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Histological and serological evidence of disease among invasive, non-native stoats *Mustela erminea*

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Abstract

Invasive, non-native species are a major threat to global biodiversity. Stoats were introduced from Britain to New Zealand in the 1880s and have since caused grave conservation problems. A histopathological and serological survey of disease and infection in stoats from New Zealand was undertaken to identify agents that might be used or modified to control this population. Of 60 stoats examined, 63% exhibited inflammation of the lung, mostly occurring as local or diffuse interstitial pneumonia, 30% showed signs of inflammatory liver disease and 14% were positive for antibodies reactive with feline calicivirus. In Britain only 11% of 44 stoats exhibited symptoms of pulmonary inflammatory disease, suggesting higher rates of infection or compromise of the pulmonary immune system among invasive stoats, possibly related to genetic founder effects or environmental variation. These findings could be exploited in biological control programmes.

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1. Introduction

Invasive non-native species are among the greatest threats to global biodiversity (Atkinson, 1996). Non-native species often increase their populations rapidly in a novel range where they may be free of the factors, such as disease and predators, that limit populations in their native range. Alternatively, they may be exposed to new pathogens that have the potential to prevent or reduce the rate of establishment (Hilker et al., 2005). This may particularly be the case when small founder populations with reduced genetic diversity render newly established populations particularly vulnerable to novel infections (Hawley et al., 2006).

The ecological impacts of invasive species are most pronounced on islands which are often home to a diversity of

endemic species that are naïve to predation risk (Simberloff, 2000). Introduced mammals are the single gravest threat to biodiversity in New Zealand (King, 1984). Predators, such as stoats *Mustela erminea*, are a particularly serious problem since many of New Zealand's endemic birds have evolved in the absence of predators (King, 1984) and there has been considerable public expenditure on large-scale culling of stoats with the aim of reducing stoat predation (McDonald and Murphy, 2000). Given that conventional methods of stoat control are limited to trapping and poisoning which are expensive, there are moves towards identifying more cost-effective methods of long term and widespread control of stoat predation. These include conventional biological control using lethal or sub-lethal pathogens and immunocontraception, potentially using a disseminating vector that would be genetically modified from a widespread, host-specific infectious agent (Norbury, 2000; McDonald and Larivière, 2001). This clearly requires knowledge and comparison of the diseases stoats encounter in their native and non-native range.

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McDonald and Larivière (2001) provided a comprehensive review of the diseases and pathogens of stoats and other mustelids, generally in their native range, but with particular reference to the control of invasive stoats in New Zealand. Studies of the general biology of stoats in New Zealand have provided basic data on the prevalence of certain parasites, such as the nematode *Skrjabinogylus nasicola*, the adults of which infects the sinuses of various mustelids, and of ectoparasites (King and Moody, 1982). A histopathological survey of naturally occurring disease among stoats living in Britain concluded that stoat populations in their native range were remarkably healthy, since 61% of stoats exhibited no significant pathological lesions at the microscopic level (McDonald et al., 2001). Among British samples, nematode parasitism was identified in the intestines of 14% of 44 stoats and in the lungs of 11%. Pulmonary granulomatous inflammation or microgranulomas were identified in 11% and blood-filled, peliosis-like cavities in the livers of two individuals.

The objective of this study was to provide information about the incidence of disease in free-living, invasive stoats in New Zealand, compare the prevalence of disease in the non-native and native ranges and to relate this to possible founder effects. This information will also inform decisions about the future direction of research into the biological control of invasive stoats.

2. Materials and methods

Samples were collected opportunistically from conservation rangers and researchers in addition to hunters and other members of the public who trapped or shot the animals as part of their normal pest control procedures. Samples were taken from a range of locations and habitats in South Island, New Zealand, between January and June 2001. Blood and tissue samples were extracted as soon as possible after death. A range of tissues was collected and fixed in 10% neutral-buffered formalin solution, including brain, heart, lung, kidney, liver, mesenteric lymph node, thymus, spleen, stomach, duodenum, pancreas and colon. Extraction and fixation of samples was undertaken by field personnel and whole animals were not available for inspection of gross pathology or of age-structure.

A haematoxylin and eosin (HE) stained section was prepared from each tissue and was examined by light microscopy for pathological change. Where HE stains clearly indicated the presence of an infectious agent, a panel of further stains (Gram, periodic acid-Schiff and Ziehl-Neelsen), was conducted in order to provide further diagnostic information. All examinations were conducted by a Board-certified veterinary pathologist (MJD).

Sera were screened with immunofluorescence assays (IFAs) using viruses grown in various cell lines using standard laboratory protocols. lymphocytic choriomeningitis virus (LCMV) IFA slides were purchased from Charles River Diagnostics (18-well slides, SL-012) and the assay was performed as per manufacturers' protocol except that bound antibodies were detected using a goat anti-ferret immunoglobulin G fluorescein isothiocyanate isomer I conjugate (IgG-FITC, Bethyl Laboratories Inc.) used at a 1/20 dilution.

All other viral IFA antigens were produced in-house. Briefly, each virus was grown to a standardised titre in a suitable cell line (see below) in a 96-well microtitre plate, and was then fixed by the addition of 70% alcohol to each well. Prior to use, wells were washed and rehydrated in phosphate buffered saline (PBS). Each serum sample was diluted to 1 in 20 and 1 in 40 in PBS and tested in duplicate: 30 µL of serum dilutions were added to each well and incubated for 1–12 h (depending on the assay) at

37 °C in a moist atmosphere. Identical dilutions of a positive control serum standard (though not from a stoat) and a negative control serum standard were also included on each plate tested. After repeat washing with PBS, 30 µL of the anti-ferret IgG-FITC conjugate (see above), diluted 1 in 20, was added to each well and plates were reincubated at 37 °C for 1 h in a moist atmosphere. Wells were then rinsed repeatedly in PBS, then loaded with 30 µL of glycerol for examination under suitable epifluorescence at 1000× magnification. Results (positive fluorescence) were assigned to each sample by reference to the positive and negative control standards.

Viruses for use in the IFAs described above were grown in the following cell lines: canine distemper virus (morbillivirus) and cowpox (orthopoxvirus) – Vero cells, canine parvovirus-2 and canine coronavirus – A72 cells, feline calicivirus – Feline embryo (FEA) cells, canine adenoviruses I and II and canine herpesvirus – Madin Derby canine kidney (MDCK) cells.

3. Results

Tissue samples from 60 stoats and sera from 57 of these were analysed. Samples were collected from nine locations (Fig. 1). The sex ratio was not significantly different from parity (overall males:females = 1.3:1, $G_c = 0.66$, $P > 0.1$). On the whole, preservation of tissue structure was remarkably good. Samples from the intestinal tract were most affected by autolytic change, but in most cases the 'ghost outline' of the underlying tissue permitted some analysis of tissue structure. Summary comments on the appearance of the major organ systems are presented below.

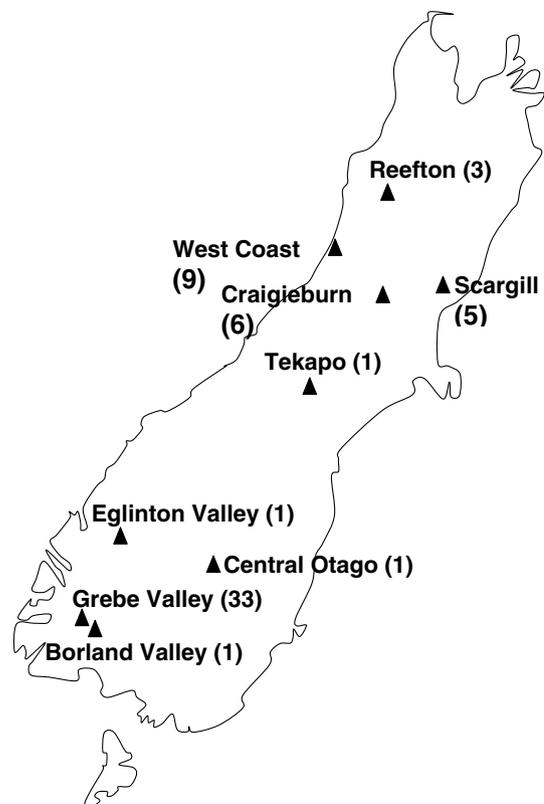


Fig. 1. Locations of sampling sites in South Island, New Zealand. Figures in parentheses are sample size.

Most lungs had evidence of either focal or diffuse congestion/haemorrhage, interpreted as agonal change. Most ($n = 38$, 63%) also had evidence of diffuse or focal interstitial pneumonia involving infiltration of neutrophils and macrophages (pyogranulomatous) (Fig. 2). In five of these cases, there was additional involvement of the bronchi with neutrophilic exocytosis into the bronchiolar lumina (bronchopneumonia). Some lungs had evidence of BALT aggregates, but this was not a consistent feature. Parasitic larvae were not identified within the pulmonary tissue. There were occasional focal and well-defined microgranulomas. One of these centred upon fragments of aspirated matter and two had large, crescentic, translucent structures at the centre reminiscent of the adiaspores of the fungus *Chrysosporium* (Fig. 3). Myocardium, endocardium and epicardium were almost invariably normal. In two stoats there was evidence of a mild and focal myocarditis – generally involving a mixed mononuclear cell infiltration.

The majority of stomachs were histologically normal. In six stoats, there were small focal granulomatous infiltrates at the base of the glandular mucosa, sometimes extending into muscularis mucosa. In several of these cases there were parasitic larvae in the centre of these aggregates, suggesting that these lesions were attributable to larval migration (Fig. 4). Sections of the duodenum, ileum, caecum and colon were largely normal. Pancreatic tissue was generally normal (both exocrine and endocrine) and it was noted that stoats have very prominent islets relative to some species. In two cases, there was evidence of interstitial pancreatitis.

The majority of samples of liver were histologically normal. Seven had evidence of diffuse hepatocyte vacuolation. Other livers had focal (centrilobular) vacuolation of hepatocytes which is a non-specific change. Occasional portal areas had a mild, mononuclear cell infiltration but this was rarely above what would be considered background

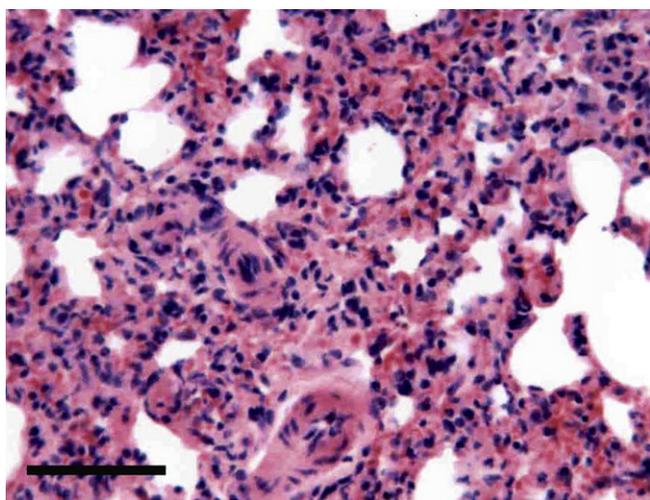


Fig. 2. Section of stoat lung parenchyma. There is loss of normal alveolar microarchitecture with infiltration of interstitial tissue by macrophages and neutrophils. Focal congestion and haemorrhage is also present. Haematoxylin and eosin, bar = 300 μ m.

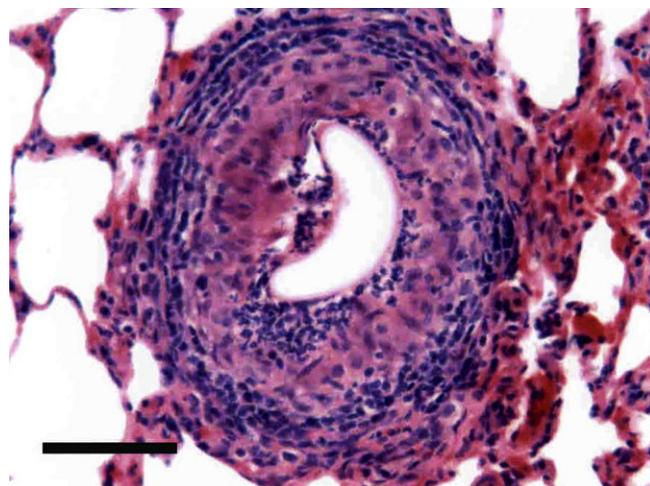


Fig. 3. Section of stoat lung. There is a distinct granuloma centred upon a translucent, crescent shaped structure consistent with an adiaspore of the fungus *Chrysosporium*. Haematoxylin and eosin, bar = 300 μ m.

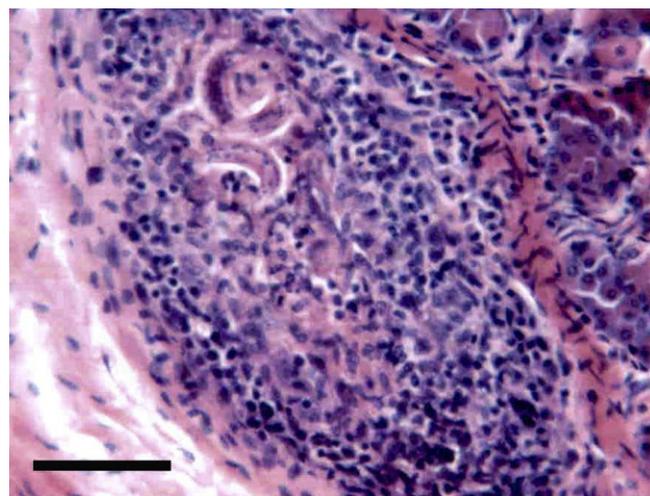


Fig. 4. Section of stoat gastric mucosa. At the base of the glandular tissue is a focal granulomatous lesion centred upon several parasitic larvae. Haematoxylin and eosin, bar = 300 μ m.

in other species. Several livers had small parenchymal (mid-zonal) foci of hepatocyte degeneration and mixed inflammation (focal hepatitis). Two had evidence of vascular telangiectasis, and in one of these cases there was a relatively large area of hepatocyte loss, blood pooling and surrounding granulomatous inflammation (Fig. 5). No significant abnormality was found in any kidney but one sample showed evidence of a single small focus of interstitial lymphoid aggregation.

Longitudinal sections of spleen were all remarkably similar in appearance and the spleen was invariably markedly active. The white pulp (lymphoid) areas were extremely prominent, often with very large secondary follicles dominating. Red pulp was not often severely congested, but was always very cellular. The cellular content of red pulp included lymphocytes, plasma cells, and megakaryocytes

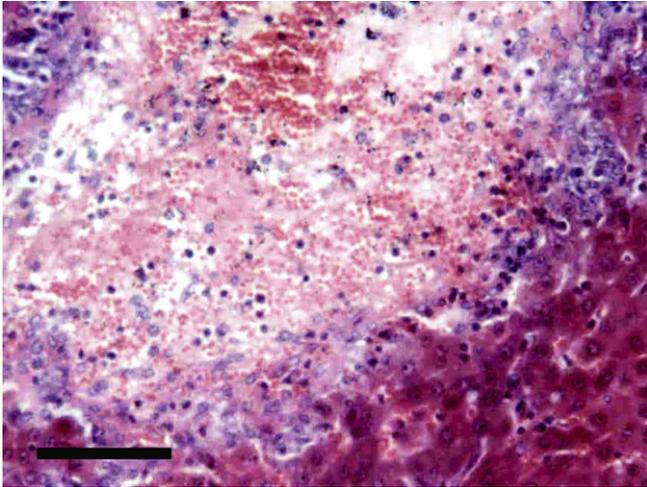


Fig. 5. Section of stoat liver. There is a large blood-filled cavity within the hepatic parenchyma, the margins of which show evidence of an early inflammatory infiltration dominated by macrophages. Haematoxylin and eosin, bar = 300 μ m.

were often prominent indicating extramedullary haematopoiesis. Also of note in red pulp was the presence of a population of very large, blastic round cells with an open-faced nucleus and prominent nucleolus. These cells may have been lymphoblastic. In one or two spleens this population dominated, and the cells were so large and pleomorphic in appearance that in other species their presence may have suggested neoplasia. Mesenteric lymph nodes were all very similar in appearance, consistent with marked immunological activity of the gut. There were very prominent secondary follicles with germinal centres and mantle zones, frequent paracortical hyperplasia and dilated medullary sinuses with sinus histiocytosis.

The brain was histologically normal in the majority of stoats, with no evidence of lesions consistent with viral infection (e.g. distemper virus). In one stoat there was a focal, granulomatous meningitis that affected the meninges ventral to hindbrain, this animal was seropositive for lymphocytic choriomeningitis virus (arenavirus), though a link between these observations is not certain. In another stoat there was mild focal gliosis within the parenchyma. Testicular structure and ovarian tissue, including the fallopian tube, were generally histologically normal. In one stoat, there were granulomatous plaques covering a number of abdominal viscera, with granulomatous disease of the lung and meninges. This animal appeared to have a systemic granulomatous inflammatory disease.

Sera from eight individuals (14%), seven of which were from one site (Grebe Valley), were positive in the feline calicivirus antibody assay. Three individuals, two of which were from a single site (Reefton), were seropositive for morbillivirus, a family that includes canine distemper virus. Two individuals were positive for parvovirus antibody, including one that was one of the three stoats that were positive for morbillivirus. Single, separate individuals were found to be positive for antibodies reactive with ortho-

poxviruses, lymphocytic choriomeningitis virus (arenavirus), and canine herpesvirus. All sera were negative for canine adenovirus and coronavirus. There was no evidence of a relationship among samples between seroprevalence of virus antibodies and histopathological evidence of disease. There was no significant relationship between the incidence of inflammation in one organ system and another ($P > 0.1$ in all cases).

4. Discussion

As in the survey of stoats in their native range (McDonald et al., 2001), many of these individuals had histologically normal tissues and were likely to have been in good health at the time of death. Opportunistic sampling, such as that used here, may not reflect the prevalence of disease among the sickest individuals in the population, since these may be less mobile and less likely to be caught or may die particularly quickly. This is a consistent but unavoidable characteristic of such screening studies of wildlife disease.

The consistently marked activity of the spleen and the prominent lymphoid areas of the spleen suggest that stoats have a functional systemic immune system and an active systemic humoral (antibody) response. In common with the British samples, no lesions of the brain were observed that would have been consistent with infection by distemper virus, though this can not be taken as evidence of the absence of this disease and three individuals in this study were seropositive for morbillivirus, which is comparable to a recent finding of two seropositive animals from a sample of 32 stoats sampled in Canterbury, New Zealand (T. Zheng, personal communication). Two cases of blood-filled cavities in the liver were observed in stoats, similar to the peliosis-like lesions observed in two British stoats. At the time, we suggested that the British samples may have been secondarily exposed to rodenticides and this remains a possibility for the New Zealand specimens (McDonald et al., 1998; Murphy et al., 1998).

Positive results for feline calicivirus were the most significant findings of the serological survey. These feline viruses are generally very host-specific and so these findings are strongly indicative of infection with a mustelid calicivirus. It is not possible at this stage completely to rule out cross-reaction with rabbit caliciviruses that include RHD, which have previously been recorded in stoats in New Zealand (Henning, 2003). However RHD is caused by a separate genus of caliciviruses *Lagovirus* whereas cat caliciviruses are *Vesivirus*. Cross-reaction across genera and such a broad host range is unlikely. Caliciviruses can cause a range of pathologies in their hosts ranging from upper respiratory tract infections in cats to gastroenteritis in humans. Four of the stoats found to be seropositive to calicivirus exhibited histopathological signs of inflammatory lung disease. However, the prevalence of these signs of disease was particularly high across all stoats examined and so it was not possible to demonstrate an association between disease and infection by calicivirus.

An earlier review of mustelid pathogens, with reference to stoats in New Zealand, (McDonald and Larivière, 2001) did not identify calicivirus in stoats, but noted the reported but unsubstantiated association of calicivirus (together with *Escherichia coli* and coronavirus) with “sticky-kit disease” in ranch mink *Mustela* (Jorgensen et al., 1996). Mink caliciviruses (MCV) have been isolated from mink on ranches with a history of haemorrhagic pneumonia (Evermann et al., 1983). However, mink enteric caliciviruses have been shown to be genetically distinct from non-enteric calicivirus (Guo et al., 2001). This is clearly, therefore, a diverse group and while it is novel that calicivirus has been detected in stoats, it is not surprising. This screening study is a first step towards evaluating the incidence and effects of calicivirus in wild stoats. However, the finding holds promise in terms of identifying potentially widespread viral infections that may have a role as biological control agents or as vectors thereof.

Our histopathological findings differed to a surprising extent from the British survey. In particular, we detected a significantly higher prevalence (63%) of pneumonia of varying degrees of severity, while pulmonary inflammatory disease was detected in only 5/44 (11%) British stoats ($\chi^2 = 26.17, P < 0.001$). While this is a screening study, it is tempting to speculate on the implications of these findings. The widespread incidence of pneumonia suggests that stoats are subject to a higher rate of infection of some agent, which may be more prevalent in New Zealand or notably less prevalent in Great Britain. Alternatively, the pulmonary immune system of stoats in New Zealand may be compromised in some way. The cause of the high incidence of pneumonia could not be detected by histopathological techniques and our serological investigation could not reveal the association of exposure to any of the tested virus groups with these signs of disease. In the development of a biological control agent or candidate vector for an immunocontraceptive, the identification of the organism causing the inflammation observed here is an obvious first step. Thereafter, a search for an organism, or strain of the same infecting organism, that invades hosts through the lungs would be especially useful.

Potential causes of pneumonia in mustelids are numerous (Fox, 1998) and there is evidence of viral-bacterial synergism in the symptoms of severe pneumonia (Jakeman et al., 1991). Mink and ferrets are also susceptible to avian influenza (Buchman et al., 1995; Englund and af Segerstad, 1998). During a review of diseases of stoats and related mustelids, McDonald and Larivière (2001) identified a range of diseases in which horizontal transmission may occur through the respiratory tract, perhaps the best known of which are tuberculosis and paratuberculosis caused by *Mycobacterium* spp. (Ragg et al., 1995; Beard et al., 1999, 2001).

Curiously, while pulmonary inflammation was common and pulmonary parasitism rare among New Zealand stoats, inflammation was rare and parasitism common in the lungs of British stoats (McDonald et al., 2001), which

is suggestive of interactions between the immune responses to different pathogens. Similarly, rates of arthropod-borne infections such as those caused by *Bartonella* spp. among New Zealand stoats are markedly lower than occur in Britain (McDonald et al., 2000, 2004), suggesting differences among pathogens in the rate at which diseases and/or their arthropod vectors were transported between native and non-native ranges. Examination of tissues from other invasive predators collected contemporaneously in New Zealand has provided an interesting comparison to samples from the stoats. Two of three weasels, a ferret and one of three feral cats examined exhibited interstitial pneumonia and granulomatous inflammation similar to that observed in the stoats (McDonald et al., 2004). Clearly, a similar range of lesions were present in these other species. This confirms the importance of investigating host specificity in the development of any putative biocontrol agent.

5. Conclusion

In summary, this survey has provided evidence of a range of tissue lesions in otherwise healthy stoats in their non-native range in New Zealand. The high frequency of pneumonia and the discovery of calicivirus among invasive stoats are perhaps the most significant findings of the study, which hold significance both as indicators of the potential relationship between genetic founder effects and susceptibility to disease and the application of these traits to the control of invasive species and their impact on biodiversity.

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