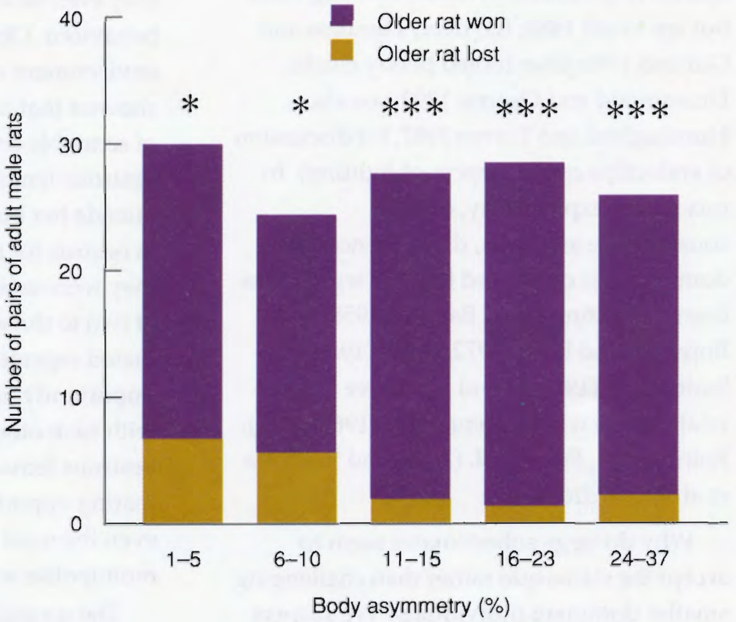


Figure 3.(b)
The effect of age asymmetry on social dominance in 132 pairs of adult male rats. There was a significantly increasing effect of age on the chance of winning contests as the weight/age asymmetries between contestants became larger (G = 12.48; d.f. = 4; *p* = 0.014). Asterisks show the statistical significance of weight asymmetry on the likelihood of winning a contest within each weight asymmetry class (ns = non significant, * = *p* < 0.05, ** = *p* < 0.01, * = *p* < 0.001, using binomial two-tailed test).**



mate selection or whether they mated promiscuously from choice.

To test these possibilities we devised an apparatus which exploited the sexual dimorphism in size amongst rats; male rats were housed in cubicles around a central arena in which a female was placed. A circular passage permitted the female access to each male's cubicle, but was too narrow for the male to pass (M. Berdoy et al., unpublished data; C. Rolland et al., unpublished data). Observations in this 'inverta-brothel' enabled us to see that females clearly exercised mate choice. Liberated from the constraints of male–male competitive behaviour, females formed enduring bonds with a single male, but nevertheless selected to mate promiscuously (although to a lesser extent) with a small number of the males available to them.

Social odours

There is a huge literature on olfactory communication amongst rats (e.g. see reviews in Brown and Macdonald 1985). Following observations of the males' assiduous sniffing of females in our enclosures, and their pursuit of those in oestrus, we began to question the significance of haunch odour. The scent of rat haunches had at least 22 volatile components, the proportions of which varied greatly between individuals. Although no single compound emerged as diagnostic, principal component analysis revealed exclusive categories in the odour profiles of males, oestrous females and dioestrous females, with the first axis explaining 80% of the variation. Haunch odours could, therefore, be separated along biologically meaningful lines, suggesting that they had the potential to signal useful information to rats (Natynczuk and Macdonald 1994a,b).

In order to receive an olfactory signal, rats must smell the encoding odour. We have investigated 'sniffing behaviour' in a group of 62 adult rats (Natynczuk and Macdonald 1994a,b). Male rats sniffed frequently at the various body zones of females but there were significant differences in the proportions of sniffs directed to the various body zones ($F = 38.21$, 32, 28 d.f., $p = 0.005$). The highest proportion of sniffs (23%) were to the females' haunches, and this behaviour was seen whether or not the female rat was related or in oestrus. However, the genital region of non-related oestrous females was investigated significantly more frequently (29% of all sniffs) than the genital region of any other group ($\chi^2 = 4.642$, 1 d.f., $p = 0.031$ for difference in proportions of sniffs for non-related oestrous and non-oestrous females). Males tended to sniff the haunch after sniffing the forequarters, as part of a sequence of sniffing along the female's body from forequarters to hindquarters. Histology of the skin sebaceous glands indicated that the secretory activity of glands in the haunch, but not those in the forequarters, changed during oestrus (Natynczuk and Macdonald 1994a,b; Natynczuk et al. 1995). A male rat will therefore smell a discontinuity or gradient in scent along the body of an oestrous female but not a dioestrous female. Rats can thus judge a female's reproductive status by calibrating the odour of her haunch against that of her forequarters.

The self-calibration model has four advantages: (a) it obviates the need for learning or inheriting responses to a pharmacopoeia of scents; (b) it minimises the difficulty posed to the recipient by variation, in quality and quantity, between the odours of different signallers; (c) social odours are inevitably mixed with variable and copious background smells, and even one individual's scent may vary over only a few days; and (d) it could work not only with complex mixes of chemicals, but also with single compounds. Self-calibration relies on assessment of the degree of difference between odours for the transmission of a signal and therefore might apply to a variety of phenomena detailed in the literature (Natynczuk et al. 1995). It is currently unclear whether a strategy employing pheromones could be used to disrupt rats mating systems and hence control population size.

FEEDING BEHAVIOUR

Foraging decisions must be carried out against a background of dominance hierarchies and competition for mates. A foraging rat must make decisions about when to feed, how to structure foraging bouts within its period of activity, and what to eat. Clearly an understanding of foraging behaviour is crucial to the development of more effective baiting systems: neophobia, food preferences and conditioned aversions associated with illness may all limit bait uptakes. As Quy et al. (1992a,b) and Cowan et al. (1994) point out, rat control operations often fail because of poor bait uptake.

Feeding decisions

Most wild rat populations are essentially nocturnal (Calhoun 1962; Taylor et al. 1991; Berdoy 1994) and therefore face two related problems. First, there is significant seasonal variation in the period for which they must fast during daylight hours. Second, and correspondingly, they must satisfy their energy requirements during nights of variable duration. The shorter the night, the more likely there is to be a conflict of interest between feeding and mating, which is also at its peak during the shortest summer nights. We attempted to determine how these conflicts are resolved.

Long-term monitoring of feeding activity in our colony of wild rats, using a purpose-built, continuously recording telemetric device (Berdoy and Evans 1990) showed that the adjustment in feeding intensity to maintain nocturnality (which rose by 65% between December and June as nights grew shorter) was not uniform (Berdoy 1994). Rats mainly compensated for the seasonal reduction in feeding time by increasing feeding activity during the last quarter of the night (Figure 4). As a result, the distribution of feeding activity through the night gradually changed from being roughly constant in winter (and comparable to that obtained in the laboratory) to being clearly skewed in summer, with a sharp peak of activity before sunrise. Feeding during the earlier part of the night remained at the same intensity as in winter. Since food availability was kept constant throughout the year why did the timing of feeding activity change so drastically between winter and summer?

First, during the first half of the night, the rats were generally busy exploring the enclosure and engaging in social activities. In males, the disruption of feeding was related to the intensity of mating effort which could result in little or no feeding at the beginning of the night. Such disturbance

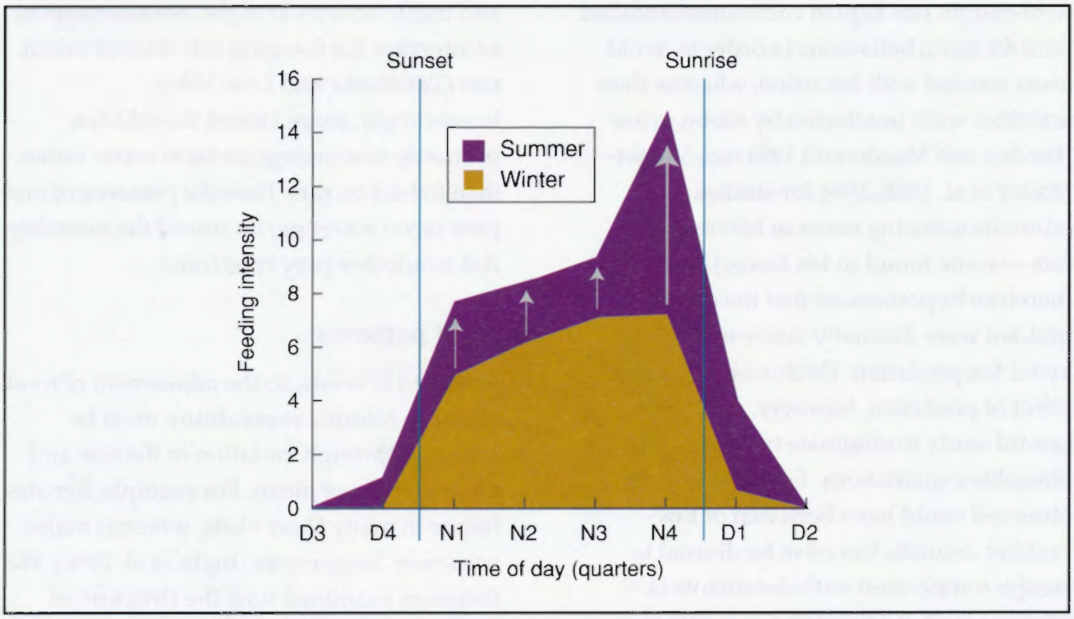


Figure 4.
Non-uniform increase in feeding activity in response to shorter summer nights. To control for the 50% reduction in night length between summer and winter, the data for day and night periods have been divided into quarters (Day = D1–D4; Night = N1–N4). The significantly greater increase in feeding intensity during the last quarter of the night (N4, thick arrow) causes an increasingly skewed feeding distribution during short summer nights. Note also the greater proportion of dawn feeding (D1) during summer.

was greatest during the summer when oestrous females were most numerous. Whereas the feeding activity of an oestrous female was principally disrupted for that night only, the presence of an oestrous female affected the feeding activity of each courting male in the colony.

Second, rats may have foraged mainly during the last portion of the night to time their feeding activity in anticipation of subsequent energy needs (Le Magnen 1985). Presumably rats needed to gather reserves that allowed them to last until the following night, particularly during periods of long daylight hours. Analyses showed that 90% of the monthly changes in the timing of activity in females, which were less disrupted by social living than were males, could be explained in terms of seasonal variation in night length.

Predator-altered behaviour

Whilst radio-tracking wild rats on farms around Oxfordshire, we identified an intriguing population of diurnal rats (Fenn and Macdonald 1995). The rats occupied a midden on one of five study farms. Coincidentally, a particularly large number of signs of the red fox (*Vulpes vulpes*) were also discovered in the vicinity of the midden, but were more or less absent from the other four sites, at which rats were typically nocturnal. We had previously found that wild-caught rats kept in enclosures modified their foraging behaviour in order to avoid areas scented with fox urine, whereas their activities were unaffected by rabbit urine (Berdoy and Macdonald 1991; see Vernet-Maury et al. 1968, 1984 for studies on odorants inducing stress in laboratory rats — some found in fox faeces). We therefore hypothesised that the rats at the midden were diurnally active in order to avoid fox predation. Establishing a direct effect of predation, however, required careful study to eliminate two other equally plausible explanations. First, the activity observed could have been that of low-ranking animals, forced to be diurnal to escape competition with dominants (a phenomenon observed in our enclosures — Berdoy 1994). Second, diurnal behaviour might have been facilitated by the lack of human disturbance at the site (see Taylor 1975).

Infra-red photo-electric cells revealed that the rats were active only when the foxes were absent. Seasonal use of the midden by foxes meant that the risk of predation to rats was greatest in summer, and correspondingly, the rats were most strictly diurnal in summer. The next step was to go beyond the correlational nature of the existing data. A fox-free enclosure was built near the midden and stocked with rats from the diurnal population. Rats in the enclosure should have been able to detect the absence of foxes though the lack of odour from fox urine and faeces. If rats were diurnally active to avoid fox predation, then in the absence of foxes we expected them to revert to nocturnal behaviour. This was exactly what happened (Fenn and Macdonald 1995). In contrast, disturbance alone, such as human and traffic activity at night, does not appear to interrupt the foraging activities of urban rats (Takahashi and Lore 1980). Interestingly, foxes visited the midden primarily to scavenge on farm waste rather than to feed on rats. Thus the presence of one prey type (scavenge) increased the mortality risk to another prey type (rats).

Meal patterns

Rats feed in bouts, so the adjustment of food intake to calorific expenditure must be achieved through variation in the size and the frequency of meals. For example, females forage in many short visits, whereas males use fewer, longer ones (Inglis et al. 1996). We therefore examined how the structure of feeding itself is influenced by variable night length and social pressures (Berdoy 1991, 1994). The feeding patterns of rats in the laboratory tend to show a positive relationship between the size of a meal and the following inter-meal interval (referred to as a 'post-prandial correlation'), but not between the size of the meal and the interval separating it from the previous one (a 'pre-prandial correlation') (Figure 5) (see Le Magnen 1985 for a review).

Colloquially, this means that rats decide on the size of a meal on the basis of how hungry they expect to be, rather than how hungry they are (Snowdon and Wampler 1974; Le Magnen 1985). A post-prandial correlation in an individual's feeding pattern is generally thought to indicate that the individual has good control of the onset and termination of its meals. On the other hand, an animal that is less able to control the onset of feeding than its termination is more likely to exhibit pre-prandial correlations. These principles have been useful in elucidating the physiology of feeding in the laboratory, but they also provided a tool for our investigation of the complex feeding patterns of rats under natural circumstances. Following Slater (1981), we tested the hypothesis that in predictable environments (such as in our enclosure) individuals would be more likely to exhibit post-prandial than pre-prandial correlations. Subordinates, on the other hand, which may be less likely to control the onset of feeding due to disturbances by dominant individuals, would effectively be living in an uncertain environment and should be more likely to exhibit pre-prandial correlations than would dominants. If so, subordinate and dominant rats might respond differently to baiting programs.

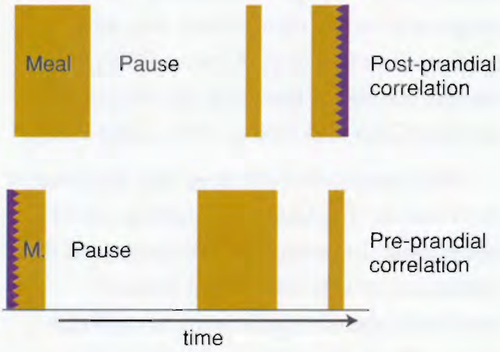


Figure 5.
Types of feeding pattern which reflect a food intake regulated from meal to meal. The upper part of the figure illustrates a positive correlation between the size of meal and the following pause. The lower part of the figure shows a positive correlation between size of meal and the preceding pause.

We analysed the feeding patterns of 12 adult rats (Berdoy 1991, 1993, 1994; Brunton 1995). First, the intervals between meals in the colony of rats appeared, on the whole, to be dependant on the size of the preceding meal (post-prandial correlation). Thus, knowledge of the length of time for which a rat had not eaten was, counter-intuitively, a poor predictor of the size of the next meal. Meal size appeared instead to be a reflection of the rat's anticipated subsequent energy need. Second, the consequences of agonistic and mating behaviour were also reflected in the values of these relationships. As predicted, dominant males, who benefited from a greater freedom of access to the feeding sites, and females (who were less disrupted by agonistic behaviour and matings than males), tended to regulate their feeding more closely than their subordinates or male counterparts (see also Slater 1981). Moreover, post-prandial correlations were more apparent in winter than in summer, supporting the idea that they were less able to regulate their meals when mating was at its peak and activity was skewed towards a pre-dawn peak.

The extent to which dietary decisions depend upon the fine structure of feeding, and the consequences of individual variation — let alone seasonal variation — on diet selection are rarely considered in the literature on rat nutrition. Yet these differences not only constitute the basis on which natural selection operates, but they may also relate to the way in which dietary decisions are made (Tempel et al. 1985; Hayne et al. 1986). They therefore affect the capacity for diet selection in general, and for aversion learning in particular.

Neophobia

Thompson's (1948) early work clearly demonstrated neophobia in wild rats in response to both new food and to lighting used to observe his experimental site (see also Barnett 1958a; Cowan 1977; Miller and

Holzmann 1981; Beck et al. 1988 and review in Royet 1983). To overcome neophobia, prebaiting is now an integral part of most rat-poisoning operations. The function of neophobia remains unproven, and it may have more to do with a general timidity than with food selection. However, while neophobia is likely to incur the cost of avoiding potentially harmless foods, it may facilitate the rat's ability to associate eating a novel food with adverse effects sometime later (Rozin and Kalat 1972; Nachman and Hartley 1975; Rescorla 1980: ability to associate illness with toxic food). Given the wide range of food qualities which a rat may encounter, and the intensity of poisoning pressure to which it is subjected, neophobia is generally assumed to be of adaptive significance (Rozin 1976). Indeed, neophobia was found to be absent amongst rats of the Hawea and Breaksea Islands, New Zealand (Taylor and Thomas 1993) which were historically uninhabited. In general, non-commensal rodent species also only show weak neophobia (Cowan 1977; Brammer et al. 1988). The transition from feeding from familiar to novel food after a period of neophobia is usually a gradual process (Chitty 1954; Barnett 1975). Wild rats may initially only sample a small amount of the food with amounts taken gradually increasing (Thompson 1948; Shepard and Inglis 1987; but see Beck et al. 1988).

It is difficult to quantify neophobia in rats in the field, because of the large number of potential confounding variables. We therefore designed a series of experiments to measure neophobic responses in wild-caught rats. The first of these housed rats in large outdoor colonies (Berdoy 1994). The rats avoided familiar foods (on which they had been fed ad libitum for two years) in a familiar feeding box when the grain was scented with natural oils. This reaction was still significant after 40 days. Even more extreme neophobia was noted when a novel food was presented. Inglis et al. (1996) undertook a similar study, but his rats were housed in concrete arenas and did not have lower intakes of novel foods, or of unfamiliar foods (foods which had previously been familiar, but had not been offered to the rats since they were caught). However, the rats did make more visits to the new foods, consuming less per trip. In contrast, the introduction of a novel food bowl, even though it contained familiar food, elicited strong neophobic responses, reducing food intake to 5% of its previous level. This neophobia, while diminished, was still evident after five days (Cowan 1976 reports similar results for the black rat, *Rattus rattus*, see also Galef and Heiber 1976; Galef 1988).

We investigated whether rats of different provenance displayed different levels of neophobia. In particular, we compared the behaviour of rats which had recently survived intensive poisoning on a Welsh farm with those which had been in the protected milieu of our enclosure for two years (Brunton and Macdonald 1996). The enclosure rats were descended from the survivors of a poisoning treatment in Hampshire two years previously. Hampshire rats had proven notoriously difficult to poison (Richards 1981; Greaves et al. 1982a,b; Quy et al. 1992b), prompting the hypothesis that they might be unusually neophobic. We designed three trials to test this. First we tested the effect of an unfamiliar odour on a familiar object (this involved handling the food container without gloves). Next, we tested the effect of replacing a familiar food container with a novel container while food and odour remained constant. Finally, we tested the effect of placing a novel food in a familiar container. In each case a familiar bowl, containing familiar food, was present throughout. The latency to feed, when confronted with each form of novelty, was allocated an ordinal score to indicate the degree of neophobia. The summed results from the three experiments were then used to create a 'neophobia index'. A score of 0 indicated no neophobia and 6 indicated extreme neophobia.

The survivors from the readily controlled Welsh populations were significantly more neophobic than enclosure rats descended from the problematic Hampshire population. Nonetheless, within both the Hampshire and the Welsh groups were some individuals that showed no neophobia (8% and 10%, respectively), and others that showed extreme neophobia (an index of 6: 4% and 10%, respectively) (Figure 6).

Perhaps the Welsh farm rats were a sample of the most neophobic individuals from the original population, the less neophobic rats having been selected out by poisoning. The rats born in the enclosure, although descended from the survivors of a poison treatment, had not themselves been subject to selection by poisoning and we hypothesise that they contained the full range of neophobic phenotypes. Thus, the differences in neophobia between the two groups is likely to be due to the presence of a higher proportion of strongly neophobic rats in the wild-caught population (Richter 1953; Barnett 1958b; Mitchell 1976; Cowan 1977). The failure of poison treatments in Hampshire is therefore not explicable by a fundamental difference in the inherited level of neophobia. In fact, like Quy et al. (1992a), we concluded that the difficulty in controlling the Hampshire rats was most likely due to the stable and abundant source of food. Various studies suggest that the strongest neophobic reaction in wild rats is to new objects in a familiar environment (Shorten 1954; Cowan and Barnett 1975; Cowan 1976, 1977; Wallace and Barnett 1990); evidence is accumulating that neophobia is related to the stability of the environment (e.g. little neophobia in land-fill site rats—Boice and Boice 1968) although unfamiliar areas with new objects are readily explored (Barnett and Spencer 1951; Cowan and Barnett 1975; Cowan 1977).

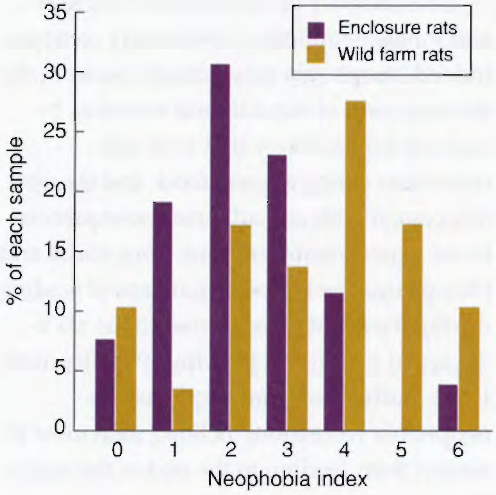


Figure 6.
Percentage of wild farm rats (from populations considered normal to control; $n = 30$) and enclosure rats (descended from Hampshire rats; $n = 30$) at each index of neophobia, where a score of 0 indicated no neophobia and 6 extreme neophobia. Overall, the farm rats were significantly more neophobic (Mann Whitney U test $Z = -3.0$; $p = 0.009$).

The interplay of conditioned aversion and innate neophobia is extremely complex. Indeed, neophobia may actually assist in the development of conditioned aversion, by making it more likely that a rat will remember eating a novel food, and thereby associate it with any adverse consequences. In the experiments with rats from Wales and Hampshire, the temporal patterns of feeding during the night were related to the rat's degree of neophobia (Berdoy 1994; Brunton 1995). Individuals that displayed no neophobia were more inclined than most to restrict their feeding to the end of the night. These associations are still largely mysterious. We can conclude, however, that subordinates may be constrained to eating shortly before dawn (Berdoy and Macdonald 1991) and may be more likely to try new foods than dominant individuals (Nott 1988). There is also evidence that the strength of taste aversions may be modulated by circadian rhythms (e.g. Infurna et al. 1979).

Social factors in food avoidance

Field studies have shown that rat colonies living in the same area can show substantial differences in food preferences not explicable by availability (Gandolfi and Parisi 1973). Subsequent laboratory studies have demonstrated that information about both the toxicity (the 'poisoned partner effect'—Bond 1984) and palatability of food can be transferred from mother to offspring, including via the mother's milk (Galef and Clark 1972; Hepper 1990). Diet preference can also be socially induced in adult laboratory rats (Galef and Wigmore 1983; Posadas-Andrews and Roper 1983). Thus, rats faced with novel foods are influenced by the olfactory information emanating from another rat which has eaten at the novel food types (the 'demonstrator effect'—Ludvigson et al. 1985; Galef 1988, 1994). However, the response of the rats receiving these cues differs according to the state of the 'demonstrator', with a dead animal producing no effects on food selection. The position of the cues is also important: residues on the anterior of the rat, but not the posterior, will elicit a response in the 'observer'. These results make functional sense since the contextual clues which induce preference are those which corroborate the circumstantial evidence of what the 'demonstrators' have eaten.

We investigated the demonstrator effect in naturalistic circumstances by manipulating the olfactory information emanating from some individuals in one of two enclosure colonies and evaluating the consequences on the diet selection of other wild colony members (Berdoy 1994). In order to produce individuals that exhibited the characteristics of having eaten cinnamon-scented food, about a dozen drops of cinnamon oil in suspension in a sugary solution were deposited on the neck, shoulders and near the mouth of 11 adult rats (informants) in one colony but not in the other. In both colonies, the rats were presented with two novel food types: peppermint and cinnamon-scented wheat grain, presented in two pairs of familiar feeding boxes. Each pair of boxes thus provided the rats with a choice between the two food types (which previous experiments had shown to be equally palatable to rats). To eliminate the possibility that food selection might be caused by neophobia to a new site rather than to the new food, the scented grain was placed in four familiar feeding boxes, previously containing non-scented wheat grain. In addition, rats could feed on non-scented grain in four other familiar feeding boxes distributed throughout the enclosure. The timing of visits to all feeding sites, as well as the identity of selected individuals within the colony, was recorded continuously using an automatic recording system (Berdoy and Evans 1990).

Before the treatment of the informants, rats in both colonies were highly and equally neophobic to both types of scented grain (Figure 7).

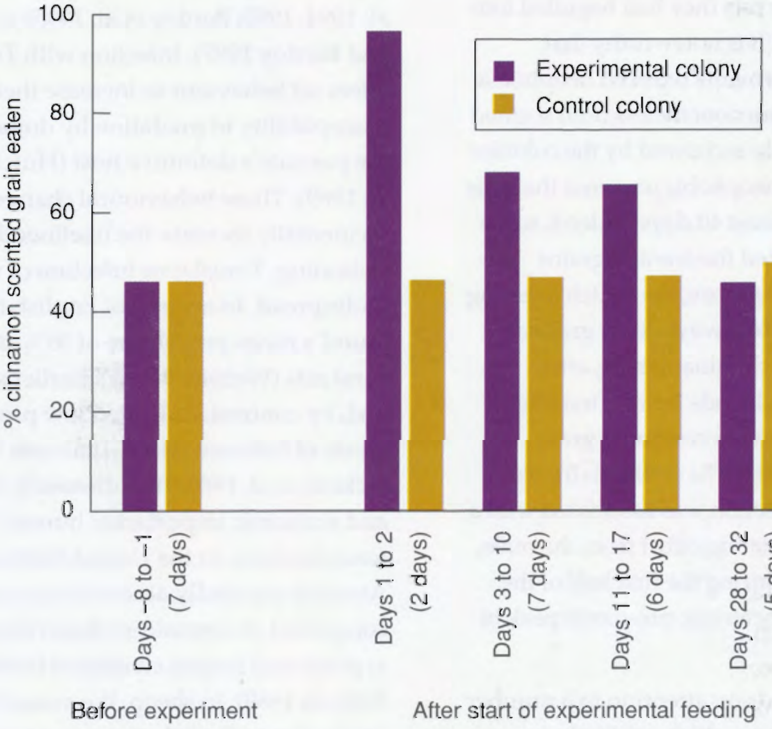


Figure 7.
Change in preference for feeding sites in two colonies of wild rats, as expressed by the proportion of cinnamon-scented grain eaten. In the control colony, the introduction of cinnamon and peppermint-scented grain was associated with no change in relative use of feeding sites. However, in the experimental colony which had 'informants' (cinnamon-scented animals) introduced to the colony before the introduction of the scented grain, there was a preference for the cinnamon over peppermint flavoured feeds. This remained statistically significant for two weeks ($p = 0.004$, permutation test).

However, following the tainting of the informants in one colony, their companions showed a significant preference for cinnamon over peppermint, whereas no such effect was observed in the control enclosure. The informants themselves were equally reluctant to eat both types of grain at first. Subsequently, however, the informants too began to show a preference for cinnamon, perhaps as they in turn were influenced by the odours on the rats they had beguiled into eating cinnamon. It is noteworthy that, although the informants reduced neophobia towards cinnamon, nonetheless both scented grains were largely eschewed by the colonies which showed a neophobic response that was significant for at least 40 days. Indeed, some individuals avoided the scented grains completely. Furthermore, the switch to eating novel foods did not always occur gradually. For example, one dominant male, after avoiding the novel foods for the first five nights, ate the cinnamon-scented grain almost exclusively (90%) on the sixth night. This new diet selection was associated with a night of intense mating effort from the male, with no feeding during the first half of the night, and a characteristic pre-dawn peak of feeding activity.

These results draw attention to a number of other parameters which are likely to influence diet selection in the wild: neophobia, the level of information, site preference, competition and the timing of feeding (Berdoy 1994). Thus while much remains to be discovered, our experiments point to the costs and benefits of sociality in decision making. And for a mammal under such heavy poisoning pressure as the rat, these decisions are of immediate practical relevance.

RAT-BORNE DISEASE

Parasite-altered behaviour

The problems of controlling rats, many of which revolve around neophobia, might seem largely separate from their role as disease vectors. However, we have revealed an intimate link between the two, at least with regard to infection with the protozoan *Toxoplasma gondii* (Webster 1994a; Webster et al. 1994, 1995; Berdoy et al. 1995b,c; Webster and Berdoy 1997). Infection with *Toxoplasma* alters rat behaviour to increase their susceptibility to predation by domestic cats, the parasite's definitive host (Hutchison et al. 1969). These behavioural changes also incidentally increase the likelihood of poisoning. *Toxoplasma* infection of rats is widespread. In a study of English farms, we found a mean prevalence of 35% among rural rats (Webster 1994a). Earlier studies had, by contrast, indicated low prevalence levels of between 0–10% (Lainson 1957; Jackson et al. 1986). The disease is of medical and economic importance: human toxoplasmosis in the United States of America reputedly accounts for more congenital abnormalities than rubella, syphilis and herpes combined (Schmidt and Roberts 1989). In sheep, the annual loss of lambs due to the infection is an estimated 100,000 in England alone (Berverley 1976). Yet despite the importance of rats in the diet of cats, and hence the subsequent opportunities for direct and indirect transmission of *Toxoplasma* to humans, wild rats have until recently been dismissed as unimportant to the dissemination of this disease. The discovery by Webster (1994a) that the infection can be perpetuated even in the absence of cats (the final hosts) (cf.

Wallace 1981) by congenital transmission, means that rats represent an important wildlife intermediate-host reservoir for toxoplasmosis.

In evolutionary terms, a parasite's goal is to increase the chances of infective stages meeting their host species. One adaptive route to this goal is host-behaviour manipulation. An obvious way of facilitating *Toxoplasma* transmission from an infected rat to a cat is to enhance the rat's activity levels: cats are attracted to moving objects and show little interest in stationary ones (Hubel and Weisel 1962; Leyhausen 1979). A study of the activities of 140 rats revealed that those infected with *Toxoplasma* were significantly more active than uninfected rats (consistent with findings in lab mice—Hutchison et al. 1980; Hay et al. 1983a,b). This was true both for rats that had acquired *Toxoplasma* as adults and for those which had acquired it congenitally. In contrast, rats harbouring parasites with direct life cycles, such as *Cryptosporidium parvum*, did not exhibit any altered activity (Webster 1994b; Webster et al. 1994). *Toxoplasma*-infected wild rats were also generally less neophobic of novel foods (see also Stretch et al. 1960a,b) (Figure 8) and, at farmsteads where the majority of the rat population could be sampled, were trapped more quickly than uninfected rats. Furthermore, they were more curious: when one of us stood in the enclosure it turned out that rats infected with *Toxoplasma* approached more closely than non-infected animals (Berdoy et al. 1995b). We further investigated whether *Toxoplasma*-infected and non-infected rats differed in their reaction to potential predation by cats. It is widely recognised that rats, including naive laboratory animals, show an innate avoidance of cat odour (Vernet-Maury et al. 1984; Blanchard et al. 1990; Berdoy and Macdonald 1991; Klein et al. 1994). In our outdoor enclosures, we presented adult rats with areas containing one of four distinct odours: the rat's own smell (own straw bedding), neutral smell (water), rabbit odour (rabbit urine) and cat odour (cat urine). As expected, non-infected rats showed a healthy avoidance of cat-scented areas, visiting them significantly less than other sites. However, in accordance with the manipulation hypothesis, infected rats however were significantly less averse and showed no overall avoidance of areas with signs of cat presence. A proportion even appeared to show a significant preference for the areas scented with odours of predators (M. Berdoy et al., unpublished data).

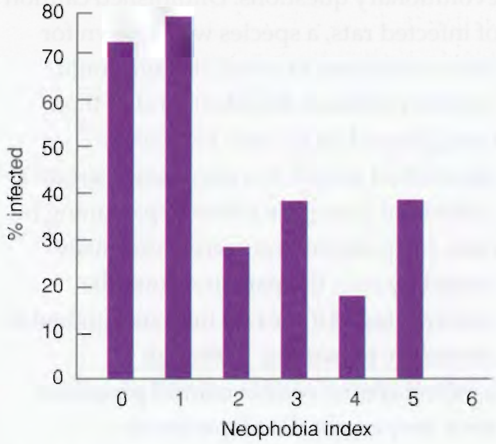


Figure 8.
Prevalence of *Toxoplasma*-positive rats in each neophobia index category, where a score of 0 indicated no neophobia and 6 extreme neophobia ($n = 36$). The figure illustrates that infected rats were less neophobic than non-infected individuals ($F_{1,34} = 5.0$, $p = 0.03$).

Alterations induced by *T. gondii* infection were confined to the predator's odour, as both types of rats behaved similarly with respect to areas containing other odours. These kamikaze rats were seemingly neither debilitated nor deranged in other respects. For example, in scramble competition for mates they secured just as many copulations as did uninfected rats, and were of equal social status (Berdoy et al. 1995b) (cf. Rau 1983, 1984; Freeland 1981: infection with *Trichinella spiralis* and *Heligmosomoides polygyrus*, respectively, prevent dominance in mice). It seems, therefore, that *Toxoplasma* affects specific behavioural traits likely to make rats more susceptible to predation by the felid definitive host. According to the saying, curiosity may or may not kill the cat ...but it is likely to kill the rat!

This kamikaze tendency raises intriguing evolutionary questions. Diminished caution of infected rats, a species well known for their fearfulness of novel stimuli, could arguably enhance the likelihood of their being preyed on by cats. However, diminished neophobia also seems certain to make wild rats more prone to poisoning by man. If a poisoned rat were more easily caught by cats, the parasite would be disadvantaged if the cats then succumbed to secondary poisoning. Although comprehensive vermin control programs are a very recent development in evolutionary terms, it is reasonable to assume that they have already begun to affect the ecology of *Toxoplasma* transmission. It is also interesting to consider the antiquity of this parasite-altered behaviour; does it have its origins in the predator–prey arms race between wild felids and rats? Has the adaptation been enhanced by the intensity of the interaction between rats and domestic cats during the last 4,000 years of shared peridomestic existence? Is the same behavioural modification found in other murine rodents?

Rural rat diseases

We investigated the prevalence of zoonoses in approximately 600 wild Norway rats captured on farms in southern England (Table 1). Thirteen zoonotic and ten non-zoonotic species of parasite were identified, many of which had never previously been recorded, or even investigated, in the United Kingdom (Webster and Macdonald 1995a). In addition to *Toxoplasma gondii*, another protozoan, *Cryptosporidium parvum*, which causes enteritis and enterocolitis in humans and other mammals (Perryman 1990) was found to be widespread (Webster and Macdonald 1995b; previously reported in Japan—Iseki 1986). The link between domestic cats and wild rats in the transmission of human disease was continued by the discovery of the rickettsian parasite, *Coxiella burnettii*, the causative agent for Q-fever (Webster et al. 1995b). Q-fever outbreaks occur sporadically (Marrie 1990a,b) and no common source has been identified. However, cases in humans are often linked with infection in domestic cats. In contrast, dogs, which occupy a similar peri-domestic niche, are rarely carriers of the parasite (Marrie et al. 1985; Baldelli et al. 1992). An obvious difference between the two, which might contribute to their radically different likelihood of acquiring *C. burnetti*, is the much more active predatory behaviour of cats.

Perhaps the most dramatic find of our surveys was the presence, for the first time in Europe, of hantavirus antibodies in wild animals (Webster and Macdonald 1995a). Hantaan viruses cause a group of illnesses in humans collectively referred to as hantaan fever or haemorrhagic fever with renal syndrome. Although a harmless, persistent infection in rodents, the disease may be extremely serious in humans. The discovery of hantavirus infection reaffirms the need to monitor the disease status of wild rats. Hantavirus transmission is strongly associated with intraspecific wounding (Glass et al. 1988). It is therefore possible that ill-planned control operations which disrupt the social hierarchy of rat groups, but fail to achieve eradication, could actually increase the prevalence of the disease (see Swinton et al. 1997). Our surveys also have demonstrated the importance of confirming rat disease status in different environments: *Leptospira* and *Salmonella* are usually considered to be highly prevalent among wild rats (e.g. Waitkins 1991; Chomel 1992), yet the former was relatively rare (14% prevalence) and the latter absent among rats studied on British farms (Webster et al. 1995a,c; see Nakashima et al. 1978). Other possible reservoirs of these diseases must therefore be investigated.

RAT CONTROL AND THE EVOLUTION OF RESISTANCE

Despite prolonged and intensive efforts to eradicate the Norway rat, the species remains a major pest. Attempts to control rat populations by poisoning tell a story of attacks and counter-attacks, currently unfolding before our eyes in a (largely unintended) billion-pound experimental demonstration of evolution in action. Rats have two main defences against poisoning campaigns: increasingly efficient behavioural adaptations, and physiological resistance to warfarin (Boyle 1960; Greaves 1985) and now, at least incipiently, resistance to second generation anticoagulants (Greaves et al. 1982a; Gill et al. 1992).

Table 1.
Prevalence of zoonoses and zoonotic agents in approximately 600 wild brown rats from farms in southern England.

Zoonoses/Zoonotic Agents	% Infected Rats
Ectoparasites	
Fleas	100
Mites	67
Lice	38
Ticks	0
Helminths	
Capillaria spp.	23
Hymenolepis diminuta	22
Toxocara cati	15
Hymenolepis nana	11
Taenia taeniaeformis	11
Bacteria	
Leptospira spp.	14
Yersinia enterocolitica	11
Listeria spp.	11
Pasteurella spp.	6
Pseudomonas spp.	4
Borrelia burgdorferi	0
Salmonella spp.	0
Protozoa	
Cryptosporidium parvum	63
Toxoplasma gondii	35
Babesia spp.	0
Sarcocystis spp.	0
Rickettsia	
Coxiella burnetti	34
Virus	
Hantavirus	4
Cowpox	0

Behavioural resistance

As mentioned above, many rats react to novel stimuli with extreme caution. Poison avoidance is also enhanced by their ability to associate the metabolic consequences of a food, long after it was ingested, and their capacity to interpret cues from other group members about the safety of foods. With these phenomena in mind, and observing that some rat populations were exceptionally difficult to poison (indeed they seemed not to eat the poison), the idea arose that they might have evolved enhanced behavioural resistance. A notable instance was the case of rats in Hampshire (Brunton et al. 1993, see also Greaves et al. 1982b). To confirm that the failure of anticoagulant poisons in Hampshire was not attributable to physiological resistance, we organised a control campaign with a non-anticoagulant poison, calciferol, to which there is no physiological resistance. Although some rats succumbed, at least 20–50% survived, despite repeated and intimate access to the poisoned bait. Clearly, they were not eating lethal doses. Three, not necessarily exclusive, factors might have explained this: (a) genetically enhanced neophobia; (b) experience; and (c) the stability of the environment. Our enclosure trials allowed us to discount the genetic explanation for hyper-neophobia: as discussed previously, the Hampshire rats were actually less neophobic than were survivors from a poisoning program in Wales. The evidence instead suggested that the abundant supply of alternative food, and the stable environment, were more likely explanations of the rats' reluctance to eat bait (Brunton et al. 1993). The farms in Hampshire, on which the 'resistant' rats were found, tended to be large and surrounded by fields of cereals, and corn is often stored loose in buildings. This provides an abundant and relatively predictable alternative food supply. Rats living in this environment can therefore 'afford' to be neophobic. By contrast, in mid-Wales, where rat control by poisoning is widely held to be successful, farms tend to be smaller with more livestock and less stored grain. In these less predictable environments, rats would need to be more opportunistic.

Physiological resistance

The introduction of the anticoagulant poisons, such as warfarin, marked a breakthrough in rat control. Due to their slow action, there is a reduced likelihood of aversion learning because the animal has already ingested a lethal dose by the time the symptoms of toxicosis develop (e.g. Nachman and Hartley 1975).

Warfarin, the most commonly used rodenticide for the last thirty years, is an anticoagulant poison which affects the biogenesis of blood clotting factors, ultimately causing death by many small internal haemorrhages. The production of several blood clotting factors is driven by a series of reactions where vitamin K is cyclically oxidised and reduced (Bell and Caldwell 1973; MacNicoll 1988). A simple vitamin K deficiency or substances disrupting the vitamin K cycle—such as warfarin — lead to the production of lower levels of blood clotting factors and therefore to poor coagulation and an increased risk of haemorrhage.

The prompt appearance of resistance to warfarin is a compelling example of the power of natural selection: by 1972, less than two decades after its introduction in England, warfarin resistance was reported in 12 areas in the United Kingdom (Greaves and Rennison 1973).

The costs of physiological resistance

The new benefits enjoyed by resistant rats involve new costs. In Welsh populations, where the mechanisms of resistance have been best studied, the altered enzyme present in warfarin-resistant individuals, whilst less affected by warfarin, is also less efficient at producing vitamin K (Greaves and Ayres 1969). This results in a natural vitamin K deficiency (Hermodsen et al. 1969) and ultimately a reduced coagulating activity in resistant individuals. Resistant rats are in a constant state of vitamin K deficiency and require a greater than normal amount of vitamin K in their diet in order to retain a normal clotting activity. Homozygous resistant rats need even more dietary vitamin K than heterozygous resistant rats (Hermodsen et al. 1969; see also Greaves and Ayres 1973; Martin 1973). The costs of resistance are substantial, particularly in homozygotes, and can lead to selective deaths during the first few weeks after birth (Bishop et al. 1977). Even when sub-lethal, resistance may also affect other components of fitness such as growth, social status and reproduction (Smith et al. 1991b).

These findings are of particular theoretical and practical relevance because the fitness costs incurred by resistant animals may affect the rate of spread and maintenance of the resistance allele in wild populations. Balanced polymorphism in wild rat populations regularly controlled with warfarin suggests the existence of a selective advantage of heterozygotes (Greaves et al. 1977). This is consistent with the interpretation that susceptible rats face reduced fitness due to the effects of the poison, and homozygous resistant rats have reduced fitness due to vitamin K deficiency. Heterozygotes, on the other hand, enjoy an efficient compromise: they are more resistant to warfarin than susceptible rats, while being less subject to vitamin K deficiency than homozygous resistant rats. In practical terms, the lower fitness of resistant genotypes (heterozygotes included) in the absence of poison (Partridge 1979) advocates a temporary relaxation of the use of warfarin to reduce the frequency of resistant individuals (Smith and Greaves 1987).

A new resistance

There may be a new strain of resistance amongst rats caught in southern England (Smith et al. 1993). We found that heterozygous resistance to warfarin poisoning was much higher than expected amongst 173 rats taken into captivity from two poplations which had resisted poisoning in the wild (71% and 76% were heterozygous resistant, versus expected values of 50% and 49%; $p < 0.001$ in both cases) (Gill et al. 1992). Moreover, while warfarin-resistant rats normally have lower body weights, these animals had slightly higher than average weights (Smith et al. 1993, 1994). These results suggest that these resistant rats, instead of suffering from the expected costs of resistance, benefited from a similar if not higher fitness than the susceptible genotypes, even in the absence of poison. Apart from its theoretical interest, these results have profound management implications. Resistance could lie dormant, if not spread, amongst rat populations living in warfarin-free environments, ready to counteract renewed poisoning efforts (Berdoy and Smith 1993). Nothing is yet known about the physiological basis of this potentially fascinating new development in the chemical arms race between humans and rats, but our results suggest that rats may currently be ahead in that race.

CONCLUSION

A full understanding of the biology of target pest species is vital to the design of effective management strategies. Nowhere, perhaps, is this better illustrated than in the case of the Norway rat, *R. norvegicus*. Not only are rats extremely widespread and adaptable, but they are also notoriously difficult to control. We have demonstrated that knowledge of the social organisation and movements of rats can assist in more effective placing of bait. Recognition of the complex feeding behaviour of rats is also of crucial importance to the success of poisoning strategies: neophobia, food preferences and aversive conditioning can all limit bait uptake. The biological approach to pest management can also reveal the need for strategies which may at first appear counter-intuitive. For example, the relaxation of poisoning pressure may actually improve the success of rat control in the long term, by reducing the frequency of resistant individuals in the population. Finally, a scientific framework will assist in the proper evaluation of the economic and public health significance of pest control operations. For example, examination of data on rat population dynamics and behaviour under conditions of high food availability suggests that new strategies are needed to complement lethal control operations. Similarly, we have demonstrated that rats may be less important than generally thought in the transmission of certain zoonoses (for example *Salmonella* sp. and *Leptospira* sp.), but that consideration must now be given to their role in the transmission of diseases such as Q-fever.

REFERENCES

Adams, N. and Boice, R. 1983. A longitudinal study of dominance in an outdoor colony of domestic rats. Journal of Comparative Psychology, 97, 24–33.

Adams, N. and Boice, R. 1989. Development of dominance in domestic rats in laboratory and semi-natural environments, Behaviour Proceedings, 19, 127–142.

Aisenstadt, D.S. 1945. Habitat distribution and ecology of the brown rat (*Rattus norvegicus*, Berkenh.) in a northwestern district of the European part of the USSR. Zool. Zh. Mosk., 24, 182–189.

Amarasekare, P. 1993. Potential impact of mammalian pest predators on endemic forest birds of western Mauria Kea, Hawaii. Conservation Biology, 7, 316–324.

Andrews, R.V., Belknap, R.W., Southard, S., Lorincz, M. and Hess, S. 1972. Physiological, demographic and pathological changes in wild Norway rat populations over an annual cycle. Comparative Biochemistry and Physiology, 41(A), 149–165.

Baenninger, L.P. 1966. The reliability of dominance orders in rats. Animal Behaviour, 14, 367–371.

Baenninger, L.P. 1970. Social dominance orders in the rat: 'spontaneous', food, and water competition. Journal of Comparative and Physiological Psychology, 71, 202–209.

Baldelli, R., Cimmino, C. and Pasquinelli, M. 1992. Dog-transmitted zoonoses: a seriological survey in the province of Bologna. Annali del Istituto Superiore di Sanita (Roma), 28, 493–496.

Barnett, S.A. 1955. Competition among wild rats. Nature, 175, 126–127.

Barnett, S.A. 1958a. An analysis of social behaviour in wild rats. Proceedings of the Zoological Society of London, 130, 107–152.

Barnett, S.A. 1958b. Experiments on 'neophobia' in wild and laboratory rats. British Journal of Psychology, 49, 195–201.

Barnett, S.A. 1975. The rat: a study of behaviour. London, University of Chicago Press.

Barnett, S.A. and Spencer, M.M. 1951. Feeding, social behaviour and interspecific competition in wild rats. Behaviour, 3, 229–242.

Beck, M., Hitchcock, C.L. and Galef, B.G. Jr. 1988. Diet sampling by wild Norway rats offered several unfamiliar foods. Animal Learning and Behaviour, 16, 224–230.

Bell, R.G. and Caldwell, P.T. 1973. Mechanism of warfarin-resistance. Warfarin and the metabolism of vitamin K_1. Biochemistry, 12, 1759–1762.

Berdoy, M. 1991. Feeding behaviour of wild rats, *Rattus norvegicus*: social and genetic aspects. PhD Thesis, Oxford University.

Berdoy, M. 1993. Defining bouts of behaviour: a three-process model. Animal Behaviour, 46, 387–396.

Berdoy, M. 1994. Making decisions in the wild: constraints, conflicts and communication in foraging rats. In: Galef, B.G. Jr. and Valsechhi, P., ed., Behavioural aspects of feeding: Ettore Majorana International Life Science Series, Vol 12. Chur, Switzerland, Harwood Academic Publishers, 289–313.

Berdoy, M. and Evans, S. 1990. An automatic recording system for identifying individual small animals. Animal Behaviour, 39, 998–1000.

Berdoy, M. and Macdonald, D.W. 1991. Factors affecting feeding in wild rats. Acta Oecologica, 12, 261–279.

Berdoy, M. and Smith, P. 1993. Arms race and rat race: adaptations against poisoning in the brown rat. Revue d'Ecologie (Terre & Vie), 48, 215–228.

Berdoy, M., Smith, P. and Macdonald, D.W. 1995a. Stability of social status in wild rats: age and the role of settled dominance. Behaviour, 132, 193–212.

Berdoy, M., Webster, J.P. and Macdonald, D.W. 1995b. Parasite-altered behaviour: is the effect of *Toxoplasma gondii* on *Rattus norvegicus* specific? Parasitology, 111, 403–409.

Berdoy, M., Webster, J.P. and Macdonald, D.W. 1995c. The manipulation of rat behaviour by *Toxoplasma gondii*. Mammalia, 59, 605–613.

Berverley, J.K.A. 1976. Toxoplasmosis in animals. Veterinary Record, 99, 123–127.

Bishop, J.A. and Hartley, D.J. 1976. The size and age structure of rural populations of *Rattus norvegicus* containing individuals resistant to the anticoagulant poison Warfarin. Journal of Animal Ecology, 45, 623–646.

Bishop, J.A., Hartley, D.J. and Partridge, G.G. 1977. The population dynamics of genetically determined resistance to warfarin in *Rattus norvegicus* from mid-Wales. Heredity, 39, 389–398.

Blanchard, R.J., Blanchard, D.C., Rodgers, J. and Weiss, S.M. 1990. The characterisation and modelling of antipredator defensive behaviour. Neuroscience and Biochemical Reviews, 14, 463–472.

Boice, R. 1969. Dominance and survival in stressed wild and domesticated populations. Proceedings of the American Psychologists' Association, 77th Annual Convention, 187–188.

Boice, R. 1972. Some behavioural tests of domestication in Norway rats. Behaviour, 42, 198–231.

Boice, R. and Boice, C. 1968. Trapping Norway rats in a land fill. Journal of Science Laboratories, Dension University, 49, 1–4.

Bond, N.W. 1984. The poisoned partner effect: some parametric considerations. Animal Learning and Behaviour, 12, 89–96.

Boreman, J. and Price, E. 1972. Social dominance in wild and domestic Norway rats (*Rattus norvegicus*). Animal Behaviour, 29, 534–542.

Boyle, C.M. 1960. Case of apparent resistance of *Rattus norvegicus* Berkenhout, to anticoagulant poisons. Nature, 188, 517.

Brammer, G., Barnett, S.A. and Marples, T.G. 1988. Responses to novelty by the Australian swamp rat, *Rattus putreolus*. Australian Mammology, 11, 63–66.

Brodie, J. 1981. Norway rats (*Rattus norvegicus*) on cereal stubble. Journal of Zoology, London, 195, 542–546.

Brown, R.E. and Macdonald, D.W. 1985. Social odours in mammals. Oxford and New York, Clarendon Press, 506p.

Brunton, C.F.A. 1995. Neophobia and its effect on the macro-structure and micro-structure of feeding in wild brown rats (*Rattus norvegicus*). Journal of Zoology (London), 235, 223–236.

Brunton, C.F.A. and Macdonald, D.W. 1996. Measuring the neophobia of individuals in different populations of wild brown rats. Journal of Wildlife Research, 1, 7–14.

Brunton, C.F.A., Macdonald, D.W. and Buckle, A.P. 1993. Behavioural resistance towards poison baits in brown rats *Rattus norvegicus*. Applied Animal Behaviour Science, 38, 159–174.

Calhoun, J.B. 1962. The ecology and sociology of the Norway rat. Public Health Service Publication No. 1008, 288p.

Chitty, D. 1954. The control of rats and mice. Oxford, Clarendon, 1041p.

Childs, J.E., Glass, G.E., Korch, G.W. and Leduc, J.W. 1987. Prospective seroepidemiology of hantaviruses and population dynamics of small mammal communities of Baltimore, Maryland (USA). American Journal of Tropical Medicine and Hygiene, 37, 648–662.

Chomel, B.B. 1992. Zoonoses of house pets other than dogs, cats and birds. Pediatric Infectious Diseases Journal, 11, 479–487.

Christian, J.J. and Davies, D.E. 1964. Endocrines, behaviour and population. Science, 146, 1550–1560.

Cowan, D.P., Bull, D.S., Inglis, I.R., Quy, R.J. and Smith, P. 1994. Enhancing rodenticide performance by understanding rodent behaviour. In: BCPC Conference—Pests and Diseases. Thornton Health, UK, BCPC (British Council of Pest Control) Publications, 1039–1046.

Cowan, P.E. 1976. The new object reaction of *Rattus rattus* L.: the relative importance of various cues. Behavioural Biology, 16, 31–44.

Cowan, P.E. 1977. Neophobia and neophilia: new object and new place reactions of three *Rattus* species. Journal of Comparative and Physiological Psychology, 91, 63–71.

Cowan, P.E. and Barnett, S.A. 1975. The new-object and new-place reactions of *Rattus rattus* L. Zoological Journal of the Linnean Society, 56, 219–234.

Cree, A., Daugherty, C.H. and Hay, J.M. 1995. Reproduction of a rare New Zealand reptile, the *Tautara sphenodon punctatus* on rat-free and rat-inhabited islands. Conservation Biology, 9, 373–383.

Davies, D.E. 1949. The weight of wild brown rats at sexual maturity. Journal of Mammalogy, 39, 125–130.

Davies, D.E. and Hall, O. 1951. The seasonal reproductive condition of the female Norway (brown) rat in Baltimore, Maryland. Physiological Zoology, 24, 9–20.

Drummond, H. and Osorno, J.L. 1992. Training siblings to be submissive losers: dominance between booby nestlings. Animal behaviour, 44, 881–893.

Emlen, J.T., Stokes, A.W. and Winsor, C.P. 1948. The rate of recovery of decimated populations of brown rats in nature. Ecology, 29, 133–145.

Errington, P.L. 1935. Wintering of field-living Norway rats in south-central Wisconsin. Ecology, 16, 122–123.

Farhang-Azad, A. and Southwick, C.H. 1979. Population ecology of Norway rats in the Baltimore Zoo and Druid Hill Park, Baltimore, Maryland. Annals of Zoology, 15, 1–42.

Fenn, M.G.P. and Macdonald, D.W. 1987. The contribution of field studies to stored product rodent control. In: Lawson, T.J., ed., Stored product pest control. Thornton Heath, UK, BCPC (British Council of Pest Control) Publications, 107–113.

Fenn, M.G.P. and Macdonald, D.W. 1995. Use of middens by red foxes: risk reverses rhythms of rats. Journal of Mammalogy, 76, 130–136.

Fenn, M.G.P., Tew, T.E. and Macdonald, D.W. 1987. Rat movements and control on an Oxfordshire farm. Journal of Zoology (London), 213, 745–749.

Freeland, W.J. 1981. Parasitism and behavioural dominance among male mice. Science, 213, 461–462.

Galef, B.G. Jr. 1988. Communication of information concerning distant diets in a social, central-place foraging species: *Rattus norvegicus.* In: Zentall, T.R. and Galef, B.G., ed., Social learning: psychological and biological perspectives. Hillside, New Jersey, Hove and London, Lawrence Erlbaum Associates, 119–139.

Galef, B.G. Jr. 1994. Olfactory communications about foods among rats: a review of recent findings. In: Galef, B.G. Jr. and Valsechhi, P., ed., Behavioural aspects of feeding. Ettore Majorana International Life Science Series, Vol 12. Chur, Switzerland, Harwood Academic Publishers, 83–103

Galef, B.G. Jr. and Clark, M.M. 1972. Mother's milk and adult presence: two factors determining initial dietary selection by weanling rats. Journal of Comparative and Physiological Psychology, 78, 220–225.

Galef, B.G. Jr. and Heiber, L. 1976. The role of residual cues in the determination of feeding site selection and exploration patterns of domestic rats. Journal of Comparative and Physiological Psychology, 90, 727–739.

Galef, B.G. Jr. and Wigmore, S.W. 1983 Transfer of information concerning distant foods: a laboratory investigation of the "information centre" hypothesis. Animal Behaviour, 31, 748–758.

Gandolfi, G. and Parisi, V. 1973. Ethological aspects of predation of rats, *Rattus norvegicus* (Berkenhout) on bivalves *Unio pictorium* and *Cerastoderma lamarcki*. Bolletino Zoologico, 40, 69–74.

Gärtner, K. Wankel, B. and Gaudszuhn, D. 1981. The hierarchy in copulatory competition and its correlation with paternity in grouped male laboratory rats. Zeitschrift Tierpsychologie, 56, 243–254.

Gill, J.E., Kerins, G.M. and MacNicoll, A.D. 1992. Inheritance of low grade brodifacoum resistance in the Norway rat. Journal of Wildlife Management, 56, 809–816.

Glass, G.E., Childs, J.E., Korch, G.W. and Le Duc, J.W. 1988. Association of intraspecific wounding with hantaviral infection in wild rats (*Rattus norvegicus*). Epidemiology of Infection, 101, 459–472.

Greaves, J.H. 1985. The present state of resistance to anticoagulants. Acta Zoologica Fennica, 173, 159–162.

Greaves, J.H. and Ayres, P.B. 1969. Linkages between genes for coat colour and resistance to warfarin in *Rattus norvegicus*. Nature, 224, 284–285.

Greaves, J.H. and Ayres, P. 1973. Warfarin-resistance and vitamin K requirement in the rat. Laboratory Animals, 7, 141–148.

Greaves, J.H., Redfern, R., Ayres, P.B. and Gill, J.E. 1977. Warfarin-resistance: a balanced polymorphism in the Norway rat. Genetics Research, Cambridge, 30, 257–263.

Greaves, J.H. and Rennison, B.D. 1973. Population aspects of warfarin-resistance in the brown rat, *Rattus norvegicus*. Mammal Review, 3, 27–29.

Greaves, J.H., Shepherd, D.S. and Gill, J.E. 1982a. An investigation of difenacoum resistance in Norway rat populations in Hampshire. Annals of Applied Biology, 100, 581–587.

Greaves, J.H., Shepherd, D.S. and Quy, R. 1982b. Field trials of second-generation anti-coagulants against difenacoum-resistant Norway rat populations. Journal of Hygiene, 89, 295–301.

Hardy, A.R. and Taylor, K.D. 1979. Radiotracking of *Rattus norvegicus* on farms. In: Amlaner, C.J., Macdonald, D.W., ed., A handbook on biotelemetry and radiotracking. Oxford, Pergamon Press, 657–665.

Hartley, D.J. and Bishop, J.A. 1979. Home range and movement in populations of *Rattus norvegicus* polymorphic for Warfarin resistance. Biological Journal of the Linnean Society, 12, 19–43.

Hay, J., Aitkin, P.P., Hutchison, W.M. and Graham, D.I. 1983a. The effect of congenital and adult acquired *Toxoplasma* infections on activity and responsiveness to novel stimulation in mice. Annals of Tropical Medicine and Parasitology, 77, 483–495.

Hay, J., Hutchison, W.M., Aitkin, P.P. and Graham, D.I. 1983b. The effect of congenital and adult acquired *Toxoplasma* infections on the motor performance of mice. Annals of Tropical Medicine and Parasitology, 77, 261–277.

Hayne, H., Rovee-Collier, C. and Gargano, D. 1986. Ambient temperature effects on energetics relations in growing chicks. Physiology and Behaviour, 37, 203–212.

Hepper, P.G. 1990. Foetal olfaction. In: Macdonald, D.W., Muller-Schwarze, D. and Natynczuk, S.E., ed., Chemical signals in vertebrates. Oxford, Oxford University Press, 282–287.

Hermodsen, M.A., Suttie, J.W. and Link, K.P. 1969. Warfarin metabolism and vitamin K requirement in the warfarin-resistant rat. American Journal of Physiology, 217, 1316–1319.

Homolka, M. 1983. On the problem of the exanthropic occurrence of *Rattus norvegicus*. Folia Zoologica, 32, 203–211.

Hubel, D.H. and Wiesel, T.N. 1962. Receptive fields, binocular interaction and functional architecture in the cat's visual cortex. Journal of Physiology, 160, 106–154.

Huntingford, F. and Turner, A. 1987. Animal conflict. London, Chapman and Hall, 448p.

Huson, L.W. and Rennison, B.D. 1981. Seasonal variability of Norway rat (*Rattus norvegicus*) infestation of agricultural premises. Journal of Zoology, London, 194, 257–289.

Hutchison, W.M., Dunachie, J.F., Siim, J.C.H.R. and Work, K. 1969. The life cycle of *Toxoplasma gondii*. British Medical Journal, 4, 806.

Hutchison, W.M., Bradley, M., Cheyne, W.M., Wells, B.W.P. and Hay, J. 1980. Behavioural abnormalities in *Toxoplasma*-infected mice. Annals of Tropical Medicine and Parasitology, 74, 507–510.

Infurna, R.N., Steinert, P. A., Freda, J.S. and Spear, N.E. 1979. Sucrose preference and LiCl illness-induced aversion as a function of drug dose and phase of the illumination cycle. Physiology and Behaviour, 22, 955–961.

Inglis, I.R., Shepherd, D.S., Smith, P., Haynes, P.J., Bull, D.S., Cowan, D.P. and Whitehead, D. 1996. Foraging behaviour of wild rats (*Rattus norvegicus*) towards new foods and bait containers. Applied Animal Behaviour Science, 47, 175–190.

Iseki, M. 1986. Two species of *Cryptosporidium* naturally infecting house rats, *Rattus norvegicus*. Japanese Journal of Parasitology, 35, 251–256.

Jackson, M.H., Hutchinson, W.M. and Siim, J.C. 1986. Toxoplasmosis in a wild rodent population of central Scotland and a possible explanation of the mode of transmission. Journal of Zoology, 209, 549–557.

Kendall, P.B. 1984. Seasonal changes of sex ratio in Norway rat (*Rattus norvegicus*) populations in Wales. Journal of Zoology, London, 203, 208–211.

Klein, S.L., Lambert, K.G., Durr, D., Schaefer, T. and Waring, R.E. 1994. Influence of environmental enrichment and sex on predator stress response in rats. Physiology and Behaviour, 56, 292–297.

Lainson, R. 1957. The demonstration of *Toxoplasma* in animals, with particular reference to members of the Mustelidae. Transactions of the Royal Society of Tropical Medicine and Hygiene, 51, 111–117.

Lattanzio, R.M. and Chapman, J.A. 1980. Reproduction and physiological cycles in an island population of Norway rats. Bulletin of the Chicago Academy of Science, 12, 1–68.

Le Magnen, J. 1985. Hunger. Cambridge, Cambridge University Press, 157p.

Leslie, P.H., Venables, U.M. and Venables, L.S. 1952. The fertility and population structure of the brown rat (*Rattus norvegicus*) in corn ricks and some other habitats. Proceedings of the Zoological Society, London, 122, 187–238.

Leyhausen, P. 1979. Cat behaviour: the predatory and social behaviour of domestic and wild cats. Garland Series in Ethology. New York, Garland STPM Press.

Ludvigson, H.W., Mathis, D.A. and Choquette, K.A. 1985. Different odours in rats from large and small rewards. Animal Learning and Behaviour, 13, 315–320.

Macdonald, D.W. and Brown, R.E. 1985. The smell of success. New Scientist, 106, 10–14.

Macdonald, D.W. and Fenn, M.P.G. 1995. Rat ranges in arable areas. Journal of Zoology (London), 236, 349–353.

MacNicoll, A.D. 1988. The role of altered vitamin K metabolism in anticoagulant resistance in rodents. In: Suttie, J.W., ed., Current advances in vitamin K research. Amsterdam, Elsevier Science, 407–417.

Marrie, T.J. 1990a. Q-fever—a review. Canadian Veterinary Journal, 3, 555–564.

Marrie, T.J. 1990b. Epidemiology of Q-fever. In: Marrie, T., ed., Q-fever: the disease, Vol. 1. Boca Raton, CRC Press, 49–70.

Marrie, T.J., Van Buren, J. and Fraser, J. 1985. Seroepidemiology of Q-fever among domestic animals in Nova Scotia. American Journal of Public Health, 75, 763–766.

Martin, A.D. 1973. Vitamin K requirement and anticoagulant response in the warfarin resistant rat. Biochemical Society Transactions, 1, 1206–1208.

McClintok, M.K., Anisko, J.J. and Adler, N.T. 1982. Group mating among Norway rats II. The social dynamics of copulation: competition, cooperation and mate choice. Animal Behaviour, 30, 410–425.

Middleton, A.D. 1954. Rural rat control. In: Chitty, D., ed., Control of rats and mice. Oxford, Clarendon Press, 414–448.

Miller, R.R. and Holzmann, A.D. 1981. Neophobia: generality and function. Behavioural and Neural Biology, 33, 17–44.

Mitchell, P. 1976. Experiments on neophobia in wild and laboratory rats; a reevaluation. Journal of Comparative and Physiological Psychology, 90, 190–197.

Nachman, M. and Hartley, P.L. 1975. Role of illness in producing taste aversion in rats: a comparison of several rodenticides. Journal of Comparative and Physiological Psychology, 89, 1010–1018.

Nakashima, R.S., Wetzler, T.F. and Nordin, J.P. 1978. Some microbial threats posed by Seattle rats to the community's health. Journal of Environmental Health, 40, 264–267.

Natynczuk, S.E. and Macdonald, D.W. 1994a. Scent, sex, and the self calibrating rat. Journal of Chemical Ecology, 20, 1843–1857.

Natynczuk, S.E. and Macdonald, D.W. 1994b. Analysis of rat haunch odour using the dynamic solvent effect and principal component analysis. Journal of Chemical Ecology, 20, 1859–1866.

Natynczuk, S.E., Macdonald, D.W. and Tattersall, F.H. 1995. Morphology and chemistry of brown rat, *Rattus norvegicus*, preputial and clitoral glands. Journal of Chemical Ecology, 21, 297–260

Nott, H.M.R. 1988. Dominance and feeding behaviour in the brown rat. PhD thesis, University of Reading.

Orians, G.H. and Pearson, N.E. 1979. On the theory of central place foraging. In: Horn, D.J., Mitchell, R. and Stair, G.R., ed., Analysis of ecological systems. Columbus, Ohio State University Press, 155–177.

Partridge, G.G. 1979. Relative fitness of genotypes in a population of *Rattus norvegicus* polymorphic for warfarin resistance. Heredity, 43, 239–246.

Perryman, L.E. 1990. *Cryptosporidium* in rodents. In: Dubey, J.P., Speer, C.A. and Fayer, R., ed., Cryptosporidiosis of man and animals. Boca Raton, CRC Press, 125–132.

Posadas-Andrews, A. and Roper, T.J. 1983. Social transmission of food preferences in adult rats. Animal Behaviour, 31, 265–271.

Price, E.O., Belanger, P.L. and Duncan, R.A. 1976. Competitive dominance of wild and domestic Norway rats (*Rattus norvegicus*). Animal Behaviour, 24, 589–599.

Quy, R.J., Cowan, D.P., Haynes, P., Inglis, I.R. and Swinney, T. 1992a. The influence of stored food on the effectiveness of farm rat control. In: BCPC Conference-Pests and Diseases. Thornton Health, UK, BCPC (British Council of Pest Control) Publications, 291–300.

Quy, R.J., Shepard, D.S. and Inglis, I.R. 1992b. Bait avoidance and effectiveness of anticoagulant rodenticides against warfarin and difenacoum-resistant populations of Norway rats (*Rattus norvegicus*). Crop Protection, 11, 14–20.

Rau, M.E. 1983. Establishment and maintenance of behavioural dominance in male mice infected with *Trichinella spiralis*. Parasitology, 86, 319–322.

Rau, M.E. 1984. Loss of behavioural dominance in male mice infected with *Trichinella spiralis*. Parasitology, 88, 371–373.

Rescorla, R.A. 1980. Pavlovian second-order conditioning: studies in associative learning. Hillside, Lawrence Earlbaum Associates.

Richards, C.G.J. 1981. Field trials of bromadiolone against infestations of warfarin-resistant *Rattus norvegicus*. Journal of Hygiene, 86, 363–367.

Richter, C.P. 1953. Experimentally produced behaviour reactions to food poisoning in wild and domesticated rats. Annals of the New York Academy of Science, 56, 225–239.

Robertson, H.A., Hay, H.J., Saul, E.K. and McCormack, G.V. 1994. Recovery of the kakeroi: an endangered forest bird of the Cook Islands. Conservation Biology, 8, 1078–1086.

Rozin, P. 1976. The selection of food by rats, humans and other animals. Advances in the Study of Behaviour, 6, 21–76.

Rozin, P. and Kalat, J. 1972. Specific hungers and poison avoidance as adaptive specializations of learning. Psychology Review, 78, 459–486.

Royet, J.P. 1983. Les aspects comportementaux de l'aversion conitionée et de la néophobia. L'année Biologique, 22, 113–167.

Rutberg, A.T. 1983. Factors influencing dominance status in American bison cows (*Bison bison*). Zeitschrift Tierpsychologie, 63, 206–212.

Schmidt, G.D. and Roberts, L.S. 1989. Foundations of parasitology, 4th edn. London, Times Mirror/Mosby, College Publishing, 750p.

Shepherd, D.S. and Inglis, I.R. 1987. Feeding behaviour, social-interactions and poison bait consumption by a family group of wild rats living in semi-natural conditions. In: Lawson, T.J., ed., Stored products pest control. Thornton Health, UK, BCPC (British Council of Pest Control) Publications, 97–105.

Shorten, M. 1954. The reaction of the brown rat towards changes in its environment. In: Chitty, D., ed., The control of rats and mice, 2. Oxford, Clarendon Press, 307–334.

Singleton, G.R. 1997. Integrated management of rodents: a Southeast Asian and Australian perspective. Belgian Journal of Zoology, 127, 157–169.

Slater, P.J.B. 1981. Individual differences in animal behaviour. Perspectives in Ethology, 4, 35–49.

Smith, P., Berdoy, M., Smith, R.H. and Macdonald, D.W. 1993. A new aspect of resistance in wild rats: benefits in the absence of poison. Functional Ecology, 7, 190–194.

Smith, P., Berdoy, M. and Smith, R.H. 1994. Body-weight and social dominance in anticoagulant-resistant rats. Crop Protection, 13, 311–316.

Smith, P., Smith, R.H. and Sibly, R.M. 1991a. Pulsed baiting: laboratory evidence for behavioural exclusion in wild rats. In: Fleurat-Lessard, F. and Ducom, P, ed., Proceedings of the 5th International Conference on Stored Product Protection, Bordeaux, France, September 1990, 1517–1526.

Smith, P., Townsend, M.G. and Smith, R.H. 1991b. A cost of resistance in the brown rat: reduced growth rate in warfarin-resistant lines. Functional Ecology, 5, 441–447.

Smith, R.H. and Greaves, J.H. 1987. Resistance to anticoagulant rodenticides: the problem and its management. In: Donahaye, E. and Navarro, S., ed., Proceedings of the 4th International Conference on Stored-Product Protection, Tel Aviv, Israel, September 1986. Bet Dagon, Israel, Agricultural Research Organisation, 302–315.

Snowdon, C.T. and Wampler, R.S. 1974. Effects of lateral hypothalmic lesions and vagotomy on meal patterns in rats. Journal of Comparative and Physiological Psychology, 87, 399–409.

Sridhara, S., Narasimham, A.U. and Krishnamoorthy, R.V. 1980. Aggressive interactions among wild and domestic rodents. Proceedings of the Indian Academy of Science (Animal Science), 89, 351–357.

Stretch, R.G.A., Leytham, G.W.H. and Kershaw, W.E. 1960a. The effect of acute schistosomiasis upon discrimination learning and activity in mice. Annals of Tropical Medicine and Parasitology, 54, 476–486.

Stretch, R.G.A., Leytham, G.W.H. and Kershaw, W.E. 1960b. The effect of acute schistosomiasis upon learning in rats under different levels of motivation. Annals of Tropical Medicine and Parasitology, 54, 487–492.

Swinton J., Tuyttens, F., Macdonald, D.W., Nokes, D.T., Cheeseman, C.L. and Clifton Hadley, R. 1997. A comparison of fertility and lethal control on bovine tuberculosis in badgers: the impact of pertubation. Transactions of the Royal Philosophical Society Series B, 352, 619–631.

Takahashi, L.K. and Lore, R.U. 1980. Foraging and food hoarding of wild *Rattus norvegicus* in an urban environment. Behavioural and Neural Biology, 29, 527–531.

Taylor, K.D. 1975. An automatic device for monitoring small mammal traffic on runways. Journal of Zoology (London), 176, 274–277.

Taylor, K.D. 1978. Range of movement and activity of common rats (*Rattus norvegicus*) on agricultural land. Journal of Applied Ecology, 15, 663–677.

Taylor, K.D., Fenn, M.P.G. and Macdonald, D.W. 1991. Common rat. In: Corbett, G.B. and Harris, S.H., ed., Handbook of British mammals. Oxford, Blackwell Scientific Publications, 248–255.

Taylor, R.H. and Thomas, B.W. 1993. Rats eradicated from Breaksea Island (170HA), Fiordland, New Zealand. Biological Conservation, 65, 191–198.

Telle, H.J. 1966. Beitrag zur Kenntis der Verhaltensweise von Ratten, vergleichen dargestelt bei *Rattus norvegicus* und *Rattus rattus*. Angewandte Zoologie, 53, 129–196 (MAFF Tolworth translation, Rodent Research reprint 4489).

Tempel, D.L., Shor-Posner, G., Dwyer, D. and Leibowitz, S.F. 1985. Nocturnal patterns of macronutrient intake in freely feeding and food deprived rats. American Journal of Physiology, 256, R541–R548.

Thompson, H.V. 1948. Studies of the behaviour of the common brown rat: 1. Watching marked rats taking plain and poisoned bait. Bulletin of Animal Behaviour, 6, 2–40.

Thouless, C.R. and Guiness, F.E. 1986. Conflict between red deer hinds: the winner always wins. Animal Behaviour, 34, 1166–1171.

Vernet-Maury, E., Le Magnen, J. and Chanel, J. 1968. Comportement émotif chez le rat: influence de l'odeur d'un prédateur et d'un non-prédateur. Paris, C.R. Acadamie Scientifique, 267, 331–334.

Vernet-Maury, E., Polak, E.H. and Demael, A. 1984. Structure–activity relationship of stress-inducing odourants in the rat. Journal of Chemical Ecology, 10, 1007–1017.

Waitkins, S.A. 1991. Rats as a source of leptospirosis—Weil's disease. Sorex Technical Paper No. 4. Cheshire, Sorex Ltd.

Wallace, G.D. 1981. Re: 'association of cats and toxoplasmosis'. American Journal of Epidemiology, 113, 198–201.

Wallace, R.J. and Barnett, S.A. 1990. Avoidance of new objects by the black rat, *Rattus rattus*, after object presentation and change. International Journal of Comparative Psychology, 3, 253–265.

Webster, J.P. 1994a. Prevalence and transmission of *Toxoplasma gondii* in wild brown rats, *Rattus norvegicus*. Parasitology, 108, 407–411.

Webster, J.P. 1994b. The effect of *Toxoplasma gondii* and other parasites on activity levels in wild and hybrid *Rattus norvegicus*. Parasitology, 110, 583–589.

Webster J. and Berdoy, M. 1997. *Toxoplasma gondii* in *Rattus norvegicus*: characteristics and evolution of parasite-altered behaviour. In:

Holland, S., ed., Modern perspectives on zoonoses. Dublin, Royal Irish Academy, 115–121.

Webster, J.P., Brunton, C.F.A. and Macdonald, D.W. 1994. Effect of *Toxoplasma gondii* on neophobic behaviour in wild brown rats, *Rattus norvegicus*. Parasitology, 109, 37–43.

Webster, J.P., Ellis, W.A. and Macdonald, D.W. 1995a. Prevalence of *Leptospira* in wild brown rat (*Rattus norvegicus*) populations in the UK. Epidemiology and Infection, 114, 195–201.

Webster, J.P., Ellis, W.A. and Macdonald, D.W. 1995b. Prevalence of *Leptospira* and other zoonoses in wild brown rats on UK farms. Mammalia, 59, 615–622.

Webster, J.P., Lloyd, G. and Macdonald, D.W. 1995c. Q-fever (*Coxiella burnetti*) reservoir in wild brown rat (*Rattus norvegicus*) populations in the UK. Parasitology, 110, 31–35.

Webster, J.P. and Macdonald, D.W. 1995a. Parasites of wild brown rats (*Rattus norvegicus*) on UK farms. Parasitology, 111, 247–255.

Webster, J.P. and Macdonald, D.W. 1995b. Cryptosporidiosis reservoir in wild brown rats (*Rattus norvegicus*): first report in the UK. Epidemiology and Infection, 115, 207–209.

Wolff, J.O. 1988. Maternal investment and sex–ratio adjustment in American bison calves. Behavioural Ecology and Sociobiology, 23, 127–133

Zapletal, M. 1964. On the occurrence of the brown rat (*Rattus norvegicus*, Berk.) under natural conditions in Czechoslovakia. Zool. Listy, 13, 125–134.

4. Models for Predicting Plagues of House Mice (*Mus domesticus*) in Australia

Roger P. Pech, Greg M. Hood, Grant R. Singleton, Elizabeth Salmon, Robert I. Forrester and Peter R. Brown

Abstract

In this chapter, the main features of current models for predicting the dynamics of house mice (*Mus domesticus*) populations are reviewed and an assessment made of their data requirements and their ability to contribute to the effective management of mice in Australia. In addition, recent progress with quantifying aspects of the dynamics of mouse populations in the Mallee region of Victoria is described.

Robust predictive models are required for the effective management of mice because plagues (massive eruptions of mice) occur at irregular intervals and farmers require early warning to implement control techniques and prevent economic losses. Nine published models have been produced for the plague-prone regions of southern and eastern Australia. Two of these aim to predict the occurrence of plagues at a regional level and five predict changes in the abundance of mice at a district, or local, scale. However, none of the current models for southern Australia include estimates of the numerical response of mouse populations. This limits their value for assessing the relative merits of control programs and new control techniques.

A model of the numerical response of mice was developed using observations of the abundance of mice over a 15-year period in the Victorian Mallee region. Rates of increase per 40 days were calculated from the smoothed abundance data and related to (i) estimates of food availability from cereal crops and grazed pasture and (ii) a density-dependent factor representing the effects of predation, disease and intrinsic regulatory processes such as dispersal and social organisation. Although the model represents reasonably well the main features of plagues, the strong seasonal variation in the modelled food supply is not matched by changes in the abundance, or rate of increase of mice, in non-plague years. These seasonal effects are likely to be more important in future models for survival and fecundity rates.

Keywords

Mus domesticus, population dynamics, predictive models, rate of increase, density-dependence, predator-regulation, disease, management

INTRODUCTION

THE INTRODUCED house mouse (*Mus domesticus*) is a major pest in the grain-growing regions of southern and eastern Australia. A wide range of climate, soils and farming regimes results in regional differences in the population dynamics of mice and in the opportunities for their management. For example, the Darling Downs in Queensland experiences relatively frequent outbreaks in contrast to the wheat belt of New South Wales, Victoria and South Australia where occasional severe eruptions are interspersed with long periods of low abundance of mice. The different dynamics imply that the major regulating factors are not uniform between regions.

There are at least nine models, with varying degrees of predictive ability, which describe the development of mouse plagues in Australia (Figure 1, Box 1). They include two broad-scale, regional models that rely on environmental or production data, five district and small-scale models that describe in detail the processes of plague formation, and two simplified 'process' models focusing on one or more mechanisms that might influence the rate of change of mouse abundance. The main features of these models are reviewed and their usefulness for predicting mouse plagues is compared.

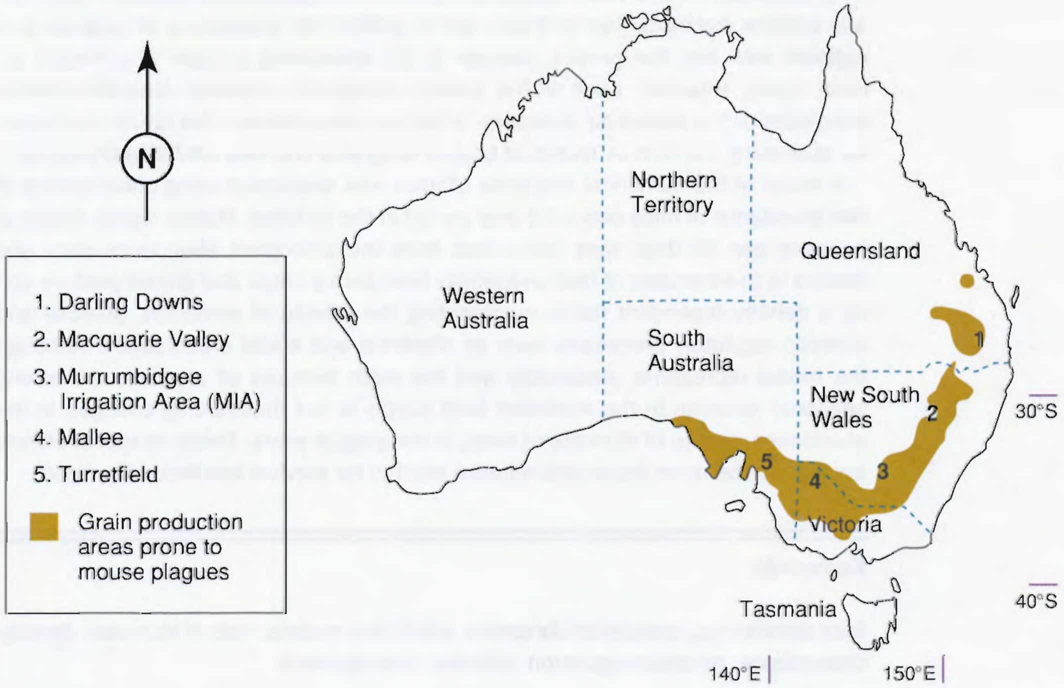


Figure 1.
Grain production areas of Australia that are prone to mouse plagues. The numbered locations correspond to the 'district' models listed in Box 1.

The model for predicting the onset, magnitude and duration of eruptions of mice in the cereal production areas of the Victorian Mallee is based on a 15-year data set that has tracked four major outbreaks of mice. In this paper we assess the usefulness of simple food-resource models for predicting the numerical response of mice during the period from 1983 to 1997. Potential areas for future development of the model for the Victorian Mallee are discussed, particularly in relation to assessing management options including the use of fertility control.

REVIEW OF CURRENT MODELS FOR MOUSE PLAGUES

Broad-scale models

New South Wales (NSW) and Victoria regional model

Saunders and Giles (1977) reviewed the historical records of mouse plagues from 1900 to 1970 for northern NSW, southern NSW and northern Victoria. Despite the difficulties in using consistent definitions for plagues and droughts, they found a strong positive correlation between eruptions of mice and the occurrence of a severe drought one or two years earlier. The model was not tested statistically but it can provide very early warning of a potential mouse plague. Predictions could be verified later by monitoring mouse populations (Saunders 1986). Saunders and Giles suggested several processes that might be responsible for the observed correlation. Few data are available to test their 'pathogen' hypothesis (see Singleton 1985). Depending on time lags in the responses of predator populations, the suggested mechanism of a drought-induced reduction in predation appears to be the converse of the *predation-regulation model* (below) and the explanation offered by Newsome and Corbett (1975) for a delayed response of mice to favourable conditions in central Australia. The emphasis on drought as a causal factor also has been criticised by Redhead (1982) on the grounds that it is confounded with the effects of drought-breaking winter–spring rainfall.

In its present form, the *NSW and Victoria regional model* is probably too vague to provide a prediction which can be the basis of management actions. The rainfall data on which the model is based are readily available but the most appropriate definition of a severe drought may require some clarification. Saunders (1986) did not specify when mouse populations should be monitored to verify the long-term prediction from the model. If coupled with additional surveys, the model's predictions, in the form of conditional probabilities suggested by Hone (1980), might constitute the first stage of a plague warning system.

South Australia regional model

Mutze (1989) documented the distribution and frequency of mouse plagues in South Australia from 1900 to 1984. These data were used first by Veitch and Anderson (1985), and then Mutze et al. (1990), to examine the relationships between the occurrence of mouse plagues and a range of independent variables including monthly rainfall, soil type, temperature indices and grain production.

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

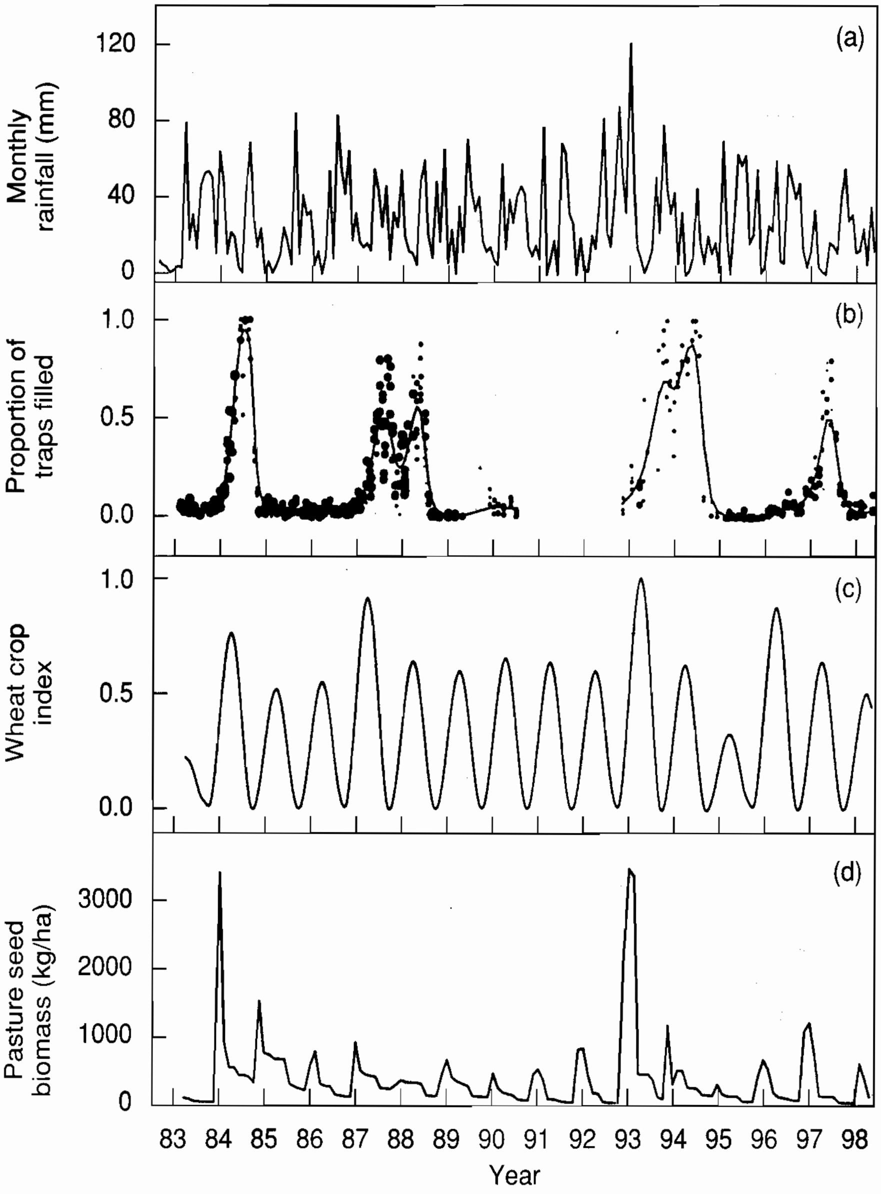


Figure 2.

(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:

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

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.

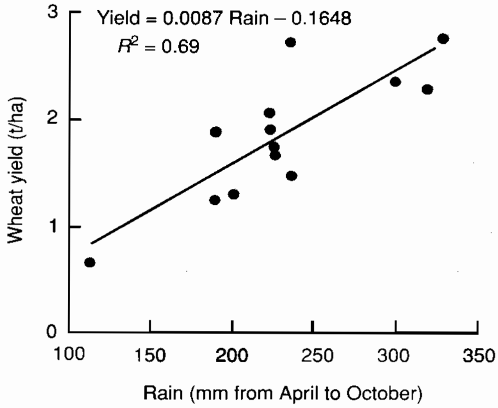


Figure 3.
Comparison of the summer wheat yields (tonnes/ha) at the Mallee Research Station with total rainfall (mm) over the preceding period from April to October. French and Schultz (1984) established a linear relationship between April to October rainfall and wheat yield using data from South Australia.

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:

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

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 $(R_{A\text{-}O}/348)^u$, 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:

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

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.

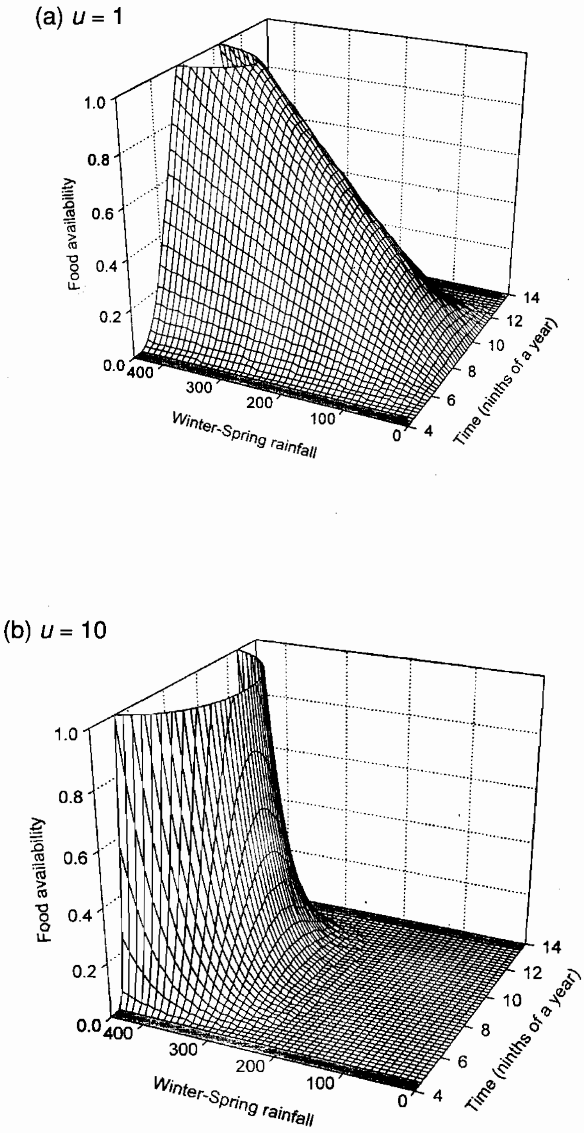


Figure 4.

The effect of the parameter, *u*, in equation (4) in modifying the index of food availability from wheat crops. Time, in units of 1/9th of a year, is measured from the 1st 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.

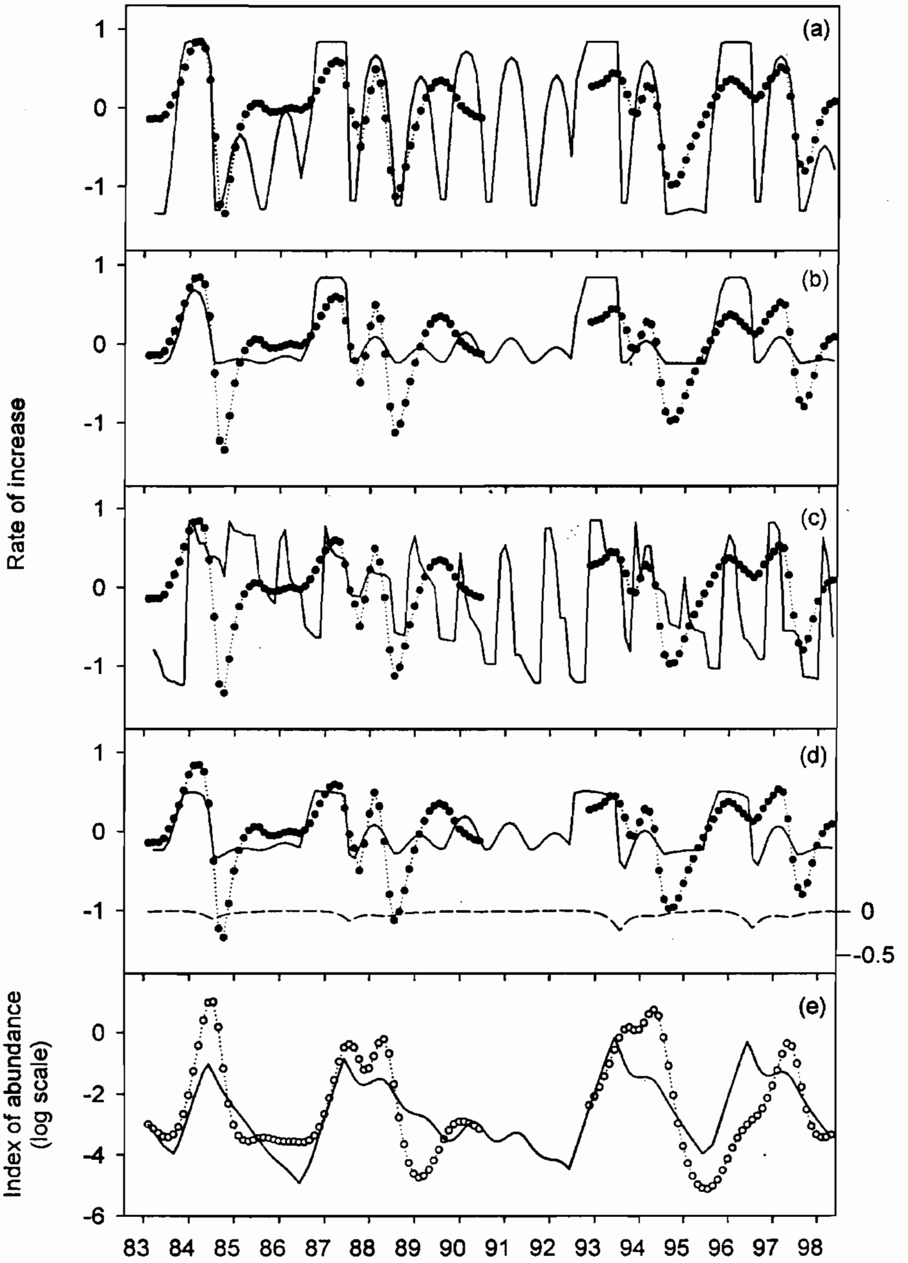


Figure 5.
(a)–(d) The observed (•) and modelled (—) rates of increase per 40 days. (e) The observed (o) and modelled (—) index of abundance of mice. For (a)–(d) the predicted trajectories correspond to the models (i)–(iv) listed in Table 2. The density-dependent contribution to *r* (- -) is shown in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

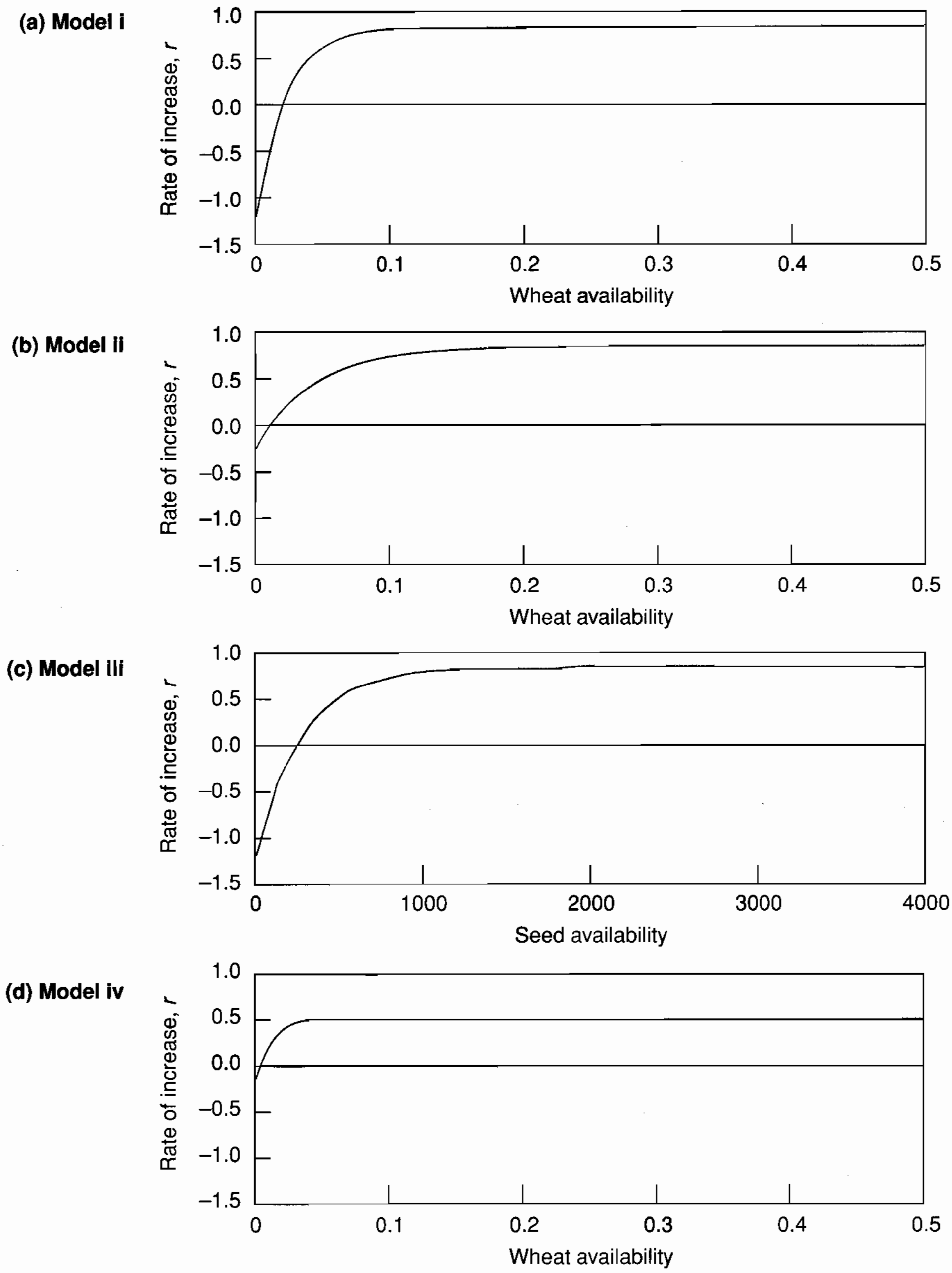


Figure 6.
The estimated numerical response functions corresponding to models (i)–(iv) in Table 2. Food is from (a) wheat crops, with r_{max} and a_W fixed at the observed values, (b) wheat crops, with only r_{max} fixed, (c) pasture seed in kg/ha, with r_{max} and a_W fixed, and (d) wheat crops with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

ACKNOWLEDGMENT

Part of this work was included in an unpublished review prepared by R. Pech for the Grains Research and Development Corporation in 1991.

REFERENCES

Barker, S.C. and Singleton, G.R. 1987. Conclusion. In: Singleton, G.R., ed., *Capillaria hepatica* (Nematoda) as a potential control agent of house mice. Proceedings of a CSIRO workshop, June 1987. Division of Wildlife and Rangelands Research, Technical Memorandum No. 28, Commonwealth Scientific and Industrial Research Organisation (CSIRO), Melbourne.

Barker, S.C., Singleton, G.R. and Spratt, D.M. 1991. Can the nematode *Capillaria hepatica* regulate abundance in wild house mice? Results of enclosure experiments in south-eastern Australia. Parasitology, 103, 439–449.

Bayliss, P. 1987. Kangaroo dynamics. In: Caughley, G., Shepherd, N. and Short, J., ed., Kangaroos: their ecology and management in the sheep rangelands of Australia. Cambridge, Cambridge University Press, 119–134.

Bomford, M. 1987a. Food and reproduction of wild house mice. I. Diet and breeding seasons in various habitats on irrigated cereal farms in New South Wales. Australian Wildlife Research, 14, 183–196.

Bomford, M. 1987b. Food and reproduction of wild house mice. II. A field experiment to examine the effect of food availability and food quality on breeding in spring. Australian Wildlife Research, 14, 197–206.

Bomford, M. 1987c. Food and reproduction of wild house mice. III. Experiments on the breeding performance of caged house mice fed rice-based diets. Australian Wildlife Research, 14, 207–218.

Bomford, M. and Redhead, T. 1987. A field experiment to examine the effects of food quality and population density on reproduction of wild house mice. Oikos, 48, 304–311.

Boonstra, R. and Redhead, T.D. 1994. Population dynamics of an outbreak population of house mice (*Mus domesticus*) in the irrigated rice-growing area of Australia. Wildlife Research, 21, 543–598.

Brown, P.R., Singleton, G.R. and Dunn, C. 1998. Best farm management practices for mouse control: testing of recommendations. In: The future of vertebrate pest management: direction for the third millennium. Proceedings of the 11th Australian Vertebrate Pest Conference, 3–8 May, Bunbury, Western Australia, 99–104.

Cairns, S.C. and Grigg, G.C. 1993. Population dynamics of red kangaroos *Macropus rufus* in relation to rainfall in the South Australian pastoral zone. Journal of Applied Ecology, 30, 444–458.

Caley, P. 1993. Population dynamics of feral pigs (*Sus scrofa*) in a tropical riverine habitat complex. Wildlife Research, 20, 625–636.

Cantrill, S. 1992. The population dynamics of the house mouse (*Mus domesticus*) in a dual crop agricultural ecosystem. PhD thesis, School of Life Science, Queensland University of Technology.

Caughley, G.C. 1977. Analysis of vertebrate populations. Chicester, John Wiley & Sons.

Caughley, G.C. 1987. Ecological relationships. In: Caughley, G., Shepherd, N. and Short, J., ed., Kangaroos: their ecology and management in the sheep rangelands of Australia. Cambridge, Cambridge University Press, 159–187.

Caughley, G.C. and Krebs, C.J. 1983. Are big mammals simply little mammals writ large? Oecologia, 59, 7–17.

Chambers, L.K., Singleton, G.R. and Hinds, L.A. 1999. Fertility control of wild mouse populations: the effects of hormonal competence and an imposed level of sterility. Wildlife Research, 26, 579–591.

Chambers, L.K., Singleton, G.R. and Hood, G.M. 1997. Immunocontraception as a potential control method of wild rodent populations. Belgian Journal of Zoology, 127, 145–156.

Choquenot, D. 1992. The outsiders: competition between introduced herbivores and domestic stock in rangeland grazing systems. In: Australian rangelands in a changing environment. Proceedings of the 7th Biennial Conference of the Australian Rangeland Society, 5–8 October 1992, Cobar, NSW. Cobar, Australian Rangeland Society, 106–116.

Choquenot, D. and Dexter, N. 1996. Spatial variation in food limitation: the effects of foraging constraints on the distribution and abundance of feral pigs in the rangelands. In: Floyd, R.B., Sheppard, A.W. and De Barro, P.J., ed., Frontiers of population ecology. Melbourne, CSIRO Publishing, 531–546.

Cornish, E.A., French, R.J. and Hill, G.W. 1980. Yield trends in the wheat belt of South Australia from 1896 to 1964. Agricultural Record No. 7. Adelaide, Government Printer.

Davey, C.C. and Fullagar, P.J. 1986. Changes in the abundance and distribution of raptors during a house mouse plague. Corella, 10, 52–54.

Foley, J.C. 1957. Droughts in Australia. Review of records from the earliest years of settlement to 1955. Melbourne, Commonwealth Bureau of Meteorology, Bulletin No. 43.

French, R.J. and Schultz, J.E. 1984. Water use efficiency of wheat in a Mediterranean-type environment. I. The relation between yield, water use and climate. Australian Journal of Agricultural Research, 35, 743–64.

Hone, J. 1980. Probabilities of house mouse (*Mus musculus*) plagues and their use in control. Australian Wildlife Research, 7, 417–420.

Jackson, R.J., Maguire, D.J., Hinds, L.A. and Ramshaw, I.A. 1998. Infertility in mice induced by a recombinant ectromelia virus expressing mouse zona pellucida glycoprotein 3. Biology of Reproduction, 58, 152–159.

Kay, B.J., Twigg, L.E., Korn, T.J. and Nicol, H.I. 1994. The use of artificial perches to increase predation on house mice (*Mus domesticus*) by raptors. Wildlife Research, 21, 95–106.

Krebs, C.J., Chitty, D., Singleton, G. and Boonstra, R. 1995a. Can changes in the social behaviour help to explain house mouse plagues in Australia? Oikos, 73, 429–434.

Krebs, C.J., Kenney, A.J. and Singleton, G.R. 1995b. Movements of feral house mice in agricultural landscapes. Australian Journal of Zoology, 43, 293–302.

Leirs, H., Stenseth, N.C., Nichols, J.D., Hines, J.E., Verhagen, R. and Verheyen, W. 1997. Stochastic seasonality and non-linear density-dependent factors regulate population size in an African rodent. Nature, 389, 176–180.

McCallum, H.I. 1993. Evaluation of a nematode (*Capillaria hepatica* Bancroft, 1893) as a control agent for populations of house mice (*Mus musculus domesticus* Schwartz and Schwartz, 1943). Revue Scientifique et Techique OIE (Office International des Epizooties), 12, 83–94.

McCallum, H.I. and Singleton, G.R. 1989. Models to assess the potential of *Capillaria hepatica* to control population outbreaks of house mice. Parasitology, 98,425–437.

Moore, A.D., Donnelly, J.R. and Freer, M. 1997. GRAZPLAN: decision support systems for Australia grazing enterprises. III. Pasture growth and soil moisture submodels, and the GrassGro DSS. Agricultural Systems, 55, 535–582.

Mutze, G.J. 1989. Mouse plagues in South Australian cereal-growing areas. I. Occurrence and distribution of plagues from 1900 to 1984. Australian Wildlife Research, 16, 677–683.

Mutze, G.J. 1991. Mouse plagues in South Australian cereal-growing areas. III. Changes in mouse abundance during plague and non-plague years, and the role of refugia. Wildlife Research, 18, 593–604.

Mutze, G.J., Veitch, L.G. and Miller, R.B. 1990. Mouse plagues in South Australian cereal-growing areas. II. An empirical model for prediction of plagues. Australian Wildlife Research, 17, 313–324.

Newsome, A.E. 1969a. A population study of house-mice temporarily inhabiting a South Australian wheatfield. Journal of Animal Ecology, 38, 341–359.

Newsome, A.E. 1969b. A population study of house-mice permanently inhabiting a reed-bed in South Australia. Journal of Animal Ecology, 38, 361–377.

Newsome, A.E. 1970. An experimental attempt to produce a mouse plague. Journal of Animal Ecology, 39, 299–311.

Newsome, A.E. 1971. The ecology of house-mice in cereal haystacks. Journal of Animal Ecology, 40, 1–15.

Newsome, A.E. and Corbett, L.K. 1975. VI. Outbreaks of rodents in semi-arid and arid Australia: causes, preventions, and evolutionary considerations. In: Prakash, I. and Ghosh, P.K., ed., Rodents in desert environments, The Hague, W. Junk, 117–153.

Newsome, A.E., Cowan, P.E. and Ives, P.M. 1982. Homing by wild house-mice displaced with or without the opportunity to see. Australian Wildlife Research, 9, 421–6.

Norton, G.A. 1988. Philosophy, concepts and techniques. In: Norton, G.A. and Pech, R.P., ed., Vertebrate pest management in Australia: a decision analysis/systems analysis approach. CSIRO Division of Wildlife and Ecology, Project Report No. 5, 1–17.

Olsen, P. 1981. The stimulating effect of a phytohormone, gibberellic acid, on reproduction of *Mus musculus*. Australian Wildlife Research, 8, 321–325.

Pech, R.P. and Hood, G.M. 1998. Foxes, rabbits, alternative prey and rabbit calicivirus disease: consequences of a new biological control agent for an outbreaking species in Australia. Journal of Applied Ecology, 35, 434–453.

Pech, R., Hood, G.M., McIlroy, J. and Saunders, G. 1997. Can foxes be controlled by reducing their fecundity? Reproduction Fertility and Development, 9, 41–50.

Pech, R.P., Sinclair, A.E. and Newsome, A.E. 1995. Predation models for primary and secondary prey. Wildlife Research, 22, 55–64.

Pech, R.P., Sinclair, A.E., Newsome, A.E. and Catling, P.C. 1992. Limits to predator regulation of rabbits in Australia: evidence from predator-removal experiments. Oecologia, 89, 102–112.

Redhead, T.D. 1982. Reproduction, growth and population dynamics of house mice in irrigated and non-irrigated cereal farms in New South Wales. PhD thesis, Department of Zoology, Australian National University, Canberra.

Redhead, T.D. 1987. Computer simulations of mouse plagues: plague formation and prevention. In: Working Papers of the 8th Australian Vertebrate Pest Control Conference, Coolangatta, Queensland, 186–194.

Redhead, T.D., Enright, N. and Newsome, A.E. 1985. Causes and predictions of outbreaks of *Mus musculus* in irrigated and non-irrigated cereal farms. Acta Zoologica Fennica, 173, 123–127.

Redhead, T.D. and Singleton, G.R. 1988a. The PICA Strategy for the prevention of losses caused by plagues of *Mus domesticus* in rural Australia. EPPO Bulletin, 18, 237–248.

Redhead, T.D., and Singleton, G.R. 1988b. An examination of the "PICA" strategy for the prevention of losses caused by plagues of the house mouse (*Mus domesticus*) in rural Australia. In: Norton, G.A. and Pech, R.P., ed., Vertebrate pest management in Australia: decision analysis/systems analysis approach. CSIRO Division of Wildlife and Ecology, Project Report No. 5, Melbourne, 18–37.

Saunders, G.R. 1986. Plagues of the house mouse in south eastern Australia. In: Salmon, T.P., ed., Proceedings of the twelfth vertebrate pest conference. Davis, University of California, 173–176.

Saunders, G.R. and Giles, J.R. 1977. A relationship between plagues of the house mouse, *Mus musculus* (Rodentia: Muridae) and prolonged periods of dry weather in southeastern Australia. Australian Wildlife Research, 4, 241–7.

Seif, E. and Pedersen, D.G. 1978. Effect of rainfall on the grain yield of spring wheat with an application to the analysis of adaptation. Australian Journal of Agricultural Research, 29,1107–1115.

Shellam, G.R. 1994. The potential of murine cytomegalovirus as a viral vector for immunocontraception. Reproduction Fertility and Development, 6, 401–409.

Sinclair, A.R.E., Olsen, P.D. and Redhead, T.D. 1990. Can predators regulate small mammal populations? Evidence from house mouse outbreaks in Australia. Oikos, 59, 382–392.

Singleton, G.R. 1985. Population dynamics of *Mus musculus* and its parasites in mallee wheatlands in Victoria during and after a drought. Australian Wildlife Research, 12, 437–445.

Singleton, G.R. 1987. Parasites of mice in the Victorian mallee wheatlands. In: Singleton, G.R., ed., *Capillaria hepatica* (Nematoda) as a potential control agent of house mice. CSIRO Division of Wildlife and Rangelands Research, Technical Memorandum No. 28, Melbourne, 12–14.

Singleton, G.R. 1989. Population dynamics of an outbreak of house mouse (*Mus domesticus*) in the mallee wheatlands of Australia—hypothesis of plague formation. Journal of Zoology (London), 219, 495–515.

Singleton, G.R. and Brown, P.R. 1998. Management of mouse plagues in Australia: integration of population ecology, bio-control and best farm practice. In: Cowan, D.P. and Feare, C.J., ed., Advances in vertebrate pest management. Fürth, Filander Verlag, 189–203.

Singleton, G.R. and Chambers, L.K. 1996. A manipulative field experiment to examine the effect of *Capillaria hepatica* (Nematoda) on wild mouse populations in southern Australia. International Journal for Parasitology, 26, 383–398.

Singleton, G.R., Chambers, L.K. and Spratt, D.M. 1995. An experimental field study to examine whether *Capillaria hepatica* (Nematoda) can limit house mouse populations in eastern Australia. Wildlife Research, 22, 31–53.

Singleton, G.R. and McCallum, H.I. 1990. The potential of *Capillaria hepatica* to control mouse plagues. Parasitology Today, 6, 190–193.

Singleton, G.R., McCallum, H.I. and Spratt, D.M. 1990. Fertility control of house mice using an hepatic nematode. Proceedings of the "Fertility Control of Wildlife" Conference, Melbourne, 21–23 November, 1990.

Singleton, G.R. and Redhead, T. 1989. House mouse plagues. In: Noble, J.C. and Bradstock, R.A., ed., Mediterranean landscapes in Australia: mallee ecosystems and their management. Melbourne, CSIRO, 418–33.

Singleton, G.R. and Redhead, T.D. 1990a. Structure and biology of house mouse populations that plague irregularly; an evolutionary perspective. Biological Journal of the Linnean Society, 41, 285–300.

Singleton, G.R. and Redhead, T.D. 1990b. Future prospects for biological control of rodents using micro- and macro-parasites. In: Quick, G.R., ed., Rodents and rice. Report and proceedings of an expert panel on rice rodent control, Los Baños, Sept. 10–14, 1990, IRRI, Philippines, 75–82.

Singleton, G.R., Smith, A.L., Shellam, G.R., Fitzgerald, N. and Müller, W.J. 1993. Prevalence of viral antibodies and helminths in field populations of house mice (*Mus domesticus*) in southeastern Australia. Epidemiology and Infection, 110, 399–417.

Singleton, G.R. and Spratt, D.M. 1986. The effects of *Capillaria hepatica* (Nematoda) on natality and survival to weaning in BALB/c mice. Australian Journal of Zoology, 34, 677–681.

Singleton, G.R. and Spratt, D.M. 1990. Biological control of mice—the challenge, progress and prospects. In: Noble, J.C., Joss, P.J. and Jones, G.K., ed., The mallee lands: a conservation perspective. Proceedings of the National Mallee Conference, Adelaide, April 1989. Melbourne, CSIRO, 238–242.

Singleton, G.R., Spratt, D.M., Barker, S.C. and Hodgson, P.F. 1991. The geographic distribution and host range of *Capillaria hepatica* (Bancroft) (Nematoda) in Australia. International Journal of Parasitology, 21, 945–957.

Smith, A.L., Singleton, G.R., Hansen, G.M. and Shellam, G. 1993. A serologic survey for viruses and *Mycoplasma pulmonis* among wild house mice (*Mus domesticus)* in southeastern Australia. Journal of Wildlife Diseases, 29, 219–229.

S-PLUS 4 Guide to Statistics. 1997. Seattle, Data Analysis Products Division, Mathsoft.

Spratt, D.M. 1990. The role of helminths in the biological control of mammals. International Journal for Parasitology, 20, 543–550.

Spratt, D.M. and Singleton, G.R. 1986. Studies of the life cycle, infectivity and clinical effects of *Capillaria hepatica* (Bancroft, 1893) (Nematoda) in mice (*Mus musculus*). Australian Journal of Zoology, 34, 663–675.

Spratt, D.M. and Singleton, G.R. 1987. Experimental embryonation and survival of eggs of *Capillaria hepatica* (Nematoda) under mouse burrow conditions in cereal-growing soils. Australian Journal of Zoology, 35, 337–341.

Tann, C.R., Singleton, G.R. and Coman, B.C. 1991. Diet of the house mouse (*Mus domesticus*) in the mallee wheatlands of north-western Australia. Wildlife Research, 18, 1–12.

Taylor, J.D., Stephens, C.P., Duncan, R.G. and Singleton, G.R. 1994. Polyarthritis in wild mice (*Mus musculus*) caused by *Streptobacillus moniliformis*. Australian Veterinary Journal, 71, 143–145.

Twigg, L.E. and Kay, B.J. 1994. The effects of microhabitat and weather on house mouse (*Mus domesticus*) numbers and the implications for management. Journal of Applied Ecology 31, 651–663.

Veitch, L.G. and Anderson, R.B. 1985, Mouse plagues in South Australia. CSIRO, Division of Mathematics and Statistics, Consulting Report No. SAC 85/17.

VENSIM® DSS Reference Supplement Version 3.0. 1997. Ventana Systems, Inc., USA.

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

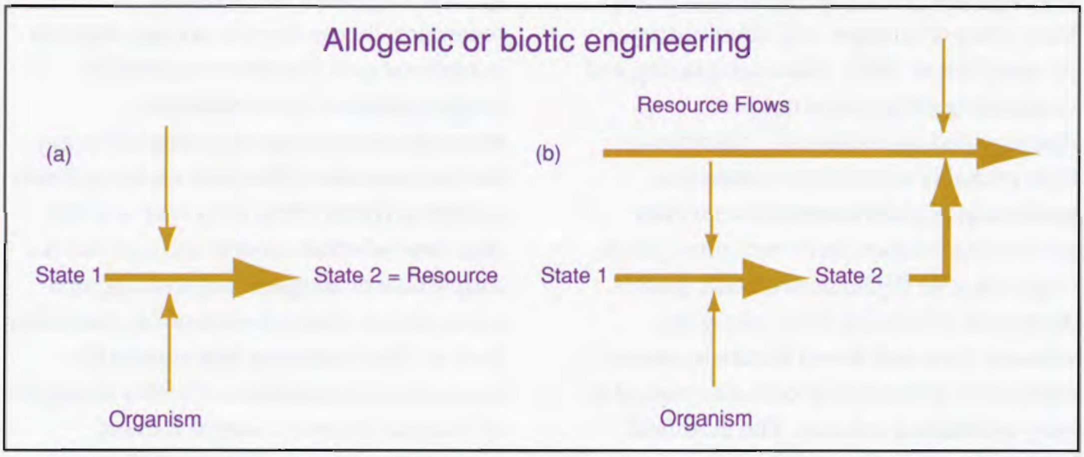


Figure 1.

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

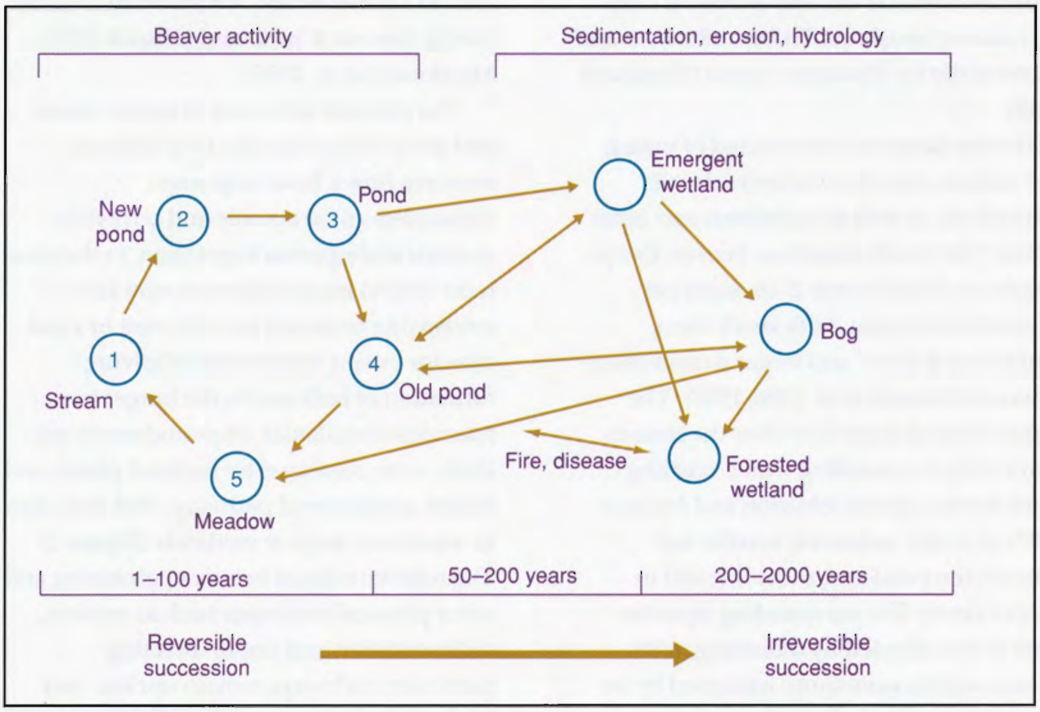


Figure 2.
Potential effects of beaver (*Castor canadensis*) on vegetation and landscape patterns, based on work by R.J. Naiman and colleagues in the boreal forests of northern Minnesota (after Naiman et al. 1988).

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).

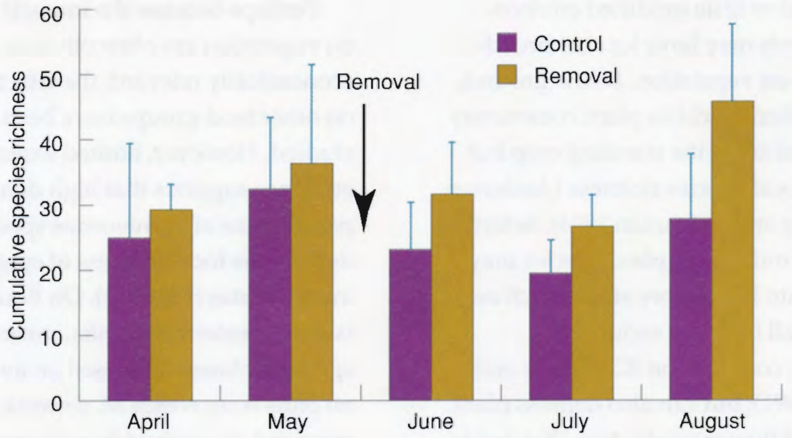
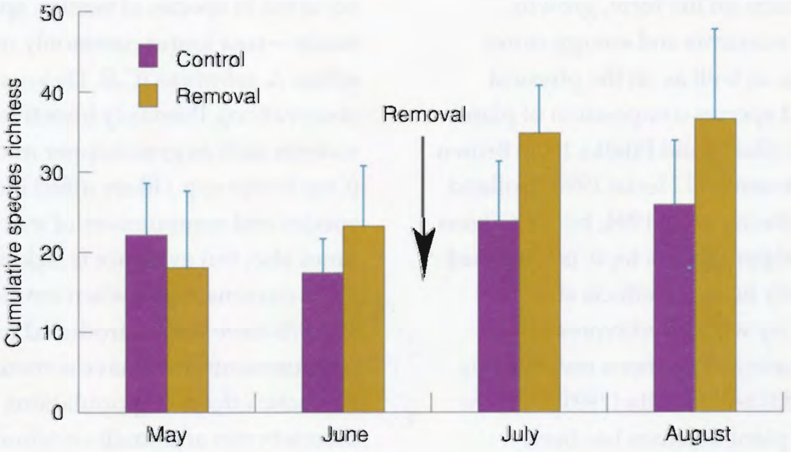


Figure 3.

Effects of rodent removal on species richness of invertebrates. (a) *Mus domesticus* was removed from trapping plots on Boullanger Island, Western Australia, and invertebrates sampled by pitfall trapping before and after removal in both the removal and control sites ($n = 3$ control, 3 removal plots, means shown ± standard error (SE); mean before/after ratios of species richness differed significantly between control and removal treatments, $P < 0.05$).



(b) *Apodemus sylvaticus* was removed from trapping plots in urban woodland in Oxford, United Kingdom, and invertebrates sampled in the same manner as in (a) ($n = 3$ control, 3 removal plots, means shown ± SE; mean before/after ratios of species richness differed significantly between control and removal treatments, $P < 0.01$). Methodological details are given in Dickman (1988), Dickman and Doncaster (1989; also unpublished data).

Extinctions and range contractions of many other species of large invertebrates and small vertebrates, including seabirds and flightless birds, have occurred on islands off the coast of New Zealand and throughout the Pacific following introductions of *R. rattus*, *Rattus exulans* and *Rattus norvegicus* (Steadman 1989; King 1990). Although rats appear to have been the only obvious threat introduced to some islands, in many cases their impact is difficult to distinguish from the effects wrought by other introduced species and by habitat change.

It remains equivocal also whether rat impacts were caused by predation, competition, introduction of diseases or other processes, although direct predation has been implicated by most authors (Smithers et al. 1977; King 1990; Atkinson 1996).

Finally, while most research has evaluated the direct trophic impacts of rodents, some recent work indicates that rodent foraging may have far-reaching indirect effects. In Californian grassland, Batzli and Pitelka (1970) showed that forb and grass cover increased in plots that excluded the herbivorous meadow vole, *Microtus californicus*, as compared with cover levels in control plots. An indirect effect of the vole exclusion was a dramatically increased abundance of the pillbug (*Armadillidium vulgare*) within two years; this species was apparently favoured by the denser vegetation or increased food resources that it contained. In analogous experiments, removal of *M. domesticus* from plots on Boullanger Island resulted in a 24% increase in litter depth within just three months, compared with a 16% decrease in litter depth on control plots over the same period (Figure 4).

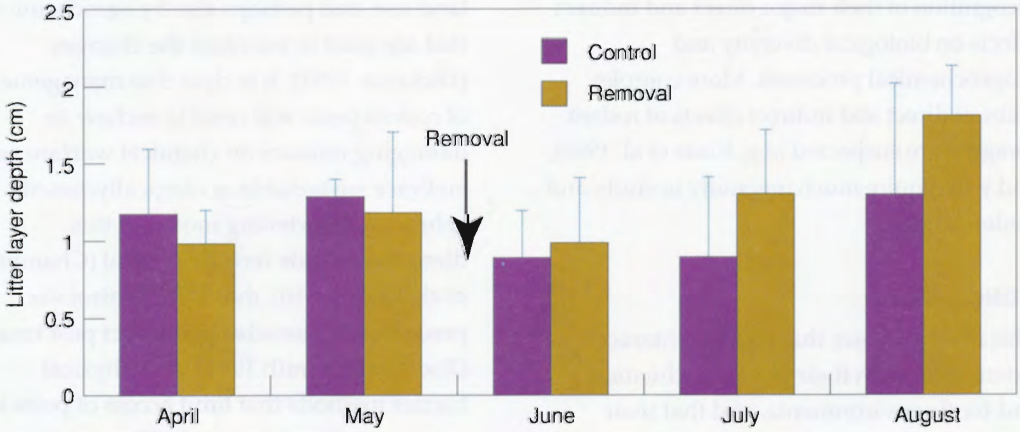


Figure 4.
Effects of removal of *Mus domesticus* on depth of the leaf litter layer on Boullanger Island, Western Australia (*n* = 3 control, 3 removal plots, means shown ± standard error; before/after ratios of mean litter depth differed significantly between control and removal treatments, $P < 0.01$). Further details are given in Dickman (1988).

Capture rates of the skinks (*Ctenotus fallens* and *Morethia lineoocellata*) increased in the *Mus*-removal plots by up to 35% (C.R. Dickman, personal observation), presumably because of the increased shelter afforded by the deep leaf litter or the more diverse food resources that were available (Figure 3a).

Over larger periods, rodent foraging can indirectly facilitate other taxa. In the Chihuahuan Desert, Thompson et al. (1991) demonstrated that the foraging activities and abundance of granivorous birds declined markedly in rodent exclusion plots over a period of 10 years. In the absence of rodents, especially kangaroo rats (*Dipodomys* spp.), litter accumulated and concealed seeds on the soil surface from the view of the visually foraging birds. In control plots by contrast, rodent foraging activities created areas of bare soil and trails, hence exposing seeds and facilitating access by birds. Brown and Heske (1990) considered kangaroo rats in the Chihuahuan Desert to be a keystone guild in recognition of their major direct and indirect effects on biological diversity and biogeochemical processes. More complex webs of direct and indirect effects of rodent foraging are suspected (e.g. Klaas et al. 1998), and will require much ingenuity to study and understand.

CONCLUSIONS

This review shows that rodents interact extensively with their physical, chemical and biotic environments, and that their activities have complex but often beneficial effects on other organisms across a broad range of spatial and temporal scales. This should not be surprising, because of the great species richness, abundance and ubiquity of rodents in terrestrial environments.

With respect to the management of rodent pests, the review also allows the conclusion to be drawn that we must be more clever and more focused in our approaches to rodent control. In many regions, broad-scale application of poison remains the favoured control method (Buckle and Smith 1994; Singleton and Petch 1994). In the 1993 mouse plague in south-eastern Australia, for example, some 350,000 ha of cropland in South Australia alone were baited with the poison strychnine (Caughley et al. 1994). Such broad-scale campaigns may reduce the numbers of the target pest, but very likely decimate populations of non-target species (Dickman 1993), including those with potentially positive effects on the physical and biotic environment. In the wheat-growing areas of New South Wales, native rodents have been virtually eliminated by introduced species, changes in land use, and perhaps also by agrochemicals that are used to maintain the changes (Dickman 1993). It is clear that management of rodent pests will need to eschew its damaging reliance on chemical warfare and embrace sustainable, ecologically-based solutions. Heartening moves in this direction include fertility control (Chambers et al., Chapter 10), mortality control via predators or parasites that target pest taxa (Buckle and Smith 1994), and physical barrier methods that limit access of pests to crop areas (Singleton et al., Chapter 8).

ACKNOWLEDGMENTS

I thank G. Singleton and L. Hinds for the opportunity to prepare this review, and

Z. Zhang and other members of the Beijing organising committee for hosting the conference on rodent biology and management. R. Baxter, C.-L. Beh, R. Shine, N. Shchipanov and G. Sjöberg helped to find elusive references or kindly provided reprints of the manuscripts, E. Gerzabek and G. McNaught assisted with preparation of this manuscript, and P. Brown, C. McKechnie and G. Singleton provided critical comments. I am grateful also to the Australian Research Council for funding my work on rodents, and to Commonwealth Scientific and Industrial Research Organisation (CSIRO) Wildlife and Ecology for funding my attendance at the Beijing conference. This paper has benefited from discussions with numerous conference participants.

REFERENCES

Abbott, R.J., Bevercombe, G.P. and Rayner, A.D.M. 1977. Sooty bark disease of sycamore and the grey squirrel. Transactions of the British Mycological Society, 69, 507–508.

Agnew, W., Uresk, D.W. and Hansen, R.M. 1986. Flora and fauna associated with prairie dog colonies and adjacent ungrazed mixed-grass prairie in western South Dakota. Journal of Range Management, 39, 135–139.

Andersen, D.C. 1987. Below-ground herbivory in natural communities: a review emphasizing fossorial animals. Quarterly Review of Biology, 62, 261–286.

Anstee, S.D., Roberts, J.D. and O'Shea, J.E. 1997. Social structure and patterns of movement of the western pebble-mound mouse, *Pseudomys chapmani*, at Marandoo, Western Australia. Wildlife Research, 24, 295–305.

Atkinson, I.A.E. 1985. The spread of commensal species of *Rattus* to oceanic islands and their effects on island avifaunas. In: Moors, P.J., ed., Conservation of island birds. Cambridge, U.K., International Council for Bird Preservation, 35–81.

Atkinson, I.A.E. 1996. Introductions of wildlife as a cause of species extinctions. Wildlife Biology, 2, 135–141.

Barnett, S.A. 1975. The rat. Chicago, U.S.A., University of Chicago Press, 318p.

Batzli, G.O. and Pitelka, F.A. 1970. Influence of meadow mouse populations on California grassland. Ecology, 51, 1027–1039.

Beuchner, H.K. 1942. Interrelationships between the pocket gopher and land use. Journal of Mammalogy, 23, 346–348.

Borghi, C.E. and Giannoni, S.M. 1997. Dispersal of geophytes by mole-voles in the Spanish Pyrenees. Journal of Mammalogy, 78, 550–555.

Bright, P.W. and Morris, P.A. 1996. Why are dormice rare? A case study in conservation biology. Mammal Review, 26, 157–187.

Bronner, G.N. 1992. Burrow system characteristics of seven small mammal species (Mammalia: Insectivora; Rodentia; Carnivora). Koedoe, 35, 125–128.

Brown, J.H. and Harney, B.A. 1993. Population and community ecology of heteromyid rodents in temperate habitats. In: Genoways, H.H. and Brown, J.H., ed., Biology of the Heteromyidae. Provo, U.S.A., The American Society of Mammalogists, 618–651.

Brown, J.H. and Heske, E.J. 1990. Control of a desert–grassland transition by a keystone rodent guild. Science, 250, 1705–1707.

Brown, J.H., Reichman, O.J. and Davidson, D.W. 1979. Granivory in desert ecosystems. Annual Review of Ecology and Systematics, 10, 201–227.

Buckle, A.P. and Smith, R.H. (ed.) 1994. Rodent pests and their control. Wallingford, U.K., CAB International, 405p.

Cantor, L.F. and Whitham, T.G. 1989. Importance of belowground herbivory: pocket gophers may limit aspen to rock outcrop refugia. Ecology, 70, 962–970.

Carthew, S.M. and Goldingay, R.L. 1997. Non-flying mammals as pollinators. Trends in Ecology and Evolution, 12, 104–108.

Caughley, J., Bomford, M., Parker, B., Sinclair, R., Griffiths, J. and Kelly, D. 1998. Managing vertebrate pests: rodents. Canberra, Australia, Bureau of Resource Sciences and Grains Research and Development Corporation, 130p.

Caughley, J., Monamy, V. and Heiden, K. 1994. Impact of the 1993 mouse plague. Canberra, Australia, Grains Research and Development Corporation, 73p.

Chew, R.M. and Whitford, W.G. 1992. A long-term positive effect of kangaroo rats (*Dipodomys spectabilis*) on creosotebushes (*Larrea tridentata*). Journal of Arid Environments, 22, 375–386.

Chitty, D. and Southern, H.N. (ed.) 1954. Control of rats and mice, 3 volumes. Oxford, U.K., Clarendon Press, 305, 532, 225p.

Cirmo, C.P. and Driscoll, C.T. 1993. Beaver pond geochemistry—acid neutralising capacity generation in a headwater wetland. Wetlands, 13, 277–292.

Claridge, A.W. and May, T.W. 1994. Mycophagy among Australian mammals. Australian Journal of Ecology, 19, 251–275.

Claridge, A.W., Tanton, M.T., Seebeck, J.H., Cork, S.J. and Cunningham, R.B. 1992. Establishment of ectomycorrhizae on the roots of two species of *Eucalyptus* from fungal spores contained in the faeces of the long-nosed potoroo (*Potorous tridactylus*). Australian Journal of Ecology, 17, 207–217.

Clifford, H.F., Wiley, G.M. and Casey, R.J. 1993. Macroinvertebrates of a beaver-altered boreal stream of Alberta, Canada, with special reference to the fauna on the dams. Canadian Journal of Zoology, 71, 1439–1447.

Coley, P.D. and Barone, J.A. 1996. Herbivory and plant defenses in tropical forests. Annual Review of Ecology and Systematics, 27, 305–335.

Contreras, L.C. and Gutiérrez, J.R. 1991. Effects of the subterranean herbivorous rodent *Spalacopus cyanus* on herbaceous vegetation in arid coastal Chile. Oecologia, 87, 106–109.

Copley, P. 1988. The stick-nest rats of Australia. Adelaide, Australia, Department of Environment and Planning, South Australia, 277p.

Coppock, D.L., Detling, J.K., Ellis, J.E. and Dyer, M.I. 1983. Plant–herbivore interactions in a North American mixed-grass prairie I. Effects of black-tailed prairie dogs on intraseasonal aboveground plant biomass and nutrient dynamics and plant species diversity. Oecologia, 56, 1–9.

Corbet, G.B. and Harris, S., (ed.) 1991. The handbook of British mammals. Oxford, U.K., Blackwell Scientific Publications, 588p.

Corbet, G.B. and Hill, J.E. 1991. A world list of mammalian species. London, U.K., Natural History Museum Publications and Oxford University Press, 243p.

Cox, G.W. and Gakahu, C.G. 1985. Mima mound microtopography and vegetation pattern in Kenyan savannas. Journal of Tropical Ecology, 1, 23–36.

Cox, G.W. and Gakahu, C.G. 1986. A latitudinal test of the fossorial rodent hypothesis of mima mound origin. Zeitschrift für Geomorphologie, 30, 485–501.

Cox, G.W., Lovegrove, B.G. and Siegfried, W.R. 1987. The small stone content of mima-like mounds in the South African Cape region: implications for mound origin. Catena, 14, 165–176.

Crawley, M.J. 1983. Herbivory: the dynamics of animal–plant interactions. Oxford, U.K., Blackwell Scientific Publications, 437p.

Dahlsted, K.J., Sather-Blair, S., Worcester, B.K. and Klukas, R. 1981. Application of remote sensing to prairie dog management. Journal of Range Management, 34, 218–223.

Dickman, C.R. 1988. Body size, prey size, and community structure in insectivorous mammals. Ecology, 69, 569–580.

Dickman, C.R. 1993. The biology and management of native rodents of the arid zone in New South Wales. Sydney, Australia, New South Wales National Parks and Wildlife Service, 149p.

Dickman, C.R. 1996. Vagrants in the desert. Nature Australia, 25, 54–62.

Dickman, C.R. and Doncaster, C.P. 1989. The ecology of small mammals in urban habitats. II. Demography and dispersal. Journal of Animal Ecology, 58, 119–127.

Dickman, C.R. and Read, D.G. 1992. The biology and management of dasyurids of the arid zone in New South Wales. Sydney, Australia, New South Wales National Parks and Wildlife Service, 112p.

Ebenhard, T. 1988. Introduced birds and mammals and their ecological effects. Viltrevy, 13, 1–107.

Emmons, L.H. 1992. The roles of small mammals in tropical rainforest. In: Ismail, G., Mohamed, M. and Omar, S., ed., Forest biology and conservation in Borneo. Kota Kinabalu, Sabah, Center for Borneo Studies, 512–513.

Evgenjeva, T.P. and Fadeeva, E.O. 1996. The distant consequence of defoliant effect on cytogenetic indexes of cornea cells in some rodents from South Vietnam. In: Sokolov, V.E. and Shilova, S.A., ed., Long-term biological consequences of the war in South Vietnam. Moscow, Russia, Tropical Department, Severtsov Institute of Ecology and Evolution, Russian Academy of Sciences, 188–200.

Fleming, T.H. and Sosa, V.J. 1994. Effects of nectarivorous and frugivorous mammals on reproductive success of plants. Journal of Mammalogy, 75, 845–851.

Francis, M.M., Naiman, R.J. and Melillo, J.M. 1985. Nitrogen fixation in subarctic streams influenced by beaver (*Castor canadensis*). Hydrobiologia, 121, 193–202.

Frey, J.K. 1992. Response of a mammalian faunal element to climatic changes. Journal of Mammalogy, 73, 43–50.

Galil, J. 1967. On the dispersal of the bulbs of *Oxalis cernua* Thunb, by mole-rats (*Spalax ehrenbergi* Nehring). Journal of Ecology, 55, 787–792.

Garkaklis, M.J., Bradley, J.S. and Wooller, R.D. 1998. The effects of woylie (*Bettongia penicillata*) foraging on soil water repellency and water infiltration in heavy textured soils in southwestern Australia. Australian Journal of Ecology, 23, 492–496.

Gibson, D.J., Freeman, C.C. and Hulbert, L.C. 1990. Effects of small mammal and invertebrate herbivory on plant species richness and abundance in tallgrass prairie. Oecologia, 84, 169–175.

Gómez-Garcia, D., Borghi, C.E. and Giannoni, S.M. 1995. Vegetation changes on subalpine plant communities induced by pine vole mounds. Vegetatio, 117, 61–67.

Gurnell, J. 1983. Squirrel numbers and the abundance of tree seeds. Mammal Review, 13, 133–148.

Gutterman, Y. 1982. Observations on the feeding habits of the Indian crested porcupine (*Hystrix indica*) and the distribution of some hemicryptophytes and geophytes in the Negev Desert highlands. Journal of Arid Environments, 5, 261–268.

Gutterman, Y. and Herr, N. 1981. Influences of porcupine (*Hystrix indica*) activity on the slopes of the northern Negev mountains—germination and vegetation renewal in different geomorphological types and slope directions. Oecologia, 51, 332–334.

Hägglund, A. and Sjöberg, G. 1999. Effects of beaver dams on the fish fauna of forest streams. Forest Ecology and Management, 115, 259–266.

Hansell, M.H. 1993. The ecological impact of animal nests and burrows. Functional Ecology, 7, 5–12.

Harris, S. and Trout, R.C. 1991. Genus *Micromys*. In: Corbet, G.B. and Harris, S., ed., The handbook of British mammals. Oxford, U.K., Blackwell Scientific Publications, 233–239.

Hartnett, D.C. and Keeler, K.H. 1995. Population processes. In: Joern, A. and Keeler, K.H., ed., The changing prairie: North American grasslands. New York, U.S.A., Oxford University Press, 82–99.

Heth, G. 1991. The environmental impact of subterranean mole rats (*Spalax ehrenbergi*) and their burrows. Symposia of the Zoological Society of London, 63, 265–280.

Hobbs, R.J. and Hobbs, V.J. 1987. Gophers and grassland: a model of vegetation response to patchy soil disturbance. Vegetatio, 69, 141–146.

Hobbs, R.J. and Mooney, H.A. 1991. Effects of rainfall variability and gopher disturbance on serpentine annual grassland dynamics. Ecology, 72, 59–68.

Hodkinson, I.D. 1975. Energy flow and organic matter decomposition in an abandoned beaver pond ecosystem. Oecologia, 21, 131–139.

Holland, E.A., Parton, W.J., Detling, J.K. and Coppock, D.L. 1992. Physiological responses of plant populations to herbivory and their consequences for ecosystem nutrient flow. American Naturalist, 140, 685–706.

Hoogland, J.L. 1994. The black–tailed prairie dog: social life of a burrowing mammal. Chicago, U.S.A., University of Chicago Press, 576p.

Huntly, N. 1991. Herbivores and the dynamics of communities and ecosystems. Annual Review of Ecology and Systematics, 22, 477–503.

Huntly, N. and Inouye, R. 1988. Pocket gophers in ecosystems: patterns and mechanisms. Bioscience, 38, 786–793.

Huntly, N. and Reichman, O.J. 1994. Effects of subterranean mammalian herbivores on vegetation. Journal of Mammalogy, 75, 852–859.

Ingham, R.E. and Detling, J.K. 1984. Plant–herbivore interactions in a North American mixed-grass prairie III. Soil nematode populations and root biomass on *Cynomys ludovicianus* colonies and adjacent uncolonized areas. Oecologia, 63, 307–313.

Inouye, R.S., Huntly, N.J. and Tilman, D. 1987a. Response of *Microtus pennsylvanicus* to vegetation fertilized with various nutrients, with particular emphasis on sodium and nitrogen concentrations in plant tissues. Holarctic Ecology, 10, 110–113.

Inouye, R.S., Huntly, N.J., Tilman, D. and Tester, J.R. 1987b. Pocket gophers (*Geomys bursarius*), vegetation, and soil nitrogen along a successional sere in east central Minnesota. Oecologia, 72, 178–184.

Inouye, R.S., Huntly, N. and Wasley, G.A. 1997. Effects of pocket gophers (*Geomys bursarius*) on microtopographic variation. Journal of Mammalogy, 78, 1144–1148.

Janzen, D.H. 1981. Patterns of herbivory in a tropical deciduous forest. Biotropica, 13, 271–282.

Jarvis, J.U.M. and Sale, J.B. 1971. Burrowing and burrow patterns of East African mole-rats *Tachyoryctes*, *Heliophobius*, and *Heterocephalus*. Journal of Zoology, London, 163, 451–479.

Jefferies, R.L., Klein, D.R. and Shaver, G.R. 1994. Vertebrate herbivores and northern plant communities: reciprocal influences and responses. Oikos, 71, 193–206.

Johnston, C.A. 1995. Effects of animals on landscape pattern. In: Hansson, L., ed., Mosaic landscapes and ecological processes. London, U.K., Chapman and Hall, 57–80.

Johnston, C.A. and Naiman, R.J. 1987. Boundary dynamics at the aquatic –terrestrial interface: the influence of beaver and geomorphology. Landscape Ecology, 1, 47–57.

Johnston, C.A. and Naiman, R.J. 1990. Browse selection by beaver: effects on riparian forest composition. Canadian Journal of Forest Research, 20, 1036–1043.

Jones, C.G., Lawton, J.H. and Shachak, M. 1994. Organisms as ecosystem engineers. Oikos, 69, 373–386.

Jones, C.G., Lawton, J.H. and Shachak, M. 1997. Positive and negative effects of organisms as physical ecosystem engineers. Ecology, 78, 1946–1957.

Karban, R. and Myers, J.H. 1989. Induced plant responses to herbivory. Annual Review of Ecology and Systematics, 20, 331–348.

Keast, A. and Fox, M.G. 1990. Fish community structure, spatial distribution and feeding ecology in a beaver pond. Environmental Biology of Fishes, 27, 201–214.

Kemper, C. 1981. Description of *Pseudomys novaehollandiae* burrows located with radioisotopes. Australian Mammalogy, 4, 141–143.

King, C.M. (ed.) 1990. The handbook of New Zealand mammals. Auckland, New Zealand, Oxford University Press, 600p.

Kiviat, E. 1978. Vertebrate use of muskrat lodges and burrows. Estuaries, 1, 196–200.

Klaas, B.A., Danielson, B.J. and Moloney, K.A. 1998. Influence of pocket gophers on meadow voles in a tallgrass prairie. Journal of Mammalogy, 79, 942–952.

Knight, M.H. 1984. The ecophysiology of the African giant rat *Cricetomys gambianus* (Waterhouse). Pretoria, South Africa, University of Pretoria, unpublished M.Sc. dissertation.

Kostelecka-Myrcha, A., Myrcha, A. and Gutkowska, H.E. 1981. Hematological and morphophysiological indices of some rodents under conditions of varying industrial pressure. Polish Ecological Studies, 7, 145–153.

Lepage, P. and Parker, G.H. 1988. Copper, nickel, and iron levels in pelage of red squirrels living near the ore smelters at Sudbury, Ontario, Canada. Canadian Journal of Zoology, 66, 1631–1637.

Lumer, C. 1980. Rodent pollination of *Blakea* (Melastomataceae) in a Costa Rican cloud forest. Brittonia, 32, 512–517.

Macdonald, D.W., Tattersall, F.H., Brown, E.D. and Balharry, D. 1995. Reintroducing the European beaver to Britain: nostalgic meddling or restoring biodiversity? Mammal Review, 25, 161–200.

MacKinnon, K., Hatta, G., Halim, H. and Mangalik, A. 1996. The ecology of Kalimantan. Hong Kong City, Hong Kong, Periplus Editions, 802p.

Madsen, T. and Shine, R. 1999. Rainfall and rats: climatically-driven dynamics of a tropical rodent population. Australian Journal of Ecology, 24, 80–89.

Martinsen, G.D., Cushman, J.H. and Whitham, T.G. 1990. Impact of pocket gopher disturbance on plant species diversity in a shortgrass prairie community. Oecologia, 83, 132–138.

Martinsen, G.D., Driebe, E.M. and Whitham, T.G. 1998. Indirect interactions mediated by changing plant chemistry: beaver browsing benefits beetles. Ecology, 79, 192–200.

Maser, C., Trappe, J.M. and Nassbaum, R.A. 1978. Fungal–small mammal interrelationships with emphasis on Oregon coniferous forests. Ecology, 54, 799–809.

McDowell, D.M. and Naiman, R.J. 1986. Structure and function of a benthic invertebrate stream community as influenced by beaver (*Castor canadensis*). Oecologia, 68, 481–489.

McNaught, G.H. 1994. The foraging behaviour of an Australian desert rodent, *Notomys alexis* (Family Muridae). Sydney, Australia, University of Sydney, unpublished Honours dissertation.

Meadows, P.S. and Meadows, A. (ed.) 1991. The environmental impact of burrowing animals and animal burrows. Oxford, U.K., Clarendon Press.

Medway, Lord 1983. The wild mammals of Malaya (Peninsular Malaysia) and Singapore. Kuala Lumpur, Malaysia, Oxford University Press, 128p.

Mielke, H.W. 1977. Mound building by pocket gophers (Geomyidae): their impact on soils and vegetation in North America. Journal of Biogeography, 4, 171–180.

Moloney, K.A., Levin, S.A., Chiariello, N.R. and Buttel, L. 1992. Pattern and scale in a serpentine grassland. Theoretical Population Biology, 41, 257–276.

Murray, B.R., Dickman, C.R., Watts, C.H.S. and Morton, S.R. 1999. Dietary ecology of Australian desert rodents. Wildlife Research, 26, 421–437.

Naiman, R.J., Johnston, C.A. and Kelly, J.C. 1988. Alteration of North American streams by beaver. Bioscience, 38, 753–762.

Naiman, R.J., Manning, T. and Johnston, C.A. 1991. Beaver population fluctuations and tropospheric methane emissions in boreal wetlands. Biogeochemistry, 12, 1–15.

Naiman, R.J., Melillo, J.M. and Hobbie, J.E. 1986. Ecosystem alteration of boreal forest streams by beaver (*Castor canadensis*). Ecology, 67, 1254–1269.

Naiman, R.J., Pinay, G., Johnston, C.A. and Pastor, J. 1994. Beaver influences on the long-term biogeochemical characteristics of boreal forest drainage networks. Ecology, 75, 905–921.

Noy-Meir, I. 1988. Dominant grasses replaced by ruderal forbs in a vole year in undergrazed mediterranean grasslands in Israel. Journal of Biogeography, 15, 579–587.

Palmer, E. 1886. Notes on a great visitation of rats in the north and north-western plain country of Queensland, in 1869 and 1870. Proceedings of the Royal Society of Queensland, 1885, 193–198.

Pastor, J., Moen, R. and Cohen, Y. 1997. Spatial heterogeneities, carrying capacity, and feedbacks in animal–landscape interactions. Journal of Mammalogy, 78, 1040–1052.

Pinay, G. and Naiman, R.J. 1991. Short-term hydrologic variations and nitrogen dynamics in beaver created meadows. Archiv für Hydrobiologie, 123, 187–205.

Predavec, M. and Dickman, C.R. 1994. Population dynamics and habitat use of the long-haired rat (*Rattus villosissimus*) in south-western Queensland. Wildlife Research, 21, 1–10.

Recher, H.F. 1981. Nectar-feeding and its evolution among Australian vertebrates. In: Keast, A., ed., Ecological biogeography of Australia. The Hague, Netherlands, Dr W. Junk, 1637–1648.

Reddell, P., Spain, A.V. and Hopkins, M. 1997. Dispersal of spores of mycorrhizal fungi in scats of native mammals in tropical forests of north eastern Australia. Biotropica, 29, 184–192.

Reichman, O.J. and Price, M.V. 1993. Ecological aspects of heteromyid foraging. In: Genoways, H.H. and Brown, J.H., ed., Biology of the Heteromyidae. Provo, U.S.A., The American Society of Mammalogists, 539–574.

Reichman, O.J. and Smith, S.C. 1990. Burrows and burrowing behavior by mammals. Current Mammalogy, 2, 197–244.

Reichman, O.J. and Smith, S.C. 1991. Responses to simulated leaf and root herbivory by a biennial, *Tragopogon dubius*. Ecology, 72, 116–124.

Ross, B.A., Tester, J.R. and Breckenridge, W.J. 1968. Ecology of mima-type mounds in north-western Minnesota. Ecology, 49, 172–177.

Schlosser, I.J. 1995. Dispersal, boundary processes, and trophic-level interactions in streams adjacent to beaver ponds. Ecology, 76, 908–925.

Sinclair, A.R.E. 1989. Population regulation in animals. In: Cherrett, J.M., ed., Ecological concepts. Oxford, U.K., Blackwell Scientific Publications, 197–241.

Singleton, G.R. and Petch, D.A. 1994. A review of the biology and management of rodent pests in southeast Asia. Canberra, Australia, Australian Centre for International Agricultural Research, 65p.

Skinner, J.D. and Smithers, R.H.N. 1990. The mammals of the southern African subregion. Pretoria, South Africa, University of Pretoria, 769p.

Smithers, C., McAlpine, D., Colman, P. and Gray, M. 1977. Island invertebrates. In: Smith, N., ed.,. Lord Howe Island. Sydney, Australia, The Australian Museum, 23–26.

Snodgrass, J.W. and Meffe, G.K. 1998. Influence of beavers on stream fish assemblages: effects of pond age and watershed position. Ecology, 79, 928–942.

Sokolov, V.E., Shilova, S.A. and Shchipanov, N.A. 1994. Peculiarities of small mammals populations as criteria for estimating anthropogenic impacts on tropical ecosystems. International Journal of Ecology and Environmental Sciences, 20, 375–386.

Steadman, D.W. 1989. Extinction of birds in eastern Polynesia: a review of the record, and comparisons with other Pacific Island groups. Journal of Archaeological Science, 16, 177–205.

Steinberger, Y. and Whitford, W.G. 1983. The contribution of rodents to decomposition processes in a desert ecosystem. Journal of Arid Environments, 6, 177–181.

Tardiff, S.E. and Stanford, J.A. 1998. Grizzly bear digging: effects on subalpine meadow plants in relation to mineral nitrogen availability. Ecology, 79, 2219–2228.

Thompson, D.B., Brown, J.H. and Spencer, W.D. 1991. Indirect facilitation of granivorous birds by desert rodents: experimental evidence from foraging patterns. Ecology, 72, 852–863.

Tory, M.K., May, T.W., Keane, P.J. and Bennett, A.F. 1997. Mycophagy in small mammals: a comparison of the occurrence and diversity of hypogeal fungi in the diet of the long-nosed potoroo *Pototous tridactylus* and the bush rat *Rattus fuscipes* from southwestern Victoria, Australia. Australian Journal of Ecology, 22, 460–470.

Twigg, G.I. 1978. The role of rodents in plague dissemination: a worldwide review. Mammal Review, 8, 77–110.

Vander Wall, S.B. 1990. Food hoarding in animals. Chicago, U.S.A., University of Chicago Press, 445p.

Vander Wall, S.B. 1997. Dispersal of singleleaf piñon pine (*Pinus monophylla*) by seed-caching rodents. Journal of Mammalogy, 78, 181–191.

Van Tets, I.G. 1997. Extraction of nutrients from *Protea* pollen by African rodents. Belgian Journal of Zoology, 127, 59–65.

Whicker, A.D. and Detling, J.K. 1988. Ecological consequences of prairie dog disturbances. Bioscience, 38, 778–785.

Whittaker, J.C., List, E., Tester, J.R. and Christian, D.P. 1991. Factors influencing meadow vole, *Microtus pennsylvanicus*, distribution in Minnesota. Canadian Field-Naturalist, 105, 403–405.

Wiens, D., Rourke, J.P., Casper, B.B., Rickard, E.A., La Pine, T.R., Peterson, C.J. and Channing, A. 1983. Non-flying mammal pollination of southern African proteas: a non-coevolved system. Annals of the Missouri Botanical Gardens, 70, 1–31.

Wilde, S.A., Youngberg, C.T. and Hovind, J.H. 1950. Changes in composition of ground water, soil fertility, and forest growth produced by the construction and removal of beaver dams. Journal of Wildlife Management, 14, 123–128.

Yair, A. and Rutin, J. 1981. Some aspects of the regional variation in the amount of available sediment produced by isopods and porcupines, northern Negev, Israel. Earth Surface Processes and Landforms, 6, 221–234.

Yavitt, J.B., Angell, L.L., Fahey, T.J., Cirmo, C.P. and Driscoll, C.T. 1992. Methane fluxes, concentrations, and production in two Adirondack beaver impoundments. Limnology and Oceanography, 37, 1057–1066.

6. The Role of Rodents in Emerging Human Disease: Examples from the Hantaviruses and Arenaviruses

James N. Mills

Abstract

Because of the severity and the dramatic nature of the diseases they cause, the rodent-borne haemorrhagic fever viruses recently have received considerable attention from ecologists and health scientists. During the past five years, researchers have identified at least 25 'new' hantaviruses and arenaviruses, all associated with murid rodents, and coevolutionary theory suggests that many additional virus–host associations await discovery. Basic research on the ecology of hantavirus and arenavirus reservoir species is providing information of practical importance for reservoir control and disease prevention. Studies of reservoir geographic distribution and habitat associations help define potential disease-endemic areas and more precisely identify the spacial variation in relative risk to humans. Cross-sectional and longitudinal studies of reservoir populations help define mechanisms of viral transmission and identify the relationship between environment, reservoir populations, and human disease. Integrated results from a variety of reservoir studies can be combined with data from satellite images to provide models that can help scientists predict specific times and places of increased risk to human populations. Biologists and pest control specialists who work with reservoir species may be at increased risk of infection with rodent-borne viruses unless appropriate safety guidelines are followed.

Keywords

Rodents, infectious disease, rodent-borne disease, zoonoses, haemorrhagic fever, arenavirus, hantavirus

INTRODUCTION

AS THE chapters in this volume amply illustrate, rodents—as pests —are responsible for considerable economic loss, through damage to crops, food stores and human property. Rodents, as carriers of diseases transmitted to humans (rodent-borne zoonoses), are also responsible for considerable economic loss in terms of decreased worker productivity and health-care costs. The most important negative impact of rodent-borne diseases, the loss of human health and lives, can be assigned no price tag.

Although the specific theme of this chapter is the application of basic research on rodent biology and ecology toward public health goals, the goals of pest management personnel and public health practitioners are similar. Whether to prevent economic loss or to prevent disease, we seek to control rodent populations, prevent their access to human food and other products, prevent their access to our dwellings, and minimise their contact with humans.

Accomplishment of these objectives is facilitated by a thorough understanding of the biology and ecology of the target species. In this chapter, I will (1) introduce the extent and severity of the human disease problem caused by the rodent-borne haemorrhagic fever viruses; (2) use examples from the hantaviruses and arenaviruses to illustrate the broad nature and potential for expansion of the rodent-borne disease problem; (3) illustrate how a structured basic research program can provide data that may assist in the management of rodent host populations and the prevention of human disease; (4) discuss the implications of our increasing awareness of severe rodent-borne diseases and the understanding of their transmission patterns in terms of the safety measures recommended for mammalogists and vertebrate pest control specialists working in disease-endemic areas; and (5) provide an up-to-date list of the rodent-borne haemorrhagic fever viruses, their reservoir hosts, and the host distributions, so that rodent biologists can assess their particular risk and take appropriate precautions.

EMERGING INFECTIOUS DISEASES

In 1967 the Surgeon General of the United States, William H. Stewart, declared that it was time to "close the book on infectious diseases" and start paying more attention to chronic ailments. In 1998, Surgeon General David Satcher, speaking to the United States Congress, addressed the "continuing threat of emerging infectious diseases", singling out infectious disease as the number one killer worldwide. Even in the United States, according to Dr. Satcher, the death rate from infectious diseases, excluding acquired immune deficiency syndrome (AIDS), rose by 22% between 1980 and 1992. What factors are responsible for this stark difference in perspective coming from two surgeons general? What are emerging infectious diseases? Why are they suddenly becoming important?

The term 'emerging infectious disease' applies to two groups of illnesses: those caused by previously unknown agents that are being recognised at an increasing rate, such as AIDS, Lyme disease and hantavirus pulmonary syndrome, and those that represent the re-emergence of previously described diseases in drug-resistant or more virulent forms, such as tuberculosis, malaria, and the illnesses due to *Escherichia coli* 0157:H7 (Institute of Medicine, 1992). The recent awareness of emerging infectious diseases is due to several factors. The development of antibiotic resistance is one. Others include rapid transportation, that quickly brings victims of remotely acquired diseases into heavily populated cities, improved diagnosis, and physician education. Another important factor is our rapidly expanding population with the resulting incursion of humans into remote, natural habitats where previously unknown diseases have existed for many years in cycles involving wild-animal hosts. Some of these diseases of wild animals, the zoonotic diseases, can be transmitted to humans. Rodents, because of their tremendous diversity, social behaviour, opportunistic life history, high reproductive potential, periodically high population densities, and peridomestic affinities, are among the most important natural reservoirs for zoonotic diseases. A review published in 1995 described approximately 60 zoonotic diseases, or groups of associated diseases, for which rodents serve as hosts for the etiologic agent (Hugh-Jones et al. 1995). The rodent-borne haemorrhagic fevers represent one such group.

Because of our phylogenetic relatedness to them, other mammals are the most likely animals with which humans may be expected to share pathogens. Rodents are the most diverse group of mammals, comprising nearly half of the 4,600 species in the class. It is likely that only a small proportion of the organisms infecting rodents would cause disease in humans. Nevertheless, the potential number of agents is vast. The disease agents that infect the house mouse (*Mus musculus*) have been relatively well studied because of its use as a laboratory animal. Although it does not claim to be comprehensive, a recent report (Committee on Infectious Diseases of Mice and Rats, 1991) provides a discussion of about 40 bacteria, viruses, and parasites that cause disease in *M. musculus*. These agents are described mostly from populations of mice that have not been outside of the relatively sterile environment of the laboratory for many generations. Thus, the potential for pathogen diversity may be much greater in populations of sylvatic species. It is certainly not unreasonable to suspect that every species of wild rodent might harbour as many, if not more, potential disease agents.

In the natural host, coevolution likely has lead to a relatively benign relationship between host and pathogen. The death or severe illness of the host is rarely to the evolutionary benefit of a parasite. When other animals (including humans) come into contact with rodents, the possibility of a cross-species infection occurs. The response of the human immune system to these novel agents is unpredictable. In many cases the pathogen will be cleared rapidly by the immune response and no disease occurs. Some pathogens may be pre-adapted to hide from the human immune system (e.g. human immunodeficiency virus). Others might elicit a strong immune response, but that response might, itself, result in severe pathology to the host. Such is the case with the rodent-borne haemorrhagic fever viruses.

THE RODENT-BORNE HAEMORRHAGIC FEVERS

The magnitude of the potential for human disease involving rodent-borne agents is largely unknown. However, one group for which considerable progress has been made in recent years is the rodent-borne haemorrhagic fever viruses. An awareness of the distribution of these viruses and their disease potential is becoming increasingly important to rodent biologists and management personnel. In this chapter I will provide brief descriptions of the most important of the known viruses, the recognised diseases caused by each, and their general distributions. Additional details concerning their importance for wildlife biologists (Childs et al. 1995), identification and distribution of the reservoir species (Mills and Childs 1999), and epidemiology of the diseases (Peters et al. 1996; Peters et al. 1999; Enría et al. 1999) can be found in other sources.

The rodent-borne viral haemorrhagic fevers are caused by two groups of viruses, the hantaviruses and the arenaviruses. Although they are both negative-stranded, enveloped ribonucleic acid (RNA) viruses, the two groups are not closely related taxonomically. The hantaviruses constitute the genus *Hantavirus*, within the family Bunyaviridae; the arenaviruses constitute the family Arenaviridae. Nevertheless, these two groups of viruses share several important characteristics. Both cause severe haemorrhagic fever in humans. The hantaviruses cause hantavirus pulmonary syndrome (HPS) in the New World and haemorrhagic fever with renal syndrome (HFRS) in the Old World. The arenaviruses cause the South American hemorrhagic fevers in the New World, and Lassa fever in the Old World. These diseases are the cause of significant morbidity and mortality. There may be 200,000 cases of HFRS each year in Asia, primarily in China and Korea (McKee et al. 1991). Lassa virus is responsible for as many as 300,000 human infections each year in West Africa (McCormick et al. 1987). Secondly, each virus in both families is usually associated with a specific rodent host, of the family Muridae, in which it establishes a chronic, persistent infection that involves the sporadic or persistent shedding of large quantities of infectious virus into the environment in urine, faeces or saliva. These characteristics of the infection are key to the transmission of the virus, both from rodent to rodent, and from rodent to human. Humans are believed to be infected most frequently via the inhalation of infectious aerosols of rodent excreta or secreta. Other likely but less frequent routes of infection include direct contact of broken skin or mucous membranes with contaminated rodent fluids or fomites, ingestion of contaminated food, or the bite of an infected rodent. Transmission within host populations may be by a variety of horizontal and vertical mechanisms, but evidence from field studies indicates that horizontal transmission, and perhaps, specifically aggressive encounters between adult male rodents, may be an important mechanism (Glass et al. 1988; Mills et al. 1992, 1997b).

HAEMORRHAGIC FEVER WITH RENAL SYNDROME

HFRS is the term applied collectively to a suite of diseases of varying severity caused by hantaviruses in Asia and Europe. The diseases are characterised by fever, chills, myalgia, and varying degrees of haemorrhage and renal compromise with 1% to 15% mortality. The viruses are carried by rodent hosts of the murid subfamilies Murinae (Old World rats and mice) and Arvicolinae (voles), and the distributions of the diseases generally coincide with the distributions of the host species (Mills and Childs 1999). A summary of the viruses, the known diseases, and approximate distribution of the reservoirs is provided (Appendix 1).

The prototype hantavirus, Hantaan virus, gained worldwide attention during the Korean conflict, when over 3,000 United Nations troops contracted a severe form of HFRS, then referred to as Korean haemorrhagic fever (KHF). However, the disease, which has variously been referred to as epidemic hemorrhagic fever, hemorrhagic nephrosonephritis, Churilov's disease, and Songo fever, has been recognised for many years in Asia (McKee et al. 1991; Peters et al. 1999). A Chinese medical text from 960 AD may describe compatible symptoms. The disease was noted by Soviet scientists as early as 1913, and outbreaks continued to be described by the Soviets as well as among Japanese troops in Manchuria during the 1930s. The etiologic agent of KHF was not described until the late 1970s when Ho Wang Lee isolated a virus from the lungs of the striped field mouse (*Apodemus agrarius*) captured on the banks of the Hantaan River near the border between North and South Korea (Lee et al. 1978). Currently, Hantaan virus is responsible for perhaps 200,000 cases of HFRS each year, in China, Korea, and the Russian Far East (McKee et al. 1991).

Epidemics are seasonal with an autumn peak, coinciding with the maximum agricultural activity and probably maximum host population density. Cases occur predominantly among adult men in rural habitats; many cases are among farmers, forest workers and soldiers in the field (McKee et al. 1991; Peters et al. 1999).

Seoul virus, which is found nearly worldwide in association with its cosmopolitan host, the Norway rat (*Rattus norvegicus*), is responsible for a relatively mild form of HFRS (Lee et al. 1980). Although Seoul virus has been detected in rats throughout most of the range of the species, most confirmed cases of HFRS caused by Seoul virus have been restricted to Korea, Russia, and China. Reasons for the apparent lack of disease in other parts of the world are unknown, but may include inadequate case finding. A search in one United States city revealed three suspected cases (Glass et al. 1994).

Dobrava virus, hosted by the yellow-necked field mouse (*Apodemus flavicollis*), is responsible for a severe form of HFRS in the Balkans. Dobrava virus may be associated with *A. agrarius* in the Baltic region (Plyusnin et al. 1997; Peters et al. 1999).

Although several hantaviruses are hosted by arvicoline rodents in Asia and Europe, only one is known to be associated with human disease. Puumala virus, carried by the bank vole (*Clethrionomys glareolus*), is the etiologic agent for a mild form of HFRS called nephropathia epidemica (NE). NE is endemic to Scandinavia, western Europe, and European Russia. Several additional hantaviruses, some only recently discovered, are associated with murine and arvicoline rodents in Asia and Europe

(Appendix 1). These viruses have not been definitively associated with human disease, but extensive studies are lacking.

HANTAVIRUS PULMONARY SYNDROME

HPS is a New World hantavirus disease characterised by a flu-like prodrome involving fever, myalgia, and malaise, which rapidly progresses to cardiopulmonary compromise that may end in death in about 50% of cases in spite of aggressive hospital care.

In early 1993, only a single autochthonous hantavirus was known from the New World: Prospect Hill virus (Lee et al. 1982), which is associated with the meadow vole, *Microtus pennsylvanicus*. It still has not been associated with any human disease. The seemingly sudden appearance of HPS in the spring of 1993 led to the isolation of Sin Nombre virus (SNV; Elliott et al. 1994) and its association with the deer mouse (*Peromyscus maniculatus*; Nichol et al. 1993; Childs et al. 1994). Armed with a specific case definition and the molecular and serologic tools and reagents necessary to detect SNV, physicians and mammalogists quickly discovered that HPS was a pan-American disease, and that numerous species of New World sigmodontine and arvicoline rodents serve as hosts for a plethora of hantaviruses. Currently, approximately 20 viruses have been described in association with about as many host species occurring from Canada to Patagonia (Appendix 1 and Figure 1). About half of these viruses are known human pathogens. All of those viruses responsible for HPS are hosted by rodents of the murid subfamily Sigmodontinae. In addition to the United States and Canada, HPS has now been documented in Argentina, Chile, Paraguay, Uruguay, Brazil, and Bolivia, and hantavirus or hantavirus antibody has been demonstrated in rodents from Peru, Venezuela, Costa Rica and Mexico.

THE SOUTH AMERICAN HAEMORRHAGIC FEVERS

The South American haemorrhagic fevers currently consist of four recognised diseases that are clinically similar. They are characterised by an insidious prodrome of fever, malaise, muscle aches, and retroorbital headache, which may be followed by hypotension, conjunctival injection, petechiae on the throat, chest, and axillary area, dizziness, and tremors. Severe cases demonstrate bleeding from gums and mucous membranes, shock, coma, and convulsions (Enría et al. 1999). Mortality may be 10% to 33%. With a single possible exception, the New World arenaviruses for which the reservoir is known are all associated with sigmodontine rodents (Appendix 2).

The first arenaviral haemorrhagic fever to be recognised and, to date, the best studied is Argentine haemorrhagic fever (AHF). The disease was described in 1953 (Arribalzaga 1955) and the etiologic agent, Junín virus, was described in 1958 (Parodi et al. 1958). Several hundred to over a thousand cases of AHF were confirmed each year on the central Argentine Pampa from the discovery of the disease until the recent development of a vaccine.

Cases are predominantly in adult men from rural areas, and epidemics are seasonal, occurring in the fall—coinciding with the harvest of principal crops (corn and soybeans) and maximum densities of the

Figure 1.
Geographic locations of the principal currently recognised New World hantaviruses (after Mills and Childs 1998).

principal reservoir, the corn mouse (*Calomys musculinus*). The introduction of an effective treatment using immune plasma decreased the mortality from 15–30% to less than 1%, and the recent use of a highly efficacious vaccine in the AHF-endemic area has resulted in a substantial reduction in the numbers of reported cases (Maiztegui et al. 1998).

Bolivian hemorrhagic fever (BHF), caused by Machupo virus, was described following several clusters of cases in 1959. The 2,000–3,000 cases of naturally acquired BHF have all been from the Beni Department of north-western Bolivia. Sporadic cases predominantly involve adult males from rural environments (Kilgore et al. 1995), but several large outbreaks have been associated with high densities of the reservoir, *Calomys callosus*, in and around villages. Unlike its congener, *C. musculinus* (which is strictly associated with grassland and agricultural habitats), *C. callosus* can be found in close association with human dwellings. An outbreak in the town of San Joaquín in 1963–1964 ended abruptly after two weeks of continuous trapping in homes, during which 3,000 *C. callosus* were captured (Kuns 1965), and an ongoing program of rodent trapping in villages in the BHF-endemic area may be, at least in part, responsible for the scarcity of cases since 1974 (PAHO 1982).

Venezuelan haemorrhagic fever, described in 1989 (Salas et al. 1991), is caused by Guanarito virus, an arenavirus hosted by the cane mouse, *Zygodontomys brevicauda*. Recognised cases have been restricted to rural areas of southern Portuguesa and northern Barinas states. The highest risk of disease is among adult male farm workers, and the greatest numbers of cases occur between November and January (Manzione et al. 1998).

Finally, an arenaviral haemorrhagic fever caused by Sabiá virus is known from a single naturally acquired case near Sao Paulo, Brazil, in 1990 (Coimbra et al. 1994). Nothing is known about the reservoir, or the potential endemic area.

OLD WORLD ARENAVIRAL HAEMORRHAGIC FEVERS

Lassa fever, which is endemic to West Africa, is the only recognised arenaviral haemorrhagic fever in the Old World. Although the magnitude and geographic extent of the cases are poorly known, Lassa virus probably causes 100,000 to 300,000 cases and 5,000 deaths annually (McCormick et al. 1987). The virus has been isolated from humans or rodents in Nigeria, Sierra Leone, Guinea, and Liberia, but serologic surveys show that Lassa or Lassa-like viruses are present in at least 10 other African countries (Peters et al. 1996; Appendix 1). Two or more species of the *Mastomys natalensis* species complex appear to serve as the reservoir for Lassa virus. At least eight species of *Mastomys* occur in Africa south of the Sahara, and their distribution and relationships are poorly understood (Robbins and Van Der Straeten 1989). A 32-chromosome species, *Mastomys huberti*, has been described as being found in dwellings, while a 38-chromosome species, *Mastomys erythroleucus*, was found in the surrounding bush areas; both species were frequently infected with Lassa virus with a prevalence of about 30% (McCormick et al. 1987).

Lymphocytic choriomeningitis (LCM), caused by the arenavirus lymphocytic choriomeningitis virus (LCMV), is associated with the house mouse (*M. musculus*) throughout much of its worldwide range. LCMV usually produces a syndrome of fever and myalgia (sometimes complicated by meningitis), which is rarely serious, but infections during pregnancy have been associated with serious, even fatal complications to neonates (Peters et al. 1996). LCM is not considered a viral haemorrhagic fever and will not be discussed further. Nevertheless the disease may be much more common than is diagnosed, and biologists and pest control practitioners should be aware of the risk. Detailed reviews have been published (Jahrling and Peters 1992; Peters et al. 1996; Enría et al. 1999).

POTENTIAL VIRUS DIVERSITY

The rate of discovery of new haemorrhagic fever viruses has increased almost exponentially in recent years. In the Americas, for instance, the number of known autochthonous hantaviruses has increased from one, in 1993, to over 20 in 1998 (Figure 1). The rate of discovery of new hantaviruses and arenaviruses is not slowing, and theoretical considerations suggest that we may recognise only a small proportion of the potential diversity. In general, each hantavirus and arenavirus appears to be associated with a single species of murid rodent host. The hantaviruses are associated with the Murinae, the Sigmodontinae, and the Arvicolinae; the arenaviruses with the Murinae and the Sigmodontinae. Furthermore, the phylogenetic relationships among the viruses (with some exceptions for the Arenaviridae) are generally mirrored by the phylogenetic relationships among the rodent hosts (Bowen et al. 1997; Mills et al. 1997a; Schmaljohn and Hjelle 1997). This pattern suggests that there was an ancestral hantavirus and arenavirus associated with an ancestral murid rodent, before the subfamilial lineages diverged, over 20 million years ago, and that the viruses have been co-speciating and co-evolving along with their rodent hosts since that time. Implicitly, the maximum potential number of hantaviruses and arenaviruses would be one for each of the 143 species of Arvicolinae, 529 species of Murinae, and 423 species of Sigmodontinae (numbers of species from Musser and Carleton 1993). Indeed, some species (e.g. *Sigmodon alstoni, Sigmodon hispidus, Bolomys obscurus;* Appendix 1) are known to host an arenavirus and a hantavirus. Nevertheless, it is unlikely that this maximum number of viral species will be found. Virus extinctions are very likely to have occurred in some murid lineages. For instance, some well-studied species (e.g. *M. musculus*, and *C. musculinus*) appear not to be associated with a hantavirus. It is also likely, however, that some trans-species 'host jumping' may have occurred over time (Bowen et al. 1997; Morzunov et al. 1998), thus further increasing the potential diversity of viruses.

ECOLOGICAL STUDIES OF RESERVOIR SPECIES

Just as control of pest populations for economic reasons depends upon an understanding of the biology and ecology of pest species, the control or prevention of rodent-borne disease largely depends upon understanding the biology and ecology of the host. Several basic research studies of the ecology of virus reservoir species during the past 12 years have provided information that potentially can be very useful for risk assessment and directed intervention in disease control.

In an earlier paper (Mills and Childs 1998) we reviewed some of these studies and outlined a series of directed goals toward the understanding of reservoir ecology as it relates to human disease. After initial identification of the reservoir host, these goals include (a) determining the potential disease-endemic area by identifying the geographic distribution of the host, and the range of infection by the pathogen within the host distribution; (b) more precisely defining relative human risk by determining the distribution of the host and pathogen among the distinct habitats on a regional scale; (c) investigating potential mechanisms of transmission of the pathogen within host populations; (d) conducting long-term prospective studies to elucidate the temporal patterns of infection in host populations; and (e) integrating data from reservoir studies toward the development of a predictive model that would allow the early identification of specific times, places, and conditions that may lead to increased rodent populations, or increased infection in rodent populations that can cause elevated risk of human disease. Although specifically directed at understanding rodent ecology in relation to human disease, many of these goals (especially a, b, d, and e) are applicable to studies of economic pests. In this section, the above-listed goals are used as a structural basis, while providing examples from studies and theoretical problems specific to the hantaviruses and arenaviruses, to illustrate the value of basic research toward the practical goal of preventing and controlling human disease caused by rodent-borne pathogens.

Defining disease-endemic areas

One of the most basic pieces of information for designing and directing a prevention program for any disease is a precise knowledge of the geographic area where a disease may occur (the potential endemic area). Prevention efforts, such as public education and reservoir control, must be directed throughout this area, while efforts outside the area represent wasted time and money. For any rodent-borne disease, the geographic distribution of the reservoir defines the maximum potential endemic area of the disease. For many rodent species in North America, the distributional ranges are precisely known and are available in the literature (Hall and Kelson 1959). Following the identification of the deer mouse as the reservoir for SNV (Childs et al. 1994), scientists consulted the published distribution of *P. maniculatus* in North America (Carleton 1989), and realised that the potential endemic area for HPS caused by SNV could encompass most of the North American continent. Education of physicians and increased surveillance soon confirmed that sporadic cases of HPS occurred throughout the range of the deer mouse in the United States. For other rodent species, in less extensively studied parts of the world, these distributions are poorly defined. This is the case with several important hantavirus and arenavirus reservoir species in South America. For example, the published distribution for *C. musculinus*, reservoir of Junín virus, includes central and northern Argentina (Redford and Eisenberg 1992). Nevertheless, during recent ecological investigations of Laguna Negra virus on the Chaco of Paraguay (Yahnke et al. 1998), *C. musculinus* was frequently captured. A concerted effort, as well as multi-disciplinary studies that include health scientists, ecologists, and systematists, will be essential to define accurately the distributional ranges of important reservoir species.

For some host–virus systems (e.g. *P. maniculatus* and SNV), the host appears to be infected throughout its geographic range. In other cases, the distribution of the virus may include only a small portion of the range of the host. In those cases, the identification of the geographic area in which the host and the pathogen both occur provides a more precise definition of the potential disease-endemic area. While *C. musculinus* occurs throughout central and northern Argentina (and apparently western Paraguay), the AHF-endemic area occupies only a very limited region of the central Argentine Pampa (Maiztegui et al. 1986). Limited searches for Junín virus in *C. musculinus* populations outside the endemic area have been unsuccessful (Mills et al. 1991). *Calomys laucha* also occurs from central Argentina through south-eastern Bolivia, western Paraguay, and west-central Brazil (Musser and Carleton 1993), yet Laguna Negra virus apparently occurs only on the Chaco of Paraguay (Johnson et al. 1997, Mills and Childs 1998). Populations of *C. laucha* in Paraguay and central Argentina appear to be disjunct, and it is possible that the populations of *C. laucha* in Argentina are genetically distinct and will not support infection with Laguna Negra virus. Populations of *C. musculinus* appear to be continuous across the boundary of an expanding AHF-endemic area (Maiztegui et al. 1986). The spatially restricted but expanding distribution of Junín virus within a continuous host population suggests recent introduction of Junín virus or recent genetic changes in the virus, host, or both populations. Reasons for the lack of coincidence in host and virus distributions are likely to be diverse and involve host genetics, geographic boundaries, and local extinctions in subpopulations. The elucidation of these factors, which will require collaboration among ecologists, virologists, geneticists, and systematists, will contribute to our understanding of host–virus coevolution, the relationships among rodent species, and the properties of host systems that are required to support long-term maintenance of viral symbionts. On the practical side, these studies will allow the precise definition of the geographic areas in which humans are at risk for specific diseases.

Defining habitat associations

In addition to the large geographic patterns discussed above, host and pathogen populations may vary on regional or local scales. Many species of rodents demonstrate distinct local habitat preferences, which may have practical implications for disease transmission as well as reservoir management. The risk of human disease may be more precisely defined by describing differences in host distribution, population densities, and prevalence of infection among the distinct habitats represented in a local area. Even for species that are considered opportunists or generalists, habitat studies may yield useful information.

The deer mouse is considered a habitat generalist and has been reported as occurring in nearly every dry-land habitat in North America (Burt and Grossenheider 1976). A habitat study conducted in the south-western United States confirmed that *P. maniculatus* occupied all of the major habitats represented (Mills et al. 1997b). Nevertheless, the prevalence of infection with SNV varied significantly among habitats, being lowest at the altitudinal and climatic extremes (desert and alpine tundra) and highest at the middle altitude habitats such as chaparral, grassland, and piñon-juniper woodland. The last habitat is where most HPS cases in the south-western United States have occurred. Similar results were demonstrated by a study in Nevada and California (Boone et al. 1998). Results such as these can be applied by public health scientists to define more precisely the relative risk of disease to humans living, working, or pursuing recreational activities in various habitats.

The corn mouse, *C. musculinus*, has historically been considered a denizen of corn fields, as its common name implies. Junín virus has been thought to be transmitted to farmers working in those crop fields during the mechanised harvesting process (Carballal et al. 1988). Recently however, habitat studies conducted in the AHF-endemic area demonstrated a distinct preference by *C. musculinus* for the relatively stable, weedy border habitats (fence lines and roadsides) adjacent to the crop fields (Figure 2).

These results suggest a need to reconsider the possible places and mechanisms of transmission of Junín virus to humans. They also suggest a specific intervention mechanism for decreasing the incidence of AHF: periodically burning or cutting the weedy border habitats to eliminate the preferred habitat for the reservoir host. Habitat studies conducted for other

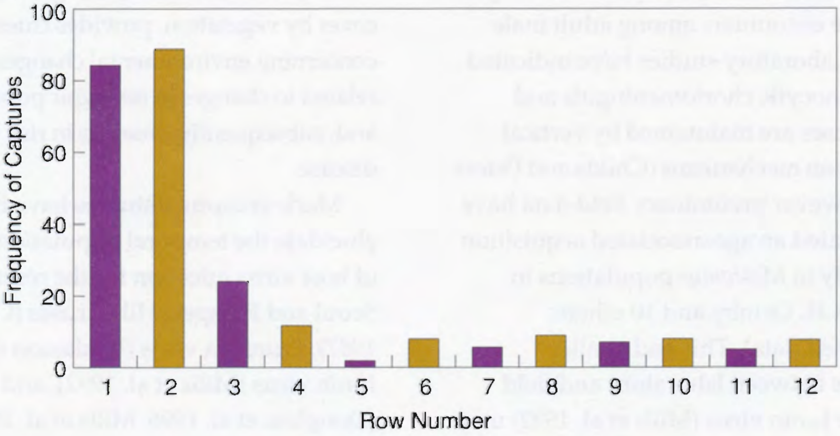


Figure 2.

Cumulative numbers of captures within each of 12 rows of traps of a 12 by 12 trapping grid located in crop fields and adjacent roadside habitat in central Argentina, March 1998 to August 1990. Rows 1 and 2 are roadside habitat; rows 3 through 12 are in crop fields.

reservoir species might suggest similar approaches.

Identifying mechanisms of transmission

Basic field studies of reservoir demography have provided important clues to the specific mechanisms of virus transmission within host populations. Field studies with SNV (Mills et al. 1997b) and Black Creek Canal virus (Glass et al. 1998) have shown a J-shaped curve of antibody prevalence with host age. This pattern suggests that the young of infected females are born with maternal antibody, which is lost within a few weeks. Infection is then acquired by some horizontal mechanism later in life. Field data have also demonstrated a positive correlation between scars and the prevalence of infection (Glass et al. 1988). The more aggressive males may have a much higher prevalence of infection than females (Mills et al. 1992; Mills et al. 1997b). Thus, an important, specific mechanism of virus transfer within reservoir populations may be aggressive encounters among adult male animals. Laboratory studies have indicated that lymphocytic choriomeningitis and Lassa viruses are maintained by vertical transmission mechanisms (Childs and Peters 1993). However preliminary field data have demonstrated an age-associated acquisition of antibody in *Mastomys* populations in Guinea (A.H. Demby and 10 others, unpublished data). This and similar disparities between laboratory and field results for Junín virus (Mills et al. 1992) may indicate that laboratory results may not always be applicable to natural field conditions and that field studies are important for testing predictions based on laboratory results.

Long-term studies

Perhaps the greatest amount of useful information about reservoir populations and host–virus dynamics is achieved through the use of longitudinal mark–recapture studies (Mills et al. 1999b). These studies involve the establishment of multiple permanent trapping plots, which are operated at defined intervals (usually monthly for disease studies) for several consecutive nights. Captured rodents are measured, sampled (e.g. blood and oral swab), identified with a permanent mark or number, and released at the site of capture. In subsequent trapping events, animals are repeatedly captured, measured, and sampled. In this way, changes in community structure and population densities — as well as individual growth rates, movement, reproductive condition, and infection status —are measured over time. Simultaneous monitoring of environmental variables, such as temperature, rainfall, and growth and cover by vegetation, provides clues concerning environmental changes that are related to changes in reservoir populations and, subsequently, changes in risk of human disease.

Mark–recapture studies have helped to elucidate the temporal population dynamics of host virus infection for the reservoirs of Seoul and Prospect Hill viruses (Childs et al. 1987), Puumala virus (Niklasson et al. 1995), Junín virus (Mills et al. 1992), and SNV (Douglass et al. 1996; Mills et al. 1999a). The cited studies and others in progress are helping to elucidate the associations among environmental conditions, rodent population densities, prevalence of infection in reservoir populations, and human disease risk.

The incidence of several rodent-borne diseases is related to changes in density of reservoir populations. Large year-to-year fluctuations in population density are characteristic of rodent populations of many species. Northern Hemisphere arvicolines, such as the reservoir for Puumala virus (*C. glareolus*), undergo regular population cycles with a periodicity of 3–4 years, although the causes for the cycles are still unclear (Krebs and Myers 1974; Niklasson et al. 1995). The year-to-year incidence of HFRS caused by Puumala virus was shown to be correlated with the density of *C. glareolus* in Russia and Scandinavia (Niklasson et al. 1995).

Regular population cycles are not known in rodents from the Northern Hemisphere tropics or anywhere in the Southern Hemisphere. However, periodic, dramatic increases in the density of some rodent populations do occur. These population irruptions are generally associated with unusual climatic conditions, which result in abundant food supplies and ideal or prolonged conditions for reproduction. A three-year longitudinal study of *C. musculinus* in Argentina demonstrated a clear positive association between reservoir population density and the magnitude of AHF epidemics (Figure 3).

The associated environmental conditions were a relatively benign winter, followed by a wet summer, which apparently resulted in

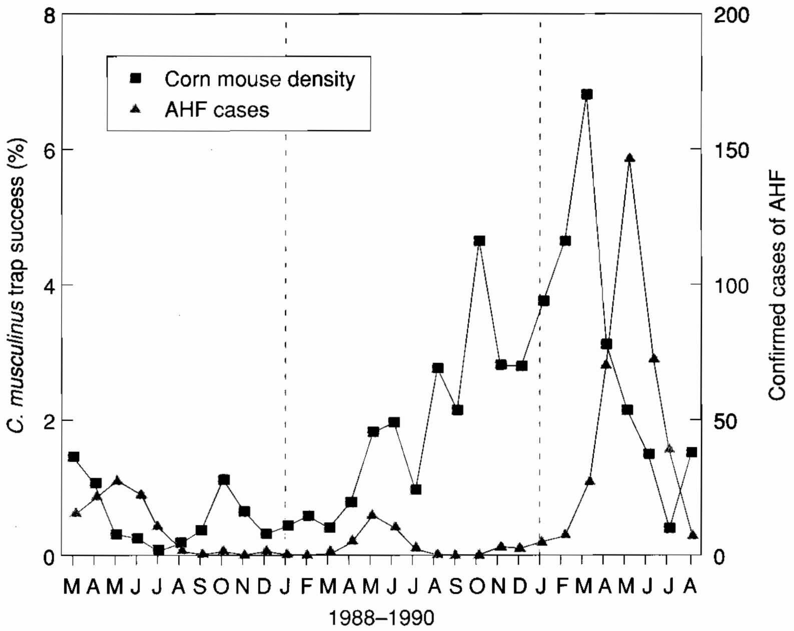


Figure 3.
Mean numbers of captures of *Calomys musculinus* per 100 trap nights (trap success) and numbers of cases of Argentine haemorrhagic fever (AHF) in central Argentina, March 1998 to August 1990. Reprinted with permission from Mills et al. (1992).

unusually lush vegetation and abundant food supplies (Mills et al. 1992).

The outbreak of HPS in the south-western United States in 1993 was preceded by an El Niño Southern Oscillation (ENSO) event, which resulted in unusually warm winters and high rainfall in affected areas. It has been hypothesised that the HPS outbreak was a direct result of increases in rodent populations and increases in prevalence of SNV infection among high-density reservoir populations (Parmenter et al. 1993). Although intuitively attractive, no longitudinal monitoring of rodent populations and infection status was in place in the area of the outbreak, so this hypothesis cannot be confirmed. Subsequent to the outbreak, however, the Centers for Disease Control and Prevention, in collaboration with several local universities, initiated a series of longitudinal mark–recapture studies in the south-western United States (Mills et al. 1999b). These studies were in place to document environmental changes and associated increases in reservoir populations in some areas of the south-west in response to an ENSO event in 1997/1998 (T.L. Yates, K.D. Abbott, C.H. Calisher and M.L. Morrison, unpublished data). These increases in reservoir populations have been associated with increased numbers of HPS cases in the south-western United States. As of August 1998, there have been about 14 cases in the four-state area of Arizona, Colorado, New Mexico, and Utah, in comparison to 2, 2, and 4, for the same time periods in 1995, 1996, and 1997, respectively (Centers for Disease Control and Prevention, unpublished data).

A recent outbreak of HPS in southern Chile was apparently preceded by a dramatic increase in local populations of the reservoir of Andes virus (*Oligoryzomys longicaudatus*). Causes for the rodent irruption are unclear, but may be related to an unusually benign winter or to the flowering of a local species of bamboo, an event that may occur only every 40 years and that provides abundant food for the granivorous *O. longicaudatus* (Murúa et al. 1996; Toro et al. 1998). Longitudinal studies are currently being planned and initiated in Chile and Argentina to follow the environmental variables associated with changes in population density and infection status in *O. longicaudatus* populations and the relation of these variables to human disease.

An important key to being able to predict the relative risk of diseases to humans is understanding the conditions that lead to increased virus transmission and increased prevalence of infection in host populations. Infection appears to be associated with behavioural events involving the interactions of individual rodents. Given the pattern of horizontal transmission demonstrated for many hantaviruses and arenaviruses, it might be predicted that increasing population densities should result in increased rodent-to-rodent contact and a higher prevalence of infection in host populations. In fact, however, investigators are frequently unable to show a correlation between rodent population density and prevalence of infection in rodent populations (Mills et al. 1992; Douglass et al. 1996; Bond et al. 1998; Boone et al. 1998). The problem with these approaches may be in seeking an instantaneous, linear relationship between density and antibody prevalence. In strongly seasonal environments, the effects of seasonal reproduction and horizontal transmission of virus may result in an alternation of peaks in population density and prevalence of infection. In Sweden, the population density of *C. glareolus* was highest in autumn (Niklasson et al. 1995), while the prevalence of antibody to Puumala virus in these populations was highest in the spring, and correlated with vole population density the previous fall. A similar pattern of alternating peaks in density and antibody prevalence was observed for populations of *Akodon azarae* infected with Pergamino virus in Argentina—rodent density was highest in the fall, antibody prevalence was highest in the spring (Schmidt et al. 1998). This pattern has also been observed for *P. maniculatus* populations infected with SNV in Colorado (Calisher et al. 1999), and *Peromyscus boylii* infected with a Sin Nombre-like virus in Arizona (Abbott et al. 1999). This delayed-density-dependent prevalence of infection may be typical for viruses transmitted by horizontal mechanisms in seasonal environments. Autumn populations display peak densities because of the culmination of the spring/summer reproductive effort; yet the population consists primarily of young of the year that have not yet been infected, or are only recently infected and do not yet have detectable antibody. The cessation of reproduction and over-winter mortality results in a population nadir in the spring, but at that time the population consists exclusively of older adults, which are more likely to be infected. In an autumn during which particularly high population levels occur, crowding would presumably lead to more intraspecific contacts, more virus transmission events, and a proportionally higher antibody prevalence the following spring (Niklasson et al. 1995; Mills et al. 1999a). Research leading to an understanding of the conditions that lead to increased virus transmission and prevalence of infection in host populations will improve the ability of public health scientists and modellers to predict increases in the risk of human disease.

Predictive models of disease risk

Perhaps the most important practical application of studies of reservoir populations is to integrate the data from these studies into a predictive model that would allow public health practitioners to identify specific times and places where conditions may pose a threat to the public health. Such a model (Figure 4) assumes that the risk of human disease is related to rodent population density and prevalence of infection; rodent populations are affected by the quality of the biotic environment (e.g. habitat quality and food supply); and the abiotic environment (e.g. edaphic factors and weather) influences rodent populations both directly (e.g. direct effects of cold temperatures on survival) and indirectly (through their effect on habitat quality and food supply; Mills and Childs 1998). It is not possible to have scientists continuously measuring rodent populations and environmental variables wherever rodent-borne diseases occur. However, this may not be necessary.

Recent studies using satellite imaging and geographic information systems have demonstrated that remotely monitored vegetation indices can help predict the changing risk of human disease in sites as far away as East Africa (Linthicum et al. 1987), and the south-western United States (Boone et al. 1998; Cheek et al. 1998). The success of these mathematical models will depend upon the accuracy of the parameter estimates, and the accuracy of the estimates, in turn, will depend upon data collected by investigators conducting basic field and laboratory research into the ecology and biology of the rodent reservoirs.

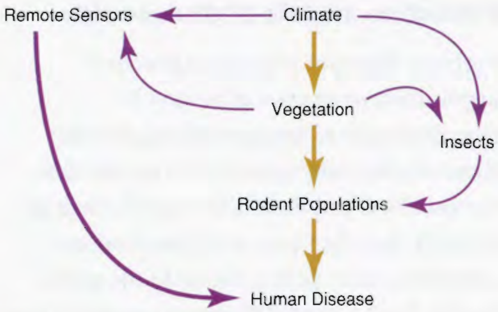


Figure 4.
Simplified schematic model of relationships among ecosystem components within an endemic area for a rodent-borne human disease. Remote sensors (satellites) may be used for detecting changes in the ecosystem components which may lead to increased risk of disease. From Mills and Childs (1998).

THE RISK TO RODENT BIOLOGISTS

In the United States, the sudden realisation that wild rodents are the reservoir for potentially lethal disease has resulted in significant changes in the way many rodent biologists conduct their research and teaching. Mammalogy classes avoid the handling of sigmodontine rodents by students, the establishment of laboratory colonies from wild captured individuals of known reservoir species is strictly controlled, and researchers who handle reservoir rodents are prudent to follow safety guidelines.

The chance of contracting HPS by handling New World sigmodontine rodents appears to be low. Nevertheless, the disease is sufficiently severe to warrant strict safety measures for the general public (CDC 1993), and there is evidence that wildlife biologists are at increased risk. Among the first 100 cases of HPS in the United States, three were in wildlife biologists, and although a recent serosurvey of over 1,000 American mammalogists demonstrated that the risk of infection with SNV appeared to be low (less than 1%), risk increased with the number of *Peromyscus* that investigators had handled during their careers (Armstrong et al. 1994). A study of Finnish mammalogists showed a more striking relationship. Although no mammalogist with less than five years of experience had antibody to Puumala virus, 40% of those who had trapped voles for more than 10 years had antibody (Brummer-Korvenkontio et al. 1982). These results suggest that Puumala virus is more easily transmitted to humans than is SNV (although because of the high mortality of SNV, about half of those infected would not be available for sampling). Fortunately, nephropathia epidemica is a relatively mild disease. The murine- and sigmodontine-associated HFRS, HPS, Lassa fever, and South American haemorrhagic fevers can be much more severe and can lead to fatalities in 15–50% of cases. Relatively simple safety precautions will minimise the risk of infection to biologists and are highly recommended for all researchers handling all known viral haemorrhagic fever reservoir species.

Standard precautions have been promulgated for investigators conducting field studies, which may involve handling reservoir species for haemorrhagic fever viruses. These guidelines, which were developed during field studies of Junín virus in Argentina and the sigmodontine hantaviruses in the Americas, have been published in English (Mills et al. 1995a,b) and in Spanish (Mills et al. 1998). Briefly, investigators should wear rubber gloves when handling traps containing captured animals, and the traps should be handled in a manner that will prevent or minimise contact with rodent excretions or secretions and inhalation of potentially infectious aerosols of these materials. If captured rodents are transported, the traps containing them should be placed in airtight plastic bags. These bags subsequently should be opened and the rodents handled only in an isolated outdoor area by personnel wearing protective equipment (latex gloves, gowns or overalls, respirators fitted with high-efficiency particulate air filters, and goggles). Handling rodents outdoors is preferred in order to take advantage of the disinfectant properties of natural ultraviolet light and the rapid dilution of aerosols in open circulating air. Rodents should be anesthetised before handling to prevent bites and production of aerosols, and the use of sharp instruments such as needles and scalpels should be avoided when possible. Instruments, working surfaces, and traps should be decontaminated using an appropriate disinfectant (e.g. 5% hospital strength Lysol, or 10% household bleach in water), and contaminated gloves, disposable gowns, and wastes should be autoclaved or burned. Rodent carcasses kept for museum specimens can be decontaminated by fixing in 10% formalin for at least 48 hours.

Pest control workers should be alert to the possibility of inhalation of infectious aerosols when working in closed structures, which may be infested by hantavirus or arenavirus reservoir species. The doors and windows of such structures should be opened, and the building allowed to air out for at least 30 minutes before beginning work. Clean-up of these structures should be conducted so as to avoid the creation of aerosols. Nesting materials or contaminated areas should be wetted down with disinfectant, and floors should be mopped, not swept (CDC 1993).

Hantavirus infection in laboratory rodent colonies has resulted in extensive outbreaks of human disease (Kulagin et al. 1962). Precautions when initiating laboratory colonies from wild rodents that are known reservoir species for hantaviruses or arenaviruses should include quarantine as defined cohorts and serologic screening upon capture, and again after 30 days (Mills et al. 1995b).

The rodent-borne haemorrhagic fever viruses are only one example of many zoonotic agents that are likely to be hosted by rodents. In this chapter they have been used as an example to illustrate the potential diversity of rodent-borne disease agents. Although the diseases they cause are dramatic, the risk to researchers can be minimised by the adherence to relatively simple safety guidelines. Researchers studying these agents have amassed a large amount of new data during the last 5–6 years. Continued basic research into the ecology of these host–virus systems promises to provide useful models for understanding host–pathogen coevolution, transmission processes in natural populations, and the relationship of environmental factors to host populations, pathogen transmission patterns, and human disease.

ACKNOWLEDGMENTS

Thanks to Barbara Ellis for graphics, and to Barbara Ellis, Jamie Childs, Tom Ksiazek, and C.J. Peters for helpful comments on the manuscript.

REFERENCES

Abbott, K., Ksiazek, T.G. and Mills, J.N. 1999. Long-term hantavirus persistence in rodent populations in central Arizona. Emerging Infectious Diseases, 5, 102–112.

Armstrong, L.R., Khabbaz, R.F. and Childs, J.E. 1994. Occupational exposure to hantavirus in mammalogists and rodent workers. American Journal of Tropical Medicine and Hygiene, 51, 94 (Abstract).

Arribalzaga, R.A. 1955. Una nueva enfermedad epidémica a germen desconocido: hipertermia nefrotóxica, leucopénica y enantemática. Día Médico, 27, 1204–1210.

Bond, C.W., Irvine, B., Alterson, H.M., Van Horn, R. and Douglass, R.J. 1998. Longitudinal incidence of hantavirus infection in deer mice. Fourth International Conference on HFRS and Hantaviruses, March 5–7 1998, Atlanta, Georgia (Abstract).

Boone, J.D., Otteson, E.W., McGwire, K.C., Villard, P., Rowe, J.E. and St. Jeor, S.C. 1998. Ecology and demographics of hantavirus infections in rodent populations in the Walker River basin of Nevada and California. American Journal of Tropical Medicine and Hygiene, 58, 445–451.

Bowen, M.D., Peters, C.J. and Nichol, S.T. 1997. Phylogenetic analysis of the Arenaviridae: patterns of virus evolution and evidence for cospeciation between arenaviruses and their rodent hosts. Molecular Phylogenetics and Evolution, 8, 301–316.

Brummer–Korvenkontio, M., Henttonen, H. and Vaheri, A. 1982. Haemorrhagic fever with renal syndrome in Finland: ecology and virology of nephropathia epidemica. Scandanavian Journal of Infectious Diseases, Suppl. 36, 88–91.

Burt, W.H. and Grossenheider, R.P. 1976. A field guide to the mammals, North America north of Mexico, third edition. Boston, Houghton Mifflin Company.

Calisher, C.H., Sweeney, W., Mills, J.N., and Beaty, B.J. 1999. Natural history of Sin Nombre virus in western Colorado. Emerging Infectious Diseases, 5, 126–134.

Carballal, G., Videla, C.M. and Merani, M.S. 1988. Epidemiology of Argentine haemorrhagic fever. European Journal of Epidemiology, 4, 259–274.

Carleton, M.D. 1989. Systematics and evolution. In: Kirkland, G.L. and Layne, J.N. ed., Advances in the study of *Peromyscus* (Rodentia). Lubbock, Texas Tech University Press, 7–141.

CDC (Centers for Disease Control and Prevention) 1993. Hantavirus infection — southwestern United States: interim recommendations for risk reduction. Morbidity and Mortality Weekly Report, 42, 113.

Cheek, J., Bryan, R. and Glass, G. 1998. Geographic distribution of highrisk, HPS areas in the U.S. Southwest. The Fourth International Conference on HFRS and Hantaviruses, March 5–7, 1998. Atlanta, Georgia, USA (Abstract).

Childs, J.E., Glass, G.E., Korch, G.W. and Le Duc, J.W. 1987. Prospective seroepidemiology of hantaviruses and population dynamics of small mammal communities of Baltimore, Maryland. American Journal of Tropical Medicine and Hygiene, 37, 648–662.

Childs, J.E., Ksiazek, T.G., Spiropoulou, C.F., Krebs, J.W., Morzunov, S., Maupin, G.O., Rollin, P.E., Sarisky, J., Enscore, R.E., Frey, J.K., Peters, C.J. and Nichol, S.T. 1994. Serologic and genetic identification of *Peromyscus maniculatus* as the primary rodent reservoir for a new hantavirus in the southwestern United States. Journal of Infectious Diseases, 169, 1271–1280.

Childs, J.E., Mills, J.N. and Glass, G.E. 1995. Rodentborne hemorrhagic fever viruses: a special risk for mammalogists? Journal of Mammology, 76, 664–680.

Childs, J.E. and Peters, C.J. 1993. Ecology and epidemiology of arenaviruses and their hosts. In: Salvato, M.S., ed., The Arenaviridae. New York, Plenum Press, 331–384.

Coimbra, T.L.M., Nassar, E.S., Burattini, M.N., de Souza, L.T.M., Ferreira, I.B., Rocco, I.M., Travassos da Rosa, A.P., Vasconcelos, P.F.C., Pinheiro, F.P., Le Duc, J.W., Rico Hesse, R., González, J.P., Jahrling, P.B. and Tesh, R.B. 1994. New arenavirus isolated in Brazil. Lancet, 343, 391–392.

Committee on Infectious Diseases of Mice and Rats 1991. Infectious diseases of mice and rats. Washington, D.C., National Academy Press, 397p.

Douglass, R.J., Van Horn, R., Coffin, K. and Zanto, S.N. 1996. Hantavirus in Montana deer mouse populations: preliminary results. Journal of Wildlife Diseases, 32, 527–530.

Elliott, L.H., Ksiazek, T.G., Rollin, P.E., Spiropoulou, C.F., Morzunov, S., Monroe, M., Goldsmith, C.S., Humphrey, C.D., Zaki, S.R., Krebs, J.W., Maupin, G., Gage, K., Childs, J.E., Nichol, S.T. and Peters, C.J. 1994. Isolation of the causative agent of hantavirus pulmonary syndrome. American Journal of Tropical Medicine and Hygiene, 51, 102–108.

Enria, D.A., Bowen, M.D., Mills, J.N., Shieh, G.J., Bausch, D. and Peters, C.J. 1999. Arenaviruses. Chapter 111. In: Guerrant, R.L., Walker, D.H. and Weller, P.F. ed., Tropical infectious diseases, principles, pathogens, and practice. New York, W.B. Saunders, 1191–1212.

Glass, G.E., Childs, J.E., Korch, G.W. and Le Duc, J.W. 1988. Association of intraspecific wounding with hantaviral infection in wild rats (*Rattus norvegicus*). Epidemiology and Infection, 101, 459–472.

Glass, G.E., Livingstone, W., Mills, J.N., Hlady, W.J., Fine, J.B., Rollin, P.E., Ksiazek, T.G., Peters, C.J. and Childs, J.E. 1998. Black Creek Canal virus infection in *Sigmodon hispidus* in southern Florida. American Journal of Tropical Medicine and Hygiene, 59, 699–703.

Glass, G.E., Watson, A.J., Le Duc, J.W. and Childs, J.E. 1994. Domestic cases of hemorrhagic fever with renal syndrome in the United States. Nephron, 68, 48–51.

Hall, E.R. and Kelson, K.R. 1959. The mammals of North America. New York, Ronald Press.

Hugh-Jones, M.E., Hubbert, W.T. and Hagstad, H.V. 1995. Zoonoses: recognition, control, and prevention. Ames, Iowa State University Press, 369p.

Institute of Medicine 1992. Emerging infections: microbial threats to health in the United States. Washington D.C., National Academy Press, 294p.

Jahrling, P.B. and Peters, C.J. 1992. Lymphocytic choriomeningitis virus: a neglected pathogen of man. Archives of Pathology and Laboratory Medicine, 116, 486–488.

Johnson, A.M., Bowen, M.D., Ksiazek, T.G., Williams, R.J., Bryan, R.T., Mills, J.N., Peters, C.J., and Nichol, S.T. 1997. Laguna Negra virus associated with HPS in western Paraguay and Bolivia. Virology, 238, 115–127.

Kilgore, P.E., Peters, C.J., Mills, J.N., Rollin, P.E., Armstrong, L., Khan, A.S. and Ksiazek, T.G. 1995. Prospects for the control of Bolivian hemorrhagic fever. Emerging Infectious Diseases, 1, 97–99.

Krebs, C.J. and Myers, J.H. 1974. Population cycles in small mammals. Advances in Ecological Research, 8, 267–399.

Kulagin, S.M., Fedorova, N.I. and Ketiladze, E.S. 1962. Laboratory outbreak of haemorrhagic fever with renal syndrome. Journal of Microbiology, Epidemiology and Immunology (Russian), 33, 121–126.

Kuns, M.L. 1965. Epidemiology of Machupo virus infection. II. Ecological and control studies of hemorrhagic fever. American Journal of Tropical Medicine and Hygiene, 14, 813–816.

Lee, H.W., Lee, P.W. and Johnson, K.M. 1978. Isolation of the etiologic agent of Korean hemorrhagic fever. Journal of Infectious Diseases, 137, 298–308.

Lee, H.W., Park, D.H., Baek, L.J., Choi, K.S., Whang, Y.N. and Woo, M.S. 1980. Korean hemorrhagic fever patients in urban areas of Seoul. Korean Journal of Virology, 10, 16.

Lee, P.-W., Amyx, H.L., Gajdusek, D.C., Yanagihara, R.T., Goldgaber, D. and Gibbs, C.J. 1982. New haemorrhagic fever with renal syndrome related virus in indigenous wild rodents in United States. Lancet, 2, 1405.

Linthicum, K.J., Bailey, C.L., Davies, F.G. and Tucker, C.J. 1987. Detection of Rift Valley fever viral activity in Kenya by satellite remote sensing imagery. Science, 235, 1656–1659.

Maiztegui, J.I., Briggiler, A., Enria, D. and Feuillade, M.R. 1986. Progressive extension of the endemic area and changing incidence of Argentine hemorrhagic fever. Medical Microbiology and Immunology, 175, 149–152.

Maiztegui, J.I., McKee, K.T., Jr., Barrera Oro, J.G., Harrison, L.H., Gibbs, P.H., Feuillade, M.R., Enria, D.A., Briggiler, A.M., Levis, S.C., Ambrosio, A.M., Halsey, N.A. and Peters, C.J. 1998. Protective efficacy of a live attenuated vaccine against Argentine hemorrhagic fever. Journal of Infectious Diseases, 177, 277–283.

Manzione, N., Salas, R.A., Paredes, H., Godoy, O., Rojas, L., Araoz, F., Fulhorst, C.F., Ksiazek, T.G., Mills, J.N., Ellis, B.A., Peters, C.J. and Tesh, R.B. 1998. Venezuelan hemorrhagic fever: clinical and epidemiological studies of 165 cases. Clinical Infectious Diseases, 26, 308–313.

McCormick, J.B., Webb, P.A., Krebs, J.W., Johnson, K.M. and Smith, E.S. 1987. A prospective study of the epidemiology and ecology of Lassa Fever. Journal of Infectious Diseases, 155, 437–444.

McKee, K.T., Le Duc, J.W. and Peters, C.J. 1991. Hantaviruses. In: Belshe, R.B. ed., Textbook of human virology. St. Louis, MO, Mosby Year Book, 615–632.

Mills, J.N., Bowen, M.D. and Nichol, S.T. 1997a. African arenaviruses—coevolution between virus and murid host? Belgian Journal of Zoology, 127, 19–28.

Mills, J.N. and Childs, J.E. 1998. Ecologic studies of rodent reservoirs: their relevance for human health. Emerging Infectious Diseases, 4, 529–537.

Mills, J.N. and Childs, J.E. 1999. Rodent-borne hemorrhagic fever viruses. In: Williams, E.S. and Barker, I. ed., Infectious diseases of wild mammals. Ames, Iowa State University Press (in press).

Mills, J.N., Childs, J.E., Ksiazek, T.G., Peters, C.J. and Velleca, W.M. 1995a. Methods for trapping and sampling small mammals for virologic testing. Atlanta, United States Department of Health and Human Services.

Mills, J.N., Childs, J.E., Ksiazek, T.G., Peters, C.J. and Velleca, W.M. 1998. Métodos para trampeo y muestreo de pequeños mamíferos para estudios virológicos, OPS/HPS/HCT98.104 edn. Washington, D.C., Organización Panamericana de la Salud.

Mills, J.N., Ellis, B.A., McKee, K.T., Calderón, G.E., Maiztegui, J.I., Nelson, G.O., Ksiazek, T.G., Peters, C.J. and Childs, J.E. 1992. A longitudinal study of Junín virus activity in the rodent reservoir of Argentine hemorrhagic fever. American Journal of Tropical Medicine and Hygiene, 47, 749–763.

Mills, J.N., Ellis, B.A., McKee, K.T.J., Ksiazek, T.G., Oro, J.G., Maiztegui, J.I., Calderon, G.E., Peters, C.J. and Childs, J.E. 1991. Junín virus activity in rodents from endemic and nonendemic loci in central Argentina. American Journal of Tropical Medicine and Hygiene, 44, 589–597.

Mills, J.N., Ksiazek, T.G., Ellis, B.A., Rollin, P.E., Nichol, S.T., Yates, T.L., Gannon, W.L., Levy, C.E., Engelthaler, D.M., Davis, T., Tanda, D.T., Frampton, W., Nichols, C.R., Peters, C.J. and Childs, J.E. 1997b. Patterns of association with host and habitat: antibody reactive with Sin Nombre virus in small mammals in the major biotic communities of the southwestern United States. American Journal of Tropical Medicine and Hygiene, 56, 273–284.

Mills, J.N., Ksiazek, T.G., Peters, C.J. and Childs, J.E. 1999a. Long-term studies of hantavirus reservoir populations in the southwestern United States: a synthesis. Emerging Infectious Diseases, 5, 135–142.

Mills, J.N., Yates, T.L., Childs, J.E., Parmenter, R.R., Ksiazek, T.G., Rollin, P.E. and Peters, C.J. 1995b. Guidelines for working with rodents potentially infected with hantavirus. Journal of Mammology, 76, 716–722.

Mills, J.N., Yates, T.L., Ksiazek, T.G., Peters, C.J. and Childs, J.E. 1999b. Long-term studies of hantavirus reservoir populations in the southwestern United States: rationale, potential, and methodology. Emerging Infectious Diseases, 5, 95–101.

Morzunov, S.P., Rowe, J.E., Ksiazek, T.G., Peters, C.J., St Jeor, S.C., and Nichol, S.T. 1998. Genetic analysis of the diversity and origin of hantaviruses in *Peromyscus leucopus* mice in North America. Journal of Virolology, 72, 57–64.

Murúa, R., Gonzáles, L.E., González, M. and Jofré, Y.C. 1996. Efectos del florecimiento del arbusto *Chusquea quila* Kunth (Poaceae) sobre la demografía de poblaciones de roedores de los bosques templados fríos del sur Chileno. Boletín de la Sociedad de Biología, Concepción, Chile, 67, 37–42.

Musser, G.G. and Carleton, M.D. 1993. Family Muridae. In: Wilson, D.E. and Reeder, D.M. ed., Mammal species of the world, a taxonomic and geographic reference, 2nd ed. Washington, D.C., Smithsonian Institution, 501–755.

Nichol, S.T., Spiropoulou, C.F., Morzunov, S., Rollin, P.E., Ksiazek, T.G., Feldmann, H., Sanchez, A., Childs, J.E., Zaki, S., and Peters, C.J. 1993. Genetic identification of a hantavirus associated with an outbreak of acute respiratory illness. Science, 262, 914–917.

Niklasson, B., Hornfeldt, B., Lundkvist, A., Bjorsten, S. and Le Duc, J. 1995. Temporal dynamics of Puumala virus antibody prevalence in voles and of nephropathia epidemica incidence in humans. American Journal of Tropical Medicine and Hygiene, 53, 134–140.

PAHO (Pan American Health Organization) 1982. Bolivian hemorrhagic fever. Epidemiological Bulletin Pan American Health Organization, 3, 1516.

Parmenter, R.R., Brunt, J.W., Moore, D.I. and Ernest, S. 1993. The hantavirus epidemic in the southwest: rodent population dynamics and the implications for transmission of hantavirus-associated adult respiratory distress syndrome (HARDS) in the four corners region. No. 41, University of New Mexico. Sevilleta LTER Publication.

Parodi, A.S., Greenway, D.J., Ruggiero, H.R., Rivero, E., Frigerio, M.J., Mettler, N., Garzon, F., Boxaca, M., de, G.L.B. and Nota, R. 1958. Sobre la etiología del brote epidémica de Junín. Día Médico, 30, 2300–2302.

Peters, C.J., Buchmeier, M., Rollin, P.E. and Ksiazek, T.G. 1996. Arenaviruses. In: Fields, B.N., Knipe, D.M. and Howley, P.M. ed., Virology. Philadelphia, Lippincott-Raven, 1521–1551.

Peters, C.J., Mills, J.N., Spiropoulou, C.F., Zaki, S.R. and Rollin, P.E. 1999. Hantaviruses. Chapter 113. In: Guerrant, R.L., Walker, D.H. and Weller, P.F. ed., Tropical infectious diseases, principles, pathogens, and practice. New York, W.B. Saunders, 1217–1235.

Plyusnin, A., Valpalahti, O., Vasilenko, V., Henttonen, H. and Vaheri, A. 1997. Dobrava hantavirus in Estonia: does the virus exist throughout Europe? Lancet, 349, 1369.

Redford, K.H. and Eisenberg, J.F. 1992. Mammals of the neotropics, the southern cone. Chicago, The University of Chicago Press.

Robbins, C.B. and Van Der Straeten, E. 1989. Comments on the systematics of *Mastomys* Thomas 1915 with the description of a new West African species. Senckenbergiana Biologica, 69, 114.

Salas, R., de Manzione, N., Tesh, R.B., Rico Hesse, R., Shope, R.E., Betancourt, A., Godoy, O., Bruzual, R., Pacheco, M.E., Ramos, B., Taibo, M.E., Tamayo, J.G., Jaimes, E., Vásquez, C., Araoz, F. and Querales, J. 1991. Venezuelan haemorrhagic fever a severe multisystem illness caused by a newly recognized arenavirus. Lancet, 338, 1033–1036.

Schmaljohn, C.S. and Hjelle, B. 1997. Hantaviruses: a global disease problem. Emerging Infectious Diseases, 3, 95–104.

Schmidt, K., Ksiazek, T.G. and Mills, J.N. 1998. Ecology and biologic characteristics of Argentine rodents with antibody to hantavirus. The Fourth International Conference on HFRS and Hantaviruses, March 5–7, 1998, Atlanta, Georgia, USA. (Abstract).

Toro, J., Vega, J.D., Khan, A.S., Mills, J.N., Padula, P., Terry, W., Yadon, Z., Valderrama, R., Ellis, B.A., Pavletic, C., Cerda, R., Zaki, S., Wun-Ju, S., Meyer, R., Tapia, M., Mansilla, C., Baro, M., Vergara, J.A., Concha, M., Calderón, G., Enria, D., Peters, C.J. and Ksiazek, T.G. 1998. An outbreak of hantavirus pulmonary syndrome, Chile, 1997. Emerging Infectious Diseases, 4, 687–694.

Wilson, D.E. and Reeder D.M. 1993. Mammal species of the world, a taxonomic and geographic reference. Washington, D.C., Smithsonian Institution Press.

Yahnke, C.J., Meserve, P.L., Ksiazek, T.G. and Mills, J.N. 1998. Prevalence of hantavirus antibody in wild populations of *Calomys laucha* in the central Paraguayan Chaco. The Fourth International Conference on HFRS and Hantaviruses, March 5–7, 1998. Atlanta, Georgia USA, (Abstract).

Appendix 1. Currently recognised hantaviruses and the diseases they produce, the small mammal host species and host distribution. Nomenclature and distributions from Wilson and Reeder (1993).

Host subfamily	Reservoir	Virus	Disease	Distribution of reservoir
Murinae	*Apodemus agrarius*	Hantaan	HFRS	C. Europe, S to Thrace, Caucasus, and Tien Mtns; Amur River through Korea, to E. Xizang and E. Yunnan, W. Sichuan, Fujiau, Taiwan.
	A. flavicollis	Dobrava	HFRS	England, Wales; NW Spain, France, Denmark, S. Scandinavia through European Russia, Italy, Balkans, Syria, Lebanon, Israel; Netherlands
	Bandicota indica	Thai	not known	Sri Lanka, India, Nepal, Burma, S. China, Taiwan, Thailand, Laos, Vietnam; introduced to Malay Peninsula and Java
	Rattus norvegicus	Seoul	not known	Nearly worldwide
Arvicolinae	*Clethrionomys glareolus*	Puumala	not known	France and Scandinavia to Lake Baikal, S to N Spain, N Italy, Balkans, W Turkey, N Kazakhstan; Britain, SW Ireland
	C. rufocanus	not named	not known	Scandinavia through Siberia to Kamchatka, S to Ural Mtns, Altai Mtns, Mongolia, Transbaikal, N. China, Korea, N. Japan
	Lemmus sibericus	Topografov	not known	Palearctic from White Sea, W Russia, to Chukotski Peninsula, NE Siberia, Kamchatka; Nearctic from W Alaska E to Baffin Island, Hudson Bay, S in Rocky Mtns to C. British Columbia
	Microtus arvalis	Tula	not known	Spain through Europe to Black Sea and Kirov region, Russia; Orkney Islands, Guernsey, and Yeu (France)
	M. rossiaemeridionalis	Tula	not known	From Finland E to Urals, S to Caucasus, through Ukraine E to Rumania, Bulgaria, S. Yugoslavia, N Greece, NW Turkey
	M. californicus	Isla Vista	not known	SW Oregon through California, USA, to N Baja California, Mexico
	M. fortis	Khabarovsk	not known	Transbaikal and Amur Region S though Nei Mongol and E China to lower Yangtze Valley and Fujian
	M. ochrogaster	Bloodland Lake	not known	EC Alberta to S Manitoba, Canada S to N Oklahoma and Arkansas E to C Tennessee and W Virginia, USA

Appendix 1. (Cont'd) Currently recognised hantaviruses and the diseases they produce, the small mammal host species and host distribution. Nomenclature and distributions from Wilson and Reeder (1993).

Host subfamily	Reservoir	Virus	Disease	Distribution of reservoir
Arvicolinae (cont'd)	*M. pennsylvanicus*	Prospect Hill	not known	C Alaska to Labrador, Newfoundland, Prince Edwards Island; S in Rocky Mtns to New Mexico, Great Plains to N Kansas, Appalachians to N Georgia, USA
Sigmodontinae	*Akodon azarae*	Pergamino	not known	NE Argentina, S Bolivia, Paraguay, Uruguay, S Brazil
	Bolomys obscurus	Maciel	not known	S Uruguay and EC Argentina
	Calomys laucha	Laguna Negra	HPS	N Argentina and Uruguay, SE Bolivia, W Paraguay, WC Brazil
	Oligoryzomys chacoensis	Bermejo	not known	W Paraguay, SE Bolivia, WC Brazil, N Argentina
	O. flavescens	Lechiguanas	HPS	SE Brazil, Uruguay, Argentina
	O. longicaudatus	Andes	HPS	Andes of Chile and Argentina
	O. longicaudatus?	Oran	HPS	Andes of Chile and Argentina
	O. microtis	Rio Mamore	not known	C Brazil, contiguous lowlands of Peru, Bolivia, Argentina
	Oryzomys palustris	Bayou	HPS	SE USA
	Peromyscus leucopus	New York	HPS	C and E USA into S and SE Canada, S to Yucatan Peninsula, Mexico
	P. maniculatus	Sin Nombre	HPS	Alaska across N Canada, S through USA to S Baja California and NC Oaxaca, Mexico
	Reithrodontomys megalotis	ElMoro Canyon	not known	SC British Columbia and SE Alberta, Canada, W and NC USA, S to N Baja California, and interior Mexico to C Oaxaca
	R. mexicanus	Rio Segundo	not known	S Tamaulipas and WC Michoacan, Mexico S to Panama; Andes of Columbia, Ecuador
	Sigmodon alstoni	Caño Delgadito	not known	NE Colombia, N and E Venezuela, Guyana, Surinam, and N Brazil
	S. hispidus	BlackCreek Canal	HPS	SE USA, interior Mexico to C Panama, N Colombia and N Venezuela
	Unknown	Juquitiba	HPS	(Human cases from Brazil)
Non-rodent	*Suncus murinus* (insectivore)	Thotopalayam	not known	Afghanistan, Pakistan, India, Sri Lanka, Nepal, Bhutan, Burma, China, Taiwan, Japan, Indomalayan region; introduced to coastal E Africa, Madagascar, Comores, Mauritius, Reunion & coastal Arabia.

Appendix 2. Currently recognised arenaviruses and the diseases they produce, small mammal host species and host distributions. Nomenclature and distributions from Wilson and Reeder (1993).

Host subfamily	Reservoir	Virus	Disease	Distribution of reservoir
Murinae	*Arvicanthus* sp.	Ippy	Not known	S Mauritania, Senegal, Gambia, E through Sierra Leone, Ivory Coast, Ghana, Burkina Faso, Togo, Benin, Nigeria, Niger, Chad, Sudan, Egypt, to Ethiopia; S through N Zaire, Uganda, S Burundi, Kenya, S Somalia & Tanzania, to E Zambia
	Mastomys natalensis	Mopeia	Not known	S Africa as far north as Angola, S Zaire, and Tanzania
	Mastomys spp.	Lassa	Lassa fever	Africa south of the Sahara
	Mus musculus	Lymphocytic choriomeningitis	LCM	Most of world in association with humans
	Praomys sp.	Mobala	Not known	C Nigeria through Cameroon Republic and Central African Republic, S. Sudan, Zaire, N Angola, Uganda, Rwanda, Kenya, south through E Tanzania to N and E Zambia
Sigmodontinae	*Bolomys obscurus*	Oliveros	Not known	S Uruguay and EC Argentina
	Calomys callosus	Machupo	Bolivian hemorrhagic fever	N Argentina, E Bolivia, W Paraguay, WC to EC Brazil
	C. callosus	Latino	Not known	N Argentina, E Bolivia, W Paraguay, WC to EC Brazil
	C. musculinus	Junín	Argentine hemorrhagic fever	N and C Argentina, E Paraguay
	Neacomys guianae	Amaparí	Not known	Guianas, S Venezuela, N Brazil
	Neotoma albigula	Whitewater Arroyo	Not known	SE California to S Colorado to W Texas, USA, south to Michoacan & W Hidalgo, Mexico
	Oryzomys buccinatus?	Paraná	Not known	E Paraguay and NE Argentina
	O. albigularis	Pichindé	Not known	N & W Venezuela, E Panama, Andes of Colombia & Ecuador to N Peru
	Oryzomys sp.?	Flexal	Not known	Not known
	S. alstoni	Pirital	Not known	NE Colombia, N and E Venezuela, Guyana, Surinam, N Brazil

Appendix 2. (Cont'd) Currently recognised arenaviruses and the diseases they produce, small mammal host species and host distributions. Nomenclature and distributions from Wilson and Reeder (1993).

Host subfamily	Reservoir	Virus	Disease	Distribution of reservoir
Sigmodontinae	*S. hispidus*	Tamiami	Not known	SE USA, Mexico to C Panama, N Colombia and N Venezuela
	Zygodontomys brevicauda	Guanarito	Venezuelan hemorrhagic fever	S Costa Rica through Panama, Colombia, Venezuela, Guianas, to N Brazil; including Trinidad & Tobago and smaller islands adjacent Panama & Venezuela
	Unknown	Sabiá	Unnamed	(Human cases from Sao Paulo State, Brazil)
Non-rodent	*Artibeus* (bats)?	Tacaribe	Not known	(Isolates from bats on Trinidad and Tobago)

Section 2

Methods of Management

7. Rodenticides — Their Role in Rodent Pest Management in Tropical Agriculture

Alan P. Buckle

Abstract

Rodents are serious pests of tropical agriculture. Most crops are attacked, particularly those grown for food by smallholders in the tropics. Globally, principal pest species include *Sigmodon hispidus*, *Arvicanthis niloticus*, *Mastomys natalensis*, *Meriones* spp., *Bandicota* spp., *Rattus argentiventer* and *Microtus* spp. Crop protection specialists usually recommend control programs based on integrated pest management (IPM) technologies involving the use of rodenticides in combination with various techniques of habitat manipulation. However, few proper IPM schemes have been developed and implemented on a wide-scale and long-term basis. Rodenticides are much used by growers. Acute compounds, such as zinc phosphide, are popular with smallholders because they are cheap but are rarely very effective. First generation anticoagulants (e.g. warfarin) are potentially effective, but only where their use is well managed because of the need for frequent applications of bait in relatively large quantities. Baits containing the potent second generation compounds (e.g. brodifacoum and flocoumafen) are likely to be the most effective because of the small amounts of bait and labour needed when they are applied, but questions remain about their potential to have adverse environmental impacts in agro-ecosystems. Rodenticides will be important in rodent pest management in tropical agriculture for the foreseeable future but much remains to be done to optimise their use. Improved decision-making methods, the wider assessment of non-target hazard, synergies between rodenticides and other rat management technologies and more sustainable extension programs are all areas requiring development. Unfortunately, few agencies now seem willing to expend effort on such research, although novel techniques to replace rodenticides still seem a long way off.

Keywords:

Rodents, rodenticides, rodent control, anticoagulants, resistance, integrated pest management, rice, sugar cane, oil palm, tropical crops

THE RODENT PESTS OF TROPICAL AGRICULTURE

FEW TROPICAL crops are free from rodent attack. Among common crops, perhaps only mature stands of rubber (*Hevea brasiliensis*) and some crops grown for fibre, such as sisal (*Agave sisalana*), are immune from damage by these ubiquitous pests. Crops grown in tropical agro-ecosystems for food, such as cereals (rice, wheat, maize, millet, barley and sorghum), roots, fruit, legumes and vegetables are particularly susceptible to rodent depredation. Also, crops cultivated on an industrial scale in plantations, such as sugarcane, coconut, cocoa and oil palm are commonly attacked. The extent of losses in these agro-ecosystems is highly variable. Two damage models may be recognised. In the first, if left unchecked by some form of management practice, rodent populations reach the carrying capacity of the standing crop they infest. This is frequently very high due to the abundant rodent food and cover that the crops offer. Economically significant losses in the region of 5–25% are often inflicted (Wood 1994). This type of damage may be overlooked both by farmers and crop protection specialists and becomes apparent only when carefully planned damage assessment programs are implemented over large crop areas (e.g. Posamentier 1989; Salvioni 1991). Within this model, patterns of the supply of irrigation water and subsequent harvesting sometimes concentrate rodent populations from a wide area into relatively small tracts of crop land at the end of the season and some farmers then suffer very heavy losses. The second pattern of damage is one in which certain overriding climatological or demographic phenomena create specific conditions for rodent populations to reach extraordinary, or plague, levels. At such times crops may be totally devastated. The development of very high populations of *Mastomys natalensis* after unseasonal rains in East African croplands is an example of this type of episode (Mwanjabe and Leirs 1997). Another is the very high populations of rodents that occur in some parts of Southeast Asia coincident with the irregular flowering of bamboo forests (Singleton and Petch 1994).

The number of tropical rodent pest species involved is very large and appears to present a bewildering challenge to those attempting to develop sound management strategies. However, global rodent pest problems were classified following work by the Expert Consultation of the Organisation for Economic Cooperation and Development, Food and Agriculture Organization and the World Health Organisation into seven key components of global significance (Drummond 1978) and this still provides a useful framework. Six of these problems are to be found in tropical and sub-tropical, food-crop, smallholder agriculture (Table 1). The seventh is the cosmopolitan problem of rodent damage to stored products, mainly by *Rattus norvegicus* and *Rattus rattus*.

The purpose of this chapter is to review some of the learnings obtained from a number of research and development projects aimed at introducing management strategies for these pests of tropical agriculture. The majority of these projects were broadly based investigations including the assessment of damage levels, studies of rodent biology and the development and implementation of rodent management methods. In relation to the latter, many studies were based on the use of rodenticides, although a number of subsidiary techniques were frequently incorporated to provide elements of integrated pest management (IPM).

INTEGRATED PEST MANAGEMENT AND THE USE OF RODENTICIDES

Few who devise and evaluate rodent management strategies fail to advocate integrated approaches as the most reliable, long-term solutions to rodent problems (see Richards and Buckle 1987; Mwanjabe and Leirs 1997, among many others). A review of the principles of rodent IPM was recently provided by Singleton (1997). This analysis indicates that strong IPM programs must be environmentally sound, cost-effective, sustainable, capable of application over large areas and recognisably advantageous, both for growers who implement them and politicians who support and fund them. However, after many years of work by a wide range of national and international agencies very few schemes currently operate to fulfil these criteria (Leirs 1997).

All too often those who conduct rodent control programs pay only lip service to IPM ideas and rely almost solely on rodenticides. There are many reasons for this but paramount is the fact that, although potentially effective, many of the techniques that complement rodenticides in IPM are labour-intensive and their impact is not immediately obvious to those who must invest scarce resources to implement them; in effect they do not satisfy Singleton's criteria. The control of rice-field rats in Southeast Asia through habitat manipulation is a case in point.

[The following is based mainly on work with *Rattus argentiventer* (Lam 1978, 1990) but may be relevant to other rice rat species in Asia, such as *Rattus flavipectus*, *Rattus losea* and *Rattus rattus mindanensis*, and also elsewhere.] Some of the conditions of rice cultivation that exacerbate rat problems have been long understood (Buckle et al.1985; Lam 1990; Leung et al., Chapter 14).

Table 1.
The world's major rodent pests of agriculture (from Drummond 1978)*.

Rodent pest species involved	Area affected	Crops attacked
Sigmodon hispidus	Central and Latin America	rice, sugar, cotton
Arvicanthis niloticus, *Mastomys (Praomys) natalensis*	sub-Saharan Africa	food crops
Meriones spp.	North Africa, Middle East	cereals
Bandicota bengalensis	Indian sub-continent, Southeast Asia	sugar, cereals, food crops
Rattus argentiventer	Southeast Asia	rice (oil palm)
Rattus rattus, *Rattus norvegicus*, *Rattus exulans*	Oceanic islands	coconuts, food crops

* For various reasons certain regions and pests were omitted in this analysis. However, a complete list of global rodent pest problems of open-field agriculture would certainly also include those caused by *Rattus flavipectus* in southern China and Indochina, *Microtus* spp. across the Holarctic and *Mus musculus* in mainland Australia.

Rats choose to build nests for breeding almost exclusively in rice-field bunds that are more than about 300 mm wide and 150 mm above water level. They breed primarily during the reproductive stages of the rice plants and asynchronous planting allows prolonged breeding by permitting rats to move from harvested fields to others nearby where rice is still at an appropriate stage for reproduction. Weedy rice fields (Drost and Moody 1982), as well as overgrown, uncultivated areas either in or nearby rice fields provide refugia for rats and supplementary sources of food. Habitat manipulation measures to overcome these problems are obvious; a reduction in bund size, synchronous sowing/transplanting and clean rice field cultivation practices, but all are almost impossible to implement on a wide scale because of other, overriding agronomic and socioeconomic factors.

In contrast, rodenticides have a high potential to contribute useful elements within rodent IPM strategies (Singleton 1997). Of particular importance is their relatively low cost, both in terms of the price of baits in relation to the value of the crop to be protected and the labour needed to apply them. Therefore, rodenticides seem likely to remain central to rodent management strategies for some time to come.

RODENTICIDES AND THEIR USE IN TROPICAL AGRICULTURE

The types of rodenticides, the techniques used in their application and some of their advantages and disadvantages were reviewed recently in a general account by Buckle (1994). A discussion of them is given here in relation, particularly, to their application in tropical agriculture.

Acute rodenticides

The fast-acting, acute rodenticides are still much used by tropical smallholders, although zinc phosphide is now the only specific rodenticide in this class that remains widely available. In the absence of alternatives, growers frequently apply as rodenticides other compounds with high mammalian toxicities (e.g. certain organochlorine and organo-phosphide insecticides) contrary to the regulatory approval of the compounds concerned.

The benefits of the acute compounds mainly lie in their ready availability, low cost and rapid action. They are favoured by tropical farmers because their effects are apparent almost immediately after application. To be set against these advantages are their disadvantages. They are sold as concentrates and before use must be mixed with bait bases, usually cereals, to the desired concentrations. Tropical smallholders are ill-equipped to do this safely and accurately and often, cereals of sufficiently high quality to provide attractive baits are scarce. Acute rodenticides are sold as powder concentrates and are particularly prone to adulteration during manufacture and distribution. These characteristics result in baits of very dubious quality. Even when they are properly made, acute rodenticide baits have the drawback of eliciting 'bait shyness'. This is where the onset of symptoms of poisoning in sub-lethally dosed animals is so rapid that rodents are able to relate them to the novel food (the bait) which has caused them. Bait shy rodents are those that will avoid contact with the poisoned bait when it is applied in future. The likelihood of this occurring may be reduced, but not eliminated, by the use of 'pre-baiting'. In this, the bait base later to be used in the poisoning campaign is first offered without poison for several days. Rodents slowly overcome their suspicion of the novel food (neophobia) and eventually feed consistently. Only then is the acute poison introduced. The use of pre-baiting to overcome neophobia and reduce bait shyness is time-consuming, poorly understood by smallholders and rarely practised.

Probably the best results that can be anticipated with the use of zinc phosphide baits, under practical conditions, were demonstrated by Rennison (1976) on farms in the United Kingdom. Zinc phosphide baits, at 2.5% concentration, were applied by trained and experienced rodent control operators. Pre-treatment population assessment was done by census baiting and this provided a form of pre-baiting. An average level of control of 84% of *R. norvegicus* was achieved. Few good studies have been conducted on the efficacy of acute rodenticides in tropical agriculture and it is unlikely that this level of success is ever achieved. Most studies have suffered from a lack of replication, plot sizes that are too small and with insufficient separation between plots receiving different treatments, poor (or no) statistical analysis and, often, a lack of detailed explanation of the methods employed (see Chia et al. 1990 for a discussion of field trial methodology). These failings are common among field studies of rodenticides and it is not surprising, therefore, that highly variable results have been obtained (West et al. 1975; Lam 1977; Mathur 1997). In spite of the shortcomings of zinc phosphide, Adhikarya and Posamentier (1987) used manufactured zinc phosphide bait cakes in a successful large-scale rodent control campaign in cereals in Bangladesh.

The recommended concentration of zinc phosphide for field use varies from 1% to 5%. Zinc baits are generally unpalatable to rodents and a compromise between the active ingredient concentration used and the quantity of bait likely to be eaten must be reached with the objective of administering the maximum quantity of the active ingredient. The preferred concentration is probably 2–2.5% (MAFF 1976). The bait bases used are locally available cereals. They may be soaked overnight in water before the zinc phosphide is added and this is thought to enhance uptake (MAFF 1976) but reduces the stability of bait. The baits are placed in small piles of 20–50 g at intervals of 5–20 m on bunds in rice fields or, in other crops, wherever rodents are active (Lam 1977; Mwanjabe and Leirs 1997). The rate of application may be varied, both by the weight of bait used and the distance between bait points, in order to accommodate different pest infestation densities. Undoubtedly, a few days of pre-baiting with the cereal to be used later as the carrier for the active ingredient will enhance effectiveness.

First generation anticoagulants

The archetypal first generation anticoagulant rodenticide is warfarin. After its introduction in the early 1950s, a number of other compounds were developed, including pival, coumachlor, coumatetralyl, and the indandiones — diphacinone and chlorophacinone. However, with the possible exception of coumatetralyl (e.g. Greaves and Ayres 1969; Buckle et al. 1982), there is little evidence that these compounds differ much from each other in their efficacy. All these compounds are most potent when administered in small daily doses. However, their most advantageous common feature is their chronic mode of action, which means that bait shyness does not arise. These novel features required the development of a different means of quantifying the potency of the first generation anticoagulants. This was done in terms of the number of days of consumption of field strength baits required to obtain a given mortality percentile and resulted in the expression 'lethal feeding period' (LFP).

Warfarin was first developed for use against the Norway rat and it is particularly effective against that species (Table 2). Used against Norway rats in commensal situations and in animal husbandry (pig/poultry sheds, dairies, beef-rearing units) and other farm buildings (mills and granaries) the virtual elimination of Norway rat infestations was possible for the first time. However, other species are less susceptible to it and among the least susceptible are some important pests of tropical agriculture, such as *Mastomys natalensis*, *Meriones* spp., *Bandicota* spp., *R. argentiventer and R. rattus*. Greaves (1985) gave data for 'natural resistance' to warfarin for 11 rodent species, of which nine were pests of agriculture (Table 2). This shows that for only three species (*R. norvegicus*, *Sigmodon hispidus* and *Arvicanthis niloticus*) is the LFP_{99} less than 14 days.

It is a reasonable conclusion that warfarin (and the other similar compounds) is unlikely to be as effective when used in agriculture as it is in commensal situations if more than two weeks of continuous no-choice feeding is required to deliver an LFP_{99}.

Table 2.

'Natural resistance' to warfarin of key rodent pest species as indicated by the number of days of no-choice feeding on 250 ppm warfarin baits to achieve lethal feeding period (LFP)50 and LFP99 percentiles (from Greaves 1985)

Rodent species	Feeding period (days)	
	LFP_{50}	LFP_{99}
Nesokia indica	1.9	3797.0
Acomys caharinus	5.4	239.3
Mus musculus	4.8	29.5
Mastomys natalensis	4.8	26.0
Bandicota indica	1.4	25.0
Rattus rattus	3.6	21.0
Tatera indica	5.8	19.2
Rattus argentiventer	3.2	15.5
Sigmodon hispidus	3.7	8.1
Arvicanthis niloticus	3.8	6.0
Rattus norvegicus	1.7	5.8

Even against susceptible species, the effective use of the first generation anticoagulants requires that baits are available for consumption by rodents, more or less continuously, for several weeks. Baiting programs were developed, primarily in the Philippines, for use in tropical agriculture with this requirement in mind (Hoque and Olvida 1987; Sumangil 1990). Baiting stations were put out at a density of two to five per hectare and supplied with about 150 g of bait. The bait used was generally whole or broken rice grains treated with anticoagulant powder concentrates and oil as a sticker. The bait stations were checked at frequent intervals (at least weekly) and the bait replenished. More bait and baiting stations were put out at sites where complete takes were encountered and baiting continued until takes of bait ceased or the crop was harvested. This technique came to be called 'sustained baiting' and its development, extension to smallholder groups and practical application on a nationwide basis is chronicled in the reports of the Rodent Research Centre, at Los Baños, through the mid and late 1970s. This technique remains the only practicable method of application of loose baits containing the warfarin-like compounds in tropical agriculture.

The sustained baiting technique was adapted for use with wax-block baits containing warfarin in oil palm plantations in Malaysia (Wood 1969). In this practice, a single 15 g block was placed in the weeded circles of each palm. The baits were checked at four-day intervals and replenished where they were taken. Baiting continued until the requirement to replenish baits declined to a predetermined percentage of bait placements, normally 20%. An important advantage of this system was that the use of wax blocks removed the need for fabricated bait stations to protect the bait.

All applications of rodenticides in agriculture are more cost effective, and their effectiveness more long lasting, when large crop areas are treated simultaneously. Thus, if large numbers of smallholders are mobilised to conduct baiting campaigns, the effort required by each farmer is minimised, the quantities of bait used are small and the duration of baiting is short (e.g. Buckle 1988). However, the sustained baiting method can be employed successfully by single smallholders in small plots, but almost continuous baiting may be needed. This creates a 'sink' into which are drawn rodents from a wide area. Clearly, this benefits more farmers than the one conducting baiting and may not be sustainable because its cost falls so inequitably, both in terms of effort and money. Using such a system, Sumangil (1990) used 44 kg of bait per treated hectare on small farms, during a 12-week rice growing season, where rats were numerous.

Second generation anticoagulants

Resistance to the first generation anticoagulants led to the development of a further series of compounds of greater potency that were effective against resistant rodents. These include difenacoum, bromadiolone, brodifacoum, flocoumafen and difethialone. A third generation of compounds is occasionally referred to in some publications. The last three compounds differ from the first two in being more potent but none differs sufficiently from any other to be considered in a class apart.

Early tests of brodifacoum focused on the objective of obtaining a degree of effectiveness against resistant animals that was equivalent to that of warfarin when used against fully susceptible ones. Very low concentrations in baits (5 to 20 ppm) fed over several days were sufficient to achieve this objective (Redfern et al. 1976). However, it was soon observed that 50 ppm brodifacoum baits were effective at providing very high levels of kill, against both susceptible and resistant rodents, when rodents fed for only one day on small amounts of bait (see, for example, Buckle et al. 1982, for *R. argentiventer*). However this benefit could not be readily realised as a practical advantage because the delayed effects of brodifacoum, as an anticoagulant, meant that given free access to bait, rodents consume much more before they die than actually needed to kill them. This resulted in the development of a technique called 'pulsed baiting' in which relatively small quantities of bait are put out at intervals between which there is a period in which bait is virtually absent; allowing rodents that have consumed a lethal dose to die before a subsequent application (see Buckle et al. 1984; Dubock 1984). The principle practical benefit to arise from the use of pulsed baiting in agriculture is that the quantity of bait used is substantially reduced. Successful campaigns have been conducted in which application rates as low as one to two kilograms of bait per hectare have been used (Buckle 1988). To the advantage of a reduction in the cost of bait and labour required to transport and apply it is added a reduction in the amount of active ingredient that enters the environment. The use of this technique, with wax-block baits containing one of the potent second generation anticoagulants, provides the most practical and cost-effective method of rodent control using rodenticides currently available.

Anticoagulant resistance

Resistance to anticoagulants is uncommon in tropical agriculture. There seems to be a relationship between the time taken for anticoagulant resistance to develop and the degree of selection pressure applied (i.e. the frequency of use of the anticoagulants and the proportion of the pest population exposed). In the tropics, only in oil palm plantations in Malaysia has this pressure been such that widespread resistance has developed to the first generation anticoagulants (Lam 1984; Wood and Chung 1990). In the United Kingdom, where resistance has arguably reached its current extreme, nowhere are resistant rodent populations impossible to control with available techniques, although there is a cost in terms of the need to use the more potent compounds, sometimes for periods longer than normal (Greaves 1994) and in greater quantities. This perspective is not intended to generate complacency. When anticoagulants are used in tropical agriculture it is essential to establish susceptibility baselines and to monitor pest populations for subsequent changes in susceptibility. Published guidelines set out how this should be done (EPPO 1995). These baseline studies would also provide preliminary performance data on active ingredients and the baits that contain them.

Decision-making

Rodent pest problems in tropical crops are rarely uniform, either in time or space. If a rodenticide (or any other control measure) is to be used cost-effectively, a process is required by which to decide when and where to apply it. Frequently in tropical smallholder agriculture this decision is made on the basis of subjective judgement, either by individuals or small groups of growers, and is often made too late. It is well established that cost-effective rodent management is most likely when efforts are co-ordinated over substantial crop areas. Surveillance and forecasting systems have been devised to assist decision-makers in these circumstances, based either on information on pest density or on meteorological observations.

Surveillance systems based on pest density have been worked out for sugarcane, oil palm and rice. In sugarcane, the 'Hawaii trapping index' (Hampson 1984) is widely used to determine the need for rodenticide applications. Snap-trap lines are set and rodent population density, expressed as an index of trapping success, is used as a decision-making tool. The pitfalls of this technique were pointed out by Hampson (1984) but no better method has been devised in spite of the great economic importance of the crop and the significance of rodent damage as a constraint to production in some areas.

The assessment of rodent damage can be used as another indirect index of rodent density and, if the relationship between damage and crop loss is understood, provides additional data on the latter important parameter. However, much work remains to be done in the majority of crops on the relationships between rodent population density, damage levels and crop losses. An aid to decision-making in this context is the establishment of the economic injury level, determined as follows (Dolbeer 1981):

$$T\% = 100(Y/bX) \tag{1}$$

where

T = economic injury level;
Y = cost of control;
X = value of potential crop loss;
b = constant representing the proportion of potential loss saved by control.

Khoo (1980) proposed a systematic damage sampling scheme for use in oil palm plantations in which the percentage of palms with fresh damage to fruit bunches provided a criterion dictating the need for the application of pulsed baiting with second generation anticoagulants. Buckle (1988) conducted large-scale pilot trials of an integrated rice rat management scheme which involved farmers undertaking frequent monitoring of the percentage of rice hills with rat damage as a trigger for the need for control action. This parameter is related to, and more easily assessed than, the percentage of rat-damaged rice tillers. The advantages of these methods are that assessments may be conducted by the growers themselves, the data obtained reflects the level of rat damage/yield loss and that it is possible to target decisions so that applications may be made, when necessary, to land parcels of moderate size (e.g. 50 to 100 ha). A disadvantage is that sampling is relatively labour intensive.

Climatic factors are of limited importance as determinants of pest population densities in seasonal irrigated crops (e.g. lowland rice) and in perennial crops (e.g. oil palm) and decision-making is then best founded on measures of pest populations. However, in rain-fed crops early-warning systems based on rainfall data may be useful in predicting rodent pest outbreaks. Mwanjabe and Leirs (1997) used such a system, combined with damage assessments, to predict outbreaks of *M. natalensis* in Tanzanian maize fields. An advantage of this approach is that useful information is obtained from established national meteorological monitoring systems. Although substantial work is required on a case-by-case basis to validate the predictive accuracy of such methodology.

The mechanisms briefly described have been developed in order to target rodent control efforts more efficiently but decision-making is always likely to carry some uncertainty. Pest populations may be low until a susceptible crop stage is reached and then there may be a rapid influx from neighbouring infested habitats. This makes it necessary to monitor pest populations in quite large areas and not exclusively in croplands. All these systems require a degree of coordination from some central agency, such as a national surveillance network or a plantation crop protection advisor. Only the method developed for use in sugarcane is widely adopted and none of those designed for tropical smallholders has yet gained general acceptance. The challenge remains to crop protection researchers and vertebrate pest managers, therefore, to develop and implement practical and effective systems for monitoring rodent pest populations to allow timely and appropriate management actions to be taken in tropical smallholder agriculture. Whether direct rodent population density measures or indirect gauges, such as damage assessments, are used as the decision-making tools, a reasonable understanding of the dynamic relationships between rodent populations, damage and yield loss is needed.

ASSESSMENT OF NON-TARGET HAZARDS

In the 'good IPM check-list' provided by Singleton (1997) it is probably the need for schemes to be environmentally sound that has caused a degree of reluctance among crop protection researchers to use methods based on rodenticides in tropical agriculture. Of course, the requirement for crop protection practices to inflict no unnecessary harm on the environment is of the highest importance but refusal to work with rodenticides has now reached the level of 'chemophobia' in some quarters. In the opening chapter of this book a 20-year hiatus is mentioned during which little progress has been made in rodent pest management in developing countries. This is partly attributed to too much an emphasis on rodenticides. However, the contention that very little innovative work on rodenticides has been done in tropical agriculture for the past 10 years is borne out by a study of the reference list of this chapter and very few projects receiving funding from international agencies have included any substantial element concerned with improving rodenticide applications.

Methods for the assessment of the potential for rodenticide applications to harm the environment are well established (Edwards et al. 1988; Brown 1994). Generally, rodenticide applications pose negligible risks to soil and aquatic systems because of the nature of the compounds and their methods of use. This is particularly the case with anticoagulants. Baits are discrete, used at low rates of application, and carry low concentrations of, usually, insoluble active ingredients, which are bound readily to soil particles and do not move into plants. However, by their nature, all rodenticides are potent vertebrate toxicants. Their principle risks lie in the potential for non-target animals to consume directly baits laid for rodents (primary poisoning) and for scavengers and predators themselves to be poisoned when consuming the bodies of contaminated rodents (secondary poisoning). Those few extensive field studies that have been performed to quantify these potential effects (Tongtavee et al. 1987; Hoque and Olvida 1988) have shown that pulsed baiting with wax blocks containing second generation anticoagulants poses few risks to wildlife populations in Southeast Asian rice fields, but more studies are needed both in rice and in other agro-ecosystems.

All rodent management techniques have the potential to affect the environment adversely and this is not restricted only to those methods based on rodenticides. For example, the habitat modification methods often recommended in rice fields (removal of weedy land patches, lowering of bunds, increasing field size, periodic deep flooding of growing areas and extensive synchronous planting) would have a significant detrimental effect on a very wide range of non-target taxa that rely on these remnant habitats as their only footholds in an otherwise ecologically barren rice monoculture. Such potential impact needs to be weighed against the possible effect of occasional rodenticide use on a limited number of predatory and scavenging species, but such thinking is seldom done.

EXTENSION

The fact that smallholders are the most likely agency by which rodent control measures, particularly rodenticides, are to be applied is often overlooked by those developing management techniques (Posamentier 1997). Conflicting pressures on smallholders' time and money, their uncertain perception of the importance of the pest problem being addressed and many other socioeconomic factors jeopardise the sustainability of otherwise well-designed and cost-effective schemes. Adhikarya and Posamentier (1987) undertook a 'knowledge, attitude and practice' (KAP) survey to establish first baseline information on rodent control practice and perceptions among smallholder cereal growers in Bangladesh. Armed with this information they designed a multi-media campaign to modify beliefs and stimulate action. This substantial program met with considerable initial success but its long-term impact is uncertain.

It is tempting to look for successful models of extension of sustainable rodent control programs and attempt to draw lessons from them. A search for such models in current smallholder tropical agriculture is largely fruitless (Leirs 1997). However, oil palm plantations in Malaysia have long benefited from well organised control programs based on anticoagulant baiting. These programs are founded on a base of long-term research on the biology and control of the pest funded by those with most to gain from its results, the plantation sector agri-businesses. As a result, *Rattus tiomanicus* is arguably the best understood rodent pest of tropical agriculture (see Wood 1984; Wood and Liau 1984 a,b). Within an estate, or estate group, rat management decisions are made by a single person or small team, on the basis of well-understood economic criteria. Resources are usually available to conduct control operations as and when necessary. Rodenticide applications are made by trained workers, with no other distracting tasks on the day of application, and baits are applied over extensive areas with reasonable expectation, therefore, that the investment will be rewarded. The situation in smallholder cropping could not be more different. Several agencies may be responsible for decisions, including government crop protection, surveillance and extension services, farmer groups and individual growers; all with their own inertia and affected by different motivational factors. The financial implications of action or inaction are poorly understood and money is rarely available when it is needed. Work is done by poorly-trained smallholders, with conflicting time constraints, and the treatments are too often made on small areas with little chance of return on investment from higher crop yields. In some respects this is an unequal comparison however. Oil palm is a perennial crop and lends itself to rodent pest management because of the constancy of conditions within crop fields. Whereas, smallholder systems based on a mosaic of crop types and pest problems present much more difficult conditions. Nevertheless, until some of the problems mentioned above are overcome the current poor status of rodent pest management in tropical agriculture mentioned by Leirs (1997) and in the first chapter of this book will remain.

CONCLUSIONS

In the medium and long-term we look forward to the introduction of novel technologies for rodent pest management The beginnings of some of these are described elsewhere in this book. Those engaged in their development must keep sight of the reasons for the past failure of what were considered to be well-designed crop protection systems but which proved to be impractical or unsustainable (Singleton 1997). Presently, however, there is an urgent need for improved rodent pest management in many smallholder agro-ecosystems in order to alleviate immediate hardship. For the time being these are best founded on IPM principles, with rodenticides used as an important element. However, more work is still needed. Decision-making systems are required to help hard-pressed crop protections workers to determine when and where management programs are needed. More extensive field studies of the non-target hazards of rodenticides are required so that objective data are available in order to dispel fears, if these prove to be unwarranted, of unacceptable adverse environmental impacts. Also, the development is needed of innovative extension technologies to motivate smallholder farming communities and to make well-designed rodent pest management programs sustainable.

REFERENCES

Adhikarya, R. and Posamentier, H. 1987. Motivating farmers for action; how multi-media campaigns can help. Deutsche Gesellschaft für Technische Zusammenarbeit (GTZ) GmbH, Eschborn, Germany, Schriftenreihe, 185, 209p.

Brown, R.A. 1994. Assessing the environmental impact of rodenticides. In: Buckle, A.P. and Smith, R.H., ed., Rodent pests and their control. Wallingford, UK, CAB International, Wallingford, 363–380.

Buckle, A.P. 1988. Integrated management of rice rats in Indonesia. FAO (Food and Agriculture Organization of the United Nations) Plant Protection Bulletin, 36, 111–118.

Buckle, A.P. 1994. Rodent control methods: chemical. In: Buckle, A.P. and Smith, R.H., ed., Rodent pests and their control. Wallingford, UK, CAB International, 127–160.

Buckle, A.P., Rowe, F.P. and Abdul Rahman, H. 1982. Laboratory evaluation of the anticoagulants coumatetralyl and brodifacoum against *Rattus argentiventer* in peninsular Malaysia. Tropical Pest Management, 28, 126–130.

Buckle, A.P., Rowe, F.P. and Abdul Rahman, H. 1984. Field trials of warfarin and brodifacoum wax block baits for the control of the rice field rat, *Rattus argentiventer*, in peninsular Malaysia. Tropical Pest Management, 30, 51–58.

Buckle, A.P., Yong, Y.C. and Rowe, F.P. 1985. Damage by rats to rice in South-East Asia with special reference to an integrated management scheme proposed for peninsular Malaysia. Acta Zoologica Fennica, 173, 139–144.

Chia, T.H., Buckle, A.P., Visvalingam, M. and Fenn, M.G.P. 1990. Methods for the evaluation of rodenticides in oil palm plantations. In: Proceedings of the Third International Conference on Plant Protection in the Tropics, 20–23 March 1990, Genting Highlands, Malaysia. Malaysian Plant Protection Society, 19–26.

Dolbeer, R.A. 1981. Cost–benefits determination of black bird damage for corn fields. Wildlife Society Bulletin, 9, 45–51.

Drost, D.C. and Moody, K. 1982. Rat damage in weed control experiments in rainfed transplanted rice. Tropical Pest Management, 28, 295–299.

Drummond, D.C. 1978. The major agricultural rodent problems of developing countries. Paper presented to the OECD/FAO/WHO Expert Consultation on Rodent Problems, Control and Research, Paris, France, 2–5 May 1978, 34p.

Dubock, A.C. 1984. Pulsed baiting—a new technique for high potency, slow acting rodenticides. In: Dubock, A.C., ed., Proceedings of the Conference on the Organisation and Practice of Vertebrate Pest Control, Elvetham Hall, UK, 30 August to 3 September 1982, 105–142.

EPPO (European Plant Protection Organization) 1995. Guideline for the evaluation of resistance to plant protection products. Testing rodents for resistance to anticoagulant rodenticides. EPPO Bulletin, 25, 575–593.

Edwards, P.J., Brown, R.A., Coulson, J.M. and Buckle, A.P. 1988. Field methods for studying the non-target hazard of rodenticides. In: Greaves, M.P., Smith, B.D. and Greig-Smith, P.W., ed., Field methods for the study of environmental effects of pesticides. BCPC Monograph No.40. Thornton Heath, UK, British Crop Protection Council, 77–88.

Greaves, J.H. 1985. The present status of resistance to anticoagulants. Acta Zoologica Fennica, 173, 159–162.

Greaves, J.H. 1994. Resistance to anticoagulant rodenticides. In: Buckle, A.P. and Smith, R.H., ed., Rodent pests and their control. Wallingford, UK, CAB International, 197–217.

Greaves, J.H. and Ayres, P. 1969. Some rodenticidal properties of coumatetralyl. Journal of Hygiene, Cambridge, 67, 311–315.

Hampson, S.J. 1984. A review of rodent damage to sugar cane with criteria for the use of rodenticides. In: Dubock, A.C., ed., Proceedings of the Conference on the Organisation and Practice of Vertebrate Pest Control, Elvetham Hall, UK, 30 August to 3 September 1982, 227–251.

Hoque, M.M. and Olvida, J.L. 1987. Comparison of baiting methods for ricefield rats in the Philippines. In: Richards, C.G.J.R. and Ku, T.Y., ed., Control of mammal pests. London, Taylor and Francis, 237–248.

Hoque, M.M. and Olvida, J.L. 1988. Efficacy and environmental impact of flocoumafen (Storm) wax block baits used for rice field rat control in the Philippines. In: Crabb, A.C. and Marsh, R.E., ed., Proceedings of the Thirteenth Vertebrate Pest Conference, 1–3 March 1988, Monterey, USA, 75–81.

Khoo, C.K. 1980. 'Pulsed baiting' with brodifacoum baits to control oil palm rats in Malaysia. Selangor Planters Associations, Annual Report/Journal, 198, 42–46.

Lam, Y.M. 1977. Zinc phosphide—an effective rodenticide for the rice field rat, *Rattus argentiventer*. Malaysian Agricultural Journal, 51, 228–237.

Lam, Y.M. 1978. The ricefield rat. Information Paper No.2. Malaysian Agriculture Research and Development Institute, 37p.

Lam, Y.M. 1984. Further evidence of resistance to warfarin in *Rattus rattus diardii*. MARDI (Malaysian Agricultural Research and Development Institute) Research Bulletin, 12, 373–379.

Lam, Y.M. 1990. Cultural control of rice field rats. In: Quick, G.R., ed., Rodents and rice. Report of an expert panel meeting on rice rodent control, 10–14 September 1990, Los Baños, Philippines, 65–72.

Leirs, H. 1997. Preface. In: Leirs, H. and Schockaert, E., ed., Proceedings of the International Workshop on Rodent Biology and Integrated Pest Management in Africa, 21–25 October 1996, Morogoro, Tanzania. Belgian Journal of Zoology, 127 (Supplement 1), 3–5.

MAFF (Ministry of Agriculture, Fisheries and Food) 1976. Control of rats and mice—reference manual for pest control personnel. United Kingdom, MAFF, 97p.

Mathur, R.P. 1997. Effectiveness of various rodent control measures in cereal crops and plantations in India. In: Leirs, H. and Schockaert, E., ed., Proceedings of the International Workshop on Rodent Biology and Integrated Pest Management in Africa, 21–25 October 1996, Morogoro, Tanzania. Belgian Journal of Zoology, 127 (supplement 1), 137–144.

Mwanjabe, P.S. and Leirs, H. 1997. An early warning system for IPM-based rodent control in smallholder farming systems in Tanzania. In: Leirs, H. and Schockaert, E., ed., Proceedings of the International Workshop on Rodent Biology and Integrated Pest Management in Africa, 21–25 October 1996, Morogoro, Tanzania. Belgian Journal of Zoology, 127 (supplement 1), 49–58.

Posamentier, H. 1989. Rodents in agriculture—review of findings in Bangladesh. Sonderpublikation der GTZ No.16. Deutsche Gesellschaft für Technische Zusammenarbeit (GTZ) GmbH, Eschborn, Germany, 108p.

Posamentier, H. 1997. Communication in national rodent management programmes. In: Leirs, H. and Schockaert, E., ed., Proceedings of the International Workshop on Rodent Biology and Integrated Pest Management in Africa, 21–25 October 1996, Morogoro, Tanzania. Belgian Journal of Zoology, 127 (supplement 1), 171–180.

Redfern, R., Gill, J.E. and Hadler, M.H. 1976. Laboratory evaluation of WBA 8119 as a rodenticide against warfarin-resistant and non-resistant rats and mice. Journal of Hygiene, Cambridge, 77, 419–426.

Rennison, B.D. 1976. A comparative field trial, conducted without pre-baiting, of the rodenticides zinc phosphide, thallium sulphate and gophacide against *Rattus norvegicus*. Journal of Hygiene, Cambridge, 77, 55–62.

Richards, C.G.J.R. and Buckle, A.P. 1987. Towards integrated rodent pest management at the village level. In: Richards, C.G.J.R. and Ku, T.Y., ed., Control of mammal pests. London, Taylor and Francis, 293–312.

Salvioni, M. 1991. Evaluation of rat damage in rice fields in Madagascar. Tropical Pest Management, 37, 175–178.

Singleton, G.R. 1997. Integrated management of rodents: a Southeast Asia and Australian perspective. In: Leirs, H. and Schockaert, E., ed., Proceedings of the International Workshop on Rodent Biology and Integrated

Pest Management in Africa, 21–25 October 1996, Morogoro, Tanzania. Belgian Journal of Zoology, 127 (supplement 1) , 157–169.

Singleton, G.R. and Petch, D.A. 1994. A review of the biology and management of rodent pests in Southeast Asia. Australian Centre for International Agriculture Research Technical Reports, 30, 65p.

Sumangil, J.P. 1990. Control of ricefield rats in the Philippines. In: Quick, G.R., ed., Rodents and rice. Report of an expert panel meeting on rice rodent control, 10–14 September 1990, Los Baños, Philippines, 35–47.

Tongtavee, K., Hongnark, S., Atrchawakom, T., Hongsbhanich, N., Brown, R.A. and Richards, C.G.J.R. 1987. The safety and efficacy of brodifacoum (Klerat) wax blocks and zinc phosphide for rodent control in Thailand. In: Richards, C.G.J.R. and Ku, T.Y., ed., Control of mammal pests. London, Taylor and Francis, 173–180.

West, R.R., Fall, M.W. and Libay, J.L. 1975. Field trial of multiple baiting with zinc phosphide to protect growing rice from damage by rats (*Rattus rattus mindanensis*). In: Proceedings 3rd Annual Scientific Meeting. Crop Protection Society of the Philippines, 143–147.

Wood, B.J. 1969. Population studies on the Malaysian wood rat (*Rattus tiomanicus*) in oil palms, demonstrating an effective new control method and assessing some older ones. Kuala Lumpur, Planter, 45, 510–525.

Wood, B.J. 1984. A long term study of *Rattus tiomanicus* populations in an oil palm plantation in Johore, Malaysia. I. Study methods and population size without control. Journal of Applied Ecology, 21, 445–464.

Wood, B.J. 1994. Rodents in agriculture and forestry. In: Buckle, A.P. and Smith, R.H., ed., Rodent pests and their control. Wallingford, UK, CAB International, 45–83.

Wood, B.J. and Chung, G.F. 1990. Warfarin resistance of *Rattus tiomanicus* in oil palms in Malaysia and the associated increase in *Rattus diardii*. In: Davis, L.R. and Marsh, R.E., ed., Proceedings of the Fourteenth Vertebrate Pest Conference, 6–8 March 1990, Sacramento, USA, 129–134.

Wood, B.J. and Liau, S.S. 1984a. A long term study of *Rattus tiomanicus* populations in an oil palm plantation in Johore, Malaysia. II. Recovery from control and economic aspects. Journal of Applied Ecology, 21, 465–472.

Wood, B.J. and Liau, S.S. 1984b. A long term study of *Rattus tiomanicus* populations in an oil palm plantation in Johore, Malaysia. III. Bionomics and natural regulation. Journal of Applied Ecology, 21, 465–472.

8. Physical Control of Rats in Developing Countries

Grant R. Singleton, Sudarmaji, Jumanta, Tran Quang Tan and Nguyen Quy Hung

Abstract

Digging, trapping, flooding, netting, rat drives and physical barriers are the norm for rodent control in rice fields in most developing countries. We provide a brief overview of physical methods of control aimed at reducing pre-harvest damage by rodents, then consider in detail the use of trap–barrier systems. An important catalyst for adoption of physical control in Southeast Asia is the use of bounties for each rat captured. In Australia, uses of bounties to control vertebrate pests have been singularly unsuccessful. Differing socioeconomics and more intense trapping may provide better results in developing countries. There is a scarcity of good data to assess whether bounties based on physical actions of control are effective. In contrast, experimental field studies support the strategic use of trap–barrier systems (TBS) using early crops ('trap crops') as a lure to rodents. Experimental studies in West Java, Indonesia, and the Mekong and Red River Deltas of Vietnam, indicate that TBS plus trap crops (TBS+TC) are cost-effective in most seasons. Yield increases of up to 1 t/ha have been recorded up to 200 m from a TBS+TC. The need to invest money into traps and fences, which protect neighbouring crops, requires a community-based approach for rodent management. An untested recommendation is that one TBS+TC (25 × 25 m) would be sufficient for every 15 ha of rice crop. Although we require more detailed knowledge of the population ecology and biology of rodent pest species, what we already know has had an important influence on the development of management strategies incorporating physical methods.

Keywords

Rice-field rat, physical control, trap–barrier system, bounty, rodent management

INTRODUCTION

IN DEVELOPING COUNTRIES, physical methods of control are probably the most commonly used approaches by farmers to combat rodent pests. This is simply because they generally cannot afford, or do not have ready access to, chemical rodenticides, fumigants, nest boxes for birds of prey, or other forms of rodent control.

Physical methods have been long recognised as effective for reducing the impact of rodents in post-harvest stores and in intensive animal production units where they damage structures and foul foods (Jenson 1965; Brooks and Rowe 1979; Meehan 1984). Actions include mechanical proofing inside and outside buildings or ships, physical barriers preventing access to an area and various means of trapping. Nevertheless, post-harvest food loss to rodents remains a substantial problem in tropical and sub-tropical regions (Morley and Humphries 1976; Elias and Fall 1988; Prakash and Mathur 1988). Post-harvest losses and impacts on livestock production will not be considered further in this chapter. Instead we refer interested readers to review articles and leaflets on rodent management in large food stores (Meyer 1994), pig production units (Brown and Singleton 1997), and small to medium-sized food stores and food processing units in developing countries (Posamentier and van Elsen 1984; Bell 1998).

This chapter will focus on physical methods aimed at reducing pre-harvest damage by rodent pests. We will provide a brief overview of physical methods of control used in developing countries, then consider in detail the use of trap–barrier systems. The latter will cover historical innovations in the use of the technique, its efficacy across different rice agro-ecosytems, benefit–cost analyses, strengths and weaknesses of the approach, research needs, and its likely role in ecological and sustainable management of rodent pests at a village and district level.

PHYSICAL METHODS—GENERAL

Many inventive techniques have been developed by farmers in developing countries to catch or kill rats or to deflect them from their crops. These include the methods outlined in Box 1.

Other methods are more peculiar to particular regions and countries. These range from placing offerings in the corner of crops to a particular god, to catching large male rats, sewing their anus closed and letting them go again. Farmers believe that 'sewn rats' will become aggressive through an inability to use their bowels and therefore scare neighbouring rats away. This latter technique is inhumane and there is no evidence that it is effective.

The efficacy of the techniques described in Box 1 for controlling rodent populations is rarely assessed. Many are inappropriate given the risk they present to humans. For example, in their desperation to protect their crops from rodents, some farmers redirect mains-power so that it flows through wire suspended centimetres above a flood-irrigated rice crop. The wire is strung around the margins of the crop, killing any rat that comes in contact with it. This method has

Box 1.
Methods promoted by farmers in developing countries to catch or kill rats or to deflect them from their crops

- Various snare and live-traps, usually made of bamboo, that garrotte a rat or break its back (see Mathur 1997; Schiller et al., Chapter 18).
- Bamboo tubes—simply offer cover for rats and either they get stuck or they are caught alive and emptied into a bag.
- Digging of burrows to kill rats in situ; occasionally dogs are used to locate burrows or to help hunt rats flushed from burrows (e.g. Posamentier and van Elsen 1984).
- Rat drives or battues—where rats are driven from cover and herded towards nets (Singleton and Petch 1994).
- Stalking at night with a kerosene light and a net at the end of a long handle—in Co Dung village of Hai Duong province in Vietnam, farmers apply this method from 1900–2200 hrs at specific times of the year and each farmer catches from 5–15 kg of rats per night.
- Electrocution—electrical wire is strung the length of a rice crop about 10–50 mm above a flooded paddy; wet rats that make contact with the wire are quickly killed. As indicated below, this method presents an unacceptably high risk to human health.
- Physical barriers—these usually consist of plastic or metal sheeting and are placed around or along the borders of crops or around areas where grain is stored (e.g. Lam 1988).
- Physical barriers plus traps—live-multiple-capture traps are inserted intermittently at the base of a physical barrier. The traps are placed against small holes in the barrier. Rats enter the traps, attracted to the developing crop or stored food that is on the other side of the barrier (e.g. Lam et al. 1990; Singleton et al. 1998).
- Metal rat guards—sheets of metal are wrapped around the trunk of a tree, higher than 1 m from the ground, to prevent rodents from climbing trees to access fruits. The design of the guards depends on the climbing habits of the rodent species; some are flat against the tree, whilst others are conical or circular metal sleeves, flush with the trunk of the tree but projecting outwards at, or less than, 90° from the trunk (e.g. Posamentier and van Elsen 1984).
- Scaring devices — white cloth or plastic is attached to a bamboo pole approximately 1.2 to 1.5 m high. The white material flapping in the wind supposedly mimics the flight of owls and therefore frightens rats away from the immediate vicinity. These 'scare-owls' are erected in ripening crops where rat damage is evident.

been observed by one of us (G. Singleton) in the Philippines and Vietnam.

In southern Luzon, Philippines, 11 human fatalities were reported in the late 1980s (Quick and Manaligod 1990). In Thai Binh province in the Mekong River Delta, three people were killed in 1997.

BOUNTY SYSTEMS

Bounty schemes in general

In developing countries, management actions are often poorly coordinated. This results in rats quickly reinvading areas where control has been conducted. Sometimes governments introduce a bounty system as an incentive for widespread concurrent control. Inherent weaknesses of bounty systems are that they require rats to be caught and they are generally invoked once densities are already high. This leads to two major problems. The first is that bounties promote inefficient reliance on physical methods of rodent control such as live-trapping, digging and rat drives, replacing management programs based on the use of rodenticides, better farm hygiene, habitat manipulation and/or changes in farm management practices. The second is that bounties encourage a crisis management mentality—acting when rat numbers are high, rather than the more appropriate use of early tactical management (see Redhead and Singleton 1988; Brown et al. 1998). Often the rationale for invoking a bounty system is more to do with political expediency rather than developing an effective, community-based management strategy. Governments have to be seen to be doing something to help farmers in their fight to save their crops from the ravages of high density rodent populations. The collection of tens of thousands of rat bodies has a strong visual effect, providing a sense of satisfaction to farming communities that they have waged a good fight against their perennial enemy.

Bounty schemes have been around for hundreds of years and have been adopted in many countries. In Australia, bounties were first introduced in 1830 for the tails of unregistered dogs in metropolitan Sydney. Since then, bounties have been used for both introduced (e.g. foxes and wild horses) and native species (e.g. dingoes, species of wallaby, Tasmanian tiger) (Breckwoldt 1988). In Australia, as elsewhere, there is no compelling evidence that bounty schemes have been successful in achieving their management aim.

A recent review of bounty schemes by Hassall and Associates (1998) identified the following reasons for their failure.

- Fraud—schemes are abused by people they are supposed to serve.
- Harvesting mentality—bounties are seen as an ongoing source of income rather than a control measure.
- Inefficiency of control—financial incentives promote management systems which provide bodies of animals; as discussed above, there are generally more efficient methods for control.
- Compensatory growth by pest populations—unless more than 50% of a pest population is removed by a bounty, then at best, the pest population will maintain numbers through enhanced survival, higher rates of immigration from uncontrolled areas and better reproductive performance (Caughley 1977; Hone et al. 1980).
- Inadequate benchmarks for success—few programs have appropriate success criteria and so they continue from one campaign to the next with the sole criteria being that they caught many animals last time through imposing a bounty.

This review primarily considered the appropriateness of bounties in Australia. It concluded that bounties were not a cost-effective system for managing vertebrate pests.

Bounty schemes for rodents

Rodents have all the life history characteristics that suggest they would not be the appropriate target for a bounty scheme. They are highly fecund, can produce a litter every three weeks, are extremely mobile and are widely distributed across a landscape. Moreover, most rat drives or bounty systems are conducted once rats have already become a significant problem. Often then it is too late to protect the ripening crop.

The issue of compensatory growth of populations, therefore, is particularly important when considering the potential effectiveness of bounties for controlling populations of rodent pests. In the case of Norway rats, *Rattus norvegicus*, in urban environments in the United States of America, populations which have been reduced to 10–25% of their pre-treatment level, double their population size within 2–4 months and are back to >75% of pre-treatment level by 6–8 months (Emlem et al. 1948). Similarly, trapping high numbers of muskrats (*Ondatra zibethicus*) in Germany, had little impact on the dynamics of their abundance. Indeed, it was estimated that annual loss due to trapping was less than the number of naturally surplus individuals in a population (Halle and Pelz 1990).

Perhaps the implementation of bounty schemes in developing countries may hold greater promise. In these countries, the density of people per hectare is up to two orders of magnitude higher and individual holdings are measured in fractions of a hectare rather than thousands of hectares.

In Lao People's Democratic Republic (PDR), the rat bounty is around 70 kip per rat tail (4,000 kip to US$1). In Indonesia, in West Java, the rat bounty is 50 rupiah for the head of a rat (9,000 rupiah to US$1). In Vietnam, in the Red River Delta, the going price during a bounty season (bounties are not available in all years) is 200 dong for a rat tail (14,000 dong to US$1). Bounty schemes have been also implemented in Cambodia and the Philippines.

In 1991 in Luang Prabang province in northern Lao PDR, a sparsely populated region by Asian standards, over 600,000 rat tails were collected in just 2–3 months (see Singleton and Petch 1994 for details). The bounty scheme stopped because the money ran out. These figures are impressive and it may have been a successful campaign. The officials that one of us (G. Singleton) spoke to were certainly impressed by the number of rats they caught and had little doubt about its success. However, there was no quantitative assessment of whether there was a substantial impact on pre-harvest losses caused by rats. In that year it was still common for growers to report losses of greater than 50% to their crops (Walter Roder, pers. comm.).

In August 1998, a rat bounty of 50 rupiah per rat was instigated in four adjoining villages in West Java, Indonesia. Over 164,000 rats were collected from 1,790 ha in less than a month. In one village of 230 ha, an average of 222 rats were caught per ha. The bounty was instigated during the land preparation for a third rice crop for 1998. A third crop is unusual for West Java and the mass action against rats was activated to guard against rat damage to the newly sown crop. The action seemed to be successful, although there was no control site for comparison and no quantification of crop damage. Nevertheless farmers were satisfied with the outcome.

More impressive still were the numbers of rats caught under a bounty system in Vietnam. In 1997, 22 provinces applied a rat bounty scheme for specific times of the year and 55 million rats were killed. The combined cost for the provincial governments involved was approximately 62 billion dong (see Table 1).

In 1988, in the first two months of the year, 8.5 million rats were killed throughout Vietnam under the bounty system. In the one province of Vinh Phuc, over 5 million rat tails were returned from January–September 1998—the bounty season closed in October. This is in a province where the human population is around 1.1 million.

Regardless of the theoretical evidence that suggests bounties may be an inefficient means of controlling rat populations, digging, trapping, flooding, fumigation, and rat drives are the norm for rodent control in rice fields in most developing countries (see Jahn et al., Chapter 17 and Schiller et al., Chapter 18). Unfortunately, there is a scarcity of good data to assess whether these physical actions of control are effective or not. In regions such as West Java, the intensity of physical activities directed at controlling rats is high. There, some people get paid a levy on the number of rats they catch, however most are locked into conducting nightly control campaigns during the generative stage of the rice crop because they can ill afford to lose much of their potential harvest to rats. These intensive physical activities and bounty schemes elsewhere need to be assessed against specific criteria of success. Apart from a simple benefit–cost analysis, it is important also to take into account whether the time, effort and resources could have been more effectively marshalled for an alternative strategy of rodent control. Such a strategy that may even centre on a coordinated, restricted, bounty season that shifts focus to earlier tactical intervention.

PHYSICAL CONTROL AS AN ADJUNCT TO RODENTICIDE BAITS

Knowledge from both the population ecology and feeding behaviour of rats indicates that the best time to use rodenticide baits in and around rice crops is at maximum tillering. This coincides with the onset of breeding and with the final weeks of a 2–6 month fallow period when food quality and quantity have been low.

Table 1.
Number of rats returned for bounty payments in three northern and three southern provinces of Vietnam for the first five months of 1997. (Source: Ministry of Agriculture and Rural Development, Vietnam.)

Province	Area rice damaged (ha)	Number of rat tails	Vietnamese dong paid for bounty
Red River Delta (North)			
Hai Duong	4 139	3 363 257	672 651 400
Hanoi	10 000	650 000	130 000 000
Vinh Phuc	6 729	9 008 700	1 801 740 000
Mekong River Delta (South)			
Long An	3 500	4 600 000	100 000 000
Quang Ngai	4 752	180 225	36 015 000
Bac Lieu	2 990	550 000	9 000 000

Hence the rat population would be at a relatively low density and bait acceptance would be high. Once panicle initiation begins, rats show low acceptance of baits (Buckle 1988). In India, local traps then become a useful control measure together with fumigation and weed control (Mathur 1997).

TRAP–BARRIER SYSTEMS

In developing countries, a common method for protecting a crop from invading rodents is to use plastic fences to deflect rats and mice away from the crop. If the rats are successfully kept out they are generally deflected into neighbouring crops. The net effect is that crop losses in a village are rarely reduced. In the 1980s, Lam (1988) developed a variation of the drift fence and pitfall method commonly used for trapping small mammals. The variation consisted of placing a plastic fence along the margin of a rice crop and placing small holes in the fence just above the irrigation water. Adjacent to each hole is a multiple-capture cage trap suspended on bamboo above the water level (on the crop side of the fence). A mud mound provides access to the hole and thence to the trap. The dimensions of the fence and trap are shown in Figure 1.

This fence plus trap method has been variably described as the 'environmentally friendly system', the 'active barrier system', the 'plastic fences and multi-capture trap' and the 'trap–barrier system' (TBS). The trap–barrier system or TBS is now the commonly accepted description used in most Southeast Asian countries and is what we will use in this chapter.

The TBS was first developed to protect crops in areas where rat damage was high (e.g. crops adjacent to abandoned agricultural land, early planted crops). In Malaysia, a TBS that extended for 5 km was used successfully to protect reclaimed cropping lands that were planted out of synchrony. The most rats caught in one night was 6,872, with 44,101 rats caught in nine weeks. Subsequent studies in Malaysia (Lam et al. 1990) and the Philippines (Singleton et al. 1994) focused on the use of small rectangular TBSs (0.25 ha to 4 ha). Again, promising results were obtained when rat densities and crop losses in surrounding areas were high. However, benefit–cost analyses indicated that losses would have to be greater than 30% for the TBS method to be cost-effective on a regular basis (Singleton et al. 1994; Lam Yuet Ming, pers. comm.).

More promising results were obtained when the TBS was used to protect a crop that was locally attractive to rats, e.g. late-harvested rice crops or vegetable crops maturing after the rice crops had been harvested (see Lam and Mooi 1994). This led to the development of a second generation TBS, consisting of an early or late planted 'trap crop' within the TBS which lures rodents to the traps. The expectation was that rats from the surrounding areas would be drawn to the trap crop and then enter the traps. The TBS plus trap crop (TBS+TC) would then provide a halo of protection to the neighbouring rice crops.

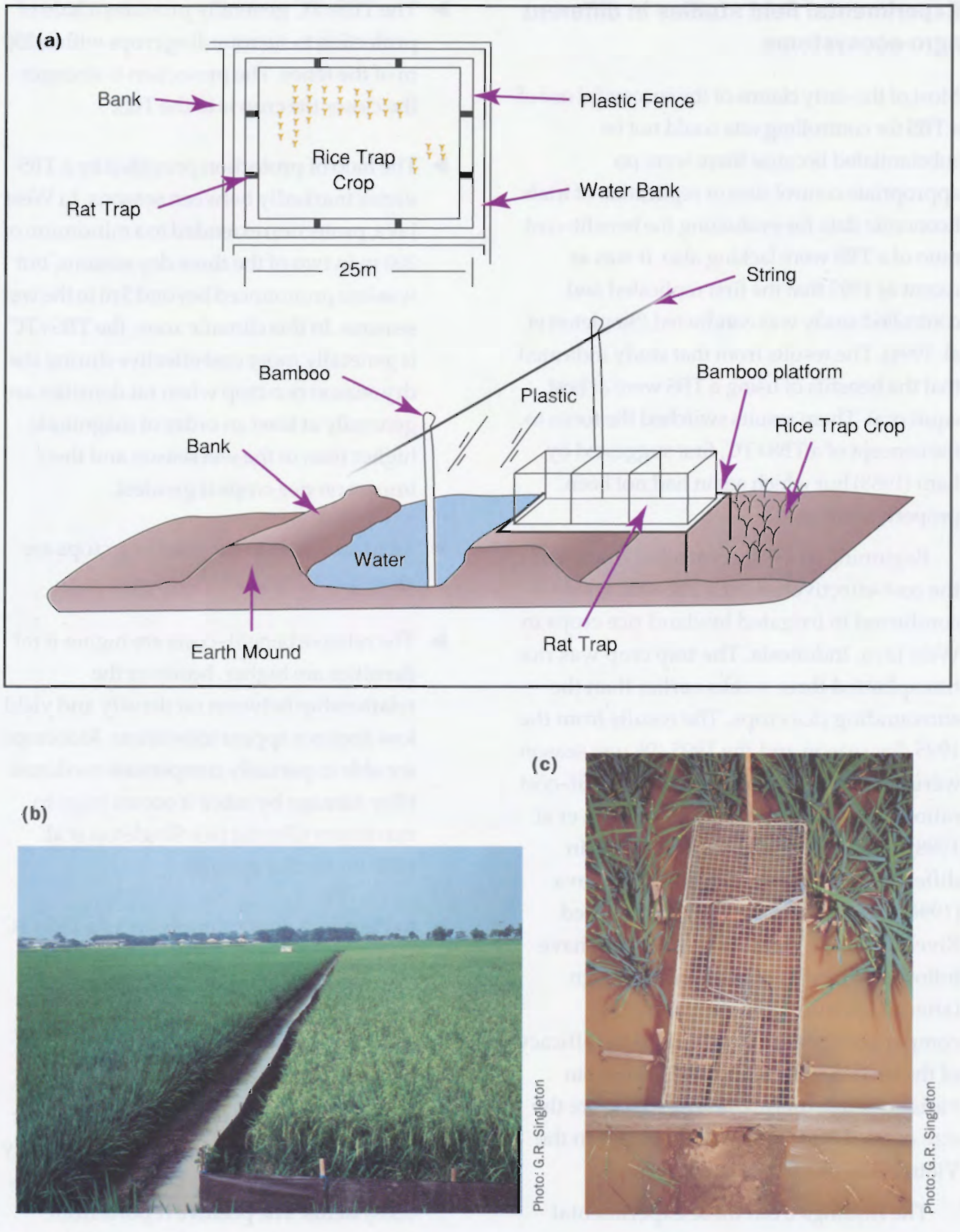


Photo: G.R. Singleton

Photo: G.R. Singleton

Figure 1.
(a) Schematic diagram showing the design of the trap–barrier system plus trap crop of rice (TBS+TC) of rice. (b) TBS and TC in Sukamandi; West Java. (c) TBS in position.

Experimental field studies in different agro-ecosystems

Most of the early claims of the successful use of a TBS for controlling rats could not be substantiated because there were no appropriate control sites or replication of trials. Economic data for evaluating the benefit–cost ratio of a TBS were lacking also. It was as recent as 1993 that the first replicated and controlled study was conducted (Singleton et al. 1994). The results from that study indicated that the benefits of using a TBS were at best equivocal. These results switched the focus to the concept of a TBS+TC, first suggested by Lam (1988) but which again had not been properly evaluated.

Beginning in 1995, controlled studies of the cost-effectiveness of a TBS+TC were conducted in irrigated lowland rice crops in West Java, Indonesia. The trap crop was rice transplanted three weeks earlier than the surrounding rice crops. The results from the 1995 dry season and the 1995/96 wet season were extremely promising with benefit–cost ratios in the vicinity of 20:1 (Singleton et al. 1998). Subsequent studies conducted in different geo-climatic zones in West Java (1996–1997) and in the Mekong and Red River Deltas in Vietnam (1997–1998), have followed a similar experimental design (after Singleton et al. 1998), allowing comparisons of the robustness of the efficacy of the second generation TBS. The main variations in experimental design were the size of the TBS and lack of replicates in the Vietnamese studies (Tables 2 and 3).

The findings from these experimental studies are summarised in Tables 2–6. The main inferences that can be drawn from these studies are as follows.

- The TBS+TC generally provides a halo of protection to surrounding crops within 200 m of the fence. The protection is stronger the closer the crop is to the TBS.
- The halo of protection provided by a TBS varies markedly between seasons. In West Java, protection extended to a minumum of 200 m in two of the three dry seasons, but was less pronounced beyond 5 m in the wet seasons. In this climatic zone, the TBS+TC is generally more cost effective during the dry season rice crop when rat densities are generally at least an order of magnitude higher than in the wet season and their impact on rice crops is greatest.
- Yield increases to surrounding crops are generally 0.3 to 1.0 t/ha.
- The relative benefit–costs are higher if rat densities are higher, however the relationship between rat density and yield loss does not appear to be linear. Rice crops are able to partially compensate moderate tiller damage by rats if it occurs prior to maximum tillering (see Singleton et al. 1998 for further details).
- In West Java, the optimum size of a TBS+TC is in the range of 20 × 20 m to 50 × 50 m. When a 10 × 20 m early trap crop was employed, there was a net loss to farmers.
- The comparative performance of the TBS+TC across the different agro-climatic regions indicates that the technique is likely to be effective in a wide range of rice agro-ecosystems. The positive reports from Malaysia (e.g. Lam and Mooi 1994), where it was first trialled, adds credence to this observation.

In Vietnam particularly, and Indonesia in 1995–96, the yield increases at the treatment sites appeared high given the relatively low number of rats caught. Given that rats weigh around 165–200 g and consume about 20–25% of their body weight per day, then an individual rat would take about 30 days to consume 1.5 kg of rice. Yet each rat represented a reduction in damage of around 3 kg per ha or 45 kg if the halo of protection to the surrounding crop extended to 15 ha. The number of rats caught during the TBS studies in Indonesia in the dry season in 1997, and in Vietnam in the summer season in 1997, provide more convincing cases for the realised increases in yield (Table 2).

Table 2.
Overview of when rats were caught in 'trap–barrier system (TBS) plus trap crop' in Indonesia during 1995–1997. Note the different sizes of TBS. See Singleton et al. 1998 for methods.

Size TBS(m)	Season	Replicate	Timing of rat captures			Total rats caught
			Tillering–Booting	Flowering–Heading	Harvest	
Site: West Java, Sukamandi						
2 500 m^2 (50 × 50)	Dry season 1995	1	63	82	40	185
		2	28	45	17	90
			Proportion of total rats			
			33.1%	46.2%	20.7%	
	Wet Season 1995/96	1	96	11	10	117
		2	42	4	9	55
			Proportion of total rats			
			80.2%	8.7%	11.1%	
200 m^2 (10 × 20)	Dry Season 1996	1	96	11	29	136
		2	16	27	26	69
			Proportion of total rats			
			54.6%	18.5%	26.8%	
	Wet Season 1996/97	1	15	6	7	28
		2	50	4	8	62
			Proportion of total rats			
			72.2%	11.1%	16.7%	
2 500 m^2 (50 × 50)	Dry Season 1997	1	75	514	117	706
		2	43	441	364	848
900 m^2 (30 × 30)		1	65	202	66	333
		2	11	86	54	151
400 m^2 (20 × 20)		1	46	248	108	402
		2	24	85	56	165
			Proportion of total rats			
			10.1%	60.5%	29.4%	

Singleton et al. (1998) proposed three factors that together may explain the apparent disparity between the number of rats caught and the resulting increase in yield on the treatment sites. Firstly, each rat is likely to have damaged many tillers during the generative stage, compounding the loss in yield. The earlier these rats are removed the greater the resulting increase in yield. Secondly, the removal of rats leads to substantially fewer females breeding in the vicinity of each TBS—an important consideration given that breeding commences during the maximum tillering stage, the average litter size is around 10 and the first litter is weaned prior to harvest. Thirdly, rats in live-capture traps provide an early visual cue to farmers to begin other rodent control activities, leading to more effective rodent control activities on the TBS plots relative to the control plots. Typically in West Java, farmers wait until there is obvious rat damage to the maturing crop before embarking on intensive rodent control activities.

Economics of a second generation TBS

Cost of a trap–barrier system for trapping rats in rice crops

The cost of the materials for a 25 × 25 m TBS with 10 cage traps (allowing for two replacement traps during a cropping season), and the labour costs required to construct a TBS, varies markedly between countries. In April 1998, the relative costs for materials were: Indonesia—US$44.75 but should last for four seasons, therefore the cost is US$11.40 (114,250 rupiah) per season; Malaysia—US$800, should last four seasons, therefore the cost is US$200 per season; Vietnam—US$80 (1,016 million dong), the traps last for minimum of two seasons but not the fence, so the average cost over two seasons is US$50. In Vietnam, this cost can be discounted because the used plastic is adapted for other purposes and the live rats are often sold to the local market for meat.

The traps are the most expensive items of a TBS. In Indonesia, they constitute about 60% of the cost. Traps also are easily removed. It is not uncommon for traps to disappear overnight, especially when the system is trialled for the first time in a district. Generally, however, peer group pressure at the village level quickly puts a stop to traps being stolen or 'borrowed'.

Staff at the Research Institute for Rice in Indonesia have been experimenting with ways of reducing the cost of traps. The most promising development is the recycling of 18–20 litre tins which previously held cooking oil or biscuits. They are about a quarter of the price of a standard cage trap, yet they catch about 90 rats for every 100 caught in a standard trap (Table 7). These recycled traps provide the added benefit of the possible development of a village-based industry for their manufacture.

Adoption rate of TBS+TC technology

The benefit–cost ratio of a TBS+TC varies from a gain of 20 times the initial investment in a TBS to a net cost when rat densities are low (Table 6). High benefit–cost ratios are only meaningful at the village level, because they only occur if there is a halo of protection extending 150 to 200 m from the TBS.

Table 3.

Overview of when rats were caught in 'trap–barrier system (TBS) plus trap crop' in Vietnam during 1997. Note the different sizes of TBS. Methods were based on Singleton et al. 1998.

Size TBS(m)	Season	Replicate	Timing of rat captures			Total rats caught
			Tillering–Booting	Flowering–Heading	Harvest	
Site: Red River Delta						
360 m^2	Spring 1997	Ha Bac	17	34	13	64
			Proportion of total rats			
			26.6%	53.1%	20.3%	
(12 × 30)	Summer 1997	Ha Bac	40	76	18	134
			Proportion of total rats			
			29.9%	56.7%	13.4%	
(12 × 30)	Summer 1998	Vinh Phuc	119	54	16	189
			Proportion of total rats			
			63.0%	28.5%	8.5%	
Site: Mekong Delta — Tra Vinh						
1 000 m^2 (30 × 30 m)	Summer –Autumn 1997	1 (Chien)	184	79	40	303
		2 (Cheng)	228	88	67	383
		3	148	154	21	323
		4	182	87	42	311
		5	151	127	34	312
			Proportion of total rats			
			54.7%	32.8%	12.5%	
	Autumn–Spring 1997	1 (Cheng)	72	67	46	185
		2	106	89	50	245
		3	118	81	62	261
		4	105	112	72	289
			Proportion of total rats			
			40.9%	35.6%	23.5%	
Site: Mekong Delta — Ho Chi Minh						
1 000 m^2	Winter–Spring 1997	1	482	355	196	1 033
		2	529	194	4	727
		3	551	266	5	822
			Proportion of total rats			
			60.5%	31.6%	7.9%	

Table 4.

Effect of the trap–barrier system (TBS) plus trap crop on rice yields (kg/ha) at various distances from the TBS, in Indonesia. These estimates were based on the weight (water content approximately 14%) of unhulled rice harvested from 10 m^2 quadrats (Repl = replicate; nth = sample from north of TBS; sth = sample from south of TBS; se = standard error of mean yield estimates for the control plots).

Site: West Java			Rice yield (kg/ha)					Control	
			5 m	50 m	100 m	150 m	200 m	Mean	se
Dry Season 1995									
	Replicate 1			5 600	4 750	3 500	4 750	2 313	98.7
	Replicate 2			5 600	3 900	3 650	4 100	4 638	74.7
		Mean		5 600	4 325	3 575	4 425	3 475	
	Yield relative to control (%)			+61%	+24%	+3%	+27%		
Wet Season 1995/96									
	Repl 1 nth		6 230	5 930	5 760	5 860	5 660	5 736	37.6
	Repl 1 sth		5 990	6 070	5 920	5 690	5 560	5 498	44.5
	Repl 2 nth		6 630	5 620	5 560	5 490	5 780	4 736	48.9
	Repl 2 sth		6 250	5 590	5 670	5 430	5 670	5 210	48.5
		Mean	6 275	5 803	5 728	5 618	5 668	5 295	88.0
	Yield relative to control (%)		+19%	+10%	+8%	+6%	+7%		
Dry Season 1996									
	Repl 1 nth		4 608	4 536	4 549	4 501	4 604	4 768	32.8
	Repl 1 sth		4 495	4 593	4 576	4 575	4 539	4 705	25.4
	Repl 2 nth		4 525	4 501	4 593	4 437	4 510	4 646	28.7
	Repl 2 sth		4 600	4 694	4 558	4 605	4 549	4 667	48.0
		Mean	4 557	4 581	4 569	4 529	4 550	4 697	19.2
	Yield relative to control (%)		–3%	–2%	–3%	–3%	–3%		
Wet Season 1996/97									
	Repl 1 nth		7 312	7 166	7 317	7 165	7 316	7 087	12.9
	Repl 1 sth		7 301	7 201	7 112	7 135	7 165	7 148	52.5
	Repl 2 nth		6 627	6 615	6 634	6 580	6 761	7 317	35.9
	Repl 2 sth		6 580	6 782	6 622	6 611	6 639	7 273	38.4
		Mean	6 955	6 941	6 921	6 873	6 970	7 206	27.4
	Yield relative to control (%)		–3%	–3%	–4%	–5%	–3%		
Dry Season 1997 (50 × 50 m only)									
	Repl 1 nth		5200	5400	5800	5400	5300	4100	114.0
	Repl 1 sth		5000	5000	4900	4900	5000	4000	70.7
	Repl 2 nth		4800	4700	4500	4600	4400	3920	58.3
	Repl 2 sth		4300	4200	4200	4300	4350	3980	86.0
		Mean	4825	4825	4850	4800	4762	4000	41.7
	Yield relative to control (%)		+21%	+21%	+21%	+20%	+19%		

Table 5.

Effect of the trap–barrier system (TBS) plus trap crop on rice yields (kg/ha) at various distances from the TBS, in Vietnam. These estimates were based on the weight (water content approximately 14%) of unhulled rice harvested from 10 m^2 quadrats (se = standard error of mean yield estimates for the control plots).

		Mean Rice yield (kg/ha)[a]					Control	
		5 m	50 m	100 m	150 m	200 m	Mean	se
Site: Red River Delta								
Spring 1997	Yield relative to control (%)		5269 +8%	5236 +7%		5028 +3%	4886	
Summer 1997	Yield relative to control (%)		3941 +9%	3888 +8%		3736 +4%	3605	
Site: Mekong Delta								
Summer–Autumn 1997	Site 1 (Chien) Yield relative to control (%)	3100 +10%	3200 +14%	3000 +6%	3200 +14%	2700 –4%	2817	
	Site 2 (Cheng) Yield relative to control (%)	3200 +14%	3200 +14%	3150 +12%	3000 +6%	3600 +28%	2817	
Winter–Spring 1997/98	(Cheng) Yield relative to control (%)	4960 +17%	4640 +9%	4410 +4%	4660 +9%	4520 +6%	4256	43.9

[a] The mean rice yields for each distance from the TBS were from two measurements, except in winter–spring 1997/98 when there were six measurements.

Table 6.

The effect of a trap–barrier system (TBS) plus trap crop on mean yield increases up to 200 m from the TBS and the associated benefit–cost ratios, in the Red River and Mekong River Deltas, Vietnam, and West Java, Indonesia. Costs were calculated from material costs of the TBS and labour costs associated with building the fence and the daily clearing of rats from traps. Benefits were based simply on the increase in yield relative to an untreated site. The dimensions of the respective TBS, the rat density during the growing season and the timing of rat damage to tillers, provides context for the variation in benefit–cost ratios.

Year and season	Dimensions of TBS	Rat density	Timing of main tiller damage	Mean yield increase (t/ha)	Benefit–cost ratio
Vietnam					
Red River Delta					
Spring 1997	12 × 30 m	Low	Flowering to harvest	0.3	
Summer 1997	12 × 30 m	Low	Flowering to harvest	0.3	
Mekong River Delta					
Summer 1997	33 × 33 m				
Site 1		Medium	No data	0.2	
Site 2		Medium	No data	0.4	
Winter 1997	33 × 33 m	Low/Med	Throughout	0.4	2.5:1

Table 6. (Cont'd)
The effect of a trap–barrier system (TBS) plus trap crop on mean yield increases up to 200 m from the TBS and the associated benefit–cost ratios, in the Red River and Mekong River Deltas, Vietnam, and West Java, Indonesia. Costs were calculated from material costs of the TBS and labour costs associated with building the fence and the daily clearing of rats from traps. Benefits were based simply on the increase in yield relative to an untreated site. The dimensions of the respective TBS, the rat density during the growing season and the timing of rat damage to tillers, provides context for the variation in benefit–cost ratios.

Year and season	Dimensions of TBS	Rat density	Timing of main tiller damage	Mean yield increase (t/ha)	Benefit–cost ratio
INDONESIA					
West Java					
1995 Dry	50 × 50 m	Very high	After booting	1.0	20:1
1995/96 Wet	50 × 50 m	Low	Maximum tillering	0.5	7:1
1996 Dry	20 × 10 m	Medium	Transplanting and tillering	–0.1	Net cost
1996/97 Wet	20 × 10 m	Low	Low damage	–0.2	Net cost
1997 Dry	50 × 50 m	Med/high	Maximum tillering to harvest (all crops)	0.8	14:1
	30 × 30 m			0.5	10:1
	20 × 20 m			0.9	24:1

Table 7.
Comparison of the efficacy of different trap designs in a trap–barrier system (TBS). See Singleton et al. (1998) for description of the 'standard trap'. Trap designs II to IV are modifications of a recycled 18 litre tin of vegetable oil (350 × 230 × 230 mm). The comparison was conducted in rice crops at Sukamandi, West Java, during the 1998 dry season. The rice crops were two weeks old and the traps were set for three weeks (May 18—June 3). There were three sample plots spaced 500 m apart. Each TBS was 50 × 100 m with eight traps per plot. One of each trap type was placed in random order along the two 100 m sides of the TBS (SE = standard error).

Trap type	Replicate	Rats captured	Total	Mean	SE	Cost (Rupiah)
I (standard trap)	1	51	317	105.7	40.18	30 000
	2	184				
	3	82				
II (wire mesh back)	1	22	193	64.3	25.46	6 000
	2	110				
	3	61				
III (wire mesh front and back)	1	50	277	92.3	32.41	8 000
	2	156				
	3	71				
IV (entrance only wire mesh)	1	24	69	23.0	7.81	4 000
	2	36				
	3	9				

In developing countries in Asia, this is well beyond the area of crop owned by an individual family. However, the results have been sufficiently promising to have the governments of both Indonesia and Vietnam express strong support for the implementation and adoption of this simple technology. For example, in the Mekong River Delta the concept of a TBS+TC was only first tested in early 1997, yet by May 1998 there were more than 100 TBSs established in five provinces. In Indonesia, the field trials on the TBS were initially conducted on a research farm (440 ha) and then on a commercial seed farm (1,000 ha with farmers share-farming areas of up to 5 ha). Following our trials, large TBS+TC (50 × 50 m or 100 × 100 m) were established and both institutions have been pleased with the returns for their outlay. At the research farm there was just one TBS+TC in 1996/97 and it caught over 26,500 rats. The next year there were three TBS+TCs and over 48,000 rats were caught. In 1998, all the plant variety trials on the research farm were conducted within a TBS, and there were more than five other large TBS+TCs.

In Malaysia, the country of its origin, the TBS is generally only used in areas that have acute rat problems (e.g. previously abandoned fields or asynchrony of cropping at borders of districts with different irrigation schedules) or high value crops (e.g. research farms).

When to use a TBS+TC?

Effective and efficient pest control strategies generally have a monitoring protocol that determines whether particular control actions need to be implemented. These protocols are based on preventing a pest population from reaching a density above which they cause unacceptable economic hardship to growers. This is referred to as the economic injury level (EIL). To prevent a species reaching its EIL, a lower population threshold is identified at which appropriate control actions are implemented.

This threshold level is relatively easy to define for actions that have a rapid impact on the pest population, such as the use of chemical rodenticides (Buckle 1988). This is not the case for the use of a TBS+TC. In this situation, the decision point is at land preparation, to enable the trap crop to be planted three weeks in advance of the main crop. By comparison, the decision of whether to use chemical rodenticides is made just before maximum tillering of the rice crop (around day 40–45 post transplanting).

An informed decision of whether or not to use a TBS+TC requires a population model that enables reasonable accurate forecasts of rodent population densities for the forthcoming cropping season. These models have been developed for some regions for mouse plague management in Australia (see Pech et al., Chapter 4), however such models in Southeast Asia are lacking, underlining the need for sound ecological studies of the principal rodent pest species in rice farming systems. Effective decision analysis on the use of TBS+TC therefore relies on the development of an ecologically-based management system for rodent pests.

Weaknesses of the TBS+TC

In weighing up the potential of the TBS+TC, an economic benefit–cost analysis is one of a number of considerations. Others include those listed in Box 2.

Whether these points are minor or major will depend on the socioeconomic context of the end-users and on the effectiveness and thoroughness of the extension campaign. Moreover, governments have shown through the implementation of bounty systems that they are prepared to invest in management of rodent pests. This raises the possibility of government subsidies for the TBS+TC at village or regional levels. Subsidising the cost of the materials for a TBS+TC would be much cheaper than funding a bounty system and grain production is likely also to be higher under a TBS+TC pest management system.

The exciting potential of the TBS+TC acting as a platform for an integrated strategy for managing rodent pests, and therefore lessening the reliance on chemical rodenticides, provides governments with another option for investing funds into rodent management.

Moving to village-level management

The impressive cost–benefit ratio for the TBC+TC needs to be viewed in the context that these were experimental studies. The challenge is to transfer this technology readily and effectively to rice farmers. An important consideration is the average size of family holdings in Southeast Asia, which is 0.5 to 1.5 ha. A TBS which encloses 0.25 ha could provide protection to neighbouring farmers without them outlaying money for materials, providing the labour required to maintain the TBS or taking the concomitant risks associated with planting an early trap crop. Therefore the TBS + TC will be most effective if it is part of a community-based approach to rodent pest management.

Box 2.
Economic benefit–cost analysis

- High initial cost—many farming families in Southeast Asia do not have the disposable income to invest in pest management methods.
- High labour involvement—the traps need to be checked every day, although stoppers (e.g. clump of straw) can be placed in the opening of the traps on days when no labour is available.
- Strong vigilance on maintenance—the fence needs to be checked daily for evidence of rats going through or under the fence; weed growth needs to be controlled near the fence.
- Early trap crop attracts avian and insect pests—this needs to be factored into a benefit–cost analysis.
- Mechanics of growing an early crop—the main difficulty is the availability of sufficient water three weeks in advance of the general irrigation schedule to maintain firstly a rice nursery and then the transplanted trap crop. An earlier maturing variety of rice may help overcome this problem.
- Non-target captures—amphibians and reptiles are caught in the traps. The experimental protocol requires these species be released. Whether farmers would release all of these species is problematical.
- Humaneness—protocols have been developed (see Singleton et al. 1998) which include the use of carbon monoxide from the exhaust of motor cycles or automobiles for killing rats. The adoption of recommended methods will depend on the operator but he/she should be encouraged to kill the rats humanely.
- Environmental contamination—proper disposal and recycling of the plastic fences are required.