Review of international literature relevant to stoat control

SCIENCE FOR CONSERVATION 170

Robbie A. McDonald and Serge Larivière

Published by
Department of Conservation
P.O. Box 10-420
Wellington, New Zealand

Science for Conservation presents the results of investigations by DOC staff, and by contracted science providers outside the Department of Conservation. Publications in this series are internally and externally peer reviewed.
This report was prepared for publication by Science Publications, Science & Research Unit; editing and layout by Geoff Gregory. Publication was approved by the Manager, Science & Research Unit, Science Technology and Information Services, Department of Conservation, Wellington.

ISSN 1173–2946
ISBN 0–478–22019-7

Cataloguing-in-Publication data
McDonald, Robbie A.
Review of international literature relevant to stoat control / Robbie A. McDonald and Serge Larivière. Wellington, N.Z. : Dept. of Conservation, 2001.
78 p. ; 30 cm. (Science for conservation, 1173-2946 ; 170.)
Includes bibliographical references.
ISBN 0478220197
1. Stoats—Control. 2.Mustela erminea—Control.
I. Larivière, Serge II. Title. Series: Science for conservation (Wellington, N.Z.) ; 170.

CONTENTS

Review of international literature relevant to stoat control

Robbie A. McDonald[1] and Serge Larivière[2]

[1]School of Biological Sciences, University of Bristol, Bristol BS8 1UG, UK; present address: Department of Biological Sciences, University of Waikato, Private Bag 3105, Hamilton, New Zealand.

[2]Department of Biology, University of Saskatchewan, 112 Science Place, Saskatoon SK S7N 5E2, Canada; present address: Delta Waterfowl Foundation, R.R. #1, Box 1, Portage La Prairie, Manitoba R1N 3A1, Canada.

ABSTRACT

Stoats present a serious threat to the avian biodiversity of New Zealand. Control measures are needed to ensure the long-term survival of several bird species. Existing control technology is labour-intensive and expensive, and so new techniques and approaches are needed to provide long-lasting solutions to conservation challenges. We reviewed the literature on the demography, captive breeding, diseases, biological and non-lethal control, metabolism and toxicology of stoats and closely related mustelids. We conclude that a long-term solution to lethal stoat control may lie with specific disease agents, such as canine distemper, Aleutian disease virus and mink enteritis virus, and toxins, such as rodenticides and mycotoxins. The role of host-specific vectors in disease transmission may represent an existing method of accurately deploying non-lethal control agents. We urge caution in developing biotechnology without a parallel investigation of the effects of control on stoat populations and the survival of threatened birds. The establishment of captive breeding facilities for stoats deserves a high priority since these will be indispensable for technological development and validation. Throughout their native range, stoats are dependent on a range of prey species. In certain areas of New Zealand, they are dependent on very few prey species, and outbreaks of house mice lead to acute conservation problems at regular and predictable intervals. We therefore advocate a research effort directed towards the control of mouse outbreaks.

Keywords: bird conservation, biocontrol, *Mustela erminea*, pest management.

McDonald, R.A.; Larivière, S. 2001. Review of international literature relevant to stoat control. *Science for Conservation 170*. 78 p.

1. Introduction

The endemic fauna of New Zealand evolved in the absence of mammalian predators and has proved particularly vulnerable to some of the mammals introduced since human settlement (King 1984). Stoats *Mustela erminea*, ferrets *M. furo*, and weasels *M. nivalis* were introduced to New Zealand in the 1880s in an attempt to control rabbits *Oryctolagus cuniculus*. Stoats in particular were almost immediately implicated in the declining abundance of native birds. They are still contributing to the decline of native fauna, and now present a particularly serious threat to the future existence of several endemic bird species (McLennan et al. 1996; O'Donnell et al. 1996; Wilson et al. 1998). McDonald & Murphy (2000) provided a recent review of the problems caused by stoats and of the steps taken so far to manage them in New Zealand.

Reducing predation by stoats is clearly essential for the survival of several endemic species on the mainland of New Zealand. The main effort so far has been put into developing and employing methods of lethal stoat control, hence current programmes rely to a great extent on 'traditional' kill trapping using steel spring traps, which is very labour-intensive. More recently the deployment of bait stations with poisoned hen eggs has become widespread. These methods have proven useful in temporarily reducing stoat abundance and enhancing the nesting success of certain birds. However, more cost effective and sustainable approaches to controlling stoats are urgently needed. In the May 1999 budget, the New Zealand Government announced that an extra $6.6 million over five years would be given to the Department of Conservation to initiate the development of an integrated stoat control research programme.

The Stoat Research Programme has identified as its vision:

That stoats will no longer be a threat to indigenous biodiversity.

In the first round of funding aimed at realising this vision, several review and screening projects have been commissioned, of which this is one. Parallel projects have been commissioned on research in the former Soviet Union, modelling stoat control and population dispersion, immunocontraception, disease screening, practical effects of stoat control, habitat use and toxin development. There is, therefore, considerable overlap between this and other projects. However, the function of this project is particularly to draw attention to studies conducted outside of New Zealand, and on species that are closely related to stoats, that may present particularly novel approaches to controlling stoat predation.

2. Objectives and outcomes sought

The objective of our project was to provide New Zealand scientists with an accessible and up-to-date review of the international literature on stoat biology and ecology with particular reference to areas that are pertinent to stoat control. This information is therefore of direct relevance to realising the vision of the Stoat Research Programme. We have highlighted existing knowledge about stoat control and have actively identified areas that, based on this knowledge, we believe will be most productive for future research.

This report forms the primary outcome of our work. We have examined five main areas:

1. Demography—a review of demographic studies of mustelids, with particular reference to the effects of control and trapping on population size and growth rates.
2. Captive breeding—a review of captive breeding in mustelids, especially those experiencing delayed implantation. We have provided data on reproductive pathology and diseases where known and detailed any information that will assist with captive breeding programmes for use in future control trials on captive animals in New Zealand.
3. Diseases—a review of all available data on diseases of stoats and other mustelids. This includes data on all known pathogens, including viruses, bacteria and parasites. We have also provided an outline of their relevance to the control and limitation of mustelid populations.
4. Biological and non-lethal control—we have reviewed options for lethal and non-lethal biocontrol of stoats and recommended areas for further investigation. This section has been expanded to include non-biological methods of control and an outline consideration of modelling approaches to understanding the feasibility and efficacy of biological control programmes.
5. Metabolism and toxicology—where possible we have identified novel approaches for toxin development based on specific aspects of mustelid metabolism. We have also expanded this section to include a wider-ranging review of the application of toxins to control stoat populations.

3. Methods

This is a literature review that takes in an extremely broad area of work. We have drawn on a wide range of sources in the published and unpublished literature. We were fortunate in being able to draw on a number of general reviews of stoat biology. The accounts provided by King & Moody (1982), King (1983a, 1989), Fagerstone (1987) and McDonald & Murphy (2000) have proven very useful. We benefited in particular from the work already undertaken and literature already reviewed by one of us for inclusion in the most recent general review of stoat biology provided by McDonald & King (in press) which will be published shortly in the new edition of *The Handbook of British Mammals*.

We conducted searches for new material pertinent to the topics under consideration. Since certain aspects of the biology of stoats have not yet been described in detail, especially in areas such as pathology and disease, metabolism and reproductive biology, we expanded our searches to take into account work on closely related species. Domestic ferrets, black-footed ferrets *Mustela nigripes,* and American mink *M. vison* have proven particularly fertile areas of research because of their significance to medical research, for conservation biology, and in the fur industry, respectively. Work on the biology of other *Mustela* spp. is comparatively scarce and so we have also drawn on literature about *Martes*, *Lontra* and other mustelids where appropriate. One of the principal aims of this review is to bring work undertaken internationally to the attention of New Zealand researchers. Therefore, we have deliberately not included all the details of the extensive work undertaken on stoats in New Zealand, except where this work is unparalleled elsewhere and is significant in a global context.

We searched the ISI Science Citation Index provided online by Bath Information Data Services. We searched for all papers published from 1981 to 2000 containing the following search terms in the title, keywords, or abstract: stoat*, ermine, *erminea*, ferret, *Gulo*, *Martes*, mink, *Mustela*, mustelid, Mustelidae, polecat, weasel* and wolverine. From an initial hit list of 6139 published works, we narrowed this to a list of 591 studies of particular relevance. We also searched *Zoological Record* from 1974 to 1999. We focused on the systematic lists and drew on work on *Mustela* spp. mainly *Mustela erminea*, *M. nivalis* and *M. frenata*. We also searched abstracts of biological journals on *BIOSIS* from 1990 to 2000.

Web searches were also conducted to locate sources of information and relevant electronic addresses. We also posted a call for information on *MAMMAL-L*, a list-server devoted exclusively to the study of mammals. E-mail responses to the posting were followed up, and individuals were contacted either by phone or by e-mail. Specific queries were directed to individuals and organisations known to have unpublished information on topics of interest.

4. Demography

4.1 INTRODUCTION AND AIMS

We have interpreted demography in the broadest sense. We have reviewed all aspects of stoat life history that are pertinent to developing methods for their control. This includes 'traditional' aspects of demography such as fecundity, fertility, productivity, survival, mortality and density dependence and the application of these data in life tables and population models. We have also considered other aspects of population ecology such as movement patterns and dispersal and have included practical methods for describing stoat populations, such as ageing and census techniques. Our aim was to provide sources of demographic data for researchers attempting to evaluate the efficacy and cost-effectiveness of various methods of stoat control and to highlight priority areas for future work.

4.2 CENSUS TECHNIQUES

Information on stoat density is required to assess the risk to native species as well as the efficacy of removal or control programmes (McDonald & Murphy 2000). However, census techniques are highly problematic because stoats live at low densities, they are very wide-ranging relative to their small body size, and their population size and social structure are highly flexible (Erlinge & Sandell 1986; King 1989). King & Edgar (1977) provided a guide for the use of live trapping and footprint tracking census methods, and this was expanded subsequently with workshop guides (King 1994; King et al. 1994). Footprint tracking in particular is becoming more widespread and, while this has been tested recently in Britain without much success (Morris & Birks 1997), tracking tunnels are becoming the standard method for monitoring mustelids in New Zealand (Murphy et al. 1999). In colder climates, tracks in snow can be counted and used as an index of activity (Nyholm 1959; Becker 1991). This method, of course, depends on long-term snow cover, a rare situation in most temperate climates (Becker 1991), and is probably of limited utility in New Zealand.

Kill-trapping can be used as a relative index of stoat densities and is the only method currently employed for stoat monitoring in Britain (Tapper 1982, 1992). For accuracy, this method depends on the recording of trapping effort (McDonald & Harris 1999), without which long-term trends in population indices are probably biased. An index of captures per 100 trap nights is an appropriate way of measuring the relative success of trapping efforts and hence presumably bears a closer relationship to actual densities. McDonald & Harris (1999) recommended that numbers of males and females are recorded separately, on the grounds that this may serve to identify resident and immigrant animals, since females are more philopatric than males.

Stoat densities can also be estimated by live trapping, and this has successfully been adopted in Sweden (e.g. Erlinge 1983), Switzerland (e.g. Debrot 1984) and

in New Zealand (e.g. King & Edgar 1977; King & McMillan 1982). In *Nothofagus* forest, stoat trappability increased during and following the post-seedfall decline phase in mouse populations. Hence stoat capture rates varied not only with their abundance, but also with that of their prey (Alterio et al. 1999). This should be borne in mind when interpreting capture rates, and indices of prey availability should be recorded during both live trapping and kill trapping campaigns.

4.3 AGEING TECHNIQUES

A range of techniques of varying accuracy is available for ageing stoats (King 1991). The most reliable is sectioning and counting annual layers in dental cementum and this has been validated on animals of known age (Grue & King 1984; King 1991). Sectioning of canine teeth from dead animals has been used effectively in several studies (including Erlinge 1977a; Powell & King 1997; McDonald 1998). If the date of death is known, the development of the post-orbital constriction, sagittal crest and other skull features, together with the weight of the baculum in males, can be used to identify young males up to approximately ten months and young females up to approximately five months (King 1991). Over 99% of adult males have enlarged testes in summer, but young of the year do not (McDonald & King in press). Adult females have visible nipples if they have borne young, but nipples of adults that have not bred and particularly the young of the year are small and inconspicuous (McDonald & King in press). Therefore, conspicuously young individuals can be identified by a combination of skull characteristics and in certain cases reproductive characters and need not be aged by cementum analysis (King 1991). However, there is little alternative to cementum analysis for ageing adult stoats in any detailed study of stoat population ecology. Fortunately, the technique is readily and economically undertaken by contract laboratories, principally Matson's Lab (Box 308, Milltown, MT, USA) who in 1999 charged approximately US$4 per sample.

All demographic studies undertaken so far have assumed a common birth date for stoats, usually 1 April in the northern hemisphere (McDonald 1998) and 1 October in the south (e.g. Powell & King 1997). Given the importance of narrow windows-in-time for the application of stoat control (see below) it would be informative to determine actual birth dates and thereby dates of implantation more accurately. This has been done in other animals such as brown hares *Lepus europaeus* by relating age to the weight of eye lenses that have been fixed in formalin (Suchentrunk et al. 1991). This technique may be suitable for mustelids but we are unaware of tests on animals of known age. This could realistically be tested in the early stages of establishing a captive colony. Towards the same aims, the length and weight of developing foetuses is normally accurately related to expected birth date, but this has only been measured in relatively few individuals and could be investigated further using carcass collections. However, pregnant females are extremely rare in any carcass collection, hence a long-term effort for this would be required.

4.4 SURVIVAL AND MORTALITY RATES

Stoats naturally experience high rates of mortality, primarily due to starvation. In Sweden, annual mortality rates in undisturbed populations measured in the field are slightly lower than in culled populations: 0.40–0.78 (males) and 0.54–0.83 (females), according to age and year (Erlinge 1983). In Switzerland, annual mortality rate for both sexes was 0.68 (Debrot 1984). Essentially, even in undisturbed populations, approximately two-thirds of all live adults in one year will die before the next year.

Few studies have taken the step of calculating life tables for stoats because of violations of the numerous assumptions inherent to the process of converting age structures to static life tables. For a detailed consideration of these problems see Caughley (1977) and Krebs (1989). The most thorough analysis of life table data for stoats, and the only study considering variation between cohorts, was based on samples collected in *Nothofagus* forest in New Zealand (Powell & King 1997). For trapped populations the approaches adopted in this study are likely to be the best attainable, given the limitations of sample size and origins. The life tables presented by Powell & King (1997) highlight the inter-annual differences in survival rates in New Zealand stoat populations, which is a factor of the fluctuations of rodent populations, which are in turn driven by the *Nothofagus* mast cycle. King et al. (1996) calculated preliminary life tables for stoats living in mixed podocarp and exotic woodland in Pureora Forest. They suggested that mortality rates were comparable to those in *Nothofagus* forest. It remains to be seen whether survival rates are as variable between years in other habitats in New Zealand.

Among 822 stoats collected by gamekeepers in Britain, McDonald (1998) identified a significant difference in the cumulative rate of survival l_x between males and females as the result of seasonal differences in their susceptibility to shooting and trapping. First-year female stoats experienced proportionally higher rates of mortality than males in the two three-month periods from July to September and October to December. In the period January–March, capture rates of females by trapping were significantly lower than expected, although mortality rates of both sexes were comparable in this period, because shooting females compensated to some extent for their lower susceptibility to trapping (McDonald 1998). This suggests that applying alternative methods of control in addition to trapping at this time of year will be effective in raising female mortality and reducing population growth.

Age structure and recruitment rates depend on breeding success in the preceding year. The proportion of young stoats in the population can vary markedly, depending on location and time of year (Table 1). Life span is short, especially where kill-trapping pressure is continuous (Debrot 1984). In undisturbed populations in southern Sweden, the proportion of young varied from 31% to 76% over five years. The average further life expectancy from age of independence (3–4 months) was 1.4 years in males and 1.1 years in females, and the maximum age observed was 4.5 years in males (n = 47) and 3.5 years in females (n = 48) (Erlinge 1983). In a similar three-year study of an undisturbed population in Switzerland the proportion of young from August to December was 55–67% and the mean age was 1.2 years (Debrot 1984). The maximum age

TABLE 1. SUMMARY OF THE AGE STRUCTURES OBSERVED IN TRAPPED SAMPLES OF STOATS AND WEASELS.

The structures of additional samples are reviewed in King (1989 p. 175).

AGE STRUCTURE						LOCATION	REFERENCE
<1 YEAR		>1–2 YEARS		>2 YEARS			
461	(64)	256	(36)	*		Great Britain	McDonald 1998
31	(56)	13	(24)	11	(20)	New Zealand	King et al. 1996
83	(60)	55	(40)	*		New Zealand	Murphy & Dowding 1995
31	(58)	22	(42)	*		New Zealand	Murphy & Bradfield 1992
81	(63)	38	(30)	9	(7)	Sweden	Erlinge 1983
113	(84)	17	(16)	*		New Zealand	King & McMillan 1982
18	(64)	5	(18)	5	(18)	Denmark	Jensen 1978
113	(75)	37	(25)	*		Netherlands	van Soest & van Bree 1970
47	(62)	29	(38)	*		Denmark	Fog 1969

* These studies combined all animals >1 year old.

attained, very infrequently, by wild stoats in temperate countries appears to be 6–8 years (Grue & King 1984), though this is probably less in the far north (Kopein 1967).

In order to derive annual rates of survival of adult stoats for entry into an extended Leslie matrix population projection model, McDonald (1998) considered that the maximum age detected by cementum analysis in a sub-sample of 40 out of approximately 800 stoats (5%) reflected the longevity of 5% of the population. Thus the annual rate of survival of stoats older than one year *P* was calculated as:

$$P = 0.05^{\frac{1}{A-1}}$$

where *A* was the maximum age observed by cementum analysis. In the sample of 822 stoats killed by gamekeepers the oldest individual was four. Hence these assumptions meant that, in Britain, adult survival approximated 0.37 and suggested that individual stoats over five years in age constituted only 1.8% of the wild population while those over six constituted 0.7% and so on (McDonald 1998). These figures are comparable to those presented for more complete samples of cementum analysis (King et al. 1996; Powell & King 1997).

4.5 REPRODUCTION

Stoats mate from April to July in the northern hemisphere and from October to January in southern hemisphere. Ovulation is induced by repeated and vigorous mating (Müller 1970; Ternovsky 1983), although copulations between neonatal females and adult males lasted less time than between adults (Ternovsky 1983). There is a post-partum oestrus in adult females (Rowlands 1972). The peak period for stoat births is April in the northern hemisphere and October in the southern hemisphere (McDonald & King in press).

Females are sexually mature as early as 2–3 weeks after birth, while they are still blind, deaf and hairless (Ternovsky 1983). They are mated by adult males and are usually fertilised before weaning. In contrast, males do not mature until they

are approximately one year old. Müller (1970), Ternovsky (1983) and others have recorded no hesitation on the part of adult males or neonatal females to mate when one was the father of the other. In wild populations, however, the lack of pair bonds between adult males and females and the rapid turnover of the population apparently reduces the chances that adult males mate with their own young (King 1989). However, levels of inbreeding in stoat populations in New Zealand or, for comparison, Britain have not been tested. Given the genetic bottleneck that stoats went through when introduced to New Zealand and this trait in their mating biology, it may be that stoats in New Zealand have experienced unusually high levels of inbreeding. In the case of black-footed ferrets, the drastic effects of disease outbreaks were blamed in part on the low level of genetic variability in the population (Fagerstone 1987). This may provide some encouragement for the potential effectiveness of novel control agents for stoats in New Zealand.

After mating, the initial development of corpora lutea and blastocysts is rapid but is interrupted after 2–3 weeks by an obligate delay in implantation of 9–10 months. Corpora lutea remain small and plasma progesterone levels stay low until March in the northern hemisphere and September in the south (Deanesly 1935, 1943; Gulamhusein & Thawley 1974). Implantation is initiated by increasing day length in spring, mediated by rise in plasma progesterone, produced by reactivated corpora lutea (Gulamhusein & Thawley 1974). The critical day length for stimulating implantation is reached earlier at lower latitudes and so births are earlier. There is a difference of about ten days between the north and the south of New Zealand, which lie seven degrees of latitude apart (King & Moody 1982). The active period of pregnancy lasts about four weeks and so the total gestation period lasts approximately 280 days (Rowlands 1972).

4.6 FECUNDITY, FERTILITY AND PRODUCTIVITY

Productivity, in terms of the number of independent young produced per female, is controlled by a range of factors (King 1989). Fecundity, i.e. the number of ova shed per female, is relatively constant, though it varies with food supplies in the year during which mating takes place, prior to the period of delayed implantation (King 1981, 1983b; Powell & King 1997). The mean ovulation rate in a colony of captive stoats in Britain was ten and the range was 6–17 (Rowlands 1972). In New Zealand the mean was also ten but the range was larger, 3–20 (King & Moody 1982). Fecundity is not related to female age as previously thought (King & Moody 1982) since this analysis was later found to be confounded by inter-annual variation in food availability (Powell & King 1997). In any case, there is no relationship between fecundity, which is set in the year of mating, and productivity the following year (King 1981). The main response to food supplies takes place in the year during which implantation and births take place, in the form of highly variable levels of intra-uterine mortality (King 1981, 1989).

Counts of embryos in the uterus take into account intra-uterine mortality of blastocysts, but not subsequent resorption of embryos, so they incorporate part, if not all, of the difference between fecundity and fertility. In captive

stoats, the average fertility when defined as the number of blastocysts in delay was 11.2 ± 0.9 (Ternovsky 1983). The mean fertility measured by embryo counts in 50 litters described in various sources was 8.9 and the range was 6–13 (Deanesly 1935; Fairley 1971; Rowlands 1972; King & Moody 1982; McDonald 1998). 25% of 17 pregnancies showed signs of resorbing embryos (Rowlands 1972).

The productivity of stoats is not well known. In wild populations so few litters have been analysed that this is the least well described, but perhaps most important, aspect of stoat demography. In practice, productivity has been measured in captive animals (see below). However, conditions in captivity, especially the provision of food *ad libitum*, suggest that these may be over-estimates of productivity in the wild. In Britain, nine young were born in one litter in captivity (East & Lockie 1965). Thirteen young were born in another captive litter, although mortality was high in the first days of life (J. Roberts pers. comm. to RM). In a captive colony in Siberia (which is described in detail below), 29 females that were mated as juveniles had a mean litter size of 7.6 ± 0.66 while 17 females that were mated as adults had a mean litter size of 6.7 ± 0.85 (Ternovsky 1983). A truer measure of productivity can be derived from differences in observed population densities and the percentage of independent young present. However, in practice this is usually unworkable in trapped populations and requires a set point in time for calculation.

4.7 DENSITY DEPENDENCE

Evidence for density-dependent population regulation is scarce in stoats, mainly because of a lack of detailed demographic studies. We are not aware of any evidence for density-dependent processes controlling fertility. The strongest indications of density-dependent mortality were derived from studies of trapped samples taken from different stages of the beech mast cycle in New Zealand (Powell & King 1997). The first-year mortality rates of the large cohorts born in years with increased food availability were higher than for the smaller cohorts born in years when food was scarce. This effect was ascribed to the high energy demands placed on young stoats, particularly males, that had grown large when food was plentiful but suffered when food became scarce. The crash phase of rodent populations and the intense intraspecific competition led to increased levels of density-dependent mortality (Powell & King 1997).

In some other mustelids, evidence of density dependence is more prevalent. In feral American mink populations in Ireland, weight loss and increased seasonal levels of mortality appeared to provide evidence of density-dependent population regulation, although strict tests for the process were not applied (Smal 1991). In Algonquin Park, Canada, the growth rate in harvested American marten *Martes americana* populations was influenced by prey availability, harvest rate and density dependence. The mechanism for density dependence may have been related to agonistic interactions between territory holders. The combined effects of prey fluctuations and intrinsic density-dependent mechanisms may have had a stabilising influence on marten populations, and local marten populations were not cyclic (Fryxell et al. 1999).

4.8 MOVEMENT PATTERNS

Stoats generally adopt a spacing pattern that follows the typical model of mustelid intrasexual territoriality (Powell 1979). Male territories generally encompass the smaller territories of several females (Simms 1979). Both males and females defend territories against intruders of the same sex (Erlinge 1977b). Territory size and ranging behaviour varies between seasons, depending on the relative importance of food and mates (Erlinge & Sandell 1986).

In a live trapping study, a single male stoat living on a plantation in Stirlingshire, Scotland, with high-density vole populations had a range of > 20 ha (Lockie 1966). One radio-tracked male living on farmland in Aberdeenshire had a range of > 250 ha, while three females had a mean range of 114 ha (Pounds 1981). However, if these calculations had taken into account only the exploited area and excluded field centres and exposed areas, ranges were much smaller, though such a correction would not be relevant to the spatial integrity of the population. In Co. Cork, Ireland, radio-tracked female stoats had ranges of 2–22 ha and a single male had a range of 11 ha (Sleeman 1991).

In open pasture and marshland in Sweden, male stoats occupied 8–13 ha and females occupied 2–7 ha (Erlinge 1977b). A later radio-tracking study on the same site revealed a mean male range of 16.7 ha (11–26 ha) in autumn and winter when not breeding but a mean male range of 733.5 ha (105–2644 ha) in spring and summer during the breeding season (Erlinge & Sandell 1986). In Switzerland, live trapping revealed ranges of 8–40 ha for males and 2–7 ha for females (Debrot & Mermod 1983). On farmland in Québec, radio-tracked male stoats had a mean range of 19.8 ha while females occupied 4.8 ha (Robitaille & Raymond 1995). In *Nothofagus* forest in New Zealand in 1990–91 four females had a mean range of 69 ± 8 ha, and three males 93 ± 7 ha (Murphy & Dowding 1994). In the same area in 1991–92, five female stoats had a mean range of 124 ± 21 ha and four males had a mean range of 206 ± 73 ha (Murphy & Dowding 1995). Inter-annual differences in ranges in this habitat were ascribed to marked changes in food availability.

Female ranges remain largely unchanged during the breeding season (Erlinge & Sandell 1986). In contrast, in order to maximise their mating success male stoats adopt one of three strategies, becoming: roamers, stayers, or transients. Dominant older males expanded their territory size up to 50 times and roamed widely spending a few days with every female they encountered. Younger males of moderate social status stayed in a small distinct range, and males of the lowest status were completely transient and had no distinct range (Erlinge & Sandell 1986; Sandell 1986; Sandell & Liberg 1992). Clearly these strategies will have a marked impact on spatial models of stoat control.

4.9 DISPERSAL

Stoats are capable of extremely long distance movements relative to their size and these can be particularly pronounced during juvenile dispersal. In Switzerland the annual turnover of resident stoat populations is also very high, at approximately 0.93, i.e. only 7% of adults remained resident for a second year

(Debrot 1984). However, the Swiss study area was rather small relative to the dispersal capabilities of stoats and so this may not be surprising. In Sweden, females tended to be philopatric while males were more prone to juvenile dispersal (Erlinge 1977b). In Sweden, 16 out of 18 females remained on their natal area for life (Erlinge 1983). This appears to be the pattern for other mustelids, since in a harvested population of fishers *Martes pennanti* the probability of dispersal in the first year of life was 73% and 100% for males and females respectively (Arthur et al. 1993). Long-distance movements by adult males can also be associated with the roaming strategy adopted by high-status males or the transient strategy adopted by low-status males during the mating season and are not necessarily related to true dispersal (Erlinge & Sandell 1986).

A single tagged stoat moved 35 km in Alaska between captures in August 1960 and March 1961 (Burns 1964). In New Zealand several long-distance movements have been recorded, particularly by young males, though many returned to their original capture sights and hence this could not be considered to be true dispersal (King & McMillan 1982). Several young males travelled 6–15 km over 2–4 weeks. At least three other males crossed from one river drainage to another, connected by a mountain pass at 530 m. Of these three males, one travelled at least 23.3 km in 39 days and another travelled at least 24.4 km in only one week (King & McMillan 1982). In Fiordland, New Zealand, a single juvenile female stoat dispersed at least 65 km in four weeks before being trapped (Murphy & Dowding 1995). To our knowledge, these are the only data on dispersal in stoats. Since this is such an important factor in spatial modelling and control studies, we advocate both a sensitivity analysis of any future spatial models to dispersal distances as well as a more detailed field study of the process.

4.10 EFFECTS OF TRAPPING

Killing stoats by trapping may reduce their population density and change age structure. In a study of 63 stoats collected from Pureora Forest in New Zealand, King et al. (1996) found that capture rate and the proportion of adults caught declined after the initiation of a regular kill trapping campaign and remained lower than the first year thereafter. They also reported similar patterns of change in density and age structure in *Nothofagus* forest (King et al 1996; Powell & King 1997). Using a population projection model, McDonald (1998) found that trapping and shooting by gamekeepers in Britain had a marginal effect on closed stoat populations (McDonald 1998). Stoat populations did not mirror the exponential growth sustained by weasel populations ($\lambda = 1.350$, $r = 0.300$) under an equally intense control regime. Stoat population growth rate was slightly less than one ($\lambda = 0.953$, $r = -0.048$), suggesting population growth was not significantly different from equilibrium. This was thought to be mainly the result of the mortality of dependent juveniles following the mortality of their mothers. The model populations were closed, and in real populations, normal levels of immigration would probably maintain growth. The importance of trapping is, however, likely to increase as populations become more isolated and levels of immigration are reduced. King & McMillan (1982) concluded that trapped stoats would commonly be replaced by dispersing individuals from up

to 20 km away. Under normal conditions and over a large scale, trapping by gamekeepers in Britain does not appear to limit population growth in stoats. This is consistent with the conclusions of King & Moors (1979) and Tapper et al. (1982) that trapping exerts only a short-term and local effect on populations.

In Algonquin Park, Canada, the growth rate of harvested marten populations was influenced at least in part by harvest rate. Harvest intensity was negatively related to population growth rate, though percentage mortality rates did not vary with population density. Hence, harvesting acted as an external variable that was additive to density-dependent and prey-dependent effects in population regulation (Fryxell et al. 1999). In a harvested population of fishers, high levels of dispersal meant that animals trapped by fur trappers were rapidly replaced (Arthur et al. 1993).

4.11 MODELS OF STOAT DEMOGRAPHY

The development of models of stoat population biology is at an early stage. Simple approaches such as Leslie matrix modelling have been attempted (McDonald 1998; C. King, M. Efford & R. Powell unpublished data). Recent developments of matrix modelling techniques include the addition of density dependence and demographic stochasticity (Tuljapurkar & Caswell 1997). A particular advantage of matrix modelling is that it allows the analytical solution of the sensitivity and proportional sensitivity (elasticity) of growth rate to individual demographic parameters. This means that the effects of perturbation of key parameters can be predicted quantitatively. Such developments have greatly expanded the utility of matrix models to conservation biologists, and the approach has gained particular favour in applications for conspicuously age- or stage-structured species. It is debatable whether stoats fit this description since the differences in the survival and fertility of adult age classes are not clear but do not seem substantial. However, the analytical simplicity of this modelling approach makes it attractive.

To our knowledge there have been no attempts, other than in the studies now being undertaken as part of the Stoat Research Programme, to construct spatially explicit models of stoat populations. This is clearly of fundamental importance to understanding the efficacy and cost effectiveness of stoat control. The basic data required to support such a modelling exercise are now available. However, we suggest that caution should be applied to the interpretation of these initial models, given the few aspects of spatial behaviour that have so far been described in detail. Provision of further information on spatial behaviour in a range of habitats and times would be of great significance to these modelling exercises.

The management of furbearers in North America has surprisingly not provided many comparable models that would be of use in managing stoats. We are aware of one recent novel development of an expert system model for managing lynx *Lynx canadensis* populations in Alaska (Golden 1999). This model formalises the decision-making process for wildlife managers in a number of districts and takes into account a range of parameters, including previous harvest rates, prey censuses and climatic factors, that impact on the

conservation of the species. The formality of an expert system model is attractive from the perspective of ensuring consistency between local conservation managers and for ensuring cost effectiveness at a large scale.

4.12 SUMMARY AND RECOMMENDATIONS

Stoats are *r*-selected predators with high rates of mortality and productivity. While this life-history pattern makes them resilient to control, there is evidence that even unfocused control efforts can affect density and age structure, at least in the short term. It is well known that most aspects of stoat demography are tightly governed by the availability of prey (King 1983b, 1983c, 1989). Food supply in late winter and early spring controls the fertility of females by influencing rates of blastocyst implantation. Later in the year, food supply is influential in controlling productivity by affecting the survival of nestlings. Eventually changes in food supply mediate the annual mortality that affects population growth rate by causing variation in the survival of newly independent young. Clearly all these processes are directly related to rates of predation on birds.

Two particular features of stoat management in New Zealand make changes in prey populations significant to bird conservation. First, the incidence of predation increases when stoat populations exhibit a numerical response to increasing prey populations during the mast years in *Nothofagus* forests. Birds may be no more important to the diet of individual stoats, but the greater abundance of foraging predators means that predation is more significant to bird populations. Second, if prey supplies are reduced, stoats may exhibit a functional response by switching prey from rodents to birds. Essentially, while control of prey populations may eventually reduce stoat survival, there is a significant risk that more birds may be killed in the immediate term.

Stoats can only have one litter per year and their fertility appears to be controlled over a short period of time in the late winter when implantation is initiated and the four weeks of pregnancy thereafter. The functional response to prey supplies after the young are born means that controlling prey populations after this period is problematic. If prey availability could be reduced in a targeted period before and during pregnancy, stoats have no potential to increase their fertility in response to the recovery of prey populations later on. This would remove the effect of the numerical response. Prey populations that are left to recover after the critical period should, however, provide sufficient prey for surviving adults. This would avoid the functional response. Therefore it is the late winter period, when fertility could potentially be manipulated, which deserves the greatest attention from researchers.

The collection of demographic data should be seen as an ongoing process. The picture we have of stoat demography is far from complete and should not be seen as such, even though certain aspects may now appear well described. To illustrate this point, it is clear that density-dependent processes are of fundamental importance in the demography of any species and are of direct significance to understanding stoat control. However, this aspect of stoat population biology has only once received attention in the literature from New

Zealand (Powell & King 1997) and cannot adequately be addressed except by one set of data collected in the early 1970s.

In order to provide an expanding database of essential data on stoat demography, we recommend that a standardised procedure be instigated for the continual collection of census and demographic information from several districts of New Zealand. This information could most easily and immediately be collected by capitalising on the control work already undertaken by DOC and other government personnel and would require a minimal budget for post-mortem examination and cementum analysis. In tandem with this long-term effort we highlight the utility of a detailed knowledge of actual birth dates in various areas and this could be elucidated by studies of accurate ageing techniques on captive animals.

Given the significance of spatial information to understanding stoat control, we suggest that large-scale capture–mark–recapture exercises be undertaken in the field. Again, these could harness the effort already being put into stoat control by local DOC personnel. At the end of intensive kill-trapping periods for bird protection when birds were less at risk, it is conceivable that live traps could be substituted and live stoats tagged and released in the expectation that they would be killed in traps the following season. Such an exercise would not greatly threaten bird survival if conducted outside of the nesting season, but if conducted at a large scale would greatly supplement the scant information currently available on dispersal (King & McMillan 1982).

In the longer term it would be desirable to standardise decision making for local managers in order to ensure the cost effectiveness and maximum efficiency of stoat control operations. This could most readily be realised by the development of expert system models as detailed by Golden (1999).

5. Captive breeding

5.1 INTRODUCTION AND AIMS

The establishment of captive colonies of stoats will be invaluable for developing novel control technology. Captive animals could be used to test lures, baits and trap designs in order to improve current trapping methods. Captive animals may provide further insights into factors affecting the control of reproduction and reproductive pathology. They may also be used in toxicological and disease trials and in studies of disease transmission and the importance of arthropod vectors. There is also some potential for the use of 'Judas' stoats, whereby captive-bred individuals might be released into a wild population and used to initiate disease transmission to wild animals.

In the past, stoats have been considered unsatisfactory to tame and no records of breeding in captivity were thought to exist (Deanesly 1935). Detailed records of attempts at farming stoats for fur are extremely scarce. Details of a few stoats held on Canadian fur farms are provided in the Reports of Fur Farms. In the

whole of Canada, only eight stoats producing a total of 18 young and nine stoats producing eight young were held in 1929 and 1930 respectively (Dominion Bureau of Statistics 1931). In 1950 only one fur farm out of 3492 in Canada held stoats. No farms with stoats were recorded in 1949 or in 1951, suggesting this was a short-lived venture (Dominion Bureau of Statistics 1951, 1952). In 1953, an unknown number of stoats was included in 98 'other fur animals' which also included fitch (ferret), nutria (coypu *Hyocastor coypus*) and skunks, out of a total of 320 000 animals harvested from fur farms (Dominion Bureau of Statistics 1954). We have not been able to locate any sources describing the methods adopted for keeping stoats in fur farms. To our knowledge there are no commercial fur farms in North America that currently keep stoats (T. Platt, Fur Commission USA pers. comm. to SL).

Stoats have received limited attention in science projects (Gulamhusein & Tam 1974; Gulamhusein & Thawley 1974; Robitaille 1989, Bern 1993, Raymond et al. 1990, Robitaille & Baron 1987, Vaudry et al. 1990). They have also been held and bred in captivity as pets (M. Gamache pers. comm. to SL; J. Roberts pers. comm. to RM) and for display in zoos (Doncarlos et al. 1986; D. Gow pers. comm. to RM). As a result of their high activity combined with extended rest bouts, stoats are frequently considered unsuitable for captive display (Crandall 1964). The International Species Information Service (ISIS) at www.worldzoo.org provides a list of the very few zoos that keep stoats in captivity. In North America, the Minnesota Zoological Garden, Apple Valley, MN, have had an exhibit of both stoats and long-tailed weasels *Mustela frenata* for more than ten years (R. Powell pers. comm. to SL). In Britain, New Forest Nature Quest, near Southampton, until recently kept approximately 12 stoats and five weasels. This collection has now been transferred to another wildlife park, Wild Wood in Kent (D. Gow pers. comm. to RM). Leningrad Zoo kept three pairs of stoats resulting from a single litter in the early 1970s, but details of this collection are scarce (Andreewskaja & Brandesowa 1977). None of the world zoos participating in the ISIS have reported births during the last six months, and to our knowledge, the Minnesota Zoological Garden and New Forest Nature Quest are the only zoos that have recently bred stoats in captivity.

We have reported the details of several attempts at keeping stoats in captivity for a diverse range of purposes. Accounts of keeping stoats in Bern (Müller 1970), at Minnesota Zoo (Doncarlos et al. 1986), and at the Siberian Branch of the Soviet Academy of Sciences in Novosibirsk (Ternovsky 1983) were particularly useful. We have supplemented this information by accounts of keeping related species where relevant. We aimed to provide a starting point for captive stoat maintenance and husbandry and to give direction to other sources of advice.

5.2 HOUSING

In Bern, stoats were kept in cages made of 10 mm mesh of 1 mm gauge wire and fibre cement (Woodcrete) bottoms covered with sheet metal or glass. The cages were either 125 cm^3, 120 × 80 × 70 cm or preferably 150 × 100 × 60 cm. The cages were furnished with a nest box with a 5 cm diameter entrance hole. The box was made of thick softwood of 35 × 25 × 25 cm on the outside with an

inner box of 20 cm^3 and fibre board insulation between the two layers. Natural moss was provided for nesting material. The bottoms of the cages were covered with a variety of natural material including sticks, moss, leaves and hollow logs which were replaced only every 1–2 months to avoid irritating the animals. Cages developed later were equipped with a nest box on the outside of the cage, the door of which could be closed without opening the cage (Müller 1970).

Stoats at Minnesota Zoo were housed individually in holding boxes 25 × 20 × 45 cm in size, with a detachable nest box of 18 × 12 × 14 cm. The nest box had a Plexiglas roof for observation of stoats and their litters and was equipped with a sliding entrance cover. The animals were exhibited in a 200 × 200 × 100 cm glass-fronted container for one hour at a time (Doncarlos et al. 1986). Owners of pet stoats have successfully kept them in commercial hamster cages that are approximately 40 × 20 × 30 cm. Provision of cotton for nesting material was sufficient, as long as stoats were kept at room temperature (M. Gamache pers. comm. to SL).

Part of the New Forest stoat collection was housed indoors in cages with wooden sides and chicken wire mesh tops, approximately 150 × 200 × 50 cm. The cages were supplied with a nest box of approximately 15 × 15 × 15 cm and ample wood shavings and hay for bedding. Other stoats were kept outdoors on the ground in metallic circular pens of approximately 600 cm diameter by 150 cm high. The outdoor pens contained a nest box and a pile of rocks in which stoats also apparently made their own nests. However, stoats occasionally escaped from these outdoor pens through mole and rodent tunnels (D. Gow pers. comm. to RM). In Sweden, stoats have been kept successfully in cages of 120 × 50 × 60 cm for up to ten days before and after their use in behavioural experiments. The experimental arenas measured approximately 30 m^2 and contained simulated habitat with tree stumps, piles of rock and brush (Erlinge 1977a). At the Wellcome Institute of Comparative Pathology, London, stoats were kept individually in anodised aluminium cages (Gulamhusein & Tam 1974).

At Exeter University captive weasels were held in a cage of 120 × 60 cm (East & Lockie 1964). In New Zealand, eight weasels were successfully kept and bred outdoors in cages of 120 × 50 × 50 cm with wooden nest boxes of 25 × 15 × 15 cm (Hartman 1964). At Aberdeen University weasels were housed individually in wire cages 60 × 30 × 20 cm with a glass-topped removable nest box 15 × 15 × 15 cm with non-absorbent cotton wool for bedding. The floor of the cage was covered with sawdust which was regularly replaced (Moors 1974). At Helsinki University, weasels were kept in a cage of 120 × 60 × 60 cm, with a Plexiglas roof and wire mesh for sides. Each cage had a nest box filled with wood chips, tubes to hide in, a running wheel and an open water dish (J. Sundell pers. comm. to SL). A single pet weasel was kept in a clear plastic rat's cage of 55 × 40 × 20 cm (Scott 1982).

Because of their long, thin shape, thermoregulation in stoats is managed behaviourally and captive stoats must be provided with ample nesting material if kept in cold temperatures. If provided with insulated boxes and adequate bedding, the stoats kept in Bern and in Siberia fared well when housed outside with no artificial heating or lighting (Müller 1970; Ternovsky 1983). The

Minnesota Zoo kept their stoat colony indoors at 21°C, with illumination provided from fluorescent and incandescent light modified weekly to mimic natural photoperiod (Doncarlos et al. 1986). At the Wellcome Institute and in the New Forest, stoats were kept in an unheated, well-lit room under normal daylight conditions (Gulamhusein & Tam 1974; D. Gow pers. comm. to RM). At the latter location artificial lighting was used when feeding the animals in the morning and evening in winter and this may have upset the animals' photoperiod (D. Gow pers. comm. to RM). At Exeter, captive weasels were initially kept indoors, then they were transferred via a cold greenhouse, to the outdoors (East & Lockie 1964). Similarly, at Aberdeen, weasels were kept in a shed with no artificial lighting or heating (Moors 1974), and in Helsinki, weasels were kept in greenhouses under normal photoperiod (J. Sundell pers. comm. to SL).

Solitary confinement of adult stoats is essential, as any combination results in fighting except during the mating season. Mixed-sex siblings may be housed together, but they need to be separated when they reach adult size (Doncarlos et al. 1986).

5.3 FEEDING

In Bern, stoats were fed mainly on ox heart, which was better than muscle, liver or offal because it kept longer. They were also fed freshly killed mice from a colony kept for the purpose. However, the stoats' requirements exceeded the breeding capacity of the mouse colony and so this was supplemented with rats, guinea pigs and hamsters from a commercial laboratory supplier. Smaller rodents were fed to the stoats live, but larger rodents were killed and cut in two. Stoats were not keen on newborn rodents, hatchling birds or foetuses. On average they ate about 50–70 g of food per day, although food was provided in excess so that starvation was not a risk (Müller 1970).

Prior to 1983, captive stoats at the Minnesota Zoo were fed canned catfood twice daily in amounts approximating 25% of body weight (Doncarlos et al. 1986). However, none of the three captive females reproduced on this diet. In 1984, one female was fed freshly killed laboratory mice and she reproduced, whereas the other two females that were still fed on canned catfood did not. In 1985, all three females were fed freshly killed mice and all three reproduced. Since then all stoats at the Minnesota Zoo have been fed freshly killed mice *ad libitum* (Doncarlos et al. 1986). At Leningrad Zoo, a female stoat and her litter of 12 young were fed mainly on live white mice, supplemented with eggs, cream, curds and a little meat with vitamin and mineral supplements (Andreewskaja & Brandesowa 1977).

In the behavioural trials in Sweden captive stoats were fed on live mice (Erlinge 1977a; Erlinge et al. 1982). Captive stoats used in behavioural experiments in Canada were also fed live mice (J.F. Robitaille pers. comm. to SL). At Exeter University, stoats and weasels were fed live mice (East & Lockie 1964). At the Wellcome Institute, stoats were fed on raw minced cow or horse flesh alternated with dead mice. They were also provided with an egg-milk mixture every other day (Gulamhusein & Tam 1974). Captive stoats and weasels at the

New Forest Nature Quest and the Wild Wood Park were fed mainly on day-old chicks (D. Gow pers. comm. to RM).

Pet stoats have been fed a variety of foods including fresh meat, from muskrats *Ondatra zibethicus*, woodchucks *Marmota monax* and rabbits to commercial catfood. Surprisingly, captive stoats that were offered simultaneously dry catfood and fresh meat from muskrats or woodchucks preferred the dry catfood (M. Gamache pers. comm. to SL).

Amongst other mustelids, 13 stoats caught in Canada were kept in captivity for trials of the effects of Aleutian Disease Virus on the Mustelidae (Kenyon et al. 1978). They were fed on a prepared diet of fish (30%), poultry (32%) cottage cheeses (15%), soyabean meal (15%) bread crumbs (5%) and corn sugar (1%) or on a complete ration (G'NF-100 supplied by National Northwood Co., a division of Cudahy Co., New Holstein, Wisconsin).

In Helsinki, captive weasels were fed dead chicks as well as laboratory mice and rats. Each weasel consumed on average one 30 g vole per day. In early attempts at establishing the colony, several weasels died in the first few days of captivity. However, no weasels died when they were fed live voles for the first few days of captivity, apparently because killing voles appeared to relax stressed weasels (J. Sundell pers. comm. to SL). In New Zealand, captive weasels were fed twice daily until three months of age and once daily after that. They were given ox heart with occasional fresh milk, egg yolk and ox liver, with vitamin supplements and cod liver oil (Hartman 1964). At Aberdeen University, captive weasels were provided with food and water *ad libitum*. Their main diet was dead day-old chicks supplemented with dead mice or portions of rabbit (Moors 1974). A single pet weasel was fed a single weaner rat every day (Scott 1982).

Caching behaviour is well developed in mustelids, and captive stoats may cache food in the nest box, resulting in rapid disappearance (not necessarily rapid consumption) of food offered (Doncarlos et al. 1986; J.F. Robitaille pers. comm. to SL). Thus, regular cleaning of the nest box is probably necessary to maintain sanitary conditions.

Stoats rapidly learn to use commercial water 'sippers' designed for gerbils and hamsters (M. Gamache pers. comm. to SL). At the Minnesota Zoo and at the Wellcome Institute, sippers provided water *ad libitum* to each captive stoat (Gulamhusein & Tam 1974; Doncarlos et al. 1986). In Helsinki, weasels were provided with an open water dish, but because they frequently urinated in the water dish, a sipper was also provided. Weasels occasionally bathed in the open water vessels, especially during hot weather, and this doubtless contributed to the enrichment of their captive environment (J. Sundell pers. comm. to SL). A pet weasel was provided with a 500 cm^3 sipper and water *ad libitum* (Scott 1982). Bissonnette & Bailey (1940) provided each captive stoat with two sippers, one for cows' milk and one for water.

In the case of hand-rearing baby stoats, it is preferable to wean them as soon as possible rather than feed them on milk. If feeding with milk is unavoidable, powdered milk formula and water is apparently preferable to cow's milk (Müller 1970).

5.4 BREEDING

Females that are caught alive in the first year of life are almost always carrying fertilised blastocysts in delay. Therefore live captures of young females will be invaluable for establishing a captive colony. Andreewskaja & Brandesowa (1977) briefly described the care at Leningrad Zoo of a single female and her litter that were caught in this way. Captive stoats kept in groups within outdoor enclosures produced litters (J.F. Robitaille pers. comm. to SL). At the Minnesota Zoological Garden, all 33 stoats born in captivity between 1983 and 1986 survived to maturity. Thus, survival of captive-born stoats does not appear especially problematic.

In Bern, adult males were kept separate from pregnant females during the period of pregnancy, birth and early stages of rearing the young. In one case, 47 days after the birth of her litter a female stoat aggressively chased away a male that had been let into her cage. At 60 days the same female allowed herself to be mated by the same male. Three days after this the same male mated with the nine-week-old females, although the mating was interrupted by the mother (Müller 1970). In a further case, a male was introduced to the cage containing three 64-day-old females. The first female was mated almost immediately in a typical copulation behaviour (Heidt et al. 1968). The male grasped the female by the neck with his teeth, gripped her body with front and hind paws and dragged her around the cage. The copulation lasted 3.5 minutes and was followed immediately by the male copulating with a second female. This second copulation lasted 19 minutes. After this, the third female arched her back like a cat and presented her genital area to the male. Her advance was ignored by the male who then returned to his cage. Successful matings also took place between adult males and young females of 36–39 days (Müller 1970).

In Siberia, 46 litters of 335 young (167F) were born to 40 captive female stoats between 1968 and 1982 (Ternovsky 1983); 29 of the breeding females were mated by adult males between 17–75 days after birth and gave birth to normal offspring the following year. The youngest 17-day-old female was still blind and helpless when mated successfully by an adult male. Females that mated when neonates were no less fertile than those mated when adults. Furthermore, the female offspring resulting from matings between neonates and adults were no less fertile when they themselves bred. Mother stoats were found to chase male suitors away only in the first 5–10 days after birth but hostility diminished rapidly thereafter. Females carrying blastocysts in delay could apparently be induced to give birth earlier than expected by manipulating the lighting regime (Ternovsky 1983).

In Minnesota, four adult males were introduced one at a time to a cage containing five young stoats (2M, 3F) of six weeks and their mother. Typical copulation between the adult male and all the young females followed. Neonatal females did not show oestrus behaviour, except when a male was in their presence. When a male was introduced, neonatal females (36–48 days of age) vocalised with high-pitched trills or chuckles, and crawled after the male and even interfered with matings with their mother or siblings. Males that were unsuccessful in copulating with adult females had greater success in copulating with neonatal females. A single neonatal female mated at 42 days of age gave

birth to ten young after a delay of 307 days. Most matings between adult males and young females took place 36–48 days after birth (Doncarlos et al. 1986).

Usually, adult males do not interact with the juvenile males. In one case in Minnesota an adult male attempted to copulate with a neonatal male, but was fought off by the kit (Doncarlos et al. 1986). In one case in Siberia, the adult male mated with all four females in a litter and killed all four males (Ternovsky 1983).

Mating between adults follows a similar pattern to that described. Males were always introduced to the cage of females, ideally by removing a partition between adjoining cages. Copulation was very vigorous, lasted between 2 and 13 minutes and was repeated four or five times over a period of about an hour (Müller 1970).

The development of a litter of young stoats in captivity has been described at Exeter University (East & Lockie 1965). Few details are provided about the captivity of the single female stoat, but in a related paper (East & Lockie 1964) the authors describe conditions for breeding weasels. Male and female weasels were kept in adjoining cages, and were introduced to one another initially under close observation and subsequently by having two 2.5 -cm holes joining the cages through which only the female could pass. When the female was pregnant the male was removed from the adjoining cage (East & Lockie 1964).

On mink farms, a common problem for breeding is the presence of sterile males. Although this has not been reported as a problem for captive stoats, if mating success is low, sperm tests can help detect and identify sterile males (Sundqvist & Gustafsson 1983).

5.5 OTHER MUSTELIDS IN CAPTIVITY

The IUCN Mustelid/Viverrid Specialist Group considered that promoting captive breeding of mustelids was a priority (Schreiber et al. 1989). At the time they recommended that a Captive Breeding Specialist Group be formed. However, we do not know whether such a group was ever formed or active. European mink *Mustela lutreola* and black-footed ferrets have been successfully raised in captivity, but tropical weasels *M. africana* and mountain weasels *M. lutreolina* have not. To our knowledge, the Colombian weasel *M. felipei* and the back-striped weasel *M. strigidorsa* have never been kept in captivity (Schreiber et al. 1989). Amstislavsky & Ternovskaya (2000) have described the reproduction of eleven species of mustelid held in captivity in Novosibirsk, Siberia. These included stoats, martens, mink, ferrets and polecats, but also sable *Martes zibellina*, mountain weasel *Mustela altaica*, and Siberian weasel *M. sibirica*. Their institution is probably the greatest source of practical expertise for breeding mustelids in captivity.

Other mustelids, primarily those for which there has been a commercial interest, have frequently been kept in captivity, and guidelines for husbandry are well described. Detailed manuals are available for raising American mink (Adams 1935; Laberee 1941; Kellogg et al. 1948; Leonard 1966). Guidelines are also available for American martens (Yerbury 1947; Ritchie 1953), ferrets

(Carpenter & Hillman 1978), long-tailed and common weasels (Wright 1948; Sanderson 1949; Short 1961; Heidt et al. 1968) and black-footed ferrets (Aldous 1940; Progulske 1969; Thorne et al. 1985). Fishers do not reproduce well in captivity (Powell 1993) but some information on farming is nonetheless available (Hodgson 1937; Laberee 1941). Guidelines for the care and handling of North American river otters *Lontra canadensis* are well detailed (Serfass et al. 1993, 1996).

In North America and elsewhere, fur-bearing carnivores have been raised extensively in captivity for fur farming. Intensive farming of mink for fur began in 1925 (Tomson 1987) and the American mink is by far the most important species in fur-farming operations (Venge 1959; Thompson 1968). For this reason there is an extensive literature on the behaviour (MacLennan & Bailey 1969), metabolism and physiology (Wamberg 1994), lactation (Clausen et al. 1996; Hansen et al. 1996), reproduction (Enders 1952; Hansson 1947; Duby and Travis 1972; Sundqvist and Gustafsson 1983; Sundqvist et al. 1988, Lagerkvist et al. 1992), selective breeding (Lagerkvist et al. 1994), stress levels (Gilbert and Bailey 1967, 1969), veterinary care (Tomson 1987), economics (Lagerkvist 1997) and welfare (Nimon & Broom 1999) of captive mink. There are also papers on techniques for increasing rates of reproduction in Siberian polecats *Mustela eversmanni* (Mead & Neirinckx 1990) and farmed mink (e.g. Lagerkvist 1992, 1997; Lagerkvist et al. 1992, 1994; Lagerkvist & Tauson 1993), and these may be useful for rapidly establishing a captive colony of stoats.

For further information, a brief list of periodicals, publications and web sites that provide help and advice on raising mustelids is provided in the Appendix.

5.6 SUMMARY AND RECOMMENDATIONS

Stoats in captivity will be useful for several aspects of the new Stoat Research Programme. Stoats do not lend themselves to display or to commercial exploitation, hence there have been relatively few attempts at captive breeding. However, a small number of detailed studies permit recommendations for the husbandry and breeding of stoats in captivity. Obviously techniques will be improved with experience.

Captive stoats should be housed individually in cages of approximately 150 × 100 × 60 cm made out of wood, fine wire mesh and glass. An insulated, removable nest box with a door that may be closed from outside the cage and an inspection window should be provided. Adequate bedding material, such as wood shavings, moss or hair, is essential and features for environmental enrichment such as a water dish, hollow logs and pipes are beneficial. Stoats can be kept outside and no artificial lighting or heating should be provided, unless active management of photoperiod is required in order to manage reproduction. Stoats should be fed *ad libitum* on live or freshly killed small rodents or ox-heart as an alternative. Provision of live rodents is particularly desirable in the early stages of captivity.

Young females caught in the wild during embryonic diapause will be invaluable for establishing colonies since they ought to give birth the next spring. For breeding in captivity, males should be introduced to females' cages. In the case

of females with young this should probably be approximately 25–45 days after giving birth, although female stoats appear able to mate or defend themselves as appropriate before this time. To ensure successful mating, several males can be introduced one after the other. Male kits could perhaps be removed from the cage during mating attempts in order to prevent the small risk of infanticide.

6. Diseases

6.1 INTRODUCTION AND AIMS

The use of diseases as agents of biocontrol is often the most appealing of a range of options for the control of pest species. While the use of *Myxoma* virus to control rabbits is a model for similar campaigns, the uncontrolled or unauthorised releases and outbreaks of *Calicivirus* causing rabbit haemorrhagic disease serve as a reminder for the need for caution when deploying agents of this kind, especially in terms of public relations. It is not our aim to enter into the ethical or political debate about using disease as a biocontrol agent. In contrast, we have drawn together a range of evidence for the importance of various diseases and parasites of stoats and other mustelids.

A range of bacterial and viral infectious diseases can affect small mustelids but they have not received as much attention as the diseases of larger carnivores (Murray et al. 1999). Consequently, the pathology and epidemiology of the numerous diseases that may affect stoats are poorly known. Fortunately the economic incentive for rearing ferrets and mink has led to a good deal of information on the diseases that afflict them. Ferrets and mink are useful models for stoat disease because of good evidence that congeneric species are similarly vulnerable to many diseases. For instance, domestic ferrets, Siberian polecats, and ferret–polecat hybrids were extensively used as disease models in planning for black-footed ferret conservation plans (Williams et al. 1991; Williams & Thorne 1996). However, conclusions of comparable susceptibility among congeners are not always supportable (Williams & Thorne 1996). Diseases of the Mustelidae in captivity have been reviewed at least in part previously (Williams & Thorne 1996), and the family has been shown to particularly susceptible to a range of viral diseases, though this review did not consider in detail bacterial, protozoan or metazoan agents of disease. The reviews provided by Davis et al. (1981) and Addison et al. (1987) are invaluable to a consideration of infectious diseases in wild mammals and furbearers.

6.2 VIRUSES

Morbilliviruses and parvoviruses

Morbilliviruses cause a range of human and wildlife diseases including measles, rinderpest and canine distemper (Barrett 1999). Parvoviruses are apparently able to spread worldwide, since most viruses are not genetically distinct even

when geographically separated (Parrish 1995). Recent epidemics of morbilliviruses and parvoviruses in wildlife and companion animals further suggest that this is a highly variable group of pathogens that are capable of rapid adaptation to alternative hosts. For instance, the incidence of feline panleukopenia virus (FPV) in captive large cats was suggestive of interspecific transmission from domestic dogs (Steinel et al. 2000). Mustelids can also be host to non-specific parvoviruses. For example FPV, but not canine parvovirus (CPV), has been found in wild honey badgers *Mellivora capensis* (Steinel et al. 2000), and North American river otters also appear to be susceptible to FPV (Harris 1968).

Isolates of phocine distemper virus (PDV) from harbour seals *Phoca vitulina* collected in Denmark, Norway, Greenland, and USA were comparatively distinct from reference strains of canine distemper virus (CDV). However, there were similarities between Danish and Norwegian isolates of PDV and morbillivirus isolates from Danish mink farms, suggesting that epizootics among farmed mink may have arisen from transmission from diseased seals to terrestrial carnivores (Blixenkrone-Moller et al. 1992).

Two parvoviruses that cause very different diseases have been described in detail in mink. Mink enteritis virus (MEV) is associated with rapid, high-level viral replication and acute disease. In contrast, infection with Aleutian mink disease parvovirus (ADV, less commonly called AMDV) is associated with persistent, low-level viral replication and chronic severe immune dysregulation (Storgaard et al. 1997).

Aleutian disease virus

Aleutian disease of mink is a naturally occurring persistent viral disease first described in 1958 (Helmboldt & Jungherr 1958). It is caused by the Aleutian mink disease parvovirus (ADV) and in adult mink results in a chronic disease that can be broadly characterised as an immune disorder with a persistent infection of lymphoid organs. ADV is particularly lethal to the Aleutian strain of mink, but all strains are susceptible to some degree (Bloom et al. 1994). The virus is transmissible to other mustelids, particularly *Mustela* spp., including ferrets and stoats (Kenyon et al. 1978; Alexandersen et al. 1985). Symptoms similar to Aleutian disease have been described in an otter *Lutra lutra*, though while the pathology was consistent with infection by ADV an absolute diagnosis was not provided (Wells et al. 1989).

When inoculated with ADV isolated from farmed mink, stoats exhibited an immune reaction to the challenge. Antibodies were detected by counter immuno-electrophoresis. However, they did not show clinical signs of the disease, i.e. abnormal accumulation of lymphocytes in kidney or liver cells or hyperplasia of lymphoid organs (Kenyon et al. 1978). In England, 8.5% of 446 domestic ferrets were seropositive for ADV (Welchman et al. 1993) implying that significant reservoirs may exist for this virus among species that are less affected by the disease.

Clinical signs of the chronic disease in adult mink include plasmacytosis, hyper-gammaglobulinaemia, high antiviral antibody titres and immune complex disease (Bloom et al. 1994). In severe cases, structural organisation of the thymus gland is destroyed and T-cells are found throughout the organ, whereas

they would normally be found in greatest numbers in the inner medulla (Chen & Aasted 1998). Immune complex derived lesions cause arteritis in which elevated levels of gammaglobulins and antigens are detectable (Kostro et al. 1999). The virus is present in faeces, saliva and, intermittently, urea, about 15 days after infection (Kostro et al. 1999). ADV can be cultured in mink lymph nodes (Jensen et al. 2000).

The disease is transmissible vertically and horizontally (Kostro et al. 1999). Airborne transmission is possible, but is less efficient than mechanical transmission (Jackson et al. 1996). In farmed mink, ADV is transferred between infected mothers and their kits, but not between infected fathers and kits (Jackson et al. 1996), suggesting that ADV crosses the endotheliochorial placental barrier between infected female mink and embryos (Broll & Alexandersen 1996). The percentage of dead and resorbed foetuses was much higher in females infected with ADV before mating than in those infected after the assumed date of implantation (Broll & Alexandersen 1996). In contrast to adult mink, infected newborn mink develop acute interstitial pneumonia that is fatal in most cases (Bloom et al. 1994). Inoculation of 449 mink kits with ADV of various strains resulted in 48% mortality, though the severity of each strain varied from 30% to 100%. In kits, high-virulence strains included ADV-K, ADV-Utah I, and ADV-DK and resulted in mortality rates of 90–100%. Low-virulence groups ADV-GL and ADV-Pullman resulted in 30–50% mortality. Kits that survived challenge by ADV developed chronic Aleutian disease as normally expressed in adult mink (Alexandersen et al. 1994). Certain strains of mink, being more susceptible to ADV, were better at transmitting the infection. However, in black mink, infected sires appeared to provide some immunity to their kits (Jackson et al. 1996).

ADV is unusually genetically variable, with at least three subgroups and many genotypes. More than one genotype has been found at one farm (Olofsson et al. 1999). Structurally, ADV is similar to human parvovirus B19, CPV, FPV and minute virus of mice (MVM). Tropism and pathogenicity of ADV is related to these structural differences at the threefold axes resulting from variable residues (Parker & Parrish 1997; McKenna et al. 1999). Differences between parvovirus types, including their host range, are due to variation in only three or four sequence differences in capsid protein genes (Parrish 1999).

The several isolates of ADV vary markedly in the severity of the disease they cause. ADV-Utah strain causes severe Aleutian disease and death in mink in 6–8 weeks. ADV-G strain does not replicate in mink, but does in cats. The difference between these two strains amounts to as little as five amino acids (Bloom et al. 1998). Changes of single amino acids at particular locations can cause changes in host-specific replication and can cause less acute, but not classical, forms of Aleutian disease, where unmodified versions were benign (Fox et al. 1999).

Mink enteritis virus

MEV is part of the feline parvovirus subgroup and as such is closely related to FPV and CPV (Bittle 1981; Steinel et al. 2000). We are not aware of any attempts to infect stoats with MEV. Clinical signs of the disease in mink include a rapid onset of depression, lethargy and high temperature. MEV replicates very rapidly, at least 20 times faster than ADV, in Crandell's feline kidney cell

cultures. This may be because MEV is constituted of a higher proportion of structural proteins whereas ADV codes for a high percentage of non-structural proteins (Storgaard et al. 1997). MEV is very robust and survives well under normal outdoor conditions. Viruses contained in mink faeces collected from infected individuals survived outdoors and were able to infect mink for up to ten months after collection, which was one month after standard cell culture tests proved negative. The virus was not tolerant of drying out but did survive well in soil and damp conditions (Uttenthal et al. 1999). Protection against MEV in mink can be induced by a single inoculation with viruses developed for protection of dogs against canine parvovirus (Langeveld et al. 1995).

Canine distemper

While all members of the Mustelidae have been reputed to suffer from distemper this has not been confirmed in many of the 67 species. Confirmation that stoats are susceptible to viral distemper has only been obtained once and is based on the death of one individual. A captive colony of seven stoats, four weasels and three least weasels experienced an outbreak of canine distemper that was diagnosed by early clinical signs including muscular spasms, reduced activity, photophobia and discharges from their eyes (Keymer & Epps 1969). Two stoats, four weasels and two least weasels showed clinical signs of the disease and of these one stoat, three weasels and the two least weasels died. Distemper was confirmed by taking inoculates from the dead animals and inoculating distemper-immune and non-immune ferrets. The immune ferrets survived while the others did not (Keymer & Epps 1969). The progression of the disease was very slow and in certain individuals lasted as long as 12 weeks (Keymer & Epps 1969). The moderate level of virulence of this disease may therefore lend itself to widespread transmission in free-living animals.

Canine distemper is caused by canine distemper virus (CDV) and has been considered the most important viral infection of mustelids (Williams et al. 1988). However, data on the fatalities caused by the disease are very scarce. The disease is particularly lethal to black-footed ferrets (Williams et al. 1988) and was responsible for the near elimination of the remnant populations of this species. In black-footed ferrets, clinical signs included pruritus, hyperkeratosis and loss of body condition (Williams et al. 1988). Out of 146 mustelids (132 stone martens *Martes foina*, five badgers *Meles meles*, five polecats and four weasels) sampled in Germany, CD antigens were found in the brains of 54 (37%) including one of the four weasels, and were mainly found in the grey matter. Histological brain lesions were detected in 45% of the CDV positive animals. The high prevalence and seasonal variation in prevalence of the antigens and lesions suggested that in Germany there was at that time an epizootic of CDV among mustelids, in particular stone martens (van Moll et al. 1995). Few differences in the structure of CDV detected in domestic dogs and martens, polecats and weasels could be detected by immunocytochemical techniques, suggesting that the mustelid virus was not antigenically distinct from the canine virus (Alldinger et al. 1993).

Inactivated canine distemper vaccines with adjuvant and a modified live virus were used comparatively successfully in vaccinating black-footed ferret × Siberian polecat hybrids. All eight animals vaccinated with the modified live virus survived and resisted virulent CDV inoculation. Of the seven hybrids given

the inactivated vaccine one was killed following the development of CDV from the vaccine. A further two developed clinical signs of the disease but survived (Williams, E.S. et al. 1996). Multivalent avian-origin vaccines for CDV induced typical clinical signs of canine distemper in four inoculated European mink *Mustela lutreola*. All four were producing CDV antigens, confirming clinical signs, and died of CDV 16–26 days after inoculation (Sutherland-Smith et al. 1997).

High levels of mortality in farmed mink can be caused by initial infections by *Pneumocystis carinii* and secondary infection by CDV (Dyer & Schamber 1999). Symptoms similar to distemper have been observed in farmed mink of particular strains. However, these can occasionally be related to Tyrosinemia II, known as pseudodistemper, which is a rapidly fatal genetic disease prevalent in standard black ranched mink and is caused by a deficiency of hepatic tyrosine aminotransferase (Sanford 1988).

Rabies

Rabies is one of the best known and most widespread zoonotic diseases. The rabies virus is a member of the family Rhabdoviridae, genus *Lyssavirus* (Dietzschold et al. 1996). In humans the virus causes an acute, incurable encephalitis that results in 40 000–100 000 deaths per year worldwide (Meslin et al. 1994; Rupprecht et al. 1995). In infected hosts, the virus is excreted mostly in the saliva and new rabies infections occur through bites from infected animals (Charlton 1994). Rabid mammals ehibiting the furious form of the disease are characterised by wide-ranging and often erratic movements, as well as aggression towards other animals including humans (Kaplan 1985). Such behavioural modification is thought to enhance rabies transmission and persistence in host populations. For wildlife species, recovery from rabies seems possible and may have occurred in a large proportion, perhaps more than 25%, of infected animals (Carey & McLean 1983).

Five rabies variants currently exist in North America: the raccoon *Procyon lotor* variant in the east, grey fox *Urocyon cinereoargenteus* rabies in the south-west, skunk *Mephitis mephitis* rabies in the west and north-west, and arctic fox *Alopex lagopus* and red fox *Vulpes vulpes* rabies in the north (Krebs et al. 1999). Although many animals may get rabies, only a few species act as reservoirs. Cases of rabid stoats have been reported (Ballantyne & O'Donoghue 1954; Plummer 1954; Rausch 1958; Johnson 1959), but such cases are insignificant compared to rabies in other species. Many stoats may die undetected of rabies, but even then the significance of the disease to stoat populations is probably minimal.

Rabies in North America is currently mostly problematic in skunks and raccoons (Charlton et al. 1988, Rupprecht & Smith 1994). Skunk rabies in the US still occupies the largest geographic area of all disease variants, and is currently expanding its distribution to include areas that previously were unaffected (Pybus 1988, Canadian Food Inspection Agency, unpubl. reports 2000). Moreover, no human mortality has been associated with raccoon rabies whereas at least ten human fatalities have been caused by skunk rabies since 1950 (e.g. Gomez et al. 1965). Currently, oral rabies vaccines have been successfully tested in the field for foxes, raccoons, and coyotes *Canis latrans*, but not for

skunks (Hanlon et al. 1999). Thus, skunk rabies remains of primary concern for human and domestic animal health and safety (Hayles and Dryden 1970, Center for Disease Control 2000).

The role of stoats in rabies transmission remains unclear (Johnson 1959) but is unlikely to be important. Following inoculation with the virus the incubation period of the disease in ferrets was 28 days when inoculated with the raccoon variant and 33 days with the skunk variant. Clinical signs were typical, including ataxia, hypothermia, tremors and lethargy. Following clinical signs, death ensued within 4–5 days. Viral excretion ranged from one day before onset of clinical signs to six days after (Niezgoda et al. 1997, 1998). While ferrets are susceptible to rabies, wild *Mustela* spp. appear to represent a 'dead end' in the propagation of the rabies virus.

Transmissible encephalopathy

Mink are susceptible to a rare transmissible mink encephalopathy (TME). This illness is similar to other conditions that cause progressive neurological disease such as scrapie in sheep. Feeding mink with material infected with transmissible spongiform encephalopathy (TSE) agents, probably cattle infected with bovine spongiform encephalopathy (BSE) or possibly sheep infected with scrapie, was the cause of original infection. TME is comparatively easily transmissible to striped skunks *Mephitis mephitis*, but not ferrets. However, changes in the back-passage re-infection of mink with material from infected ferrets suggested that the agent is highly variable in the short term. This may be the result of molecular variation in the protease-resistant proteins (PrP) that are thought to have a role in the disease (McKenzie et al. 1996; Bartz et al. 1994). A range of TME agents can also be used to re-infect cattle with more serious results than back-passage infections of cattle with scrapie agents (Robinson et al. 1995).

Ferrets are also susceptible to chronic wasting disease (CWD), another form of TSE. Clinical signs include spongiform degeneration of brain tissue and reactive astrocytosis. Experimental inoculation of ferrets led to shortening of incubation periods after several passages and unlike the original CWD isolated from deer, the ferret form was transmissible to rodents (Bartz et al. 1998).

Other viruses

Inoculation of mink with the herpesvirus that causes Aujeszky's disease resulted in salivation, vomiting and coma after an incubation period of 72–96 hours. The clinical signs of Aujeszky's disease are similar to rabies, hence the disease is sometimes termed pseudo-rabies. Lesions were detected in the brainstem in the form of a non-suppurative encephalitis, and degeneration of vessel walls was widespread. The disease virus was detected in the central nervous system (Quiroga et al. 1997). Contrary to previous thinking, replication of the related canine herpes virus-1 is supported in cells other than of canine-origin, e.g. in foetal mink lung cells. This property has been used to develop immunoassays for canine herpes virus infection in kennel dogs (Reading & Field 1999).

Coronavirus antisera have been isolated in mink and this virus apparently occurs at up to 100% prevalence in mink on farms in Denmark. The putative

mink coronavirus (MCV) is similar in structure to transmissible gastro-enteritis virus (TGEV) and porcine epidemic diarrhoea virus (PEDV). MCV may have a role in acute enteritis in young mink kits that may be related to 'sticky kit disease' (Have et al. 1992). However, sticky kits do not have any greater prevalence of coronavirus, rotavirus or calicivirus than normal kits, nor any difference in *E. coli* communities (Jorgensen et al. 1996).

Mink are susceptible to avian influenza-A virus (subtype H10), particularly strain mink/84 (H10N4), which caused pneumonia, lower rates of weight gain and higher rates of viral expression (Englund & af Segerstad 1998). In fact, mink lung cells were more sensitive than other more commonly used cells for rapid detection of influenza and other respiratory viruses (Huang & Turchek 2000). Ferrets are also susceptible to influenza A-virus (Buchman et al. 1995).

Infectious canine hepatitis (ICH) is caused by an adenovirus and, while canids are most directly susceptible, mustelids including mink, skunks (Addison et al. 1987) and otters (Harris 1968) can also contract the disease. In foxes and skunks, progression of the disease is very rapid, and clinical signs include convulsions and lethargy, rapidly progressing to coma after which death occurs in minutes to hours (Cabasso 1981). A screening exercise of 10 514 animals from all over Russia for haemorrhagic fever with renal syndrome, revealed that none of four weasels was positive for the disease antigens (Tkachenko et al. 1983).

6.3 BACTERIA

Stoats and, more commonly ferrets, are competent hosts for *Mycobacterium bovis*, which causes bovine tuberculosis (Tb) in cattle. Clinical signs of Tb in stoats have been recorded in stoats in the former Soviet states (Lavrov 1944) but accurate diagnostic tools were not then available. None of 33 stoats examined in Britain between 1971 and 1986 tested positive for *M. bovis* (MAFF 1987), but a new study is currently undertaking a further screening exercise (CSL, York, UK). In samples collected on farms experiencing bovine Tb in cattle in New Zealand, one out of 62 stoats (1.6%) and 17.9% of ferrets exhibited tuberculous lesions (Ragg et al. 1995a). Ferrets infected with *M. bovis* most commonly had lesions in the mesenteric (35%), retrophasyngeal (17%) and prescapular (16%) lymph nodes (Ragg et al. 1995b). *Mycobacterium avium paratuberculosis* has been isolated in stoats in Scotland. In cattle, this causes Johne's disease, which is a chronic enteritis that is frequently lethal. In stoats, the effects are presently unknown (Beard et al. 1999), but in ferrets, *M. avium* causes granulomatous enteritis (Schultheiss & Dolginow 1994).

Stoats can apparently be resistant to tularaemia, but the disease is thought to cause significant mortality (Lavrov 1944). The susceptibility of stoats to the disease was demonstrated by experimental infection, although the disease is found naturally in a range of other mustelids (Bell & Reilly 1981). It is caused by *Francisella tularensis* and can be transmitted by arthropod vectors, mainly rabbit fleas, or through water, hence it is also known as a 'swamp fever' (Addison et al. 1987). In carnivores, including weasels, the disease presumably originates from eating infected prey, although the role of arthropod vectors is not clear. It causes a plague-like acute febrile infection frequently resulting in

rapid mortality. The disease has not been recorded in Britain (Bell & Reilly 1981), hence is unlikely to be present in New Zealand stoats. It is transmissible to humans and so may present a significant risk in certain areas, either by contaminating water supplies or by arthropod transmission from rodents to humans.

Pseudotuberculosis or yersiniosis, caused by *Yersinia pseudotuberculosis* has similar clinical signs to tularemia and plague. The disease has been reported in American martens, mink and otters, but not stoats (Wetzler 1981). The disease is transmitted via the oral–faecal route, although again arthropod vectors may have a role in transmission. While acute and highly fatal outbreaks have occurred in laboratory animals, epizootics in wild animals have not been reported (Wetzler 1981). Plague, caused by infections of *Yersinia pestis*, has been detected in captive colonies of black-footed ferrets and may have a role in controlling wild populations, given the high prevalence of *Yersinia* among wild prairie dog populations (Williams et al. 1994; Dyer & Huffman 1999). However, earlier attempts to inoculate ferrets and Siberian polecats with *Yersinia pestis* failed to evoke clinical signs of the disease (Williams et al. 1991). American badgers *Taxidea taxus* are also susceptible to bubonic plague (Dyer & Huffman 1999). In North Dakota, the black-tailed prairie dog *Cynomys ludovicianus* is the most likely primary reservoir of bubonic plague.

Erysipelothrix rhusiopathiae has been recorded in a number of mustelids, including Siberian polecats, American mink, sable, and Siberian weasels. American mink were apparently resistant to the affects of the bacterium. However, in other hosts it causes a septicaemia of varying severity. Transmission is by consuming infected prey (Wood & Shuman 1981). Between one out of three (Twigg et al. 1968) and one out of eight weasels (Michna & Campbell 1970) sampled in Britain was positive for *Leptospira* spp. which causes leptospirosis or Weil's disease in humans. However, none of nine stoats, nine ferrets or four weasels was serologically positive for *Leptospira* in New Zealand (Hathaway & Blackmore 1981). *Pasteurella multocida,* which causes a haemorrhagic septicaemia in domestic stock, has been isolated from weasels *Mustela* spp. (Rosen 1981).

The prevalence of gut bacteria (anaerobes, aerobes and staphylococci but not enterobacteriacieae) increases with age in farmed mink, but total levels of bacterial occurrence were much lower than for other mammals, perhaps because of the rapid passage of food through the gut. *Campylobacter* was rare and *Salmonella* and *Shigella* were not detected (Williams et al. 1998). Specific strains of *Staphylococcus intermedius* have been identified in mink (Hesselbarth & Schwarz 1995). This bacterium has been identified as the cause of acute adenitis of the cervical apocrine glands in neonatal farmed mink and of vaginitis and mastitis in adult females (Schneider & Hunter 1993a; Hunter & Prescott 1991). Furthermore, urolithiasis (disease caused by kidney stones) may also be linked to bacterial infections of *Staphylococcus* (Zimmermann & Witte 1988).

Ferrets are a common model for studies of *Helicobacter pylori* since they are commonly host to the related *Helicobacter mustelae*. In humans *H. pylori* causes stomach ulcers of varying severity. In ferrets the infection is associated

with gastric lymphoma in the pyloric antrum with characteristic epithelial lesions (Erdman et al. 1997) and adenocarcinoma (Fox et al. 1997).

A Siberian polecat in captivity failed to reproduce after mating; the uterus was infected with heavy growth of *Enterococcus faecalis* causing reproductive failure by filling the uterus with purulent material, a condition known as pyometra. The strain of *Enterococcus* isolated from this polecat was resistant to a wide range of standard antibiotics (Johnson et al. 1999).

6.4 PROTOZOA

We are not aware of any specific investigations of protozoan infestations in stoats. *Pneumocystis* spp. can cause pneumonia in a range of hosts that may include stoats. *Pneumocystis* is a variable organism that exists in several host-specific 'special forms' (Wakefield 1998), the genetic differences between which are pronounced (Stringer & Cushion 1998). Two out of 46 least weasels in Finland were host to *Pneumocystis carinii* (Laakkonen et al. 1998). High levels of mortality in farmed mink are caused by initial infections by *Pneumocystis carinii* and secondary infection by CDV or other viruses (Dyer & Schamber 1999).

Of wild mink sampled in Kansas and Missouri, 66% had antibodies to *Toxoplasma gondii*, a protozoan that causes abortion in sheep but rarely causes clinical disease in its main hosts. However, attempts to culture the parasite from positive individuals were largely unsuccessful (Smith & Frenkel 1995). Similarly, in Ireland seven out of 15 wild mink caught had positive titres for *Toxoplasma gondii*, but cysts were not detected (O'Crowley & Wilson 1991). Although the prevalence of *Toxoplasma* in farmed mink was low (3%), the large numbers dealt with presented a substantial risk to their handlers and to furriers (Henriksen et al. 1994)

Two species of *Eimeria* and other Coccidian oocysts were detected in black-footed ferrets that had died from canine distemper (Williams et al. 1988). Various life stages of *Eimeria* spp. were located in the epithelium of bile ducts and gallbladder of a ferret (Williams, B.H. et al. 1996). *Eimeria ictidea* and *E. furonis* were located in the faeces and intestinal contents of wild and captive black-footed ferrets (Jolley et al. 1994). *Giardia* spp. were also found in black-footed ferrets (Jolley et al. 1994). Infection by the Coccidian *Cryptosporidium* spp. from goats caused several fatalities in a colony of captive ferrets (Gomez-Villamandos et al. 1995). Captive stone martens shed *Cryptosporidium*, probably *C. parvum*, oocysts during temporary diarrhoea episodes (Rademacher et al. 1999).

Mink are susceptible to infection by *Sarcocystis* causing muscular sarcocysts. Infection in two mink was associated with meningoencephalitis and meningomyelitis (Ramos-Vara et al. 1997). Four out of 42 (10%) American martens collected in Washington were host to *Sarcocystis* spp. (Foreyt & Lagerquist 1993). In Japa, 67 out of 70 wild-caught martens *Martes melampus* were host to schizonts or gametocytes of *Hepatozoon* spp. that caused nodular lesions, most commonly in the heart (Yanai et al. 1995). Antibodies to *Encephalitozoon cuniculi*, which causes abdominal distension, paralysis and

other symptoms similar to rabies, were found to be widespread in mammals, including mink, in Iceland (Hersteinsson et al. 1993). In farmed mink, *Encephalitozoon* causes cataracts and renal lesions (Zhou et al. 1992). A sample of mustelids including stoats, long-tailed weasels and ferrets were found not to host *Neospora caninum,* which causes abortion in cattle (McAllister et al. 1999).

6.5 FUNGI

Stoats are not thought to host dermatophytes, e.g. *Trichophyton*, that cause ringworm, though only small numbers of samples were tested (English 1969). In Finland, 21 out of 46 (46%) examined least weasels were infected by adiaspores of *Chryosparium* sp. This fungal infection caused granulomas around the adiaspores (Laakkonen et al. 1998).

6.6 HELMINTHS

The best known parasite of stoats is the nematode *Skrjabingylus nasicola,* which appears to affect all species of *Mustela* (Dougherty & Hall 1955). The parasite is common throughout the Holarctic and elsewhere including New Zealand (King 1974). It causes skull deformity by eroding bones of nasal sinuses presumably leading to pressure on the brain. Rates of infestation range from 17–31% in Britain (Lewis 1967; van Soest et al. 1972) to 50% in Ireland (Sleeman 1988), but can be as much as 100% in North America (Dougherty & Hall 1955; Jennings et al. 1982). In New Zealand, there is an average prevalence of 10% infestation (King & Moody 1982). The obligate intermediate hosts of *Skrjabingylus* are terrestrial snails and the paratenic hosts were once thought to be shrews (Hansson 1967). However, shrews are rare in stoat diet, and invasive third-stage *S. nasicola* larvae have been found encapsulated in *Apodemus*, which readily eat molluscs both in the wild and in captivity, and in experiments infected stoats within 24 days (Weber & Mermod 1985). Heavy infestations of *Skrjabingylus* were believed to affect skull size adversely on Terschelling Island in the Netherlands (van Soest et al. 1972) and density and fertility in Russia (Popov 1943; Lavrov 1944). However, stunting of infested individuals was not detected in a sample of 1492 stoats examined in New Zealand (King & Moody 1982). *Skrjabingylus* may induce fits or spasms and has been associated with 'dancing' behaviour or playing dead under stress (King 1989). *Skrjabingylus* is not thought to cause significant mortality in stoat populations and so is not a likely candidate for control in itself. However, bacterial or viral infections associated with *Skrjabingylus* may be sufficiently specific that they may hold promise as vectors for biocontrol agents.

Out of 22 stoats from Washington State, USA, 41% were infected by one or more of five helminth species: *Taenia mustelae*, *Alaria mustelae*, *Molineus patens*, *M. mustelae* and *Trichinella spiralis* (Hoberg et al. 1990). The trematode fluke *Troglotrema acutum* has been identified in Swedish stoats but appears to be rare (Hansson 1968). A survey of common helminths in a Russian sample of stoats included: nematodes *Capillaria putorii, Molineus patens* and

Strongyloides martis, cestodes *Taenia tenuicollis* and *Mesocestoides lineatus* and, rarely, Acanthocephala *Acanthocephalus* spp. (Lavrov 1944). *Filaroides martis* has been recorded in a single adult male stoat in New Zealand (McKenna et al. 1996). Guinea worm *Dracunculus* has also been recorded in stoats and other mustelids in Canada (Crichton & Beverly-Burton 1974). Out of 40 stoats screened in Canada, *Taenia mustelae* was identified in eight (20%), *Capillaria* larvae in 16 (40%) and *Aelurostrongylus pridhami* in five (13%) (Jennings et al. 1982).

The cestode tapeworm *Taenia mustelae* has been recorded in weasels in Japan (Iwaki et al. 1995). *T. mustelae* has also been recorded in black-footed ferrets (Rockett et al. 1990). In weasels, larvae of the spiroroid nematode worm *Gnathostoma nipponicum* are ingested and migrate from the stomach to the muscle of weasels, where they enlarge and mature into adult worms. The adults invade the oesophageal wall and cause tumours (Ando et al. 1994). Another spiroroid nematode *Physaloptera* sp. was found in black-footed ferrets (Jolley et al. 1994). In ferrets, infection by gut parasites, such as the nematode *Trichinella spiralis,* that cause enteritis can lead to changes in neuromuscular function, particularly muscle contractability and gut neurotransmission, that persist after the symptoms of infection have been cleared up (Venkova et al. 1999).

Filaroides infections have been detected in two feral ferrets in New Zealand (McKenna et al. 1996). Ferrets are susceptible to infection by *Dirofilaria* (McCall 1998) and are a competent host for *Dracunculus insignis* (Eberhard et al. 1988). Wild polecats are host to the trematode fluke *Troglotrema acutum* that infects nasal sinuses and causes cranial lesions similar to *Skrjabingylus* (Artois et al. 1982).

Out of 259 stone martens in Germany that were screened for helminth infection, 87% were host to *Capillaria putorii*, 27% were infected by *Taenia martis* and 10.4% were host to *Molineus europacus*. *Toxocara* spp. larvae were detected in only seven individuals. None was infected by *Skrjabingylus* (Schoo et al. 1994), though *Skrjabingylus petrovi* has been identified as the principal *Skrjabingylus* infecting *Martes* spp. in France (Gérard & Barrat 1986) and in Sweden (Hansson 1968). Japanese martens *Martes melanpus* were host to *Mesocestoides paucitesticulus* (Sato et al. 1999a) and to *Aonchotheca putorii* in the stomach, *Concinnum* in the pancreatic duct, and *Molineus* and *Euryhelmis costaricensis* in the small intestine. *Eucoleus aerophilus*, and *Soboliphyme baturini* have also been identified in Japanese martens (Sato et al. 1999b). Out of 42 American martens that were screened, 36 were host to *Capillaria putorii* in their stomachs, *Mesocestoides lineatus* were in the small intestines of 14 (33%), and *Trichinella spiralis* in the tongues of two (5%). Unusually, the prevalence of *Mesocestoides lineatus* was significantly higher in juveniles than in adults (Foreyt & Lagerquist 1993).

The cestode *Dioctophyma renale* has been recorded in mink (Wren et al. 1986), while recorded nematodes include *Bayliascaris devosti*, *Capillaria mucronata*, *Euparyphium melis*, *Filaroides martis*, *Spirometra erinacei* (Sidorovich & Savcenko 1992; Dunstone 1993) and *Skrjabingylus nasicola* (Hansson 1967). In 50 mink from Illinois, the following helminths were recorded: *Filaroides martis* (62%), *Capillaria putorii* (34%), *Paragonimus*

kellicotti (14%), *Dirofilaria immitis* (2%) and *Molineus* sp. (2%) (Zabiega 1996). North American river otters host numerous endoparasites including cestodes (Greer 1955), nematodes, trematodes, sporozoans *Isopora* and acanthocephalans (Hoberg et al. 1997; Hoover et al. 1984).

6.7 ECTOPARASITES

The specific louse *Trichodectes* (*Stachiella*) *ermineae* has been recorded in Canada (Jennings et al. 1982), Ireland (Sleeman 1989) and New Zealand (King 1989). There is also a specific flea *Nearctopsylla brooksi* that has been recorded in arctic Canada and northern Fennoscandia, but apparently not in Britain or New Zealand (Holland 1964; King 1976). Stoats are host to several ectoparasites associated with their prey species and nest parasites from species that are not eaten. European records list a total of 26 flea species (Debrot & Mermod 1982). *Rhadinopsylla pentacantha*, an uncommon flea specific to vole nests, *Megabothris rectangulatus* specific to voles, *Orchopeas howardi* to squirrels and *Spilopsyllus cuniculus* to rabbits have all been recorded in Britain (King 1976; Mardon & Moors 1977). *Ctenophthalamus nobilis, Dasypsyllus gallinulae, Nosopsyllus fasciatus* and *S. cuniculi* have been recorded in Ireland (Sleeman 1989). In Canada, *Monopsyllus vison* infested eight out of 40 stoats from Newfoundland (Jennings et al. 1982). In New Zealand, rat fleas *N. fasciatus* make up 97% of records, but *Leptopsylla segnis*, *Ceratophyllus gallinae* and *Parapsyllus nestoris* have also been recorded (King & Moody 1982). *Amphipsylla kuznetzovi* and *Ctenopsyllus bidentatus* have been collected from stoats in Kazakhstan (Lavrov 1944).

In Ireland, stoats are host to ticks *Ixodes canisuga, I. hexagonus* and *I. ricinus*, lice *Mysidea picae* and *Polyplax spinulosa,* and the mite *Neotrombicula autumnalis* (Sleeman 1989). Mites *Demodex erminae, Eulaelaps stabulans, Gymnolaelaps annectans, Hypoaspis nidicorva, Haemophysalis longicornis* and *Listrophorus mustelae* have also been recorded on stoats (King 1989). Canadian stoats were also host to the mites *Laelaps multispinosus* and *Androlaelaps fahrenholzi* (Jennings et al. 1982). Symptoms of mange caused by the mites *Sarcoptes* spp. and *Demodex* have also been recorded (Lavrov 1956). In Canada, mink, martens and weasels were recently found to host a new species of tick *Ixodes* (*Pholeoixodes*) *gregsoni* sp. nov. (Lindquist et al. 1999).

Out of 1391 mink sampled in England and Wales, *Ixodes hexagonus* and *I. canisuga* were found on 40% and 2.5%, respectively; *I. ricinus* and *I. acuminatus* were also found. Infestation rates were lower in summer and male mink had more nymphs than females (Page & Langton 1996). Mink fleas include *Ctenophthalmus*, *Megabothris*, *Malareus*, *Nosopsyllus*, *Paleopsylla* and *Typhloceras* (Chanin 1983; Fairley 1980). Domestic ferrets are susceptible to subcutaneous infection by *Demodex* spp. mites causing alopecia and pruritus, classical symptoms of demodicosis also known as demodectic mange (Noli et al. 1996). Sarcoptic mange caused by *Sarcoptes scabiei* has also been recorded in domestic ferrets (Phillips et al. 1987). The ear canker mite, *Otodectes cynotis* has been recorded in wolverines *Gulo gulo* (Wilson & Zarnke 1985). Ectoparasites of North American river otters include ticks (Eley 1977; Serfass et

al. 1992), the louse *Latagophthirus rauschi* (Kim & Emerson 1974) and the flea *Oropsylla arctomys* (Serfass et al. 1992).

6.8 NEOPLASIA

There have been no screening studies for neoplasia (tumour growth) in stoats, although a preliminary study is under way (R. McDonald & M. Day, University of Bristol). However, there is an extensive literature on neoplasia in laboratory and domestic ferrets.

In a 30-year study, 574 out of 4774 ferrets had 639 tumours of various types occurring in all systems. The most common tumours were pancreatic islet cell (22%), adrenocortical cell (17%) and lymphoma (12%). Affected animals ranged in age from one month to 15 years, but incidence was highest in animals 4–7 years old. No sex bias was detected, although speyed animals were more likely to have tumours (Li et al. 1998). Out of 57 ferrets diagnosed of having pancreatic islet cell tumours, 34 had only pancreatic carcinoma, while 23 had both carcinoma and hyperplasia or adenoma. Despite treatment, long-term survival of ferrets with pancreatic carcinoma was low, since the tumours were malignant (Caplan et al. 1996).

A single spayed female ferret was found to have epitheliotropic lymphoma leading to a range of skin disorders and other conditions, including renal disease, though this may have been the result of inappropriate treatment with corticosteroids (Rosenbaum et al. 1996). Two five-year-old ferrets had malignant lymphoma resulting in a cranial mediastinal mass of thymic cells, leading to a diagnosis of thyoma (Taylor & Carpenter 1995). Synovial sarcoma has been recorded in a laboratory ferret (Lloyd & Wood 1996). Domestic ferrets are occasionally susceptible to prostatitis consisting of prostatic cysts and/or prostatic squamous metaplasia. The disease was commonly associated with proliferative adrenal lesions and adrenal gland-associated endocrinopathy (Coleman et al. 1998). Five out of six ferrets with urogenital cysts were found to have adrenocortical hyperplasia or neoplasia (Li et al. 1996). A ferret has been diagnosed with Hodgkins-like lymphoma involving lung, liver, kidneys and lymph nodes. Similar to humans, the disease was associated with eosinophilic granulomas caused by abnormal proliferation of T-lymphocytes (Blomme et al. 1999).

Perhaps significantly from the perspective of biocontrol, inoculation of ferrets with non-cellular extracts from ferrets suffering from lymphoma resulted in inoculated ferrets developing lymphoma. Evidence of reverse transcriptase activity in inoculated animals suggested retrovirus activity. Therefore, viral agents may have a role in horizontal transmission of infectious lymphomas (Erdman et al. 1995). This led to the idea that high incidences of lymphoma and leukaemia in cohabiting ferrets may be associated with infection by ADV or FPV. However, the incidence of these viruses in 35 ferrets living in three groups with 21 cases of lymphoma was not significantly different from the incidence in 52 ferrets living in three groups with no lymphoma. Hence these agents could not be shown to be involved in the disease, though other viruses may be (Erdman et al. 1996). Similar inconclusive results arose from a similar study of a

cluster of cases of juvenile mediastinal lymphoma in ferrets (Batchelder et al. 1996).

6.9 NURSING SICKNESS

Nursing sickness is a major cause of mortality among breeding female mink, causing 56% of mortalities during the lactation period in a sample from farms in Ontario (Schneider & Hunter 1993a, b; Schneider et al. 1992) and up to 14% total mortality among 1774 lactating females in Denmark (Clausen et al. 1992).

Increasing risk of nursing sickness is related to increasing litter size, but not to the age of the mother (Schneider et al. 1992). Sick mothers have significantly larger litters (5.4 kits) than healthy mothers (5.0). However, age, litter size and female weight loss were all major determinants of the risk of nursing sickness. In the last two weeks of lactation, healthy females lost about 14% of their body weight, whereas sick females lost about 31%. Sick females exhibited signs of advanced dehydration, emaciation and other indicators of progressive catabolism (Clausen et al. 1992). In the advanced stage of the disease, coma and death appear to be the inevitable outcome of the strain of continuing milk production (Wamberg et al. 1992).

Nursing sickness appears to be alleviated by supplementary salt, but it is unknown whether the salt is actively involved in avoiding the disease or whether it is a dietary stimulant that prevents starvation (Clausen et al. 1996). Recent studies indicate that low sodium is a symptom rather than a cause of the disorder (Hansen et al. 1996). The various clinical signs of nursing sickness are essentially metabolic indicators of energy stress. The disease results in lethargy, emaciation, dehydration and other symptoms including eventual mortality of lactating female mink. However, there are few indications of the cause of the disease. Viral screening has proven negative for ADV. *Campylobacter* was isolated from affected individuals, but controls were also positive. The basic cause of the disease appears to be the energy demands placed on the mother by lactation, which, even in normal cases during lactation in female mink, are very high. Despite increases in energy intake during the early weeks of lactation, females lose body weight by using up their body reserves. This is especially true in the final part of the lactation period, when they have reached a maximum of energy intake (Hansen 1999). Nursing sickness appears particularly prevalent in farmed mink because of the large numbers of females observed and the susceptibility of a minority of individuals (Schneider & Hunter 1993b).

Pregnancy toxaemia is a common cause of mortality among pregnant female ferrets and their young. Clinical signs of the disease include anaemia, hypoproteinaemia, azotaemia, hypocalcaemia, hyperbilirubinaemia and high liver enzyme activities. Hepatic lipidosis was observed during histological examination. Causes of the disease are unclear, though they may be nutrition- and stress-related (Batchelder et al. 1999).

6.10 SUMMARY AND RECOMMENDATIONS

While the incidence of various pathogens of stoats is not poorly described, the effect of disease on stoat populations is relatively sparse. Although Lavrov (1941) asserted that declines in ermine harvests were brought about by a combination of infectious and helminth diseases, there is little contemporary evidence to support this view. Regrettably, the present lack of an economic incentive in understanding stoat disease appears to have halted the work initiated in the former Soviet Union. Of the major diseases reviewed here, several are clearly unacceptable as agents of biocontrol of stoats because of conflicts with the health of humans, domestic and companion animals. These include rabies, Aujeszky's disease, tuberculosis, plague and leptospirosis. There is insufficient information about the epidemiology of nursing sickness, transmissible encephalopathy, and fungal and protozoan parasites to advocate further work.

In terms of population regulation, Soviet research implicated the disease tularemia or other similar bacterial fevers in crashes of ermine harvests. Further investigation of the limited amount of Soviet research into this bacterium may be warranted. However, much of this work is now so dated that modern techniques have not been applied to diagnosis, description of pathogenicity, or epidemiology. *Erysipelothrix rhusiopathiae* is a common bacterium in free-living mustelids and, although its effects and epidemiology are largely unknown, it could prove useful.

Of the diseases described here, viral agents appear to hold the greatest promise for control of stoats. Although described mainly in mink, ADV and MEV may be transmissible to stoats, and clinical trials of these viruses would be productive. Repeated re-infection of highly variable viruses results in increasing specificity and pathogenicity to the new hosts over only a few 'generations'. Being tolerant of outdoor conditions, MEV may hold promise in establishing itself as an enzootic disease in wild stoats. The dangers of using these agents lie primarily in transmission to non-target species, either wild species of conservation concern or domestic and companion animals. Therefore, an extensive and inevitably costly programme of research into the stability and specificity of disease agents is absolutely unavoidable.

Disease agents that are already enzootic in wild stoats in New Zealand or in their ancestral population in Great Britain, perhaps including distemper, do not appear to have a limiting influence on stoat populations. However, their role in a future biocontrol programme should not necessarily be underestimated. If bacteria, viruses or parasites are already widespread in wild stoats they may be used as vectors for the transmission of other agents of biocontrol. Therefore we advocate a more detailed study of already widespread disease agents, particularly those that are highly specific. The transmission and epidemiology of *Skrjabingylus* has already been studied in detail but there is no information about the micro-organisms associated with this nematode. The ecology and epidemiology of the specific louse *Trichodectes ermineae* and its associated bacteria and viruses are, to our knowledge, largely unknown and warrant further investigation. We look forward to reporting on the findings of preliminary screening study of arthropod-borne diseases in British stoats

undertaken as part of a related project (R. McDonald & R. Birtles, University of Bristol).

For a programme of captive breeding stoats, precautions can be taken to prevent stock loss. However, DOC may wish to consider that unexpected disease outbreaks in a newly established captive colony may reveal further putative disease or biocontrol agents. Under intensive production management systems, mustelids are likely to contract diseases, especially if subjected to dense groupings, rigorous breeding schedules, and restricted space. Thus, preventive vaccination and sanitary conditions are required for maintaining healthy captive animals. The following recommendations were developed for captive mink (Tomson 1987) but could be followed for captive stoats:

1. Vaccinations should be given at ten weeks of age against distemper, viral enteritis, haemorrhagic pneumonia and botulism, caused by contamination of food supplies.
2. Incoming animals should be tested for common diseases, such as Aleutian disease.
3. Storage of feed must prevent oxidation and bacterial spoilage or contamination.
4. Proper cage and utensil sanitation is essential.
5. Precautions must be taken to exclude wild rodents, pets, and other animals from the vicinity of cages.

7. Biological and non-lethal control

7.1 INTRODUCTION AND AIMS

The use of biological and chemical agents to control stoat populations appears at the outset to provide the most promising suite of long-term options for reducing predation on New Zealand birds. Biological control technology has been successfully applied to the control of invertebrate crop pests and has become standard material for crop protection degree programmes (van Driesche & Bellows 1996). Biological control is particularly attractive in some quarters because of its reputed humaneness, though this is not a universal quality of all biocontrol agents. Unfortunately, research into putative biological control agents for vertebrate pests is at an early stage.

Norbury (2000) has provided a targeted review of options for the biocontrol of stoats, and it is not our intention to repeat this work. Bomford (1990) has also reviewed practical options for fertility control in a range of vertebrate pests. We have collated information that is of direct relevance to biocontrol, particularly fertility control, and have addressed some of the issues raised by Norbury. This work should be read alongside the sections of this report dealing with disease and toxins. Inevitably there is a great deal of information yet to be collected on

options for biocontrol for stoats. Therefore we concur with Norbury's conclusion that a large and costly research effort will be required to arrive at a practical biocontrol programme. In order to focus this development work on the most productive areas, we have highlighted priority areas for further investigation. In particular we have outlined the value and necessary approaches for modelling exercises aimed at describing the logistical and economic feasibility of biocontrol options. We have also briefly considered a range of non-lethal, non-biological control methods for reducing predation on native birds.

7.2 FERTILITY CONTROL

Options for fertility control in stoats were reviewed in part by Norbury (2000) and are the subject of a parallel review under the current Stoat Research Programme (Hinds et al. 2000). Most chemical agents that have been tested for fertility control in wild mammals have to be administered directly into the animal. A few exceptions exist, notably cabergoline, which has been tested for use in baits aimed at controlling fertility in foxes *Vulpes vulpes* (Marks et al. 1996). Synthetic mimics of reproductive compounds can induce reproductive failure in many mammals (Norbury 2000). Some, such as synthetic oestrogens, are acutely toxic to most mammals, including mustelids (Hart 1987). However, they usually require repeated exposure to high doses, in the same way that human reproduction can be controlled by daily pills containing synthetic oestrogens and progesterones. Such compounds are not likely to be ingested by stoats in large enough doses in baits, and because of their physiological effects they can even induce conditioned taste aversion to baits (see below) though this could be avoided by encapsulating the oestrogen compounds (Norbury 2000).

Immunocontraception using porcine zona pellucida (PZP) proteins has been successful in captive otters but has not been tested in stoats or other *Mustela* spp. (Kirkpatrick et al. 1996; J. Kirkpatrick pers. comm. to RM). Research indicates that PZP may have some utility for controlling possum *Trichosurus vulpecula* populations in New Zealand (Cowan 1996). However, the immuno-contraceptive effect of PZP is short-lived and the fact that oestrus is induced by mating means that stoats could be capable of mating successfully after the effects of PZP have worn off. Furthermore, the rapid turnover in stoat populations suggests that the administration of PZP to wild stoats would be prohibitively expensive. There is little promise of developing this comparatively crude technique for deployment against wild stoats, except perhaps as a supplementary technique to be applied at particularly crucial periods.

Bradley et al. (1999) have reviewed the use of other 'vaccines' for fertility regulation in wild species. They emphasised that to be cost effective such agents should be long-lasting and capable of delivery to a large part of the population. They concluded that such agents were scarce and that the most realistic options centred on the use of engineered recombinant organisms, probably viruses but possibly bacteria or 'ghost' bacteria. These agents could theoretically deliver vaccines that could induce long-lasting immune responses to sperm or placental proteins. However, the development of any such agent is

still distant, despite concerted efforts being undertaken in Australia and elsewhere (Bradley et al. 1999). A further major problem with such biotechnological developments is their lack of specificity and, while congeneric species in New Zealand are also pests, these techniques may present some risk to companion animals. The deployment of putative vaccines for fertility control would be enhanced by a more detailed knowledge of the currently benign bacteria and viruses that are naturally enzootic in wild stoat populations.

7.3 DELAYED IMPLANTATION

Norbury (2000) highlighted the lack of knowledge about the control of delayed implantation in stoats. He suggested that with a better understanding of the physiological mechanisms controlling implantation the delivery of fertility control agents could be better targeted. We concur, although we would emphasise that the relevance of understanding these mechanisms extends beyond their application to chemical fertility control. There have been few detailed studies of the physiology of delayed implantation in stoats and fewer that have been conducted recently. It is probable that these mechanisms are similar to those controlling implantation in mink, even though the period of delay in this species is much shorter. Mead (1986, 1993) and Sundqvist et al. (1989) have provided useful reviews of the control of reproduction in mink and other mustelids.

Delayed implantation, or embryonic diapause, in stoats, mink and fishers is maintained by involution of the corpus luteum and secretion of basal progesterone until the point of implantation (Deanesly 1935; Gulamhusein & Thawley 1974; Mead 1986, 1993; Frost et al. 1997). In mink, delay is terminated and implantation is initiated when levels of prolactin receptor mRNA activity in the ovaries are increased, leading to increased prolactin binding to the ovaries. Prolactin also maintains luteinising hormone receptors in the corpora lutea. Both processes can be inhibited by the administration of bromocriptine at 2 mg/day (Douglas et al. 1997, 1998). It has also been demonstrated that treatment of farmed mink with bromocriptine and/or monoclonal antiserum for gonadotrophin-releasing hormone compromises post-implantation corpus luteum function by inducing regression of the corpus luteum (Murphy et al. 1993).

Norbury (2000) followed Mead (1986, 1993) and pointed towards the role of an unidentified agent in the control of implantation that acts in conjunction with progesterone. Leukemia inhibitory factor (LIF) is essential for embryo implantation in animals with direct implantation, such as mice. Polymerase chain reaction (PCR) analysis of mink uteri has found that LIF is expressed in the uterine epithelial glands just prior to implantation and for two days after implantation but not before or after. Therefore, LIF appears to be a maternal signal that terminates the period of delay (Song et al. 1998a). Similarly, cyclooxygenase-2 (COX2), an enzyme involved in the production of prostaglandins, is expressed in mink uterine epithelium, stroma and at the endometrial glands at implantation sites. Levels of COX2 increase up to 3–5 days after implantation, then decrease towards day nine, and the enzyme is absent thereafter. COX2 expression is induced by the embryo and clearly has

some role in implantation and placentation (Song et al. 1998b). These two chemicals may be the unidentified non-steroidal compounds hypothesised by Mead (1986, 1993) and Norbury (2000).

7.4 EVALUATING STOAT BIOCONTROL

The resilience of animal populations to trapping, either for pest control or commercial harvest, is highly instructive when investigating the amount of effort that would be required to achieve successful control by other methods. For example, to limit the population growth rate of a large carnivore, such as the grey wolf *Canis lupus*, requires an annual harvest in excess of 35%, although harvest rates of more than 70% may be sustainable with immigration into the controlled population (Larivière et al. 2000). For animals with larger litters, the rates of removal required to keep populations low will undoubtedly be much higher. McDonald & Murphy (2000) have re-emphasised the exceptional tolerance of stoat populations to centuries of concerted trapping effort in Great Britain (King & Moors 1979; Tapper et al. 1982). Stoats are short-lived, produce large litters, and most individuals are unlikely to make more than one reproductive attempt, thus the life-history strategy of stoats makes them extremely resilient to harvest or control (see above and King & Moors 1979; McDonald & Murphy 2000).

Even if stoat densities can be reduced significantly, predation may still have a substantial effect on native birds. Therefore, any evaluation of control must be expanded to incorporate the impact of stoats living at various densities on birds living at various densities and following variable patterns of dispersion. Uncommon birds living at low density will be threatened by relatively few predation events that could be perpetrated by stoats living at even the lowest densities. This has been persuasively demonstrated by the high levels of nest predation by small numbers of stoats on curlew *Numenius arquata* living on moorland in England, despite intensive programmes of stoat trapping and shooting (Robson 1998). Models of stoat control for the maintenance of brown kiwi *Apteryx mantelli* populations suggest that stoat populations must be reduced by about 80%, and kept at densities of less than two stoats per km^2 until kiwis are large enough to avoid predation (Basse 1999).

7.5 BEHAVIOURAL EFFECTS

Behavioural aspects of stoat foraging must be considered in evaluating the likely success of biocontrol. The ability to conduct area-restricted searching following the detection of prey is of key importance in understanding the impact of a predator on resources dispersed according to varying spatial patterns. A simulation model of foraging predators has suggested that the sinuosity and path length of searches strongly affect search efficiency. The model predicted that, for a rate-maximising forager, path length should increase and search sinuosity should decrease as prey becomes less clumped. Foraging animals may learn the appropriate path length and sinuosity of searches required to locate prey living at variable degrees of clumping (Haskell 1997). In experimental

arenas, ferrets have confirmed the suggestions of the model and learnt to adopt longer and less sinuous paths as prey became more dispersed. Therefore mustelid searching behaviour can be fine-tuned in order to exploit efficiently prey living according to variable patterns of dispersion (Haskell 1997).

Stoats may also indulge in surplus killing when prey is abundant. This is an adaptive response to small size, high metabolic needs and the demands of living in a cold climate as well as a strategy for utilising unpredictable and/or otherwise indefensible resources (Oksanen 1983; Oksanen et al. 1985). Surplus killing will increase rates of predation beyond those expected from food requirements. This will be particularly significant in birds with a clumped distribution such as colonially nesting yellow-eyed penguins *Megadyptes antipodes*.

7.6 INTERSPECIFIC EFFECTS

Stoats are not alone in influencing the breeding success of native birds. Ferrets, weasels, cats, rats and possums all have a greater or lesser influence on bird abundance. King et al. (1996) concluded that management of all predatory species in this system was necessary until technology for more targeted control was available. The need for a consideration of interspecific effects has recently been emphasised by a series of models examining nesting success of native birds under dual threat from two species. In the first model, cats eat both birds and rats, which also eat birds (Courchamp et al. 1999a). This is paralleled by the situation in New Zealand where stoats, and indeed cats, eat birds and rats, which in turn also eat birds. The models indicated that removing cats led to increased levels of predation on nesting birds by the rats, which increased in the absence of their main predator. Therefore the continuing presence of cats prevented a mesopredator effect that would lead to the extinction of the native birds (Courchamp et al. 1999a). By the same token, populations of predators, such as cats, can be sustained by apparently benign introduced prey species, such as rabbits. Introduced prey species have often evolved to withstand high rates of predation and so remain common while the predators they sustain drive native birds to extinction. This is known as the hyperpredation process (Courchamp et al. 1999b, 2000). This situation is mirrored by the importance of rabbits and house mice *Mus musculus* to stoats in New Zealand, even though neither of these species directly threatens bird survival to any great extent. To conserve island birds under this scenario, simultaneous control of both introduced predator and introduced prey was necessary (Courchamp et al. 1999b, 2000). Clearly, the complexities of interspecific interactions between a tier of predators and vulnerable and resilient prey species requires a cautious approach to management. In the long run, a holistic approach is essential.

This conclusion has been supported by empirical studies in New Zealand. While predation is the proximate cause of decline in kokako *Callaeas cinerea wilsoni* populations, experimental reduction of both browsing brushtail possums and ship rats *Rattus rattus* resulted in significant increases in kokako chick output and adult density. This effect was a result of increased nesting success and increased numbers of breeding pairs (Innes et al. 1999). Management should

aim not only to reduce predator abundance but also populations of other introduced pests for several consecutive years (Innes et al. 1999).

7.7 TIMING OF CONTROL

Variation in the time of applying biocontrol agents can significantly affect the success of control efforts. Introduced coypus naturally experienced high mortality during winter in England as they thermoregulated poorly in cold weather. Coypus were exterminated by focusing control efforts during the time of year when their populations were most vulnerable (Gosling & Baker 1989). By comparison, trapping and shooting of stoats by gamekeepers in England in early April has a particularly marked effect on stoat population growth rate, since if females are trapped or shot while foraging, their dependent young will probably also die of starvation (McDonald 1998). In contrast, trapping undertaken before females give birth to their young will have a less marked effect on population growth since females are particularly hard to catch at this time due to their reduced patterns of activity (McDonald 1998). From the perspective of cost-efficiency these time-dependent factors should clearly be incorporated into models evaluating control.

There are also suggestions that short-term or local variation in prey availability can affect the timing of reproduction, even in mustelids with delayed implantation. Mating and implantation in wild American mink appears to be affected by seasonal changes in food supply. In Alaska, where seasonal peaks in salmon carcasses result in increased food supply at times inconsistent with photoperiodic stimuli related to latitude, wild mink exhibit late mating (April–May) and parturition in late June to early July. Male mating behaviour was controlled by photoperiod, but female implantation was delayed more than expected in order that lactation coincided with increased availability of salmon carcasses (Ben-David 1997). This process seems plausible in stoats but has not been investigated in any detail, but could again affect the best timing for application of control.

7.8 NON-LETHAL CONTROL

Although not part of the suite of techniques available for biological control, we have included some suggestions of non-lethal techniques for controlling stoat predation that may be of practical use. Reynolds & Tapper (1996) have considered the relative merits of a range of non-lethal approaches to the control of mammalian predation, though these were mainly with a view to reducing predation of game birds by canids, especially foxes.

Volatile repellents may be a useful way of reducing wolverine predation on sheep in Norway (Landa & Tommeras 1997). Following trials in captivity, tea-tree and peppermint oil applied on meat inhibited consumption by captive wolverines. The application of such substances to free-ranging sheep may decrease predation by wolverines. Similar trials in North America failed to reduce predation of sheep by coyotes (Linhart et al. 1997), although the use of

neck collars containing either poison or repellent substances may reduce predation locally (Savarie & Sterner 1979). For nesting birds suffering from stoat predation, the application of aversive agents near nests could be attempted. However, effective repellents have yet to be identified for stoats and the use of aversive agents around bird nests could result in nest abandonment. More importantly, the costs of such a labour-intensive technique for widespread nest protection are likely to be prohibitive, except perhaps in extreme cases. Currently, the use of aversive agents holds little promise as a long-term solution for reducing stoat predation in New Zealand.

Conditioned taste aversion (CTA) has often been tried as a field method to reduce nest predation (Nicolaus et al. 1983; Conover 1990). CTA works by treating otherwise palatable foods, such as eggs, with foul-tasting chemicals such as emetine dihydrochloride, carbamyl choline chloride, quinine, nicotine sulphate and numerous other substances (Conover 1989, 1990). CTA is effective only when the treated foods are encountered more frequently than untreated foods. In practice this means that to reduce egg predation, treated eggs deployed by managers must be encountered more often than the untreated eggs of nesting birds. In North America, CTA was considered as a strategy to alleviate predation of duck eggs by raccoons, striped skunks and American crows *Corvus brachyrhynchos*. It did not work with skunks (R. Penner pers. comm. to SL) and worked only marginally with crows and raccoons (K. Guyn & B. Semel pers. comm. to SL). In most cases, animals learn to avoid eggs while in captivity, but soon resume egg consumption when presented with untainted eggs. In the wild, effective CTA would require deployment of a high density of tainted eggs, so that the rate of encounter with tainted eggs remained much higher than the rate of encounter with untainted eggs. The efficacy of CTA could be enhanced if predators associated certain cues with certain egg types. For example, an aversion for green eggs could be accentuated using CTA if rare birds laid green eggs while 'less important' species laid blue eggs (Bogliani & Bellinato 1998).

Rusiniak et al. (1976) found evidence of CTA in laboratory ferrets, but to our knowledge this has not been tested in stoats. Elsewhere, CTA was tested as a method of reducing egg predation by mongooses but results were disappointing (Nicolaus & Nellis 1987). As a result of rapid population turnover and the range of species threatened by stoats, the potential of CTA to mitigate egg predation by stoats is not great and we cannot advocate further development of this technique.

In England, predation by weasels on tits *Parus* spp. was effectively eliminated by replacing trunk-mounted wooden nest boxes with free-hanging concrete boxes, that were essentially weasel-proof (McCleery & Perrins 1991; McCleery et al. 1996). It is hard to conceive of a similar technique for protecting ground-nesting birds. However, this technique may have some potential for protecting hole-nesting species such as mohua *Mohoua ochrocephala* or yellow-crowned parakeets *Cyanoramphus auriceps* and may be worthy of some further investigation. The degree of uptake of nest boxes by wild birds is the primary factor affecting the success of this technique.

To protect endangered tree-cavity-nesting birds on Mauritius, high-grade polythene has been used to wrap the bases of tress containing nests. This

technique apparently prevents ship rats climbing trees and has reportedly been successful in preventing predation of echo parakeet *Psittacula echo* and pink pigeon *Columba mayeri* nests (D. Hall pers. comm. to RM). This cost-effective and long-lasting technique is dependent on forest structure, since rats can jump from unprotected shrubs in the lower levels of the canopy on to the protected tree. The deterrent provided by the polythene may be sufficient to reduce significantly the predation rates on birds nesting higher in the forest canopy. On the same principal, inverted conical metal guards mounted on tree trunks have been used to reduce access by squirrels to protected trees and to feeding stations and may be worth investigating.

Two ultrasonic devices, Transonic ESP and Yard Gard (Weitech Inc., USA) have been tested to see if they repelled captive stoats from a food source. Although stoats were repelled initially, the effect was only short-lived (Spurr 1997).

7.9 SUMMARY AND RECOMMENDATIONS

Despite its appeal, biological control is currently a distant target for controlling stoats. We caution that the development of models investigating the utility of putative agents is a clear requirement before the expenditure of substantial resources on biotechnology. These models will need to explicitly consider numerous complexities of stoat biology, including spatial, temporal, behavioural and physiological factors.

Fertility control is particularly challenging for stoats. The current lack of a detailed understanding of reproductive physiology should be addressed urgently, not least because this knowledge is pertinent to all aspects of controlling stoat populations. Short life-span, high productivity and rapid population turnover conspire to confound techniques for fertility control as much as they confound other control techniques. Apparently the only realistic possibility for administering control agents to stoats hinges on the use of viral and bacterial agents as vectors. This supports our earlier recommendation of a screening exercise for naturally occurring but widespread and benign viruses and bacteria in free-living stoats.

Non-lethal methods of controlling predation appear to be of limited utility although certain types of physical barriers are worthy of further investigation.

8. Metabolism

8.1 INTRODUCTION AND AIMS

We were asked to review the metabolism of stoats and other mustelids and to identify potential metabolic ‘weaknesses’ that may be useful in stoat control. Where possible we have identified potentially novel approaches for toxin development based on specific aspects of mustelid metabolism. In order to

explore the possibilities in this broad area, we have also reviewed existing knowledge of the use of toxins against stoats and other mustelids and attempted to provide direction for future work in this area.

There has been a considerable amount of recent work on the use of toxins to control vertebrate pests, including stoats, and much of this has originated in New Zealand (see the special 1999 edition of *New Zealand Journal of Ecology 23*: 111–292). Since we were briefed to review international studies, this work forms only a small part of the following review.

8.2 GENERAL METABOLISM

The long, thin shape of stoats allows efficient pursuit of prey but makes them vulnerable to extreme temperatures. Therefore thermoregulation is achieved through behaviour instead of morphology (Brown & Lasiewski 1972; Segal 1972) and their metabolic rate is higher than would be expected for animals of similar body mass (Iversen 1972). In Britain, minimum estimates of daily food consumption for male stoats were 57 g per day (23% body weight) and for females were 33 g per day (14%) (Day 1963). In Switzerland, 19–32% of body weight was eaten daily by males and 23–27% for females (Müller 1970). In captive mink, a protein supply of 25–30% of metabolisable energy supports normal growth, but 30–35% is required for pregnant females or juvenile mink (Tomson 1987; Damgaard et al. 1998). Food passage in mustelids is rapid. In American mink, food passage is 187 minutes for mink and 182 minutes for ferrets (Bleavins & Aulerich 1981). Deprivation of food results in death within 24 hr in captive stoats (J.F. Robitaille pers. comm. to SL).

Energy demands during lactation in female mustelids are particularly high. During lactation female stoats require 2–3 times more food but whilst feeding weaned young they need 5–10 times more (Müller 1970). Despite increases in energy intake during the early weeks of lactation, female mink lose body weight by using up their reserves, first of fat but then of protein. This is especially true in the final part of the lactation period, when they have reached a maximum of energy intake (Hansen 1999). In farmed mink the demands of lactation can result in fatal nursing sickness (see above).

8.3 METABOLIC 'WEAKNESSES'

Studies of the metabolism and physiology of stoats are very scarce, so we have detailed a small number of studies of congeners and potential aspects of their physiology that may be relevant.

While ferrets have often been used as a model for medical and veterinary studies of vitamin A metabolism, they are unusually inefficient at converting dietary beta-carotene into vitamin A. Even high intakes of beta-carotenes are insufficient for maintaining hepatic vitamin A stores (Lederman et al. 1998).

High doses (6 mg/kg) of synthetic oestrogens caused marked haematological changes in domestic ferrets, resulting in liver damage, body weight loss,

nasal bruising, incontinence and other symptoms of oestrogen toxicity. Oestrus ferrets typically do not survive four weeks of treatment with oestrogen (Hart 1987).

Certain strains of farmed mink appear to have high susceptibility to severe hyperlipaemia suggestive of a recessive inheritance. On a normal diet, the affected animals had grossly elevated levels of triglycerides in plasma and had lipogranulomas in mesenteries and the pancreas. The condition was caused by an absence of lipoprotein lipase activity, despite the presence of lipoprotein lipase mass, suggesting that they had inherited a point mutation for the lipase gene (Christophersen et al. 1997).

Gastric lipase is important for fat digestion in neonatal mammals. In contrast, the role of pepsin, a proteinase enzyme, is poorly understood in neonatal animals. Newborn ferrets depend on the production of lipase enzymes before birth so that they can more easily digest milk. They also depend on not producing protein-digesting pepsins in the first few days of life. This is in order to maintain the structural integrity of proteins in milk, especially those involved in immunity (Hamosh et al. 1998). While this process is probably common to all neonatal mammals, the use of ferrets as a model species in this study reveals the range of metabolic processes that could potentially be affected using novel control agents.

In mink as in other mammals, the conversion of haemoglobin to methaemoglobin instead of oxyhaemoglobin results in inefficient transport of oxygen in the respiratory system. The resulting condition is known as methaemoglobulinaemia and results in inhibited respiration and inactivity. The condition can be heritable, but can also be induced by the action of oxidative agents, such as nitrites and chlorates, on the blood (Calabrese et al. 1995).

In ferrets, vomiting and associated behaviours that may be induced by loperamide or cisplatin can be inhibited by administration of neuro-receptor antagonists (Minami et al. 1998; Zaman et al. 2000). It is remotely possible that this property may have some utility in reducing vomiting following the administration of other toxins such as 1080.

8.4 TOXINS

The use of toxins is often the most effective but frequently the most controversial method of controlling animal populations. Poisoning is effective because it requires low investment in labour or equipment and can easily be applied to large areas. For these same reasons, it is also the most risky because of the potential impact on non-target species. Consumption of poison by species not targeted by the control efforts can amplify conservation problems, and secondary poisoning may cascade into higher trophic levels eventually resulting in potential threats to humans. In New Zealand, other introduced mammals such as cats, possums and ferrets are as undesirable as stoats and thus secondary poisoning of mammals is probably not an issue of great concern. However, recent studies have demonstrated the potential effects of secondary poisoning on birds of conservation concern, notably a native owl, the morepork *Ninox novaeseelandiae* (Stephenson et al. 1999). Wren (1987) provides a brief

review of toxic substances in fur bearers. We have provided an overview of chemical agents that can affect and control mustelid populations.

In any field trials, a preliminary test of bait uptakes may be undertaken by marking baits with tetracycline, distributing the bait, then capturing a variety of species and looking for tetracycline fluorescence in teeth and bone (Johnston et al. 1987). These methods have been extensively used to evaluate bait uptake for rabies control programmes in foxes (Nunan et al. 1994).

Environmental contaminants

Semi-aquatic mustelids such as otters and mink are highly sensitive to pollution and readily accumulate high levels of mercury, organochlorine compounds and other chemicals (Francis & Bennett 1994; Halbrook et al. 1994, 1996). Because of their position at the top of the food chain in aquatic ecosystems, mink and otters are often used as bioindicators of pollution in aquatic environments (Aulerich & Ringer 1979; Halbrook et al. 1996; Smits et al. 1996a, b; Stevens et al. 1997).

Mink are particularly sensitive to low quantities of dietary PCBs (<1 ppm in diet) and intoxication leads to weight loss, discoloration and necrosis of the liver, fibrosis of coronary arteries and stillbirths (Platonow & Karstad 1973). Dietary exposure to PCBs impairs implantation and causes foetal death in mink by causing placental degeneration and vascular lesions in the placenta (Backlin et al. 1998). Daily exposure of female mink to PCBs in the diet resulted in 50% lower litter sizes and 20% reductions in the birth weight of kits. Survival of kits in the first two weeks of life was reduced from 73% to 47%. PCBs were transported from females to kits and accumulated by kits during pregnancy and lactation (Lund et al. 1999).

Depending on the degree of exposure, hexachlorobenzene in contaminated water can cause declines in mink reproductive success (Moore et al. 1997). Persistent multigenerational dietary exposure of mink to the pesticide lindane led to a reduction in the proportion of mated females giving birth, a reduction in male testis size and an overall reduction in litter size of 60% (Beard & Rawlings 1998). Mink can tolerate low levels (<1.0 ppm) of mercury contamination but at higher levels (>1.8 ppm) severe lesions or death occurred (Wobeser & Swift 1976). Clinical signs of heavy mercury intoxication included anorexia, loss of weight, loss of co-ordination, tremors and convulsions (Aulerich et al. 1974). Exposure of mink to heavy metals also resulted in an increased incidence of morphological abnormalities, parasitism and lower body mass (Sidorovich & Savcenko 1992). Fluorine in contaminated water supplies also had a chronic toxicity to farmed mink (Aulerich et al. 1987) while other chemical pollutants such as dioxin compounds are as toxic to mustelids as they are to other mammals (Hochstein et al. 1988).

In comparison to semi-aquatic mink and otters, the reliance of stoats on terrestrial environments makes them less vulnerable to accumulation of PCB and heavy metals. Thus, environmental pollution is unlikely to alter the physiology of stoats enough to affect their productivity or populations. PCBs may have some potential as biomarkers in studies of bait uptake and exposure.

Vertebrate control agents

The toxicity of anticoagulant rodenticides has been clearly demonstrated for stoats (Grolleau et al. 1989; Alterio 1996; Alterio et al. 1997; Brown et al. 1998; Murphy et al. 1998a, etc.). Other mustelids, including weasels (Harradine 1976; Townsend et al. 1984), polecats (Shore et al. 1996; Birks 1998) and martens (Lund & Rasmussen 1986; Berny et al. 1997), are also vulnerable. Despite the evidence that exposure is common in Britain (McDonald et al. 1998) and occurs frequently in New Zealand, the limited studies of rodenticide contamination in small mustelids provide little indication of the scale of lethal or sub-lethal effects upon populations.

The potential of brodifacoum for intentionally secondarily poisoning stoats and other predators has recently been investigated (Alterio 1996; Alterio et al. 1997; Brown et al. 1998). Although rodenticide usage is an effective method for killing resident stoats, human health concerns and non-target effects may prevent its widespread use (Eason et al. 1999a). In areas where brodifacoum has been used for possum and rat control, residues have been detected in a range of non-target species including pigs *Sus scrofa* (Murphy et al. 1998b; Eason et al. 1999a), red deer *Cervus elaphus scoticus* (McDonald & Murphy 2000), kiwi and morepork (Murphy et al. 1998b; Robertson et al. 1999; Stephenson et al. 1999).

Weasels are susceptible to poisoning by the rodenticide crimidine. This is a convulsivant agent that is similar in action to strychnine, metaldehyde and methiocarb and is commercially available under the brand name Castrix. It is used in some countries for killing voles, although concerns about its humaneness have prevented widespread usage. In laboratory trials, four out of four weasels, but none of three kestrels, died after being fed on voles exposed to a diet of 0.5% crimidine bait (Tkadlec 1993). Apart from this suggestion of differential toxicity to birds and mammals, this agent has no particular qualities that we are aware of that commend it over anticoagulant rodenticides.

Sodium fluoroacetate (1080) is a highly toxic vertebrate control agent for which there is no antidote. It is virtually tasteless and odourless, making its presence extremely difficult to detect. It has been used extensively for the control of coyotes and wolves in North America and for possum and rabbit control in New Zealand (Eason et al. 1999b). Death may result from cardiac failure, progressive failure of the central nervous system or respiratory arrest following prolonged convulsions. The compound is moderately persistent in the environment, though its degradation is facilitated by dilution and microbial activity and accelerated by high rainfall and warm temperatures (Eason et al. 1999b). In cold, dry conditions the compound may persist for several weeks to several months (Eason et al. 1999b). Where it persists it can pose a potential threat for non-target species. Compound 1080 is infamous outside of New Zealand because of secondary poisoning of non-target species, particularly raptors. It is a potentially useful candidate for the intentional control of stoats when deployed in a highly specific way, such as in poisoned eggs. Furthermore, secondary poisoning of stoats after widespread deployment of 1080 baits for possum and rodent control has been demonstrated in several cases (Gillies & Pierce 1999; Murphy et al. 1999). The fact that in stoats it is faster-acting than anticoagulants lends 1080 some appeal (Murphy et al. 1999). For more

information on the use and risks of 1080, detailed reviews are presented by Eason et al. (1999b) and other authors cited therein.

Mycotoxins

McDonald & Murphy (2000) suggested that a toxin that was less persistent than anticoagulant rodenticides may be a more acceptable method of secondarily poisoning predators in New Zealand. It is possible that naturally occurring toxins, such as those produced by fungi, may fall into this category. No mycotoxins have thus far been tested on stoats. However, concerns about the effect of a range of biological toxins on the economics of mink production have led to a number of studies of the effect of mycotoxins on farmed mink (e.g. Aulerich et al. 1993).

Mink are particularly susceptible to moniliformin, a toxin derived from the fungus *Fusarium fujikuroi*. Experimental intra-peritoneal exposure resulted in an LD_{50} of 2.2–2.8 mg/kg in nine-month-old female mink. Sub-acute exposure (1.5–3.2 mg/kg) resulted in malformation of the right side of the heart, by damaging myofibres, mitochondria, nuclei, Z- and M-lines and sarcoplasmic reticula, and increasing extracellular collagen deposition (Morgan et al. 1999). The effects of dietary exposure of mink to moniliformin were tested at concentrations of 10–240 ppm wet weight in feed. Mink did not eat feed containing more than 40 ppm. Diets of 8–17 ppm moniliformin produced no significant effects on feed consumption or body condition. However, when breeding female mink were given feed containing 17 ppm of the toxin, adult survival was not affected but neonatal mortality was increased and kit weight at birth was reduced (Morgan et al. 1998).

Similarly, long-term dietary exposure to fumonisin, another mycotoxin produced by *Fusarium* spp., is not lethal to adult mink but can lead to physiological disorders, indicated by alterations in ratios of free sphinganine to free sphingosine in liver and kidneys (Restum et al. 1995). This effect also has the result that exposure to fumonisins can be monitored non-invasively by monitoring concentrations of free sphinganine and sphingosine, two sphingolipids, in mink urine, but not in hair (Morgan et al. 1997). Ingestion of fumonisin can also affect reproduction in female mink; 58% of females fed the high-dose diet (254 ppm) produced kits compared to 100% of control or low-dose (115 ppm) samples. Kit weight at birth was negatively related to fumonisin dose, but litter size was not significantly affected by dose, though there was a negative trend. The percentage of kits that were stillborn was also directly proportional to fumonisin concentration (Powell et al. 1996).

The mycotoxin zearalenone, which is also produced by *Fusarium* spp., causes hyperoestrogenism in mink. At dietary doses of 20 mg/kg, 25% of mated females whelp successfully. Zearalenone acts by causing mild to severe endometrial hyperplasia, uterine atrophy, endometritis, pyometra and atrophy and degeneration of ovarian follicles (Yamini et al. 1997). Dietary zearalenone given to female mink did not affect the percentage of the population that had kits, but did significantly increase gestation length, decrease litter size, and increase kit mortality compared to females given control diets or a diet containing zearalenone and sodium calcium aluminosilicate (Bursian et al. 1992).

There are further possible biological toxins that are known to have biochemical effects on mustelids, but may also affect other mammals. These include jellyfish toxins (Mustafa et al. 1994) and extracts of foxgloves *Digitalis purpurea* (Allen et al. 1985).

8.5 SUMMARY AND RECOMMENDATIONS

We have found relatively few aspects of stoat physiology and metabolism that may help control their populations. A more detailed investigation of vitamin A metabolism could provide the only conspicuously novel insight into the metabolic 'weaknesses' of *Mustela* spp. As a novel approach, mycotoxins that are known to be toxic to mink may hold promise as agents for the lethal or sub-lethal reproductive control of stoats.

Clearly, high metabolic rates, high costs of thermoregulation and rapid food passage are symptoms of the importance of food supplies to survival of adults and recruitment of young. This is the most pronounced of the metabolic weaknesses from which stoats are known to suffer. Large-scale reduction of food supplies in the wild is theoretically possible, by such drastic measures as widespread deployment of rodenticides or potentially even interfering with the control of the beech mast cycle. Such options are unlikely to be attractive to conservation managers.

There are undoubtedly times of the year when the overall abundance of mammalian prey is lower and these will also be the most critical times for females to control their ultimate fertility. The end of winter and very early spring will be the most energy-demanding time of year for wild stoats. At this time, a large part of their small mammalian prey base has died over winter, few birds are nesting, and individual prey animals that remain may be those that are particularly hard to catch. Winter and very early spring is likely to provide the best temporal window of opportunity to control stoat populations. First, animals are more likely to respond to bait and lures when overall food availability is low. Second, animals are already stressed so they may be vulnerable to lower levels of toxins. Third, removal of any animals during this time of year is likely to cause additional, rather than compensatory mortality, because any animal alive at this period is likely to survive until its reproductive attempt in the spring. The duration of this critical period for stoats needs to be defined precisely so that managers can determine when their control actions have the highest impact on stoat populations.

9. Conclusions

The problem of nest predation by opportunist predators is not a simple one. The restricted temporal availability of nests means that few predators rely on eggs or chicks as food, even when they are available in quantity. Instead, most carnivores are opportunistic predators that encounter eggs while searching for their main food items. The intensity of predation is therefore directly related to the total number of predators present and the amount of time that these predators spend foraging in habitats used by nesting birds. Mitigation of predation can therefore act by reducing the number of predators or by altering their foraging behaviour.

At a large scale, neither of these two options is logistically easy. Contemporary methods for controlling predator abundance over large areas typically involve poisoning, as trapping is labour-intensive and rarely effective unless intensive. Manipulation of foraging behaviour may be even harder, especially where nesting areas for endangered species are not clearly delineated. In such cases, modifying stoat behaviour through techniques such as food supplementation, repellent use, conditioned taste aversion or by artificially increasing the perception of predation risk will probably not help towards long-term objectives. Physical barriers to discourage predation of tree-nesting species may hold some promise, since one-off visits by managers to install the device could confer long-term protection and so may be particularly cost effective.

The situation in New Zealand is complicated by cycles in *Nothofagus* masting, which lead to high density of rodents (King 1982; Murphy 1992), birds and invertebrates (Murphy & Dowding 1995) at intervals of 3–7 years. Increases in prey abundance lead to rapid outbreaks in stoat populations. It is intuitive that the impact of stoat control applied during the low phases in this cycle would be multiplicative because removed animals will not contribute to the production of offspring during peak years. However, the life span of stoats is typically so short and their potential productivity so high that pre-emptive strikes in low phases are unlikely to be especially effective, except perhaps in the year immediately prior to an expected mast season.

The long-term control of stoats in New Zealand requires an aggressive approach that will not only reduce future populations, but must have an immediate impact on current densities. From intuition, techniques aimed at physiologically impairing reproduction appear unlikely to produce the best results. To impair reproduction using existing agents would require stoats to be given a reproductive depressant, either by injection or by oral administration via treated baits. Both of these procedures would allow managers simply to kill the stoat, thereby reducing that individual's future chances of reproduction and that individual's contemporary impact on native fauna by 100%. Furthermore, their high reproductive potential suggests that the failure to treat even a small percentage of stoats would compensate for the effects of treatment. From this intuitive standpoint, it is hard to conceive of any justification for developing methods of reproductive control, except for humaneness. However, the maintenance of a stable system of female territoriality while eliminating recruitment into the population during the nesting season could mitigate the

drastic effects of the numerical response of the stoat population to the peak points of the mast cycle. The conflict between the intuitive and this more involved perspective highlights the urgency of a predictive modelling exercise that can investigate the relative merits of technological development targeted at lethal control over reproductive control.

The best prospects for controlling stoats in New Zealand in the immediate future lie in the integration of existing labour-intensive technologies, mainly trapping and poisoning. However, a more specifically targeted protocol for the use of these techniques could produce a markedly more cost effective path towards the desired levels of stoat control. Such a successfully targeted protocol is likely to incorporate specific, short-term attempts to drastically but temporarily reduce prey populations. To develop these strategies we urge managers to develop protocols for monitoring stoat population density and demography and for these data to be made available for models of stoat control. It is essential that control operations are closely monitored, and that results from current trials be used to test the predictions of models.

In the long term and depending on the outcome of well-informed modelling exercises, stoat populations could be controlled by the deployment of novel pathogens. While knowledge of existing stoat pathogens is scant, there is sufficient research available on the viruses that cause problems for farmed mink and ferrets to warrant early investigations of their pathogenicity to stoats. It is clear that Aleutian disease virus, mink enteritis virus, and canine distemper virus all hold potential for stoat control. To avoid potential problems associated with the spread of these diseases to non-target species, this research could potentially be conducted overseas.

Finally and to take the most lateral approach, it is clear throughout the literature that stoat abundance is fundamentally related to the availability of food. In most parts of their natural range, several prey species sustain the stoat population through the low points in the abundance of individual prey species. The release of myxomatosis in Britain effectively depressed stoat populations for about two decades, despite the availability of alternative prey. In New Zealand, stoats are dependent on a very narrow range of prey species. In *Nothofagus* forest, stoat abundance is most directly related to the breeding success of house mice and to a lesser extent ship rats. House mice and ship rats are obligately commensal throughout their range in temperate regions, hence the metabolic 'weaknesses' of these rodents could be more easily exploited in the climates encountered in New Zealand than would those of stoats that have evolved in temperate and polar conditions.

Control methods for stoats could alternatively focus on reducing their populations indirectly by reducing the availability of prey. Manipulations of food supply should be applied at the most critical time of the year when stoats are already energetically stressed and when females are in the process of 'deciding' how many blastocysts to implant or how many embryos to resorb. With existing technology this conclusion highlights the utility of rodent poisoning campaigns in late winter before birds are nesting and before the reproductive cycle of the rodents is under way. This should avoid the functional response of prey-switching exhibited by stoats later in the year when feeding their young or when young become independent. With future technology, this

conclusion emphasises that development of techniques for managing prey populations could be of similar value to those for controlling the predators.

10. Acknowledgements

This report originated from work carried out under Department of Conservation research investigation 3358, funded as part of the first phase of the Stoat Research Programme. R. McDonald is supported by a Royal Society Postdoctoral Fellowship, hosted by the Centre for Biodiversity and Ecology Research, University of Waikato. We are very grateful to N. Vaughan for translating papers from German. Thanks also to M. Artois, M. Jackson and C. King for providing reprints and to G. Bolduc, M. Day, M. Gamache, D. Gow, K. Guyn, D. Hall, E. Murphy, G. Norbury, R. Penner, T. Platt, R. Powell, J. Roberts, J.F. Robitaille, S. Roy, B. Semel, and J. Sundell for personal communications and advice.

11. References

Adams, L.H. 1935: *Mink raising*. A.R. Harding Publishing Co., Columbus, Ohio. 222 p.

Addison, E.M.; Barker, I.K.; Hunter, D.B. 1987: Diseases and parasites of furbearers. Pp. 893–909 *in*: Novak, M.; Baker, J.A.; Obbard, M.E.; Malloch, B. (eds): *Wild furbearer conservation and management in North America*. Ontario Ministry of Natural Resources, Toronto.

Aldous, S.E. 1940: Notes on a black-footed ferret raised in captivity. *Journal of Mammalogy 21*: 23–26.

Alexandersen, S.; Larsen, S.; Aasted, B.; Uttenthal, A.; Bloom, M.E.; Hansen, M. 1994: Acute interstitial pneumonia in mink kits inoculated with defined isolates of Aleutian mink disease parvovirus. *Veterinary Pathology 31*: 216–228.

Alexandersen, S.; Uttenthal-Jensen, A.; Hansen, M.; Aasted, B. 1985: Experimental transmission of Aleutian disease virus (ADV) to different animal species. *Acta Pathologica Microbiologica et Immunologica Scandinavica Section B-Microbiology 93*: 195–200.

Alldinger, S.; Baumgartner, W.; Van Moll, P.; Orvell, C. 1993: In-vivo and in-vitro expression of canine distemper viral proteins in dogs and non-domestic carnivores. *Archives of Virology 132*: 421–428.

Allen, D.G.; Eisner, D.A.; Smith, G.L.; Wray, S. 1985: The effects of an extract of the foxglove (*Digitalis purpurea*) on tension and intracellular calcium concentration in ferret papillary-muscle. *Journal of Physiology, London 365*: P55.

Alterio, N. 1996: Secondary poisoning of stoats (*Mustela erminea*), feral ferrets (*Mustela furo*), and feral house cats (*Felis catus*) by the anticoagulant poison, brodifacoum. *New Zealand Journal of Zoology 23*: 331–338.

Alterio, N.; Brown, K.; Moller, H. 1997: Secondary poisoning of mustelids in a New Zealand *Nothofagus* forest. *Journal of Zoology, London 243*: 863–869.

Alterio, N.; Moller, H.; Brown, K. 1999: Trappability and densities of stoats (*Mustela erminea*) and ship rats (*Rattus rattus*) in a South Island *Nothofagus* forest, New Zealand. *New Zealand Journal of Ecology 23*: 95–100.

Amstislavky, S.; Ternovaskaya, Y. 2000: Reproduction in mustelids. *Animal Reproductive Science 60–61*: 571–581.

Ando, K.; Sato, Y.; Miura, K.; Matsuoka, H.; Chinzei, Y. 1994: Migration and development of the larvae of *Gnathostoma nipponicum* in the rat, second intermediate or paratenic host, and the weasel, definitive host. *Journal of Helminthology 68*: 13–17.

Andreewskaja, W.S.; Brandesowa, E.G. 1977: Zucht des Hermelins (*Mustela erminea*) unter häuslichen Bedingungen. *Zoologische Garten N.F., Jena 47*: 365–368.

Arthur, S.M.; Paragi, T.F.; Krohn, W.B. 1993: Dispersal of juvenile fishers in Maine. *Journal of Wildlife Management 57*: 868–874.

Artois, M.; Blancou, J.; Gerard, Y. 1982: Parasitisme du putois (*Mustela putorius*) par *Troglotrema acutum*: étude bibliographique et enquete preliminaire dans l'est de la France. *Revue De Medecine Veterinaire 133*: 771–777.

Aulerich, R.J.; Bursian, S.J.; Watson, G.L. 1993: Effects of sublethal concentrations of aflatoxins on the reproductive performance of mink. *Bulletin of Environmental Contamination and Toxicology 50*: 750–756.

Aulerich, R.J.; Napolitano, A.C.; Bursian, S.J.; Olson, B.A.; Hochstein, J.R. 1987: Chronic toxicity of dietary fluorine to mink. *Journal of Animal Science 65*: 1759–1767.

Aulerich, R.J.; Ringer, R.K. 1979: Toxic effects of dietary polybrominated biphenyls on mink. *Archives of Environmental Contamination and Toxicology 8*: 487–498.

Aulerich, R.J.; Ringer, R.K.; Iwamoto, S. 1974: Effects of dietary mercury on mink. *Archives of Environmental Contamination and Toxicology 2*: 43–51.

Backlin, B.M.; Persson, E.; Jones, C.J.P.; Dantzer, V. 1998: Polychlorinated biphenyl (PCB) exposure produces placental vascular and trophoblastic lesions in the mink (*Mustela vison*): a light and electron microscopic study. *APMIS 106*: 785–799.

Ballantyne, E.E.; O'Donoghue, J.G. 1954: Rabies control in Alberta. *Journal of the American Veterinary Medicine Association 125*: 316–326.

Barrett, T. 1999: Morbillivirus infections, with special emphasis on morbilliviruses of carnivores. *Veterinary Microbiology 69*: 3–13.

Bartz, J.C.; Marsh, R.F.; McKenzie, D.I.; Aiken, J.M. 1998: The host range of chronic wasting disease is altered on passage in ferrets. *Virology 251*: 297–301.

Bartz, J.C.; McKenzie, D.I.; Bessen, R.A.; Marsh, R.F.; Aiken, J.M. 1994: Transmissible mink encephalopathy species barrier effect between ferret and mink – PrP gene and protein analysis. *Journal of General Virology 75*: 2947–2953.

Basse, B.; McLennan, J.A.; Wake, G.C. 1999: Analysis of the impact of stoats, *Mustela erminea*, on northern brown kiwi, *Apteryx mantelli*, in New Zealand. *Wildlife Research 26*: 227–237.

Batchelder, M.A.; Bell, J.A.; Erdman, S.E.; Marini, R.P.; Murphy, J.C.; Fox, J.G. 1999: Pregnancy toxemia in the European ferret (*Mustela putorius furo*). *Laboratory Animal Science 49*: 372–379.

Batchelder, M.A.; Erdman, S.E.; Li, X.T.; Fox, J.G. 1996: A cluster of cases of juvenile mediastinal lymphoma in a ferret colony. *Laboratory Animal Science 46*: 271–274.

Beard, A.P.; Rawlings, N.C. 1998: Reproductive effects in mink (*Mustela vison*) exposed to the pesticides Lindane, Carbofuran and Pentachlorophenol in a multigeneration study. *Journal of Reproduction and Fertility 113*: 95–104.

Beard, P.M.; Henderson, D.; Daniels, M.J.; Pirie, A.; Buxton, D.; Greig, A.; Hutchings, M.R.; McKendrick, I.; Rhind, S.; Stevenson, K.; Sharp, J.M. 1999: Evidence of paratuberculosis in fox (*Vulpes vulpes*) and stoat (*Mustela erminea*). *Veterinary Record 145*: 612–613.

Becker, E.F. 1991: A terrestrial furbearer estimator based on probability sampling. *Journal of Wildlife Management 55*: 730–737.

Bell, J.F.; Reilly, J.R. 1981: Tularemia. Pp. 213–231 *in*: Davis, J.W.; Karstad, L.H.; Trainer, D.O. (eds) *Infectious diseases of wild mammals*. 2nd edn. Iowa State University Press, Ames, Iowa.

Ben-David, M. 1997: Timing of reproduction in wild mink: The influence of spawning Pacific salmon. *Canadian Journal of Zoology 75*: 376–382.

Bern, C. 1993: Behavioral development and maternal care in captive ermine. MS Thesis, University of Tennessee, Knoxville.

Berny, P.J.; Buronfosse, T.; Buronfosse, F.; Lamarque, F.; Lorgue, G. 1997: Field evidence of secondary poisoning of foxes (*Vulpes vulpes*) and buzzards (*Buteo buteo*) by bromadiolone, a 4-year survey. *Chemosphere 35*: 8.

Birks, J.D.S. 1998: Secondary rodenticide poisoning risk arising from winter farmyard use by the European polecat *Mustela putorius*. *Biological Conservation 85*: 233–240.

Bissonette, T.H.; Bailey, E.E. 1940: Den and runway system for weasels and other small mammals in the laboratory. *American Midland Naturalist 24*: 761–763

Bittle, J.L. 1981: Feline panleukopenia. Pp. 97–101 *in*: Davis, J.W.; Karstad, L.H.; Trainer, D.O. (eds) *Infectious diseases of wild mammals*. 2nd edn. Iowa State University Press, Ames, Iowa.

Bleavins, M.R.; Aulerich, R.J. 1981: Feed consumption and food passage time in mink and European ferrets. *Laboratory Animal Science 31*: 268–269.

Blixenkrone-Moller, M.; Svansson, V.; Appel, M.; Krogsrud, J.; Have, P.; Orvell, C. 1992: Antigenic relationships between field isolates of morbilliviruses from different carnivores. *Archives of Virology 123*: 279–294.

Blomme, E.A.G.; Foy, S.H.; Chappell, K.H.; La Perle, K.M.D. 1999: Hypereosinophilic syndrome with Hodgkin's-like lymphoma in a ferret. *Journal of Comparative Pathology 120*: 211–217.

Bloom, M.E.; Fox, J.M.; Berry, B.D.; Oie, K.L.; Wolfinbarger, J.B. 1998: Construction of pathogenic molecular clones of Aleutian mink disease parvovirus that replicate both in vivo and in vitro. *Virology 251*: 288–296.

Bloom, M.E.; Kanno, H.; Mori, S.; Wolfinbarger, J.B. 1994: Aleutian mink disease—puzzles and paradigms. *Infectious Agents and Disease-Reviews, Issues and Commentary 3*: 279–301.

Bogliani, G.; Bellinato, F. 1998: Conditioned aversion as a tool to protect eggs from avian predators in heron colonies. *Colonial Waterbirds 21*: 69–72.

Bomford, M. 1990: *A role for fertility control in wildlife management?* Bureau of Rural Resources, Canberra. 50 p.

Bradley, M.P.; Eade, J.; Penhale, J.; Bird, P. 1999: Vaccines for fertility regulation of wild and domestic species. *Journal of Biotechnology 73*: 91–101.

Broll, S.; Alexandersen, S. 1996: Investigation of the pathogenesis of transplacental transmission of Aleutian mink disease parvovirus in experimentally infected mink. *Journal of Virology 70*: 1455–1466.

Brown, J.M.; Lasiewski, R.C. 1972: Metabolism of weasels: the cost of being long and thin. *Ecology 53*: 939–943.

Brown, K.P.; Alterio, N.; Moller, H. 1998: Secondary poisoning of stoats (*Mustela erminea*) at low mouse (*Mus musculus*) abundance in a New Zealand *Nothofagus* forest. *Wildlife Research 25*: 419–426.

Buchman, C.A.; Swarts, J.D.; Seroky, J.T.; Panagiotou, N.; Hayden, F.; Doyle, W.J. 1995: Otologic and systemic manifestations of experimental influenza-a virus-infection in the ferret. *Otolaryngology-Head and Neck Surgery 112*: 572–578.

Burns, J.J. 1964: Movements of a tagged weasel in Alaska. *Murrelet 45*: 10.

Bursian, S.J.; Aulerich, R.J.; Cameron, J.K.; Ames, N.K.; Steficek, B.A. 1992: Efficacy of hydrated sodium calcium aluminosilicate in reducing the toxicity of dietary zearalenone to mink. *Journal of Applied Toxicology 12*: 85–90.

Buskirk, S.W.; Lindstedt, S.L. 1989: Sex biases in trapped samples of Mustelidae. *Journal of Mammalogy 70*: 88–97.

Cabasso, V.J. 1981: Infectious canine hepatitis. Pp. 191–195 *in*: Davis, J.W.; Karstad, L.H.; Trainer, D.O. (eds) *Infectious diseases of wild mammals*. 2nd edn. Iowa State University Press, Ames, Iowa.

Calabrese, E.J.; Leonard, D.A.; Zhao, X. 1995: Susceptibility of mink to methemoglobin formation. *Bulletin of Environmental Contamination and Toxicology 55*: 439–445.

Caplan, E.R.; Peterson, M.E.; Mullen, H.S.; Quesenberry, K.E.; Rosenthal, K.L.; Hoefer, H.L.; Moroff, S.D. 1996: Diagnosis and treatment of insulin-secreting pancreatic islet cell tumors in ferrets: 57 cases (1988–1994). *Journal of the American Veterinary Medical Association 209*: 1741–1745.

Carey, A.B.; McLean, R.G. 1983: The ecology of rabies: evidence of co-adaptation. *Journal of Applied Ecology 20*: 777–800.

Carpenter, J.W.; Hillman, C.N. 1978: Husbandry, reproduction, and veterinary care of captive ferrets. *Annual Proceedings of the American Association of Zoo Veterinarians 1978*: 36–47.

Caughley, G. 1977: *Analysis of vertebrate populations*. John Wiley and Sons, London. 234 p.

Center for Disease Control. 2000: Public health importance of rabies. URL: http: //www.cdc.gov/ncidod/dvrd/rabies/introduction/intro.htm.

Chanin, P. 1983: Observations on two populations of feral mink in Devon, U.K. *Mammalia 47*: 464–476.

Charlton, K.M. 1994: The pathogenesis of rabies and other lyssaviral infections: recent studies. *Current Topics in Microbiology and Immunology 187*: 95–119.

Charlton, K.M.; Webster, W.A.; Casey, G.A.; Rupprecht, C.E. 1988: Skunk rabies. *Reviews of Infectious Diseases 10*: s626–s628.

Chen, W.S.; Aasted, B. 1998: Analyses of leucocytes in blood and lymphoid tissues from mink infected with Aleutian mink disease parvovirus (AMDV). *Veterinary Immunology and Immunopathology 63*: 317–334.

Christophersen, B.; Nordstoga, K.; Shen, Y.; Olivecrona, T.; Olivecrona, G. 1997: Lipoprotein lipase deficiency with pancreatitis in mink: Biochemical characterization and pathology. *Journal of Lipid Research 38*: 837–846.

Clausen, T.N.; Olesen, C.R.; Hansen, O.; Wamberg, S. 1992: Nursing sickness in lactating mink *(Mustela vison)* I. Epidemiological and pathological observations. *Canadian Journal of Veterinary Research 56*: 89–94.

Clausen, T.N.; Wamberg, S.; Hansen, O. 1996: Incidence of nursing sickness and biochemical observations in lactating mink with and without dietary salt supplementation. *Canadian Journal of Veterinary Research 60*: 271–276.

Coleman, G.D.; Chavez, M.A.; Williams, B.H. 1998: Cystic prostatic disease associated with adrenocortical lesions in the ferret (*Mustela putorius furo*). *Veterinary Pathology 35*: 547–549.

Conover, M.R. 1989: Potential compounds for establishing conditioned food aversions in raccoons. *Wildlife Society Bulletin 17*: 430–435.

Conover, M.R. 1990: Reducing mammalian predation on eggs by using a conditioned taste aversion to deceive predators. *Journal of Wildlife Management 54*: 360–365.

Courchamp, F.; Langlais, M.; Sugihara, G. 1999a: Cats protecting birds: modelling the mesopredator release effect. *Journal of Animal Ecology 68*: 282–292.

Courchamp, F.; Langlais, M.; Sugihara, G. 1999b: Control of rabbits to protect island birds from cat predation. *Biological Conservation 89*: 219–225.

Courchamp, F.; Langlais, M.; Sugihara, G. 2000: Rabbits killing birds: modelling the hyperpredation process. *Journal of Animal Ecology 69*: 154–164.

Cowan, P.E. 1996: Possum biocontrol: prospects for fertility regulation. Marsupial gametes and embryos. *Reproduction Fertility and Development 8*: 655–660.

Crandall, L.S. 1964: *The management of wild mammals in captivity*. University of Chicago Press, Illinois. 761 p.

Crichton, V.F.J.; Beverley-Burton, M. 1974: Distribution and prevalence of *Dracunculus* spp. (Nematoda: Dracunculoidea) in mammals in Ontario. *Canadian Journal of Zoology 52*: 163–167.

Damgaard, B.M.; Clausen, T.N.; Dietz, H.H. 1998: Effect of dietary protein levels on growth performance, mortality rate and clinical blood parameters in mink (*Mustela vison*). *Acta Agriculturae Scandinavica Section A. Animal Science 48*: 38–48.

Davis, J.W.; Karstad, L.H.; Trainer, D.O. (eds) 1981: *Infectious diseases of wild mammals*. Iowa State University Press, Ames, Iowa. 446 p.

Day, M.G. 1963: An ecological study of the stoat (*Mustela erminea* L.) and the weasel (*Mustela nivalis* L.) with particular reference to their food and feeding habits. PhD Thesis, University of Exeter.

Deanesly, R. 1935: The reproductive processes of certain mammals. Part IX—Growth and reproduction in the stoat (*Mustela erminea*). *Philosophical Transactions of the Royal Society of London, series B 225*: 459–492.

Deanesly, R. 1943: Delayed implantation in the stoat (*Mustela mustela* [sic]). *Nature 151*: 365–366.

Debrot, S. 1984: Dynamique du renouvellement et structure d'age d'une population d'hermines *Mustela erminea*). *La Terre et la Vie 39*: 75–88.

Debrot, S.; Mermod, C. 1982: Quelques siphonaptères de mustélides, dont *Rhadinopsylla pentacantha* (Rothschild, 1897), nouvelle espèce pour la Suisse. *Révue Suisse de Zoologie 89*: 27–32.

Debrot, S.; Mermod, C. 1983: The spatial and temporal distribution pattern of the stoat (*Mustela erminea* L.). *Oecologia 59*: 69–73.

Dietzschold, B.; Rupprecht, C.E.; Fu, Z.F.; Koprowski, H. 1996: Rhabdoviruses. Pp.1137–1159 *in*: Fields, B.N.; Knipe, D.M.; Howley, P.M. (eds) *Virology*. 3rd edn. Lippincott-Raven Publishers, Philadelphia.

Dominion Bureau of Statistics 1931: Report of the fur farms of Canada 1930. Dominion Bureau of Statistics, Ottawa.

Dominion Bureau of Statistics 1951: Report of fur farms 1950. Dominion Bureau of Statistics, Ottawa.

Dominion Bureau of Statistics 1952: Report of fur farms 1951. Dominion Bureau of Statistics, Ottawa.

Dominion Bureau of Statistics 1954: Report of fur farms 1953. Dominion Bureau of Statistics, Ottawa.

Doncarlos, M.W.; Petersen, J.S.; Tilson, R.L. 1986: Captive biology of an asocial mustelid; *Mustela erminea. Zoo Biology 5*: 363–370.

Dougherty, E.C.; Hall, E.R. 1955: The biological relationships between American weasels (genus *Mustela*) and nematodes of the genus *Skrjabingylus* Petrov, 1927, (Nematoda: Metastrongylidae) the causative organisms of certain lesions in weasel skulls. *Revista Parasitologica Iberica, special edition*: 531–576.

Douglas, D.A.; Houde, A.; Song, J.H.; Farookhi, R.; Concannon, P.W.; Murphy, B.D. 1998: Luteotropic hormone receptors in the ovary of the mink (*Mustela vison*) during delayed implantation and early-postimplantation gestation. *Biology of Reproduction 59*: 571–578.

Douglas, D.A.; Song, J.H.; Houde, A.; Cooke, G.M.; Murphy, B.D. 1997: Luteal and placental characteristics of carnivore gestation: expression of genes for luteotrophic receptors and steroidogenic enzymes. *Journal of Reproduction and Fertility*: 153–166.

Duby, R.T.; Travis, H.F. 1972: Photoperiodic control of fur growth and reproduction in the mink (*Mustela vison*). *Journal of Experimental Zoology 182*: 217–226.

Dunstone, N. 1993: *The mink*. T. & A.D. Poyser Natural History, London. 232 p.

Dyer, N.W.; Huffman, L.E. 1999: Plague in free-ranging mammals in western North Dakota. *Journal of Wildlife Diseases 35*: 600–602.

Dyer, N.W.; Schamber, G.J. 1999: Pneumocystosis associated with canine distemper virus infection in a mink. *Canadian Veterinary Journal 40*: 577–578.

Eason, C.T.; Milne, L.; Potts, M.; Morriss, G.; Wright, G.R.G.; Sutherland, O.R.W. 1999a: Secondary and tertiary poisoning risks associated with brodifacoum. *New Zealand Journal of Zoology 23*: 219–224.

Eason, C.T.; Wickstrom, M.; Turck, P.; Wright, G.R.G. 1999b: A review of regulatory and environmental toxicology studies on 1080: results and implications. *New Zealand Journal of Zoology 23*: 129–138.

East, K.; Lockie, J.D. 1964: Observations on a family of weasels (*Mustela nivalis*) bred in captivity. *Proceedings of the Zoological Society of London 143*: 359–363.

East, K.; Lockie, J.D. 1965: Further observations on weasels (*Mustela nivalis*) and stoats (*Mustela erminea*) born in captivity. *Journal of Zoology, London 147*: 234–238.

Eberhard, M.L.; Ruiztiben, E.; Wallace, S.V. 1988: *Dracunculus insignis*—experimental infection in the ferret, *Mustela putorius furo*. *Journal of Helminthology 62*: 265–270.

Eley, T.J. Jr. 1977: *Ixodes uriae* (Acari: Ixodidae) from a river otter. *Journal of Medical Entomology 13*: 506.

Enders, R.K. 1952: Reproduction in the mink (*Mustela vison*). *Proceedings of the American Philosophical Society 96*: 691–755.

English, M.P. 1969: Ringworm in wild mammals: further investigations. *Journal of Zoology, London 159*: 515–522.

Englund, L.; af Segerstad, C.H. 1998: Two avian H10 influenza A virus strains with different pathogenicity for mink (*Mustela vison*). *Archives of Virology 143*: 653–666.

Erdman, S.E.; Correa, P.; Coleman, L.A.; Schrenzel, M.D.; Li, X.T.; Fox, J.G. 1997: *Helicobacter mustelae*-associated gastric MALT lymphoma in ferrets. *American Journal of Pathology 151*: 273–280.

Erdman, S.E.; Kanki, P.J.; Moore, F.M.; Brown, S.A.; Kawasaki, T.A.; Mikule, K.W.; Travers, K.U.; Badylak, S.F.; Fox, J.G. 1996: Clusters of lymphoma in ferrets. *Cancer Investigation 14*: 225–230.

Erdman, S.E.; Reimann, K.A.; Moore, F.M.; Kanki, P.J.; Yu, Q.C.; Fox, J.G. 1995: Transmission of a chronic lymphoproliferative syndrome in ferrets. *Laboratory Investigation 72*: 539–546.

Erlinge, S. 1977*a*: Agonistic behaviour and dominance in stoats (*Mustela erminea* L.). *Zeitschrift für Tierpsychologie 44*: 375–388.

Erlinge, S. 1977*b*: Spacing strategy in stoat *Mustela erminea*. *Oikos 28*: 32–42.

Erlinge, S. 1983: Demography and dynamics of a stoat *Mustela erminea* population in a diverse community of vertebrates. *Journal of Animal Ecology 52*: 705–726.

Erlinge, S.; Sandell, M. 1986: Seasonal changes in the social organisation of male stoats *Mustela erminea*: an effect of shifts between two decisive resources. *Oikos 47*: 57–62.

Erlinge, S.; Sandell, M.; Brinck, C. 1982: Scent-marking and its territorial significance in stoats, *Mustela erminea*. *Animal Behaviour 30*: 811–818.

Fagerstone, K.A. 1987: Black-footed ferret, long-tailed weasel, short-tailed weasel, and least weasel. Pp. 548–573 *in*: Nowak, R.; Baker, J.A.; Obbard, M.E.; Malloch, B. (eds) *Wild furbearer management and conservation in North America*. Ontario Ministry of Natural Resources, Toronto.

Fairley, J.S. 1971: New data on the Irish stoat. *Irish Naturalists Journal 17*: 49–57.

Fairley, J.S. 1980: Observations on a collection of feral Irish mink *Mustela vison* Schreber. *Proceedings of the Royal Irish Academy Section B-Biological Geological and Chemical Science 80*: 79–90.

Fog, M. 1969: Studies on the weasel (*Mustela nivalis*) and the stoat (*Mustela erminea*) in Denmark. *Danish Review of Game Biology 6*: 1–14.

Foreyt, W.J.; Lagerquist, J.E. 1993: Internal parasites from the marten (*Martes americana*) in eastern Washington. *Journal of the Helminthological Society of Washington 60*: 72–75.

Fox, J.G.; Dangler, C.A.; Sager, W.; Borkowski, R.; Gliatto, J.M. 1997: *Helicobacter mustelae*-associated gastric adenocarcinoma in ferrets (*Mustela putorius furo*). *Veterinary Pathology 34*: 225–229.

Fox, J.M.; Stevenson, M.A.M.; Bloom, M.E. 1999: Replication of Aleutian mink disease parvovirus in vivo is influenced by residues in the VP2 protein. *Journal of Virology 73*: 8713–8719.

Francis, D.R.; Bennett, K.A. 1994: Additional data on mercury accumulation in northern Michigan river otters. *Journal of Freshwater Ecology 9*: 1–5.

Frost, H.C.; Krohn, W.B.; Wallace, C.R. 1997: Age-specific reproductive characteristics in fishers. *Journal of Mammalogy 78*: 598–612.

Fryxell, J.M.; Falls, J.B.; Falls, E.A.; Brooks, R.J.; Dix, L.; Strickland, M.A. 1999: Density dependence, prey dependence, and population dynamics of martens in Ontario. *Ecology 80*: 1311–1321.

Gérard, Y.; Barrat, J. 1986: Parasitisme des mustélidés par *Skrjabingylus petrovi*: premier rapport en Europe occidentale. *Annales de Parasitologie Humaine et Comparee 61*: 575–579.

Gilbert, F.F.; Bailey, E.D. 1967: The effect of visual isolation on reproduction in the female ranch mink. *Journal of Mammalogy 48*: 113–118.

Gilbert, F.F.; Bailey, E.D. 1969: Visual isolation and stress in female ranch mink particularly during the reproductive season. *Canadian Journal of Zoology 47*: 209–212.

Gillies, C.A.; Pierce, R.J. 1999: Secondary poisoning of mammalian predators during possum and rodent control operations at Trounson Kauri Park, Northland, New Zealand. *New Zealand Journal of Ecology 23*: 183–192.

Golden, H.N. 1999: An expert-system model for lynx management in Alaska. Pp. 205–231 *in*: Proulx, G. (ed.) *Mammal Trapping*. Alpha Wildlife Research & Management Ltd., Sherwood Park, Alberta.

Gomez, M.R.; Siekert, R.G.; Herrmann, E.C. 1965: A human case of skunk rabies. Case report with comment on virological studies and the prophylactic treatment. *Journal of the American Medical Association 194*: 333–335.

Gomez-Villamandos, J.C.; Carrasco, L.; Mozos, E.; Hervas, J. 1995: Fatal cryptosporidiosis in ferrets (*Mustela putorius furo*): A morphopathologic study. *Journal of Zoo and Wildlife Medicine 26*: 539–544.

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

Greer, K.R. 1955: Yearly food habits of the river otter in the Thompson Lakes region, Northwestern Montana, as indicated by scat analyses. *American Midland Naturalist 54*: 299–313.

Grolleau, G.; Lorgue, G.; Nahas, K. 1989: Toxicité secondaire, en laboratoire, d'un rodenticide anticoagulant (bromadiolone) pour des prédateurs de rongeurs champêtres: buse variable (*Buteo buteo*) et hermine (*Mustela erminea*). *EPPO Bulletin 19*: 633–648.

Grue, H.E.; King, C.M. 1984: Evaluation of age criteria in New Zealand stoats (*Mustela erminea*) of known age. *New Zealand Journal of Zoology 11*: 437–443.

Gulamhusein, A.P.; Tam, W.H. 1974: Reproduction in the male stoat. *Mustela erminea*. *Journal of Reproduction and Fertility 41*: 303–312.

Gulamhusein, A.P.; Thawley, A.R. 1974: Plasma progesterone levels in the stoat. *Journal of Reproduction and Fertility 36*: 405–408.

Halbrook, R.S.; Jenkins, J.H.; Bush, P.B.; Seabolt, N.D. 1994: Sublethal concentrations of mercury in river otters: monitoring environmental contamination. *Archives of Environmental Contamination and Toxicology 27*: 306–310.

Halbrook, R.S.; Woolf, A.; Hubert, G.F., Jr.; Ross, S.; Braselton, W.E. 1996: Contaminant concentrations in Illinois mink and otter. *Ecotoxicology 5*: 103–114.

Hamosh, M.; Henderson, T.R.; Hamosh, P. 1998: Gastric lipase and pepsin activities in the developing ferret: Nonparallel development of the two gastric digestive enzymes. *Journal of Pediatric Gastroenterology and Nutrition 26*: 162–166.

Hanlon, C.A; Childs, J.E.; Nettles, V.F. 1999: Rabies in wildlife. Recommendations of a national working group on prevention and control of rabies in the United States. *Journal of the American Veterinary Medicine Association 215*: 1612–1618.

Hansen, B.K. 1999: Mink dam weight changes during the lactation period. 2. Energy consumption and plasma concentrations of thyroid hormones and insulin. *Acta Agriculturae Scandinavica Section A-Animal Science 49*: 65–72.

Hansen, O.; Wamberg, S.; Clausen, T.N. 1996: Failure of loop diuretics to induce nursing sickness in mink at weaning. *Canadian Journal of Veterinary Research 60*: 277–280.

Hansson, A. 1947: The physiology of reproduction in mink (*Mustela vison*, Schreb.) with special reference to delayed implantation. *Acta Zoologica 28*: 1–136.

Hansson, I. 1967: Transmission of the parasitic nematode *Skrjabingylus nasicola* (Leuckart 1842) to species of *Mustela* (Mammalia). *Oikos 18*: 247–252.

Hansson, I. 1968: Cranial helminth parasites in species of Mustelidae I. Frequency and damage in fresh mustelids from Sweden. *Oikos 19*: 217–233.

Harradine, J.P. 1976: Anticoagulant rodenticides and non-target wildlife: An ecological evaluation of permanent baiting in rural rat control. PhD Thesis, University of Edinburgh.

Harris, C.J. 1968: *Otters: a study of the recent Lutrinae*. Weidenfeld & Nicholson, London. 236 p.

Hart, J. 1987: Estrogen toxicity in the ferret. *British Journal of Experimental Pathology 68*: 601–602.

Hartman, L. 1964: The behaviour and breeding of captive weasels (*Mustela nivalis* L.). *New Zealand Journal of Science 7*: 147–156.

Haskell, D.G. 1997: Experiments and a model examining learning in the area-restricted search behavior of ferrets (*Mustela putorius furo*). *Behavioral Ecology 8*: 448–455.

Hathaway, S.C.; Blackmore, D.K. 1981: Failure to demonstrate the maintenance of leptospires by free-living carnivores. *New Zealand Veterinary Journal 29*: 115–116.

Have, P.; Moving, V.; Svansson, V.; Uttenthal, A.; Bloch, B. 1992: Coronavirus infection in mink (*Mustela vison*)—serological evidence of infection with a coronavirus related to transmissible gastroenteritis virus and porcine epidemic diarrhea virus. *Veterinary Microbiology 31*: 1–10.

Hayles, L.B.; Dryden, I.M. 1970: Epizootiology of rabies in Saskatchewan. *Canadian Veterinary Journal 11*: 131–136.

Heidt, G.A.; Petersen, M.K.; Kirkland, G.L. Jr. 1968: Mating behavior and development of least weasels *(Mustela nivalis)* in captivity. *Journal of Mammalogy 49*: 413–419.

Helmboldt, C.F.; Jungherr, E.L. 1958: The pathology of Aleutian disease in mink. *American Journal of Veterinary Research 19*: 212–222.

Henriksen, P.; Dietz, H.H.; Uttenthal, A.; Hansen, M. 1994: Seroprevalence of *Toxoplasma gondii* in Danish farmed mink (*Mustela vison)* S.). *Veterinary Parasitology 53*: 1–5.

Hersteinsson, P.; Gunnarsson, E.; Hjartardottir, S.; Skirnisson, K. 1993: Prevalence of *Encephalitozoon cuniculi* antibodies in terrestrial mammals in Iceland, 1986 to 1989. *Journal of Wildlife Diseases 29*: 341–344.

Hesselbarth, J.; Schwarz, S. 1995: Comparative ribotyping of *Staphylococcus intermedius* from dogs, pigeons, horses and mink. *Veterinary Microbiology 45*: 11–17.

Hinds, L.A.; Williams, C.K.; Pech, R.P.; Spratt, D.M.; Robinson, A.J.; Reubel, G.H. 2000. Feasibility of immunocontraception for managing stoats in New Zealand. *Science for Conservation 158*. 109 p.

Hoberg, E.P.; Aubry, K.B.; Brittell, J.D. 1990: Helminth parasitism in martens (*Martes americana*) and ermines (*Mustela erminea*) from Washington, with comments on the distribution of *Trichinella spiralis*. *Journal of Wildlife Diseases 26*: 447–452.

Hoberg, E.P.; Henny, C.J.; Hedstrom, O.R.; Grove, R.A. 1997: Intestinal helminths of river otters (*Lutra canadensis*) from the Pacific southwest. *Journal of Parasitology 83*: 105–110.

Hochstein, J.R.; Aulerich, R.J.; Bursian, S.J. 1988: Acute toxicity of 2,3,7,8-tetrachlorodibenzo-para-dioxin to mink. *Archives of Environmental Contamination and Toxicology 17*: 33–37.

Hodgson, R.G. 1937: Fisher farming. *Fur Trade Journal of Canada 15*: 21.

Holland, G.P. 1964: Evolution, classification and host relationships of Siphonaptera. *Annual Review of Entomology 9*: 123–146.

Hoover, J.P.; Root, C.R.; Zimmer, M.A. 1984: Clinical evaluation of American river otters in a reintroduction study. *Journal of the American Veterinary Medical Association 185*: 1321–1326.

Huang, Y.T.; Turchek, B.M. 2000: Mink lung cells and mixed mink lung and A549 cells for rapid detection of influenza virus and other respiratory viruses. *Journal of Clinical Microbiology 38*: 422–423.

Hunter, D.B.; Prescott, J.G. 1991: Staphylococcal adenitis in ranch mink in Ontario. *Canadian Veterinary Journal 32*: 354–356.

Innes, J.; Hay, R.; Flux, I.; Bradfield, P.; Speed, H.; Jansen, P. 1999: Successful recovery of North Island kokako *Callaeas cinerea wilsoni* populations, by adaptive management. *Biological Conservation 87*: 201–214.

Iversen, J.A. 1972: Basal energy metabolism of Mustelids. *Journal of Comparative Physiology 81*: 341–344.

Iwaki, T.; Abe, N.; Shibahara, T.; Oku, Y.; Kamiya, M. 1995: New distribution record of *Taenia mustelae* Gmelin, 1790 (Cestoda) from the least weasel *Mustela nivalis* in Hokkaido, Japan. *Journal of Parasitology 81*: 796.

Jackson, M.K.; Winslow, S.G.; Dockery, L.D.; Jones, J.K.; Sisson, D.V. 1996: Investigation of an outbreak of Aleutian disease on a commercial mink ranch. *American Journal of Veterinary Research 57*: 1706–1710.

Jennings, D.H.; Threlfall, W.; Dodds, D.G. 1982: Metazoan parasites and food of short-tailed weasels and mink in Newfoundland, Canada. *Canadian Journal of Zoology 60*: 180–183.

Jensen, B. 1978: Resultater af fangst med kassefaelder. *Natura Jutlandica 20*: 129–136.

Jensen, K.T.; Wolfinbarger, J.B.; Aasted, B.; Bloom, M.E. 2000: Replication of Aleutian mink disease parvovirus in mink lymph node histocultures. *Journal of General Virology 81*: 335–343.

Johnson, H.N. 1959: The role of the spotted skunk in rabies. *Proceedings of the Annual Meeting of the US Livestock Sanitary Association 63*: 267–274.

Johnson, J.D.; Biggins, D.E.; Wrigley, R.H.; Mangone, B.A.; Wimsatt, J. 1999: Pyometra in a Siberian polecat (*Mustela eversmanni*). *Contemporary Topics in Laboratory Animal Science 38*: 39–41.

Johnston, D.H.; Joachim, D.G.; Bachmann, P.; Kardong, K.V.; Stewart, R.E.; Dix, L.M.; Strickland, M.A.; Watt, I.D. 1987: Ageing furbearers using tooth structure and biomarkers. Pp. 228–243 *in*: Nowak, M.; Baker, J.A.; Obbard, M.E.; Malloch, B. (eds) *Wild Furbearer Management and Conservation in North America*. Ontario Ministry of Natural Resources, Toronto.

Jolley, W.R.; Kingston, N.; Williams, E.S.; Lynn, C. 1994: *Coccidia*, *Giardia* sp., and a physalopteran nematode parasite from black-footed ferrets (*Mustela nigripes*) in Wyoming. *Journal of the Helminthological Society of Washington 61*: 89–94.

Jorgensen, M.; Scheutz, F.; Strandbygaard, B. 1996: *Escherichia coli* and virus isolated from 'sticky kits'. *Acta Veterinaria Scandinavica 37*: 163–169.

Kaplan C. 1985: Rabies: a worldwide disease. Pp. 1–20 *in*: Bacon, P.J. (ed.) *Population dynamics of rabies in wildlife*. Academic Press, London.

Kellogg, C.E.; Bassett, C.F.; Enders, R.K. 1948: Mink raising. *Contributions from the Bureau of Animal Industry to the USDA Circular Series 801*: 1–42. Library of Congress LCCN #agr49-519.

Kenyon, A.J.; Kenyon, B.J.; Hahn, E.C. 1978: Protides of the Mustelidae: Immunoresponse of mustelids to Aleutian mink disease virus. *American Journal of Veterinary Research 39*: 1011–1015.

Keymer, I.F.; Epps, H.B.G. 1969: Canine distemper in the family Mustelidae. *Veterinary Record 85*: 204–205.

Kim, K.C.; Emerson, K.C. 1974: *Latagophthirus rauschi*, new genus and new species (Anoplura: Echinophthiriidae) from the river otter (Carnivora: Mustelidae). *Journal of Medical Entomology 11*: 442–446.

King, C.M. 1974: The nematode *Skrjabingylus nasicola* (Metastrongyloidea) in mustelids: a new record for New Zealand. *New Zealand Journal of Zoology 1*: 501–502.

King, C.M. 1976: The fleas of a population of weasels in Wytham Woods, Oxford. *Journal of Zoology, London 180*: 525–535.

King, C.M. 1981: The reproductive tactics of the stoat (*Mustela erminea*) in New Zealand forests. Pp. 443–468 *in*: Chapman, J.A.; Pursley, D. (eds) *Proceedings of the first worldwide furbearer conference*. The Worldwide Furbearer Conference, Inc., Frostburg, Maryland.

King, C.M. 1982: Age structure and reproduction in feral New Zealand populations of the house mouse (*Mus musculus*), in relation to seedfall of southern beech. *New Zealand Journal of Zoology 9*: 467–480.

King, C.M. 1983a: Mustela erminea. *Mammalian Species 195*: 1–8.

King, C.M. 1983b: The relationships between beech (*Nothofagus* sp.) seedfall and populations of mice (*Mus musculus*), and the demographic and dietary responses of stoats (*Mustela erminea*), in three New Zealand forests. *Journal of Animal Ecology 52*: 141–166.

King, C.M. 1983c: Factors regulating mustelid populations. *Acta Zoologica Fennica 174*: 217–220.

King, C.M. 1984: *Immigrant killers: Introduced predators and the conservation of birds in New Zealand.* Oxford University Press, Auckland. 224 p.

King, C. 1989: *The natural history of weasels and stoats*. Christopher Helm, London. 253 p.

King, C.M. 1991: A review of age-determination methods for the stoat *Mustela erminea*. *Mammal Review 21*: 31–49.

King, C.M. 1994: Monitoring and control of mustelids on conservation lands. 1. Planning and assessing an operation. *Department of Conservation Technical Series 3*. 36 p.

King, C.M.; Edgar, R.L. 1977: Techniques for trapping and tracking stoats (*Mustela erminea*): a review and a new system. *New Zealand Journal of Zoology 4*: 193–212.

King, C.M.; Flux, M.; Innes, J.G.; Fitzgerald, B.M. 1996: Population biology of small mammals in Pureora forest park: 1. Carnivores (*Mustela erminea, M. furo, M. nivalis,* and *Felis catus*). *New Zealand Journal of Ecology 20*: 241–251.

King, C.M.; McMillan, C.D. 1982: Population structure and dispersal of peak year cohorts of stoats (*Mustela erminea*) in two New Zealand forests, with especial reference to control. *New Zealand Journal of Ecology 5*: 59–66.

King, C.M.; Moody, J.E. 1982: The biology of the stoat (*Mustela erminea*) in the national parks of New Zealand. *New Zealand Journal of Zoology 9*: 49–144.

King, C.M.; Moors, P.J. 1979: The life history tactics of mustelids, and their significance for predator control and conservation in New Zealand. *New Zealand Journal of Zoology 6*: 619–622.

King, C.M.; O'Donnell, C.F.J.; Phillipson, S.M. 1994: Monitoring and control of Mustelids on conservation lands. 2. Field and workshop guide. *Department of Conservation Technical Series 4*. 36 p.

Kirkpatrick, J.F.; Turner, J.W.; Liu, I.K.M.; Fayrer-Hosken, R. 1996: Applications of pig zona pellucida immunocontraception to wildlife fertility control. *Journal of Reproduction and Fertility Supplement 50*: 183–189.

Kopein, K.I. 1967: Analysis of the age structure of ermine populations. Pp. 158–169 *in*: King, C.M. (ed.) 1975: *Biology of mustelids: Some Soviet research*. British Library Lending Division, Boston Spa.

Kostro, K.; Wojcicka-Lorenowicz, K.; Siemionek, J. 1999: Aleutian disease of mink. *Medycyna Weterynaryjna 55*: 423–429.

Krebs, C.J. 1989: *Ecological Methodology*. Harper Collins, New York. 654 p.

Krebs, J.W.; Smith, J.S.; Rupprecht, C.E.; Childs, J.E. 1999: Rabies surveillance in the United States during 1998. *Journal of the American Veterinary Medicine Association 214*: 1713–1728.

Laakkonen, J.; Sundell, J.; Soveri, T. 1998: Lung parasites of least weasels in Finland. *Journal of Wildlife Diseases 34*: 816–819.

Laberee, E.E. 1941: Breeding and reproduction in fur bearing animals. *Fur Trade Journal of Canada 1941*: 1–166.

Lagerkvist, G. 1992: Selection for fertility, body size and pelt quality in mink and effects of crossing. *Norwegian Journal of Agricultural Sciences, Supplement 9*: 39–48.

Lagerkvist, G. 1997: Economic profit from increased litter size, body weight and pelt quality in mink (*Mustela vison*). *Acta Agriculturae Scandinavica, Section A, Animal Sciences 47*: 57–63.

Lagerkvist, G.; Einarsson, E.J.; Forsberg, M.; Gustafsson, H. 1992: Profiles of oestradiol-17ß and progesterone and follicular development during the reproductive season in mink *(Mustela vison)*. *Journal of Reproduction and Fertility 94*: 11–21.

Lagerkvist, G.; Johansson, K.; Lundeheim, N. 1994: Selection for litter size, body weight, and pelt quality in mink (*Mustela vison*): correlated responses. *Journal of Animal Science 72*: 1126–1137.

Lagerkvist, G.; Tauson, A.-H. 1993: Effect of selection on digestibility and carcass composition in mink. *Archives of Animal Nutrition 45*: 155–160.

Landa, A.; Tommeras, B.A. 1997: A test of aversive agents on wolverines. *Journal of Wildlife Management 61*: 510–516.

Langeveld, J.P.M.; Kamstrup, S.; Uttenthal, A.; Strandbygaard, B.; Vela, C.; Dalsgaard, K.; Beekman, N.J.C.M.; Meloen, R.H.; Casal, J.I. 1995: Full protection in mink against mink enteritis virus with new-generation canine parvovirus vaccines based on synthetic peptide or recombinant protein. *Vaccine 13*: 1033–1037.

Larivière, S.; Jolicoeur, H.; Crête, M. 2000: Status and conservation of the gray wolf *Canis lupus* in wildlife reserves of Québec. *Biological Conservation 94*: 143–151.

Lavrov, N.P. 1941: Methods for forecasting population changes in the ermine (*Mustela erminea* L.). *Trudi Tsentral'noi Laboratorii Biologii i Ochotaichego Promisla 5*: 60–77. Bureau of Animal Population Translation 131, Elton Library, University of Oxford.

Lavrov, N.P. 1944: Effect of helminth invasions and infectious disease on variations in numbers of the ermine. Pp. 170–187 *in*: King, C.M. (ed.) 1975: *Biology of mustelids: some Soviet research.* British Library Lending Division, Boston Spa.

Lavrov, N.P. 1956: Characteristics and causes of the prolonged depression in numbers of the ermine in forest steppe and steppe zones of USSR. Pp. 188–215 *in*: King, C.M. (ed.) 1975: *Biology of mustelids: some Soviet research*. British Library Lending Division, Boston Spa.

Lederman, J.D.; Overton, K.M.; Hofmann, N.E.; Moore, B.J.; Thornton, J.; Erdman, J.W. 1998: Ferrets (*Mustela putorius furo*) inefficiently convert beta-carotene to vitamin A. *Journal of Nutrition 128*: 271–279.

Leonard, A.H. 1966: *Modern mink management*. Ralston Purina Co., St. Louis. 206 p. Library of Congress LCCN #67-3437.

Lewis, J.W. 1967: Observations on the skull of Mustelidae infected with the nematode *Skrjabingylus nasicola*. *Journal of Zoology, London 153*: 561–564.

Li, X.; Fox, J.G.; Erdman, S.E.; Lipman, N.S.; Murphy, J.C. 1996: Cystic urogenital anomalies in ferrets (*Mustela putorius furo*). *Veterinary Pathology 33*: 150–158.

Li, X.T.; Fox, J.G.; Padrid, P.A. 1998: Neoplastic diseases in ferrets: 574 cases (1968–1997). *Journal of the American Veterinary Medical Association 212*: 1402–1407.

Lindquist, E.E.; Wu, K.W.; Redner, J.H. 1999: A new species of the tick genus *Ixodes* (Acari: Ixodidae) parasitic on mustelids (Mammalia: Carnivora) in Canada. *Canadian Entomologist 131*: 151–170.

Linhart, S.B.; Dasch, G.J.; Roberts, J.D.; Savarie, P.J. 1997: Test methods for determining the efficiency of coyote attractants, and repellents. Pp. 114–122 *in*: Jackson, W.B.; Marsh, R.E. (eds) *Test methods for vertebrate pest control and management plans*. ASTM Special Technical Publication 625. American Society for Testing and Materials, Philadelphia.

Lloyd, M.H.; Wood, C.M. 1996: Synovial sarcoma in a ferret. *Veterinary Record 139*: 627–628.

Lockie, J.D. 1966: Territory in small carnivores. *Symposia of the Zoological Society of London 18*: 143–165.

Lund, B.O.; Orberg, J.; Bergman, A.; Larsson, C.; Bergman, A.; Backlin, B.M.; Hakansson, H.; Madej, A. 1999: Chronic and reproductive toxicity of a mixture of 15 methylsulfonyl-polychlorinated biphenyls and 3-methylsulfonyl-2,2-bis-(4-chlorophenyl)-1,1-dichloroethene in mink (*Mustela vison*). *Environmental Toxicology and Chemistry 18*: 292–298.

Lund, M.; Rasmussen, A.M. 1986: Secondary poisoning hazards to stone martens (*Martes foina*) fed bromadiolone-poisoned mice. *Nordisk Veterinaer Medicin 38*: 241–243.

MacLennan, R.R.; Bailey, E.D. 1969: Seasonal changes in aggression, hunger, and curiosity in ranch mink. *Canadian Journal of Zoology 47*: 1395–1404.

MAFF. 1987: Bovine tuberculosis in badgers, 11th Report. Ministry of Agriculture, Fisheries and Food, London.

Mardon, D.K.; Moors, P.J. 1977: Records of fleas collected from weasels (*Mustela nivalis*) in North-east Scotland. (Siphonaptera: Hystrichopsyllidae and Ceratophyllidae). *Entomologists Gazette 28*: 277–280.

Marks, C.A.; Nijk, M.; Gigliotti, F.; Busana, F.; Short, R.V. 1996: Preliminary field assessment of a cabergoline baiting campaign for reproductive control of the red fox (*Vulpes vulpes). Wildlife Research 23*: 161–168.

McAllister, M.; Wills, R.A.; McGuire, A.M.; Jolley, W.R.; Tranas, J.D.; Williams, E.S.; Lindsay, D.S.; Bjorkman, C.; Belden, E.L. 1999: Ingestion of *Neospora caninum* tissue cysts by *Mustela* species. *International Journal for Parasitology 29*: 1531–1536.

McCall, J.W. 1998: Dirofilariasis in the domestic ferret. *Seminars in Veterinary Medicine and Surgery-Small Animal 13*: 109–112.

McCleery, R.H.; Clobert, J.; Julliard, R.; Perrins, C.M. 1996: Nest predation and delayed cost of reproduction in the great tit. *Journal of Animal Ecology 65*: 96–104.

McCleery, R.H.; Perrins, C.M. 1991: Evidence of the impact of predators on the population of great tits (*Parus major*) in Wytham wood. Pp. 129–147 *in*: Perrins, C.M.; LeBreton, J.D.; Hirons, G.J.M. (eds) *Bird population studies: Their relevance for conservation and management.* Oxford University Press, Oxford.

McDonald, R.A. 1998: The effects of wildlife management on stoats (*Mustela erminea*) and weasels (*Mustela nivalis*) in Great Britain. PhD Thesis, University of Bristol.

McDonald, R.A.; Harris, S. 1999: The use of trapping records to monitor populations of stoats *Mustela erminea* and weasels *M. nivalis*: the importance of trapping effort. *Journal of Applied Ecology 36*: 679–688.

McDonald, R.A.; Harris, S.; Turnbull, G.; Brown, P.; Fletcher, M. 1998: Anticoagulant rodenticides in stoats (*Mustela erminea*) and weasels (*M. nivalis*) in England. *Environmental Pollution 103*: 17–23.

McDonald, R.A.; King, C.M. in press: Stoat. *In*: Harris, S. (ed.) *Handbook of British Mammals*. 4th edn. T. & A.D. Poyser, London.

McDonald, R.A.; Murphy, E.C. 2000: A comparison of the management of stoats and weasels in Great Britain and New Zealand. Pp. 21–40 *in*: Griffiths, H.I. (ed.) *Mustelids in a modern world*. Backhuys Publishers, Leiden.

McKenna, P.B.; Cooke, M.M.; Harper, P.R. 1996: *Filaroides* infections in wild ferrets (*Mustela putorius*) and stoats (*Mustela erminea*). *New Zealand Veterinary Journal 44*: 203.

McKenna, R.; Olson, N.H.; Chipman, P.R.; Baker, T.S.; Booth, T.F.; Christensen, J.; Aasted, B.; Fox, J.M.; Bloom, M.E.; Wolfinbarger, J.B.; Agbandje-McKenna, M. 1999: Three-dimensional structure of Aleutian mink disease parvovirus: Implications for disease pathogenicity. *Journal of Virology 73*: 6882–6891.

McKenzie, D.; Bartz, J.C.; Marsh, R.F. 1996: Transmissible mink encephalopathy. *Seminars in Virology 7*: 201–206.

McLennan, J.A.; Potter, M.A.; Robertson, H.A.; Wake, G.C.; Colbourne, R.; Dew, L.; Joyce, L.; McCann, A.J.; Miles, J.; Miller, P.J.; Reid, J. 1996: Role of predation in the decline of kiwi, *Apteryx* spp., in New Zealand. *New Zealand Journal of Ecology 20*: 27–35.

Mead, R.A. 1986: Role of the corpus-luteum in controlling implantation in mustelid carnivores. *Annals of the New York Academy of Sciences 476*: 25–35.

Mead, R.A. 1993: Embryonic diapause in vertebrates. *Journal of Experimental Zoology 266*: 629–641.

Mead, R.A.; Neirinckx, S. 1990: Photomanipulation of sexual maturation and breeding cycle of the steppe polecat (*Mustela eversmanni*) and other techniques for more rapid propagation of the species. *Journal of Experimental Zoology 255*: 232–238.

Meslin, F.X.; Fishbein, D.B.; Matter, H.C. 1994: Rationale and prospects for rabies elimination in developing countries. Pp. 1–26 *in*: Rupprecht, C.E.; Dietzschold B.; Koprowski, H. (eds) *Lyssaviruses*. Springer-Verlag, New York.

Michna, S.W.; Campbell, R.S.F. 1970: Leptospirosis in wild animals. *Journal of Comparative Pathology 80*: 101–106.

Minami, M.; Endo, T.; Kikuchi, K.; Ihira, E.; Hirafuji, M.; Hamaue, N.; Monma, Y.; Sakurada, T.; Tan No, K.; Kisara, K. 1998: Anti-emetic effects of sendide, a peptide tachykinin NK1 receptor antagonist, in the ferret. *European Journal of Pharmacology 363*: 49–55.

Moore, D.R.J.; Breton, R.J.; Lloyd, K. 1997: The effects of hexachlorobenzene on mink in the Canadian environment: an ecological risk assessment. *Environmental Toxicology and Chemistry 16*: 1042–1050.

Moors, P.J. 1974: The annual energy budget of a weasel (*Mustela nivalis*) population in farmland. PhD Thesis, University of Aberdeen.

Morgan, M.K.; Bursian, S.J.; Rottinghaus, G.E.; Bennett, G.A.; Render, J.A.; Aulerich, R.J. 1998: Subacute and reproductive effects in mink from exposure to *Fusarium fujikuroi* culture material (M-1214) containing known concentrations of moniliformin. *Archives of Environmental Contamination and Toxicology 35*: 513–517.

Morgan, M.K.; Fitzgerald, S.D.; Rottinghaus, G.E.; Bursian, S.J.; Aulerich, R.J. 1999: Toxic effects to mink of moniliformin extracted from *Fusarium fujikuroi* culture material. *Veterinary and Human Toxicology 41*: 1–5.

Morgan, M.K.; Schroeder, J.J.; Rottinghaus, G.E.; Powell, D.C.; Bursian, S.J.; Aulerich, R.J. 1997: Dietary fumonisins disrupt sphingolipid metabolism in mink and increase the free sphinganine to sphingosine ratio in urine but not in hair. *Veterinary and Human Toxicology 39*: 334–336.

Morris, C.J.; Birks, J.D.S. 1997: Preliminary monitoring work on stoats and weasels. Pp. 39–41 *in*: The *Vincent Wildlife Trust Review of 1996*. The Vincent Wildlife Trust, London.

Müller, H. 1970: Beiträge zur Biologie des Hermelins, *Mustela erminea* Linné 1758. *Säugetierkundliche Mitteilungen 18*: 293–380.

Murphy, B.D.; Rajkumar, K.; Reyna, A.G.; Silversides, D.W. 1993: Control of luteal function in the mink (*Mustela vison*). *Journal of Reproduction and Fertility*: 181–188.

Murphy, E.C. 1992: The effects of a natural increase in food supply on a wild population of house mice. *New Zealand Journal of Ecology 16*: 33–40.

Murphy, E.; Bradfield, P. 1992: Change in diet of stoats following poisoning of rats in a New Zealand forest. *New Zealand Journal of Ecology 16*: 137–140.

Murphy, E.C.; Clapperton, B.K.; Bradfield, P.M.F.; Speed, H.J. 1998a: Effects of rat-poisoning operations on abundance and diet of mustelids in New Zealand podocarp forests. *New Zealand Journal of Zoology 25*: 315–328.

Murphy, E.C.; Clapperton, B.K.; Bradfield, P.M.F.; Speed, H.J. 1998b: Brodifacoum residues in target and non-target animals following large-scale poison operations in New Zealand podocarp-hardwood forests. *New Zealand Journal of Zoology 25*: 307–314.

Murphy, E.C.; Dowding, J.E. 1994: Range and diet of stoats (*Mustela erminea*) in a New Zealand beech forest. *New Zealand Journal of Ecology 18*: 11–18.

Murphy, E.C.; Dowding, J.E. 1995: Ecology of the stoat in *Nothofagus* forest: Home range, habitat use and diet at different stages of the beech mast cycle. *New Zealand Journal of Ecology 19*: 97–109.

Murphy, E.C.; Robbins, L.; Young, J.B.; Dowding, J.E. 1999: Secondary poisoning of stoats after an aerial 1080 poison operation in Pureora Forest, New Zealand. *New Zealand Journal of Ecology 23*: 175–182.

Murray, D.L.; Kapke, C.A.; Evermann, J.F.; Fuller, T.K. 1999: Infectious diseases and the conservation of free-ranging large carnivores. *Animal Conservation 2*: 241–254.

Mustafa, M.R.; Hongo, K.; Othman, I.; White, E.; Orchard, C.H. 1994: The effect of toxins from the jellyfish *Chironex fleckeri* on cardiac muscle isolated from rat and ferret hearts. *Journal of Physiology, London 475P*: P87.

Nicolaus, L.K.; Cassel, J.F.; Carlson, R.B.; Gustavson, C.R. 1983: Taste-aversion conditioning of crows to control predation on eggs. *Science 220*: 212–214.

Nicolaus, L.K.; Nellis, D.W. 1987: The 1st evaluation of the use of conditioned taste-aversion to control predation by mongooses upon eggs. *Applied Animal Behaviour Science 17*: 329–346.

Nieto, J.M.; Vázquez, S.; Quiroga, M.I.; López-Peña, M.; Guerrero, F.; Gruys, E. 1995: Spontaneous AA-amyloidosis in mink (*Mustela vison*). Description of eight cases, one of which exhibited intracellular amyloid deposits in lymph node macrophages. *European Journal of Veterinary Pathology 1*: 99–103.

Niezgoda, M.; Briggs D.J.; Shaddock J.; Dreeden, D.W.; Rupprecht C.E. 1997: Pathogenesis of experimentally induced rabies in domestic ferrets. *American Journal of Veterinary Research 58*: 1327–1331.

Niezgoda, M.; Briggs, D.J.; Shaddock, J.; Rupprecht, C.E. 1998: Viral excretion in domestic ferrets (*Mustela putorius furo*) inoculated with a raccoon rabies isolate. *American Journal of Veterinary Research 59*: 1629–1632.

Nimon, A.J.; Broom, D.M. 1999: The welfare of farmed mink (*Mustela vison*) in relation to housing and management: A review. *Animal Welfare 8*: 205–228.

Noli, C.; van der Horst, H.H.A.; Willemse, T. 1996: Demodicosis in ferrets (*Mustela putorius furo*). *Veterinary Quarterly 18*: 28–31.

Norbury, G. 2000: The potential for biological control of stoats (*Mustela erminea*). *New Zealand Journal of Zoology 27*: 145–163.

Nunan, C.P.; MacInnes, C.D.; Bachmann, P.; Johnston, D.H.; Watt, I.D. 1994: Background prevalence of tetracycline-like fluorescence in teeth of free ranging red foxes (*Vulpes vulpes*), striped skunks (*Mephitis mephitis*) and raccoons (*Procyon lotor*) in Ontario, Canada. *Journal of Wildlife Diseases 30*: 112–114.

Nyholm, E.S. 1959: Stoats and weasels in their winter habitat. Pp. 118–131 in: King, C.M. (ed.) 1975: *Biology of mustelids: some Soviet research*. British Library Lending Division, Boston Spa.

O'Crowley, K.; Wilson, J.G. 1991: Feral mink (*Mustela vison*) and their potential as disease vectors in Ireland—an investigation in Co. Wicklow. *Irish Veterinary Journal 44*: 71–74.

O'Donnell, C.F.J.; Dilks, P.J.; Elliott, G.P. 1996: Control of a stoat (*Mustela erminea*) population irruption to enhance mohua (yellowhead) (*Mohoua ochrocephala)* breeding success in New Zealand. *New Zealand Journal of Zoology 23*: 279–286.

Oksanen, T. 1983: Prey caching in the hunting strategy of small mustelids. *Acta Zoologica Fennica 174*: 197–199.

Oksanen, T.; Oksanen, L.; Fretwell, S.D. 1985: Surplus killing in the hunting strategy of small predators. *American Naturalist 126*: 328–346.

Olofsson, A.; Mittelholzer, C.; Berndtsson, L.T.; Lind, L.; Mejerland, T.; Belak, S. 1999: Unusual, high genetic diversity of Aleutian mink disease virus. *Journal of Clinical Microbiology 37*: 4145–4149.

Page, R.J.C.; Langton, S.D. 1996: The occurrence of ixodid ticks on wild mink *Mustela vison* in England and Wales. *Medical and Veterinary Entomology 10*: 359–364.

Parker, J.S.L.; Parrish, C.R. 1997: Canine parvovirus host range is determined by the specific conformation of an additional region of the capsid. *Journal of Virology 71*: 9214–9222.

Parrish, C.R. 1995: Molecular epidemiology of parvoviruses. *Seminars in Virology 6*: 415–418.

Parrish, C.R. 1999: Host range relationships and the evolution of canine parvovirus. *Veterinary Microbiology 69*: 29–40.

Phillips, P.H.; O'Callaghan, M.G.; Moore, E.; Baird, R.M. 1987: Pedal *Sarcoptes scabiei* infestation in ferrets (*Mustela putorius furo*). *Australian Veterinary Journal 64*: 289–290.

Platonow, N.S.; Karstad, L.H. 1973: Dietary effects of polychlorinated biphenyls on mink. *Canadian Journal of Comparative Medicine 37*: 391–400.

Plummer, P.J.G. 1954: Rabies in Canada, with special reference to wildlife reservoirs. *Bulletin of the World Health Organization 10*: 767–774.

Popov, V.A. 1943: Numerosity of *Mustela erminea* as affected by *Skrjabingylus* worms invasion. *Comptes Rendus, Académie des Sciences, Paris 39*: 160-162.

Pounds, C.J. 1981: Niche overlap in sympatric populations of stoats (*Mustela erminea*) and weasels (*Mustela nivalis*) in northeast Scotland. PhD Thesis, University of Aberdeen.

Powell, D.C.; Bursian, S.J.; Bush, C.R.; Render, J.A.; Rottinghaus, G.E.; Aulerich, R.J. 1996: Effects of dietary exposure to fumonisins from *Fusarium moniliforme* culture material (M-1325) on the reproductive performance of female mink. *Archives of Environmental Contamination and Toxicology 31*: 286–292.

Powell, R.A. 1979: Mustelid spacing patterns: variations on a theme by *Mustela*. *Zeitschrift für Tierpsychologie 50*: 153–165.

Powell, R.A. 1993: *The fisher: life history, ecology, and behavior*. 2nd edn. University of Minnesota Press, Minneapolis. 237 p.

Powell, R.A.; King, C.M. 1997: Variation in body size, sexual dimorphism and age-specific survival in stoats, *Mustela erminea* (Mammalia: Carnivora), with fluctuating food supplies. *Biological Journal of the Linnean Society 62*: 165–194.

Progulske, D.R. 1969: Observations of a penned, wild-captured black-footed ferret. *Journal of Mammalogy 50*: 619–621.

Pybus, M.J. 1988: Rabies and rabies control in striped skunks *(Mephitis mephitis)* in three prairie regions of western North America. *Journal of Wildlife Diseases 24*: 434–449.

Quiroga, M.I.; Lopez-Pena, M.; Vazquez, S.; Nieto, J.M. 1997: Distribution of Aujeszky's disease virus in experimentally infected mink (*Mustela vison*). *Deutsche Tierarztliche Wochenschrift 104*: 147–150.

Rademacher, U.; Jakob, W.; Bockhardt, I. 1999: *Cryptosporidium* infection in beech martens (*Martes foina*). *Journal of Zoo and Wildlife Medicine 30*: 421–422.

Ragg, J.R.; Moller, H.; Waldrup, K.A. 1995a: The prevalence of bovine tuberculosis (*Mycobacterium bovis*) infections in feral populations of cats (*Felis catus*), ferrets (*Mustela furo*) and stoats (*Mustela erminea*) in Otago and Southland, New Zealand. *New Zealand Veterinary Journal 43*: 333–337.

Ragg, J.R.; Waldrup, K.A.; Moller, H. 1995b: The distribution of gross lesions of tuberculosis caused by *Mycobacterium bovis* in feral ferrets (*Mustela furo*) from Otago, New Zealand. *New Zealand Veterinary Journal 43*: 338–341.

Ramos-Vara, J.A.; Dubey, J.P.; Watson, G.L.; Winn-Elliot, M.; Patterson, J.S.; Yamini, B. 1997: Sarcocystosis in mink (*Mustela vison*). *Journal of Parasitology 83*: 1198–1201.

Rausch, R. 1958: Some observations of rabies in Alaska, with special reference to wild Canidae. *Journal of Wildlife Management 22*: 246–260.

Raymond, M.; Robitaille, J.F.; Lauzon, P.; Vaudry, R. 1990: Prey-dependent profitability of foraging behaviour of male and female ermine, *Mustela erminea*. *Oikos 58*: 323–328.

Reading, M.J.; Field, H.J. 1999: Detection of high levels of canine herpes virus-1 neutralising antibody in kennel dogs using a novel serum neutralisation test. *Research in Veterinary Science 66*: 273–275.

Restum, J.C.; Bursian, S.J.; Millerick, M.; Render, J.A.; Merrill, A.H.; Wang, E.; Rottinghaus, G.E.; Aulerich, R.J. 1995: Chronic toxicity of fumonisins from *Fusarium moniliforme* culture material (m-1325) to mink. *Archives of Environmental Contamination and Toxicology 29*: 545–550.

Reynolds, J.C.; Tapper, S.C. 1996: Control of mammalian predators in game management and conservation. *Mammal Review 26*: 127–156.

Ritchie, J.W. 1953: Raising marten for twenty-four years. *Fur Trade Journal of Canada 30*: 10.

Robertson, H.A.; Colbourne, R.M.; Graham, P.J.; Miller, P.J.; Pierce, R.J. 1999: Survival of brown kiwi (*Apteryx mantelli*) exposed to brodifacoum poison in Northland, New Zealand. *New Zealand Journal of Zoology 23*: 225–232.

Robinson, M.M.; Hadlow, W.J.; Knowles, D.P.; Huff, T.P.; Lacy, P.A.; Marsh, R.F.; Gorham, J.R. 1995: Experimental infection of cattle with the agents of transmissible mink encephalopathy and scrapie. *Journal of Comparative Pathology 113*: 241–251.

Robitaille, J.F. 1989: Distribution spatio-temporelle des activités de l'hermine, *Mustela erminea* L., en captivité. PhD Thesis, University of Montreal.

Robitaille, J.F.; Baron, G. 1987: Seasonal changes in the activity budget of captive ermine, *Mustela erminea* L. *Canadian Journal of Zoology 65*: 2864–2871.

Robitaille, J.F.; Raymond, M. 1995: Spacing patterns of ermine, *Mustela erminea* L., in a Quebec agrosystem. *Canadian Journal of Zoology 73*: 1827–1834.

Robson, G. 1998: The breeding ecology of curlew *Numenius arquata* on north Pennine moorland. PhD Thesis, University of Sunderland.

Rockett, J.; Seville, R.S.; Kingston, N.; Williams, E.S.; Thorne, E.T. 1990: A cestode, *Taenia mustelae*, in the black-footed ferret (*Mustela nigripes*) and the white-tailed prairie dog (*Cynomys leucurus*) in Wyoming. *Journal of the Helminthological Society of Washington 57*: 160–162.

Rosen, M.N. 1981: Pasteurellosis. Pp. 244–252 *in*: Davis, J.W.; Karstad, L.H.; Trainer, D.O. (eds): *Infectious diseases of wild mammals*. 2nd edn. Iowa State University Press, Ames, Iowa.

Rosenbaum, M.R.; Affolter, V.K.; Usborne, A.L.; Beeber, N.L. 1996: Cutaneous epitheliotropic lymphoma in a ferret. *Journal of the American Veterinary Medical Association 209*: 1441–1445.

Rowlands, I.W. 1972: Reproductive studies in the stoat. *Journal of Zoology, London 166*: 574–576.

Rupprecht, C.E.; Smith, J.S. 1994: Raccoon rabies—the re-emergence of an epizootic in a densely populated area. *Seminal Virology 5*: 155–164.

Rupprecht, C.E.; Smith, J.S.; Fekadu, M.; Childs, J.E. 1995: The ascension of wildlife rabies: a cause for public health concern or intervention? *Emerging Infectious Diseases 1*: 107–114.

Rusiniak, K.W.; Gustavson, C.R.; Hankins, W.G.; Garcia, J. 1976: Prey-Lithium aversions. II: Laboratory rats and ferrets. *Behavioral Biology 17*: 73–85.

Sandell, M. 1986: Movement patterns of male stoats *Mustela erminea* during the mating season: differences in relation to social status. *Oikos 47*: 63–70.

Sandell, M.; Liberg, O. 1992: Roamers and stayers: a model on mating tactics and mating systems. *American Naturalist 139*: 177–189.

Sanderson, G.C. 1949: Growth and behavior of a litter of captive long-tailed weasels. *Journal of Mammalogy 30*: 412–415.

Sanford, S.E. 1988: Tyrosinemia-II (pseudodistemper) in mink. *Canadian Veterinary Journal 29*: 298–299.

Sato, H.; Ihama, Y.; Inaba, T.; Yagisawa, M.; Kamiya, H. 1999a: Helminth fauna of carnivores distributed in north-western Tohoku, Japan, with special reference to *Mesocestoides paucitesticulus* and *Brachylaima tokudai*. *Journal of Veterinary Medical Science 61*: 1339–1342.

Sato, H.; Inaba, T.; Ihama, Y.; Kamiya, H. 1999b: Parasitological survey on wild Carnivora in north-western Tohoku, Japan. *Journal of Veterinary Medical Science 61*: 1023–1026.

Savarie, P.J.; Sterner, R.T. 1979: Evaluation of toxic collars for selective control of coyotes that attack sheep. *Journal of Wildlife Management 43*: 780–783.

Schneider, R.R.; Hunter, D.B. 1993a: A survey of the causes of mortality in adult mink, with emphasis on the lactation period. *Canadian Veterinary Journal 34*: 103–108.

Schneider, R.R.; Hunter, D.B. 1993b: Nursing disease in mink—clinical and post-mortem findings. *Veterinary Pathology 30*: 512–521.

Schneider, R.R.; Hunter, D.B.; Waltnertoews, D. 1992: Nursing disease in mink—individual-level epidemiology. *Preventive Veterinary Medicine 14*: 167–179.

Schoo, G.; Pohlmeyer, K.; Stoye, M. 1994: A contribution to the helminth fauna of the stone marten (*Martes foina* Erxleben 1777). *Zeitschrift für Jagdwissenschaft 40*: 84–90.

Schreiber, A.; Wirth, R.; Riffel, M.; Van Rompaey, H. 1989: *Weasels, civets, mongooses and their relatives. An action plan for the conservation of mustelids and viverrids*. IUCN, Gland, Switzerland. 100 p.

Schultheiss, P.C.; Dolginow, S.Z. 1994: Granulomatous enteritis caused by *Mycobacterium avium* in a ferret. *Journal of the American Veterinary Medical Association 204*: 1217–1218.

Scott, S.A.J. 1982: The weasel (*Mustela nivalis*). *Journal of the Institute of Animal Technicians 33*: 29–33.

Segal, A.N. 1972: Ecological thermoregulation in the American mink. *Soviet Journal of Ecology 3*: 453–456.

Serfass, T.L.; Brooks, R.P.; Swinley, T.J.; Rymon, L.M.; Hayden, A.H. 1996: Considerations for capturing, handling, and translocating river otters. *Wildlife Society Bulletin 24*: 25–31.

Serfass, T.L.; Randall, L.P.; Whary, M.T.; Brooks, R.P. 1993: River otter (*Lutra canadensis)* reintroduction in Pennsylvania: prerelease care and clinical evaluation. *Journal of Zoo and Wildlife Medicine 24*: 28–40.

Serfass, T.L.; Rymon, L.M.; Brooks, R.P. 1992: Ectoparasites from river otters in Pennsylvania. *Journal of Wildlife Diseases 28*: 138–140.

Shore, R.F.; Birks, J.D.S.; Freestone, P.; Kitchener, A.C. 1996: Second generation rodenticides and polecats (*Mustela putorius*) in Britain. *Environmental Pollution 91*: 279–282.

Short, H.L. 1961: Food habits of a captive least weasel *Mustela rixosa* Bangs. *Journal of Mammalogy 42*: 273.

Sidorovich, V.E.; Savcenko, V.V. 1992: The effect of pollution on the population of the American mink (*Mustela vison*). *Semiaquatische Säugetiere 1992*: 305–315.

Simms, D.A. 1979: Studies of an ermine population in southern Ontario. *Canadian Journal of Zoology 57*: 824–832.

Sleeman, D.P. 1988: *Skrjabingylus nasicola* (Leuckhart) (Metastrongylidae) as a parasite of the Irish stoat. *Irish Naturalists Journal 22*: 525–527.

Sleeman, D.P. 1989: Ectoparasites of the Irish stoat. *Medical and Veterinary Entomology 3*: 213–218.

Sleeman, D.P. 1991: Home ranges of Irish stoats. *Irish Naturalists Journal 23*: 486–488.

Smal, C.M. 1991: Population studies on feral American mink *Mustela vison* in Ireland. *Journal of Zoology, London 224*: 233–249.

Smith, D.D.; Frenkel, J.K. 1995: Prevalence of antibodies to *Toxoplasma gondii* in wild mammals of Missouri and east central Kansas—biologic and ecologic considerations of transmission. *Journal of Wildlife Diseases 31*: 15–21.

Smits, J.E.G.; Blakley, B.R.; Wobeser, G.A. 1996a: Immunotoxicity studies in mink (*Mustela vison*) chronically exposed to dietary bleached kraft pulp mill effluent. *Journal of Wildlife Diseases 32*: 199–208.

Smits, J.E.G.; Haines, D.M.; Blakley, B.R.; Wobeser, G.A. 1996b: Enhanced antibody responses in mink (*Mustela vison*) exposed to dietary bleached-kraft pulp mill effluent. *Environmental toxicology and Chemistry 15*: 1166–1170.

Song, J.H.; Houde, A.; Murphy, B.D. 1998a: Cloning of leukemia inhibitory factor (LIF) and its expression in the uterus during embryonic diapause and implantation in the mink (*Mustela vison*). *Molecular Reproduction and Development 51*: 13–21.

Song, J.H.; Sirois, J.; Houde, A.; Murphy, B.D. 1998b: Cloning, developmental expression, and immunohistochemistry of cyclooxygenase-2 in the endometrium during embryo implantation and gestation in the mink (*Mustela vison*). *Endocrinology 139*: 3629–3636.

Spurr, E.B. 1997: Assessment of the effectiveness of Transonic™ ESP and Yard Guard™ ultrasonic devices for repelling stoats (*Mustela erminea*). *Conservation Advisory Science Notes 151*. Department of Conservation, Wellington, New Zealand.

Steinel, A.; Munson, L.; van Vuuren, M.; Truyen, U. 2000: Genetic characterization of feline parvovirus sequences from various carnivores. *Journal of General Virology 81*: 345–350.

Stephenson, B.M.; Minot, E.O.; Armstrong, D.P. 1999: Fate of moreporks (*Ninox novaeseelandiae*) during a pest control operation on Mokoia Island, Lake Rotorua, North Island, New Zealand. *New Zealand Journal of Zoology 23*: 233–240.

Stevens, R.T.; Ashwood, T.L.; Sleeman, J.M. 1997: Mercury in hair of muskrats (*Ondatra zibethicus*) and mink (*Mustela vison*) from the U. S. Department of Energy Oak Ridge Reservation. *Bulletin of Environmental Contamination and Toxicology 58*: 720–725.

Storgaard, T.; Oleksiewicz, M.; Bloom, M.E.; Ching, B.; Alexandersen, S. 1997: Two parvoviruses that cause different diseases in mink have different transcription patterns: Transcription analysis of mink enteritis virus and Aleutian mink disease parvovirus the same cell line. *Journal of Virology 71*: 4990–4996.

Stringer, J.R.; Cushion, M.T. 1998: II. The genome of *Pneumocystis carinii*. *FEMS Immunology and Medical Microbiology 22*: 15–26.

Suchentrunk, F.; Willig, R.; Hartl, G.B. 1991: On eye lens weights and other age criteria of the brown hare (*Lepus europaeus* Pallas, 1778). *Zeitschrift für Säugetierkunde 56*: 365–374.

Sundqvist, C.; Amador, A.G.; Bartke, A. 1989: Reproduction and fertility in the mink (*Mustela vison*). *Journal of Reproduction and Fertility 85*: 413–441.

Sundqvist, C.; Ellis, L.C.; Bartke, A. 1988: Reproductive endocrinology of the mink (*Mustela vison*). *Endocrine Reviews 9*: 247–266.

Sundqvist, C.; Gustafsson, M. 1983: Sperm test—a useful tool in breeding work of mink. *Journal of the Scientific Agricultural Society of Finland 55*: 119–131.

Sutherland-Smith, M.R.; Rideout, B.A.; Mikolon, A.B.; Appel, M.J.G.; Morris, P.J.; Shima, A.L.; Janssen, D.J. 1997: Vaccine-induced canine distemper in European mink, *Mustela lutreola*. *Journal of Zoo and Wildlife Medicine 28*: 312–318.

Tapper, S. 1982: Using estate records to monitor population trends in game and predator species, particularly weasels and stoats. *International Congress of Game Biologists 14*: 115–120.

Tapper, S. 1992: *Game Heritage*. Game Conservancy Ltd., Fordingbridge. 140 p.

Tapper, S.C.; Green, R.E.; Rands, M.R.W. 1982: Effects of mammalian predators on partridge populations. *Mammal Review 12*: 159–167.

Taylor, T.G.; Carpenter, J.L. 1995: Thymoma in two ferrets. *Laboratory Animal Science 45*: 363–365.

Ternovsky, D.V. 1983: Biology of reproduction and development of *Mustela erminea* (Carnivora, Mustelidae). *Zoologichesky Zhurnal 62*: 1097–1105.

Thompson, H. 1968: British wild mink. *Annals of Applied Biology 61*: 345–349.

Thorne, E.T.; Schroeder, M.H.; Forrest, S.C.; Campbell, T.M. III; Richardson, L.; Biggins, D.; Hanebury, L.R.; Belitsky, D.; Williams, E.S. 1985: Pp. 9.1–9.8 *in*: Anderson, S.H.; Inkley, D.B. (eds) Capture, immobilization, and care of black-footed ferrets for research. *Proceedings of the black-footed ferret workshop, University of Wyoming, Laramie*.

Tkachenko, E.A.; Ivanov, A.P.; Donets, M.A.; Miasnikov, Y.A.; Ryltseva, E.V.; Gaponova, L.K.; Bashkirtsev, V.N.; Okulova, N.M.; Drozdov, S.G.; Slonova, R.A.; Somov, G.P.; Astakhova, T.I. 1983: Potential reservoir and vectors of haemorrhagic fever with renal syndrome (HFRS) in the USSR. *Annales de la Societe Belge de Medecine Tropicale 63*: 267–269.

Tkadlec, E. 1993: Crimidine hazards to scavengers. *Folia Zoologica 42*: 289–293.

Tomson, F.N. 1987: Mink. *Veterinary Clinics of North America*: *Small Animal Practice 17*: 1145–1153.

Townsend, M.G.; Bunyan, P.J.; Odam, E.M.; Stanley, P.I.; Wardall, H.P. 1984: Assessment of secondary poisoning hazard of warfarin to least weasels. *Journal of Wildlife Management 48*: 628–632.

Tuljapurkar, S.; Caswell, H. (eds) 1997: *Structured population models in marine, terrestrial, and freshwater systems*. Chapman & Hall, New York. 643 p.

Twigg, G.M.; Cuerden, C.M.; Hughes, D.M. 1968: Leptospirosis in British wild mammals. *Symposia of the Zoological Society of London 24*: 75–98.

Uttenthal, A.; Lund, E.; Hansen, M. 1999: Mink enteritis parvovirus—Stability of virus kept under outdoor conditions. *APMIS 107*: 353–358.

van Driesche, R.G.; Bellows, T.S. 1996: *Biocontrol*. Chapman & Hall, New York. 539 p.

van Moll, P.; Alldinger, S.; Baumgartner, W.; Adami, M. 1995: Distemper in wild carnivores—an epidemiologic, histological and immunocytochemical study. *Veterinary Microbiology 44*: 193–199.

van Soest, R.W.M.; van Bree, P.J.H. 1970: Sex and age composition of a stoat population (*Mustela erminea* Linnaeus, 1758) from a coastal dune region of the Netherlands. *Beaufortia 17*: 51–77.

van Soest, R.W.M.; van der Land, J.; van Bree, P.J.H. 1972: *Skrjabingylus nasicola* (Nematoda) in skulls of *Mustela erminea* and *Mustela nivalis* (Mammalia) from the Netherlands. *Beaufortia 20*: 85–97.

Vaudry, R.; Raymond, M.; Robitaille, J.F. 1990: The capture of voles and shrews by male and female ermine *Mustela erminea* in captivity. *Holarctic Ecology 13*: 265–278.

Venge, O. 1959: Reproduction in the fox and mink. *Animal Breeding Abstracts 27*: 129–145.

Venkova, K.; Palmer, J.M.; Greenwood-van Meerveld, B. 1999: Nematode-induced jejunal inflammation in the ferret causes long-term changes in excitatory neuromuscular responses. *Journal of Pharmacology and Experimental Therapeutics 290*: 96–103.

Wakefield, A.E. 1998: I. Genetic heterogeneity in *Pneumocystis carinii*: An introduction. *FEMS Immunology and Medical Microbiology 22*: 5–13.

Wamberg, S. 1994: Rates of heat and water loss in female mink (*Mustela vison*) measured by direct calorimetry. *Comparative Biochemistry and Physiology 107A*: 451–458.

Wamberg, S.; Clausen, T.N.; Olesen, C.R.; Hansen, O. 1992: Nursing sickness in lactating mink *(Mustela vison)* II. Pathophysiology and changes in body fluid composition. *Canadian Journal of Veterinary Research 56*: 95–101.

Weber, J.M.; Mermod, C. 1985: Quantitative aspects of the life cycle of *Skrjabingylus nasicola*, a parasitic nematode of the frontal sinuses of mustelids. *Zeitschrift für Parasitenkunde 71*: 631–638.

Welchman, D.D.; Oxenham, M.; Done, S.H. 1993: Aleutian disease in domestic ferrets—diagnostic findings and survey results. *Veterinary Record 132*: 479–484.

Wells, G.A.H.; Keymer, I.F.; Barnett, K.C. 1989: Suspected Aleutian disease in a wild otter (*Lutra lutra*). *Veterinary Record 125*: 232–235.

Wetzler, T.F. 1981: Pseudotuberculosis. Pp. 253–262 *in*: Davis, J.W.; Karstad, L.H.; Trainer, D.O. (eds) *Infectious diseases of wild mammals*. 2nd edn. Iowa State University Press, Ames.

Williams, B.H.; Chimes, M.J.; Gardiner, C.H. 1996: Biliary coccidiosis in a ferret (*Mustela putorius furo*). *Veterinary Pathology 33*: 437–439.

Williams, C.; Elnif, J.; Buddington, R.K. 1998: The gastrointestinal bacteria of mink (*Mustela vison* L.): Influence of age and diet. *Acta Veterinaria Scandinavica 39*: 473–482.

Williams, E.S.; Anderson, S.L.; Cavender, J.; Lynn, C.; List, K.; Hearn, C.; Appel, M.J.G. 1996: Vaccination of black-footed ferret (*Mustela nigripes*) x Siberian polecat (*M. eversmanni*) hybrids and domestic ferrets (*M. putorius furo*) against canine distemper. *Journal of Wildlife Diseases 32*: 417–423.

Williams, E.S.; Mills, K.; Kwiatkowski, D.R.; Thorne, E.T.; Boergerfields, A. 1994: Plague in a black-footed ferret (*Mustela nigripes*). *Journal of Wildlife Diseases 30*: 581–585.

Williams, E.S.; Thorne, E.T. 1996: Infectious and parasitic diseases of captive carnivores, with special emphasis on the black-footed ferret (*Mustela nigripes*). *Revue Scientifique et Technique de l'Office International des Epizooties 15*: 91–114.

Williams, E.S.; Thorne, E.T.; Appel, M.J.G.; Belitsky, D.W. 1988: Canine distemper in black-footed ferrets (*Mustela nigripes*) from Wyoming. *Journal of Wildlife Diseases 24*: 385-398.

Williams, E.S.; Thorne, E.T.; Quan, T.J.; Anderson, S.L. 1991: Experimental-infection of domestic ferrets (*Mustela putorius furo*) and Siberian polecats (*Mustela eversmanni*) with *Yersinia pestis*. *Journal of Wildlife Diseases 27*: 441–445.

Wilson, N.; Zarnke, R.L. 1985: Occurrence of the ear canker mite, *Otodectes cynotis* (Hering), on the wolverine, *Gulo gulo* (L.). *Journal of Wildlife Diseases 21*: 180.

Wilson, P.R.; Karl, B.J.; Toft, R.J.; Beggs, J.R.; Taylor, R.H. 1998: The role of introduced predators and competitors in the decline of kaka (*Nestor meridionalis*) populations in New Zealand. *Biological Conservation 83*: 175–185.

Wobeser, G.; Swift, M. 1976: Mercury poisoning in a wild mink. *Journal of Wildlife Diseases 12*: 335–340.

Wood, R.L.; Shuman, R.D. 1981: *Erysipelothrix* infection. Pp. 297–305 *in*: Davis, J.W.; Karstad, L.H.; Trainer, D.O. (eds) *Infectious diseases of wild mammals*. 2nd edn. Iowa State University Press, Ames, Iowa.

Wren, C.D. 1987: Toxic substances in furbearers. Pp. 930–936 *in*: Nowak, R.; Baker, J.E.; Obbard, M.E.; Malloch, B. (eds) *Wild furbearer management and conservation in North America.* Ontario Ministry of Natural Resources, Toronto.

Wren, C.D.; Stokes, P.M.; Fischer, K.L. 1986: Mercury levels in Ontario mink and otter relative to food levels and environmental acidification. *Canadian Journal of Zoology 64*: 2854–2859.

Wright, P.L. 1948: Breeding habits of captive long-tailed weasels *(Mustela frenata). American Midland Naturalist 39*: 338–344.

Yamini, B.; Bursian, S.J.; Aulerich, R.J. 1997: Pathological effects of dietary zearalenone and/or tamoxifen on female mink reproductive organs. *Veterinary and Human Toxicology 39*: 74–78.

Yanai, T.; Tomita, A.; Masegi, T.; Ishikawa, K.; Iwasaki, T.; Yamazoe, K.; Ueda, K. 1995: Histopathologic features of naturally occurring hepatozoonosis in wild martens (*Martes melampus*) in Japan. *Journal of Wildlife Diseases 31*: 233–237.

Yerbury, H. 1947: Raising marten in captivity. *Fur Trade Journal of Canada 25*: 14.

Zabiega, M.H. 1996: Helminths of mink, *Mustela vison*, and muskrats, *Ondatra zibethicus*, in southern Illinois. *Journal of the Helminthological Society of Washington 63*: 246–250.

Zaman, S.; Woods, A.J.; Watson, J.W.; Reynolds, D.J.M.; Andrews, P.L.R. 2000: The effect of the NK1 receptor antagonist CP-99,994 on emesis and c-fos protein induction by loperamide in the ferret. *Neuropharmacology 39*: 316–323.

Zhou, Z.Y.; Nordstoga, K.; Bjerkas, I. 1992: Extraglomerular lesions in kidneys of mink with encephalitozoonosis. *Acta Veterinaria Scandinavica 33*: 33–41.

Zimmermann, H.; Witte, W. 1988: Bacteriological investigations on urolithiasis of mink. *Monatshefte für Veterinarmedizin 43*: 314–315.

Appendix

KEY ADDRESSES AND PERIODICALS

Addresses and web sites

Minnesota Zoological Garden
Apple Valley, Minnesota 55124, USA
http://www.mnzoo.com

International Species Information System (ISIS)
Nathan R. Flesness, Director
12101 Johnny Cake Ridge Road
Apple Valley, Minnesota 55124, USA
Telephone (+1) 612-431-9295
Fax (+1) 612-431-2757
E-mail isis@maroon.tc.edu
http://www.worldzoo.org

Fur Commission USA
826 Orange Avenue, #506
Coronado, CA 92118, USA
Telephone (+1) 619-575-0139
Fax (+1) 619-575-5578
E-mail furfarmers@aol.com
http://www.furcommission.com

The American Ferret Association
P.O. Box 255
Crownsville, MD 21032, USA
E-mail afa@ferret.org
http://www.ferret.org

The World Ferret Union
c/o Sabine van Voorn
Vijverstaat 30
9321 XJ Altena
Netherlands
http://home-1.worldonline.nl/~wfu/index.html

Periodicals on fur farming

Fur Farm Letter, published by Fur Commission USA, California.
Fur Animal Research, published by Mink Farmers Research Foundation, a committee of Fur Commission USA, California.
Fur Rancher, published by Becker Publishing, Minnesota