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The impact of sarcoptic mange *Sarcoptes scabiei* on the British fox *Vulpes vulpes* population

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ABSTRACT

1. Disease epizootics can significantly influence host population dynamics and the structure and functioning of ecological communities. Sarcoptic mange *Sarcoptes scabiei* has dramatically reduced red fox populations *Vulpes vulpes* in several countries, including Britain, although impacts on demographic processes are poorly understood. We review the literature on the impact of mange on red fox populations, assess its current distribution in Britain through a questionnaire survey and present new data on resultant demographic changes in foxes in Bristol, UK.

2. A mange epizootic in Sweden spread across the entire country in < 10 years resulting in a decline in fox density of up to 95%; density remained lowered for 15–20 years. In Spain, mange has been enzootic for > 75 years and is widely distributed; mange presence was negatively correlated with habitat quality.

3. Localized outbreaks have occurred sporadically in Britain during the last 100 years. The most recent large-scale outbreak arose in the 1990s, although mange has been present in south London and surrounding environs since the 1940s. The questionnaire survey indicated that mange was broadly distributed across Britain, but areas of perceived high prevalence (> 50% affected) were mainly in central and southern England. Habitat type did not significantly affect the presence/absence of mange or perceived prevalence rates. Subjective assessments suggested that populations take 15–20 years to recover.

4. Mange appeared in Bristol's foxes in 1994. During the epizootic phase (1994–95), mange spread through the city at a rate of 0.6–0.9 km/month, with a rise in infection in domestic dogs *Canis familiaris c*. 1–2 months later. Juvenile and adult fox mortality increased and the proportion of females that reproduced declined but litter size was unaffected. Population density declined by > 95%.

5. In the enzootic phase (1996–present), mange was the most significant mortality factor. Juvenile mortality was significantly higher than in the pre-mange period, and the number of juveniles classified as dispersers declined. Mange infection reduced the reproductive potential of males and females: females with advanced mange did not breed; severely infected males failed to undergo spermatogenesis. In 2004, Bristol fox population density was only 15% of that in 1994.

Keywords: Canidae, demographic changes, enzootic, epizootic, red fox, wildlife disease

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INTRODUCTION

Wild mammals act as vectors and hosts for a range of viral, bacterial and parasitic infections that can be transmitted to humans, livestock and companion animals (Williams & Barker, 2001; Samuel, Pybus & Kocan, 2001) and *vice versa* (Funk *et al*., 2001; Cleaveland *et al*., 2002; Woodroffe *et al*., 2004). From an applied perspective, the pre- and post-exposure management of such wildlife diseases can be difficult, controversial and result in significant economic costs (e.g. Flamand *et al*., 1992; Krebs & the Independent Scientific Review Group, 1997; Aubert, 1999; Donnelly *et al*., 2003), although, conversely, diseases have also been utilized in the management of problems caused by some feral or wild species (Fenner & Fantini, 1999; Bester *et al*., 2002). Furthermore, wildlife diseases can also exert significant impacts on host population dynamics (Tompkins *et al*., 2002) and therefore the structure and functioning of ecological communities. These trophic relationships are perhaps most noticeable as the result of disease epizootics, which can cause pronounced declines in the abundance of host species in space and/or time and concomitant changes in the abundance of prey species, predators and/or competitors (Sumption & Flowerdew, 1985; Danell & Hörnfeldt, 1987; McNaughton, 1992; Lindström *et al*., 1994, 1995; Selås, 1998; Moreno *et al*., 2004). In addition, epizootics also generate conditions for the rapid development of traits that allow host(s) and pathogen to coexist, i.e. epizootic phases are characterized by high mortality rates and rapid declines in host population size, whereas enzootic phases are generally typified by lower mortality rates and less pronounced effects on host population size. Disease outbreaks therefore provide an opportunity to study a number of key evolutionary and ecological processes.

However, epizootics in wild animals are not common. In particular, it is rare for outbreaks to arise in well-studied host populations, enabling comparisons of key demographic processes in the presence and absence of disease. Yet, just such a chance arose recently following an outbreak of sarcoptic mange in red foxes *Vulpes vulpes* in the city of Bristol, UK, during a long-term study of their behaviour and ecology. Furthermore, given the intense interest in foxes in Britain, we have been able to investigate the pattern of spread of mange in this country using a questionnaire distributed to animal welfare organizations and members of the general public. In this paper, we have three key aims. First, we give a brief overview of the characteristics of sarcoptic mange infection and a recent history of the disease in fox populations in Europe. Second, we present new data on the pattern of spread of the disease in mainland Britain and the city of Bristol and, third, we quantify the effect of mange on key demographic processes and individual patterns of behaviour in Bristol.

Disease characteristics of sarcoptic mange

Sarcoptic mange is a highly contagious skin infection caused by the mite *Sarcoptes scabiei* (Pence & Ueckermann, 2002). Mites burrow into the epidermis of the skin, forming tunnels into which antigenic material (e.g. eggs, faeces, shed exoskeletons, digestive secretions) is deposited, causing intense irritation to the skin. The mite lifecycle lasts *c*. 2 weeks and heavy infections can build up rapidly, with densities reaching 5000 mites/cm^2 in some species (Bornstein, Mörner & Samuel, 2001). Lightly infected individuals may suffer only short-term effects, whereas heavily infected individuals suffer hyperkeratosis, alopecia, general wastage and ultimately muscle catabolism as the host responds to the destruction of the epidermal cells in the skin (Burgess, 1994; Newman, Baker & Harris, 2002). The inflammatory response can also lead to self-inflicted physical trauma, which can result in secondary infections. Death may arise from a wide variety of causes (Arlian *et al*., 1990), including starvation and hypothermia.

Mange has been reported in 104 domestic and wild mammal species and humans (Bornstein *et al*., 2001; Pence & Ueckermann, 2002) and infection may be spread readily between species (McCarthy, 1960; Stone *et al*., 1974), but inter-species infection can be limited in other instances (Stone *et al*., 1974; Samuel, 1981). Mites are transmitted directly through contact between infected and susceptible individuals or indirectly via the environment (Arlian & Vyszenski-Moher, 1988). The potential importance of indirect transmission in the spread of the disease is illustrated by the relative ease with which it can pass between some species that have only minimal direct contact (Mörner, 1992). Transfer among sympatric species can be of conservation concern for small fragmented populations that are vulnerable to extinction (Henricksen *et al*., 1993). All parasite life-stages can move onto the outer surface of the host's skin, facilitating deposition on substrates and transmission between individuals (Bornstein *et al*., 2001). Mites can persist in the environment for long periods of time, but survival off the host is dependent on ambient environmental conditions and substrate type (Arlian, Vyszenski-Moher & Pole, 1989). Mange may also cause significant alteration of host behaviours, in some cases increasing the likelihood of transmission, but in others leading to a dramatic decline in activity (Overskaug, 1994; Skerrat, Middleton & Beveridge, 1999; Shelley & Gehring, 2002; Newman *et al*., 2003; Skerrat *et al*., 2004).

Sarcoptic mange in red foxes in Europe: a brief history

The red fox exhibits the widest geographical range of any extant carnivore and can be found in a wide diversity of habitats. Throughout much of its range it is a significant vector of diseases of importance to human and domestic animal health, including rabies and echinococcosis (e.g. Chautan, Pontier & Artois, 2000; Deplazes & Eckert, 2001; Deplazes *et al*., 2004), as well as host to a range of other parasitic infections (Smith *et al*., 2003). Red foxes also appear particularly susceptible to sarcoptic mange, and typically suffer very high parasite burdens. In naïve populations, death usually occurs within 3–4 months from first infection (Stone *et al*., 1974).

Mange epizootics have been known to cause substantial population declines in fox populations in Australia, Denmark, England, Italy, North America and Scandinavia (Storm *et al*., 1976; Mörner, 1992; Lindström *et al*., 1994; Baker *et al*., 2000; Forchhammer & Asferg, 2000; Newman *et al*., 2002). For example, sarcoptic mange spread rapidly across Scandinavia in the 1970s–80s, initially travelling from Estonia through Finland before becoming firmly established in Sweden *c*. 1975 (Henriksson, 1972; Borg *et al*., 1976; Mörner, 1992), from where it spread into Norway during the early 1980s (Mörner, 1992). At a regional level, mange first appeared in scattered local populations before spreading through intervening areas (Lindström, 1991), with the rate of spread being slower in high-density habitats (Lindström & Mörner, 1985). Overall, population decline was in the region of 50–90% (Lindström, 1991; Mörner, 1992), and populations in Sweden did not begin to recover until the late 1980s (Lindström *et al*., 1994). The dramatic reduction in fox numbers had substantial effects on community dynamics, leading to an apparent increase in the abundance of a number of other species (Danell & Hörnfeldt, 1987; Lindström *et al*., 1994, 1995; Selås, 1998). Whilst the density of fox litters declined significantly, litter size did not appear to change during the epizootic (Lindström, 1991).

In contrast to the outbreak in Scandinavia, based on oral history, sarcoptic mange appears to have been present in Spanish fox populations since the 19th century. In Spain it has a broad geographical distribution but is locally absent, being present in only 22.5% of localities sampled (Gortázar *et al*., 1998). Furthermore, there appears to be no direct relationship between mange prevalence and altitude, rainfall or prey (rabbit *Oryctolagus cuniculus*) abun-

dance (Gortázar *et al*., 1998). The overall prevalence of mange was higher in areas of low fox density and was linked to poorer quality habitat (Gortázar *et al*., 1998); in other species, increased sarcoptic mange infection is often coincident with food shortages or when the expression of immunity is reduced due to the prioritization of nutrition for other purposes such as reproduction or lactation (Skerrat *et al*., 1999; Fthenakis *et al*., 2001).

At least five outbreaks of mange have been reported in Britain in the last century: Somerset, 1906; the south coast of England, 1914; south-west England, 1918; west Midlands and Wales, 1931; and the Sussex/Surrey border and London continuously since the 1940s (Vesey-Fitzgerald, 1965; Teagle, 1967; Burrows, 1968; Lloyd, 1980). The earlier outbreaks tended to be small and localized, and in some instances were associated with mounted foxhunts buying and releasing foxes: this need to release foxes for hunting suggests that foxes were locally very rare and this is likely to have limited the spread of mange over larger areas.

The most recent large-scale outbreak of mange in Britain appears to have arisen in the early 1990s, having spread to a large proportion of England and Wales in just a few years (Simpson, 2002, 2003; Bates, 2003; Clayton, 2003). However, it is clear that mange did not first appear at this time, as has been suggested by some authors (Simpson, 2002; Bates, 2003). It is known to have been present in London since at least the 1940s, and dramatically reduced fox numbers in some parts of the city in the 1970s and was also present in parts of Kent and Surrey at that time (S. Harris, unpublished data). Yet, it did not radiate significantly until the 1990s, and the reasons for this failure to spread are unclear. One factor widely stated in the popular press as possibly contributing to this outbreak was the large number of foxes living in urban areas at that time, and which were described as being in poor nutritional status and more susceptible to mange infection: a particularly widely disseminated point of view was that urban foxes obtained the majority of their food by scavenging from dustbins, and that the introduction of 'wheelie bins' (large refuse containers that foxes are not able to gain access to) had severely lowered food availability.

Yet, such assertions are unfounded. First, foxes in urban areas very rarely rifle dustbins (Harris, 1981a). The majority of their food consists of wild rodents and birds, invertebrates, fruit and food deliberately supplied by householders (Harris, 1981b; Doncaster, Dickman & Macdonald, 1990; Saunders *et al*., 1993; Baker *et al*., 2000; Ansell, 2004). Thus, the introduction of wheelie bins would not have lowered the amount of food available. Second, urban foxes are not in poor nutritional status. Prior to the outbreak of mange, of those animals caught or recovered dead as part of the Bristol fox study (see below), none was thought to have died directly of starvation (although some had starved as a result of, e.g. disease or other injury), and many had visible fat reserves, indicating that they were obtaining more than enough food to meet their immediate nutritional requirements. What is clear is that the majority of urban fox populations in Britain at the time of the mange epizootic in the mid-1990s were at reasonably high densities and had not previously been exposed to the disease, such that it spread rapidly once it reached a conurbation, with spread between conurbations mediated by foxes in rural areas. Foxes in urban areas are also more readily visible than in rural areas, potentially giving the biased impression that the disease is more rife than in rural populations. Fortuitously, however, this increased visibility has facilitated the study of mange in Bristol's foxes.

The Bristol fox population has been studied continuously since 1977 (see Methods for study protocols). Of over 4500 foxes caught or recovered dead within the city between 1977 and 1993, none had sarcoptic mange. The first known case of mange involved a subadult (6–12 months) male that dispersed out of the north-west of the city in winter 1993, and then returned to his natal group in spring 1994. During winter 1993, he was sighted in the rural fringes to the west

of the city by a member of the public (radio-collars were marked to aid individual identification) where sarcoptic mange was believed to be prevalent in the rural fox population and was the probable source of infection. He was subsequently recaptured in Bristol in May 1994 showing advanced stages of the disease, and was euthanased (Baker *et al*., 2000).

As far as we are aware, this is the only instance where a population of red foxes has been studied intensively prior to, during and following an outbreak of sarcoptic mange. Consequently, this has given us an unparalleled opportunity to quantify the effect of mange on (i) fox density; (ii) mortality and juvenile recruitment; (iii) female reproduction; (iv) male reproductive potential; and (v) dispersal. In addition, we summarize the temporal and spatial pattern of mange prevalence and spread in Britain using data generated by a postal questionnaire.

METHODS

Spread of mange in mainland Britain

Information on the pattern of spread of mange in Britain was obtained from a questionnaire survey distributed by The Mammal Society, UK, in 2001 to all its members and the members of four other organizations with an interest in fox welfare: (i) The Fox Project, a charitable organization based in south-east England that runs a wildlife ambulance network for sick and injured foxes, and which also provides an advisory service on problems caused by foxes; (ii) the League Against Cruel Sports, a national organization that campaigns for the abolition of 'blood-sports'; (iii) the National Fox Welfare Society, a national organization based in Northamptonshire that treats sick and injured foxes and which provides an advisory service on problems caused by foxes; and (iv) the Universities Federation for Animal Welfare, a national charity that promotes the welfare of animals as pets, in zoos, laboratories, on farms and in the wild. Additional questionnaires were sent to inspectors from the Royal Society for the Prevention of Cruelty to Animals and the Scottish Society for the Prevention of Cruelty to Animals. A website run by The Mammal Society was used to collect data from non-members.

Each questionnaire requested information on: habitat (city, town, village, woodland, moorland, heathland, pastoral, arable); the frequency with which foxes were observed by the respondent (every week, every month, every 3 months, every 6 months, less than every 6 months); the current abundance of foxes in the respondent's area (very common, common, uncommon, rare, unknown); whether mange was present at the current time (yes, no, unknown); when (month and year) mange arrived, if applicable; fox abundance prior to the arrival of mange (very common, common, uncommon, rare, unknown); the pattern of change in the fox population prior to the arrival of mange (increasing, decreasing, stable, unknown); and the prevalence of mange in the population at the present time (< 25% infected, 25–50% infected, 50–75% infected; > 75% infected). Finally, each respondent was asked to indicate how the fox population prior to the arrival of mange had changed at each of three time periods: within (i) 2 and (ii) 5 years of mange arriving, and (iii) the current situation. For each time period, respondents were asked whether the pre-mange population had increased, decreased by < 25%, decreased by 25–50%, decreased by 50–75%, decreased by > 75%, remained unchanged, or they did not know. Due to small sample sizes in some categories, habitats were grouped as urban (city, town, village), semi-natural habitats (woodland, moorland, heathland) and agricultural (pastoral, arable) habitats, or urban and non-urban (semi-natural, agricultural) habitats.

For analyses, respondents were assigned to one of 11 regions: south-west England, south-central England, south-east England, eastern England, central England, western England,

north-west England, north-east England, Wales, Greater London and Scotland. The proportion of respondents stating mange was present in each habitat in each region was compared using a Friedman test. Mean perceived prevalence in each habitat was calculated by assigning scores of 1–4 to the respective prevalence classes listed on the questionnaire (< 25% infected, 25–50% infected, 50–75% infected; > 75% infected), and dividing the sum of these responses by the total number of respondents answering this question.

To investigate the time taken for populations to recover following an outbreak, we considered respondents' perceptions of current population size relative to population size at the time mange first arrived at their locality. Data were coded as: increased = 6, remained unchanged = 5, decreased by < 25% = 4, decreased by 25–50% = 3, decreased by 50–75% = 2 and decreased by > 75% = 1. Mean scores were compared at four time periods: 0–5 years, 6–10 years, 11–20 years and > 20 years since mange arrived.

Bristol's foxes

The Bristol study commenced in 1977 with the principal goals of determining fox distribution across the city and quantifying key demographic parameters such as survival, fecundity and dispersal rates (e.g. Harris, 1981a; Harris & Smith, 1987; Trewhella, Harris & McAllister, 1988). From 1990, the study has focused on the behaviour and ecology of a small number of social groups in the north-west of the city that originally covered an intensively studied area of approximately 1.5 km^2 (Baker *et al.*, 1998, 2000). Between 1992 and 1994 juveniles and adults from a surrounding area of approximately 14 km^2 were also captured. Since the outbreak of mange the study area has been expanded to cover this larger area because of the dramatic decline in fox numbers.

Three key principle methods have been utilized throughout the study: (i) a capture-mark-recapture programme; (ii) radio-tracking; and (iii) necropsy of animals recovered dead. Adult and juvenile animals were captured using cage-traps placed in residential gardens (Baker *et al.*, 2001a): young cubs (< 3 months) were also captured by flushing them into nets from den sites. Foxes captured in extremely poor condition were humanely euthanased according to Schedule 1 of the UK Animal (Scientific Procedures) Act 1986. All animals were marked with numbered ear tags (Rototags©, Dalton Supplies Ltd, Henley-on-Thames, Oxfordshire, UK). Following the outbreak of mange, individuals were marked with unique colour combinations of tags to aid identification in the field. Animals were aged by incisor wear (Harris, 1978). All animals were assumed to have been born on 1 April each year (Harris & Trewhella, 1988). Cubs, subadults and adults are defined as animals aged 0–6, 6–12 and > 12 months, respectively. Collectively, cubs and subadults are referred to as juveniles.

Trapped foxes and cadavers were allocated to one of three classes of infection based upon hair loss and presence/absence of hyperkeratonic crusts: (i) uninfected; (ii) Class I mange – no hyperkeratosis present; and (iii) Class II mange – hyperkeratosis present (Newman *et al.*, 2002). Patterns of prevalence of mange in animals captured in each season were used to define pre- and mange-affected periods: the mange-affected period was further divided into epizootic and enzootic periods.

Disease spread

The pattern of spread of mange through the Bristol fox population was estimated from (i) reports of infected individuals by resident householders; and (ii) the recovery of dead individuals. Throughout the mange-affected period, we also regularly received telephone calls from members of the general public asking for advice concerning the treatment of and the risk posed by foxes in their garden, in particular to their pet dogs; dogs (*Canis familiaris*) are

susceptible to mange infection (Bornstein *et al.*, 2001) and, on the basis of the phone calls received, it appeared that transfer from foxes to dogs was occurring. Therefore, we also (iii) analysed veterinary records for patterns of infection in domestic dogs over the period January 1990–December 1996. Veterinary surgeons operating within the city limits were contacted and asked whether we could collate their records on the date and address of dogs treated for mange. These three data sources were then merged to give an indication of the extent of the area of the city affected by mange over time. In addition, the pattern of coincidence between mange in foxes and in dogs was determined by non-parametric rank correlations between (i) the number of cases of mange in foxes and in dogs in the same month in the north-west of the city; (ii) the number of cases in foxes and in dogs 1 month later (i.e. a time lag of 1 month from foxes to dogs); and (iii) the number of cases in dogs and in foxes 1 month later (time lag of 1 month from dogs to foxes).

Mortality and juvenile recruitment

The mean recruitment rate of juveniles (i.e. survival of animals in their first year) was compared between the pre-mange, epizootic and enzootic phases using data for those animals captured and tagged as cubs; to increase sample sizes, we have used cub mortality data from the whole city for the years 1980–93 inclusive. For each cohort of cubs we quantified the number of animals known to have died as a juvenile (J) and the number known to have survived to adulthood (A); juvenile mortality rate was calculated as (J/(J + A)). Differences in annual juvenile mortality rates between the pre-mange and enzootic periods were compared using Mann–Whitney *U*-tests: data from the epizootic period were excluded as this only lasted 2 years.

Change in density

Full-grown foxes were fitted with radio-collars, and foxes were tracked according to a standard 8-hour regime with locations (fixes) taken every 5 minutes (White & Harris, 1994). Tracking data were collected and analysed on a seasonal basis, with seasons defined to encompass key periods of the annual reproductive cycle: spring, March–May (birth and nutritional dependence of the cubs); summer, June–August (attainment of nutritional independence); autumn, September–November (onset of dispersal period); winter, December–February (main dispersal period and mating season). Asymptotic home range estimates were derived from a minimum of 200 (pre-mange period) or 250 (mange-affected period) active fixes.

The density of resident adult animals was assessed seasonally from those data on home range size and minimum group size. Minimum group size was assessed using information from collared individuals, observations of unmarked animals during tracking sessions, recovery of dead individuals and information received from resident householders. See Baker *et al.* (2000) for details of criteria used to assign animals to social groups.

Female reproduction

The impact of mange on female reproduction was determined by comparing litter size and the proportion of females breeding in the pre-mange, epizootic and enzootic periods. Litter size was estimated from capture and observational data after cubs had emerged from the natal den: where two females had bred on the same territory in the same year, the total number of cubs present was divided by two.

The proportion of females breeding was calculated from individuals captured between February and June each year from 1990 to 1999. Females were examined for physical signs

of breeding such as pregnancy, loss of belly fur and/or lactation. The number of breeding and non-breeding females in each of the time periods was compared using a chi-squared test. The effect of mange severity on the likelihood of females breeding was determined by comparing the number of females that did and did not breed in the pre-mange, epizootic and enzootic periods; these differences were considered in the context of severity of infection.

Male reproductive potential

The possible impact of mange on male reproductive potential was investigated by comparing mean testes mass for uninfected males with that for animals with Class I and Class II mange recovered dead in London during the breeding season (January) during 1971–77. To determine whether males infected with mange were capable of reproducing, their mean testes mass was compared with that of uninfected males outside the breeding season (February–December inclusive), since spermatogenesis results in an increase in testes mass during the breeding season.

Dispersal

Individuals tagged as cubs and subsequently recovered dead as a subadult or recaptured/recovered dead as an adult were classified as dispersing if they were recovered or recaptured > 1 territory diameter from the location where they had been tagged as a cub: this critical distance varied between years in accordance with average home range size as determined by radio tracking. The proportion of males and females classified as dispersers was calculated for each year from 1980 to 1993 inclusive: for the enzootic period, data from all years were pooled due to small sample sizes.

RESULTS

Spread of mange in mainland Britain

A total of 2311 questionnaires were completed, although not all respondents answered all questions. In broad terms, mange was present near the English–Welsh border and in and around London prior to 1985 before spreading westwards from London along the south coast (Fig. 1). By 2001, mange was broadly distributed across Britain (Fig. 1), but was reported as present by fewer respondents in northern regions and east of the Pennines (Table 1). The highest estimates of prevalence (> 50% of the population infected) were reported in central and southern areas of England (Fig. 1d).

Within regions, a greater proportion of respondents stated mange was present in urban and semi-natural habitats than agricultural habitats (Table 1), although this difference was not quite significant (Friedman test: $\chi^2_2 = 5.28$, $P = 0.07$; Greater London excluded because no responses for agricultural habitats were recorded). There was no significant difference in the mean perceived prevalence scores between urban and non-urban habitats in each region (Wilcoxon matched-pairs test: $Z = -0.93$, $P > 0.05$; Table 2), but there was a significant difference in the perceived change in fox abundance at different times following an outbreak of mange (Kruskal–Wallis test: $\chi^2_3 = 14.35$, $P < 0.01$), with respondents stating that populations took > 20 years to recover to pre-mange levels (Fig. 2).

Bristol's foxes

Spread of mange in Bristol's foxes

Following the initial detection of the disease in spring 1994, the proportion of individuals infected with mange at capture on the study site had increased to 55% by autumn 1994, rising to 100% in autumn/winter 1995 and then declining to 15–30% in 1996 (Table 3). This pattern

Fig. 1. Temporal changes in the distribution of mange in Britain based on a questionnaire survey. Points indicate the position of respondents stating mange was present: (a) prior to 1985; (b) up to 1990; (c) in 2001; and (d) in 50–100% of foxes in 2001.

describes three distinct phases: a pre-epizootic (pre-mange) phase up to and including winter 1993; an epizootic phase lasting from spring 1994 to winter 1995; and an enzootic phase from spring 1996 onwards (Newman, 2001).

On the basis of information derived from householders, veterinary surgeons and the recovery of cadavers, mange had spread to an area of approximately 21 km^2 by July 1994 and the entire city (144 km^2) by March 1995. Taking 1 May 1994 as time zero (the first infected animal was re-sighted for the first time on 8 May following his initial disappearance) and assuming a circular rate of spread from a focal point, these figures equate to rates of spread of 0.9 and 0.6 km/month, respectively. There was a significant positive correlation between the number of cases of mange in foxes and in dogs in the same month (Spearman rank correlation: $r_s = 0.793$, $P < 0.001$, $n = 24$) (Fig. 3). This correlation was marginally stronger for the number of cases in dogs 1 month later ($r_s = 0.796$, $P < 0.001$, $n = 23$) and weaker for the number of cases in dogs in the preceding month ($r_s = 0.604$, $P < 0.01$, $n = 23$), consistent with mange being generally spread from foxes to dogs rather than *vice versa*. Furthermore,

Table 1. Results of a questionnaire survey showing the pattern of perceived mange presence in Britain by region and habitat

Region	Urban habitats	Semi-natural habitats	Agricultural habitats	Total
South-west England	86% (29)	92% (12)	62% (29)	77% (70)
South-central England	83% (349)	75% (28)	68% (34)	81% (411)
South-east England	78% (291)	66% (35)	60% (30)	75% (356)
Eastern England	0% (11)	33% (3)	0% (12)	4% (26)
Central England	47% (58)	50% (2)	45% (11)	46% (71)
Western England	87% (113)	45% (11)	54% (24)	78% (148)
North-west England	83% (40)	100% (1)	44% (9)	76% (50)
North-east England	49% (41)	15% (13)	17% (12)	36% (66)
Wales	67% (9)	33% (3)	43% (14)	50% (26)
Greater London	84% (225)	100% (6)	–	84% (231)
Scotland	31% (16)	0% (18)	10% (10)	14% (44)
Mean	*63% (1182)*	*55% (132)*	*37% (185)*	*56% (1499)*

Figures are the percentage of respondents who reported the presence of mange (figures in parentheses are the number of respondents answering this question).

Table 2. Results of a questionnaire survey showing the mean perceived mange prevalence scores in urban and semi-natural habitats in Britain

Region	Urban habitats	Semi-natural habitats	Total
South-west England	2.1 (17)	2.0 (18)	2.0 (35)
South-central England	1.7 (245)	1.9 (40)	1.8 (285)
South-east England	1.7 (201)	1.8 (32)	1.7 (233)
Eastern England	–	–	–
Central England	1.6 (19)	1.3 (7)	1.5 (26)
Western England	2.3 (82)	1.6 (14)	2.2 (96)
North-west England	2.1 (28)	2.0 (5)	2.1 (33)
North-east England	2.2 (16)	2.3 (4)	2.2 (20)
Wales	1.8 (6)	1.0 (6)	1.8 (12)
Greater London	1.8 (161)	1.5 (6)	1.8 (167)
Scotland	1.3 (4)	2.0 (1)	1.4 (5)
Mean	*1.7 (779)*	*1.6 (133)*	*1.6 (912)*

Prevalence scores were calculated by assigning values of 1–4 for the respective categories of mean perceived mange prevalence of < 25%, 25–50%, 50–75% and > 75% on the questionnaire and dividing by the number of respondents answering this question (figures in parentheses indicate the number of respondents answering this question).

mange may take 4–6 weeks to form visible signs of infection in foxes and to become infective (Bornstein, Zakrisson & Thebo, 1995), but is likely to be detected much earlier in pet dogs (e.g. because of increased scratching). Consequently, the detection of mange in dogs is likely to have occurred once foxes had progressed to Class II infection, further suggesting that transmission was predominantly from foxes to dogs.

Mortality and juvenile recruitment

Of those animals resident in the main study groups as of spring 1990 and those animals subsequently captured and tagged during spring 1990–winter 2003 inclusive ($n = 618$), 243

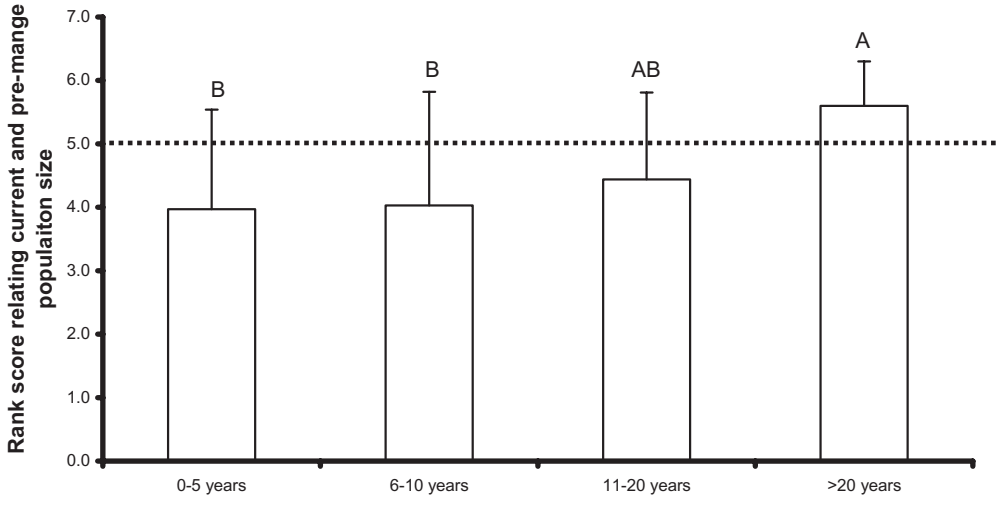

Fig. 2. Mean (± S.D.) respondent ($n = 368$) scores for the relationship between current fox population size relative to fox abundance at the time when mange first appeared in the population. Scores were calculated by assigning values to the respective categories outlined on the questionnaire (6 = increased, 5 = unchanged, 4 = decreased by < 25%, 3 = decreased by 25–50%, 2 = decreased by 50–75% and 1 = decreased by > 75%) and dividing by the number of respondents answering this question. Dotted line indicates a score equivalent to 'no change'. Letters denote *post hoc* groupings (after Siegel & Castellan, 1988).

Table 3. Prevalence of mange in tagged foxes in Bristol from summer 1994 to autumn/winter 1996

	Adults & subadults		Cubs		
Season	♂	♀	♂	♀	Total
Summer 1994	0.00 (7)	0.00 (2)	0.00 (5)	0.00 (6)	0.00 (20)
Autumn 1994	0.67 (15)	0.20 (5)	– (–)	– (–)	0.55 (20)
Winter 1994	0.78 (27)	0.56 (13)	– (–)	– (–)	0.65 (40)
Spring 1995	1.00 (11)	0.92 (26)	0.35 (23)	0.20 (15)	0.61 (75)
Summer 1995	1.00 (10)	1.00 (21)	0.43 (7)	0.90 (10)	0.90 (48)
Autumn/winter 1995	1.00 (1)	1.00 (8)	– (–)	– (–)	1.00 (9)
Spring/summer 1996	0.00 (1)	1.00 (2)	0.00 (8)	0.00 (2)	0.15 (13)
Autumn/winter 1996	0.25 (4)	0.33 (9)	– (–)	– (–)	0.31 (13)

Figures are the proportion of animals captured exhibiting signs of mange infection (sample sizes are in parentheses).

were recovered dead. In the pre-mange period, road traffic accidents were the commonest cause of death, accounting for 62% of known mortality (Table 4). During the epizootic and enzootic phases, mange was the most commonly recorded cause of death accounting for 64% and 56% of identified deaths, respectively. However, the cause of death could not be identified for 30% and 39% of the animals recovered in these two periods, respectively, generally because the animals were recovered in an advanced state of decomposition. Furthermore, animals infected with mange often seek shelter in the terminal stages of the disease, including in buildings and in underground refugia (Overskaug, 1994; Wydeven *et al.*, 2003), thereby limiting the degree to which they can be recovered. Consequently, these figures should be viewed as minimum estimates of the relative importance of mange as a mortality factor. On

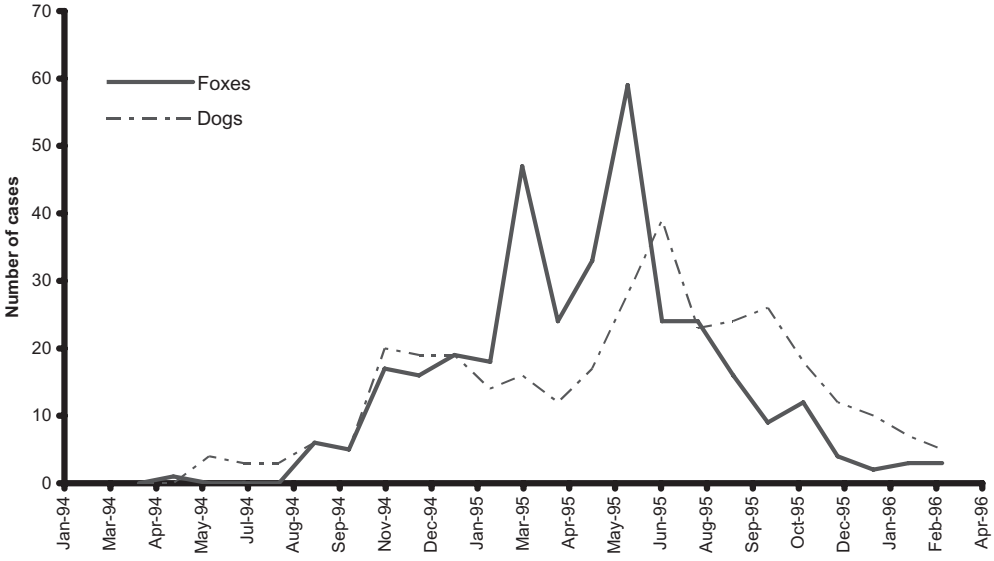

Fig. 3. The pattern of coincidence between mange in foxes and in dogs in the north-west of Bristol during April 1994–March 1996.

Table 4. Cause of death for foxes in Bristol during the pre-mange (spring 1990–winter 1993), epizootic (spring 1994–winter 1995) and enzootic (post spring 1996) periods

Cause of death	Pre-mange	Epizootic	Enzootic
Road traffic accident	36 (62)	16 (24)	13 (32)
Injury	5 (9)	2 (3)	2 (5)
Dogs	6 (10)	1 (1)	0 (0)
Drowned	1 (2)	1 (1)	1 (2)
Illness other than mange	10 (17)	4 (6)	2 (5)
Sarcoptic mange	0 (0)	43 (64)	23 (56)
Unknown	22	29	26
Total	80	96	67

Data include only those individuals captured and tagged on the main study site between spring 1990 and winter 2003. Figures are the number of animals recovered dead (figures in parentheses are the percentage of known causes of death).

the intensive study site, all resident adults and offspring died within the course of the 2 years of the epizootic period, and city-wide the fox population was reduced by more than 95% (Baker *et al.*, 2000).

The median annual mortality rate (R) of juveniles was significantly higher in the post-mange period [$R = 0.62$, interquartile range (IQR) = 0.44–0.86, $n = 8$ years, 64 recoveries] than in the pre-mange period ($R = 0.48$, IQR = 0.41–0.51, $n = 14$ years, 803 recoveries) (Mann–Whitney test: $W = 124.0$, $P < 0.05$). The mortality rate in the 2 years of the epizootic period was substantially higher ($R = 0.82$) than in either period, but this could not be tested statistically. There was no difference in the average age of tagged adult animals ($n = 127$) recovered dead in the three time periods (Kruskal–Wallis test: $\chi^2_2 = 0.094$, $P > 0.05$), although the age at death of animals during the epizootic period was complicated by the fact that many were 2 years of age or older at the time of the outbreak. As outlined above, all adult animals

Table 5. Summary of changes in adult fox density, social group size and territory size in Bristol in selected seasons in the period 1980–2004

	Pre-mange period			Epizootic period			
	Spring 1980	Spring 1990	Spring 1993	Spring 1994	Summer 1995	Autumn 1995	Winter 1995
Mean territory size (ha)	24	29	18	18	27	67	70
Mean group size (adults)	3.4	2.3	4.6	6.6	2.7	1.8	1.7
Adult density (adults/km^2)	13.9	7.8	25.8	37.0	10.0	2.7	2.4

	Enzootic period					
	Spring 1996	Summer 1998	Autumn 1999	Autumn 2002	Autumn 2003	Spring 2004
Mean territory size (ha)	–	131	169	63	58	–
Mean group size (adults)	–	1.9	1.8	2.4	2.5	–
Adult density (adults/km^2)	0.7	1.5	1.1	4.0	4.5	5.5

Reproduced with permission from Baker, Newman & Harris (2001b) with updated information.

resident on the study site perished during the epizootic period. Mean (± S.D.) age at death for adults was 1006 ± 533 days.

Changes in density

The high mortality caused by mange resulted in marked changes in social and spatial organization. Prior to the outbreak of mange, fox density increased markedly as a consequence of a reduction in territory size and increased group size due to increased food availability (Table 5). Territory size increased substantially during the epizootic phase, with animals apparently increasing range size in response to the loss of neighbouring social groups, and average group size declined. By spring 1996, the study area was occupied by a single individual that had immigrated into the site following the demise of all the resident adult animals. Consequently, population density had declined from 37.0 adults per km^2 at the onset of the epizootic to just 0.7 adults per km^2 in spring 1996, a decrease of 98%. Density in spring 2004 was approximately 15% of the density in spring 1994.

Female reproduction

There was a significant difference in the proportion of females breeding annually during the pre-mange, epizootic and enzootic periods ($\chi^2_2 = 10.52$, $P < 0.01$) (Table 6). No females with Class II mange were recorded as breeding, whereas 46–48% of non-breeding females had this degree of infection during the epizootic and enzootic periods (Table 6), consistent with severe infection being sufficiently debilitating to prevent females from breeding. However, litter size did not change significantly across the three periods (ANOVA: $F_{2,22} = 0.16$, $P > 0.05$).

Male reproductive potential

Mean (± S.D.) testes mass of uninfected males in January (11.9 ± 3.1 g, $n = 104$) was significantly greater than males with Class I (8.8 ± 3.7 g, $n = 5$) and Class II (5.0 ± 2.4 g, $n = 7$) mange ($F_{2,113} = 18.43$, $P < 0.01$). Testes mass of Class I infected males was also significantly greater than that of males with no mange outside the reproductive season (5.8 ± 2.6 g, $n = 121$) ($t_{125} = -2.8$, $P < 0.01$), consistent with spermatogenesis occurring in Class I males

Table 6. The proportion of female foxes breeding in Bristol in the pre-mange, epizootic and enzootic periods

	Pre-mange	Epizootic	Enzootic
No. of ♀s captured	51	32	26
Proportion of ♀s breeding	0.71	0.34	0.58
Proportion of breeding ♀s with			
No mange	1.00	0.18	0.67
Class I	–	0.82	0.33
Class II	–	0.00	0.00
Proportion of non-breeding ♀s with			
No mange	1.00	0.04	0.27
Class I mange	–	0.48	0.27
Class II mange	–	0.48	0.46

and their potential breeding status. However, there was no significant difference in testes mass between Class II males during the breeding season and uninfected males outside the breeding season ($t_{126} = 0.91$, $P = 0.36$), consistent with males with Class II mange being incapable of breeding.

Dispersal

The mean (± S.D.) proportion of juveniles classified as dispersers was lower in the enzootic period (males = 37%, $n = 30$ individuals; females = 14%, $n = 22$; data pooled across years) compared with the pre-mange period (males = 63 ± 7% per annum; females = 36 ± 10%: $n = 14$ years). Furthermore, there was a significant difference in the median distance dispersed by males in the pre-mange (median distance equivalent to 3.6 territories, IQR = 1.6–5.1, $n = 212$ individuals) and enzootic periods (median = 1.7 territories, IQR = 1.5–2.3, $n = 11$ individuals) (Mann–Whitney test: $W = 235.0$, $P < 0.05$).

DISCUSSION

Although based on a questionnaire requesting information from a range of people with a spectrum of expertise, and hence potentially susceptible to biases, the results of the survey presented here suggest that, in 2001, mange was widespread across much of Britain. There were, however, regional differences in the proportion of foxes affected, with areas of higher estimated prevalence being confined mainly to central and southern England. Fewer reports of mange were recorded east of the Pennines and in Scotland and Wales, which have lower fox densities (Webbon, Baker & Harris, 2004). However, mange does appear to be present in these areas, and the reduced reporting rate may simply reflect observer bias and/or that mange was still spreading into these areas.

Mange was more frequently cited as being present by respondents from urban and semi-natural habitats vs. those in agricultural habitats. Yet, foxes in urban habitats are more likely to be visible to respondents than those in agricultural habitats, potentially leading to perceptual biases. However, contrary to views expressed in the popular press, there was no significant difference in perceived prevalence of infection between urban and non-urban habitats. Furthermore, it is clear that, as in other countries (Lindström, 1991; Gortázar *et al*., 1998), mange has spread rapidly through a range of habitats and populations at different densities.

The outbreak of sarcoptic mange has had a profound impact on the Bristol fox population. Overall, population density declined by > 95% in 2 years (Baker *et al*., 2000), which is comparable to levels of change seen in other fox populations (e.g. Mörner, 1992; Lindström *et al*.,

1994; Gortázar *et al*., 1998). Population recovery following an epizootic tends to be slow at both local and regional scales. For example, populations in Scandinavia took approximately 20 years to recover to pre-mange levels (Lindström *et al*., 1994), a figure directly comparable to the perceived recovery time cited by respondents in the national questionnaire in the present study. In Bristol, fox population density in 2004 was still only 15% of that observed at the outbreak of the disease in 1994, although density at that time was exceedingly high because of the increased availability of anthropogenic food sources (Baker *et al*., 2000); fox density in 2004 was equivalent to 71% of that in 1990.

The initial decline in fox numbers was due to an increase in the mortality rate of both juveniles and adults (see also Pence, Windberg & Sprowls, 1983). Juvenile foxes may be particularly susceptible to infection since they experience a high degree of physical contact during their early development (Pryor, 1956; Meyer & Weber, 1996), such that whole litters are likely to be affected once a littermate is infected. Similarly, direct transmission between adults within social groups, and from adult to cubs, is also likely to be common (see White & Harris, 1994; Baker *et al*., 1998; Baker & Harris, 2000).

Indirect transmission is also likely to occur, for example, as foxes scrape under fences and through the shared use of dens. The importance of den sites as a route of transmission is well known (Talbot, 1906). For example, the spread of mange in Russia was controlled by fumigating dens with acaricides (Gerasimoff, 1958). Adults within the same social group also utilize the same den sites for resting during the day (Baker *et al*., 2000), although this may change with fox density (Newman *et al*., 2003). The importance of indirect transmission in the urban environment is illustrated by the concordance between levels of mange in foxes and in domestic dogs.

Population recovery during the enzootic phase has been slow, as mange infection has caused a reduction in the proportion of females able to breed and increased juvenile mortality. The pathology of mange stems from the nutritional stress caused by high parasite loads (Newman *et al*., 2002). In this study, severe levels of infection were associated with an absence of breeding in females and an apparent failure to undergo spermatogenesis in males. Similar patterns have also been observed in coyotes *Canis latrans* (Pence *et al*., 1983) and male wombats *Vombatus ursinus* (Skerrat *et al*., 1999). Consequently, in advanced stages, mange is sufficiently debilitating to limit individual reproduction. However, in the present study, there was no reduction in litter size for those female foxes that did breed. However, juvenile mortality was significantly higher in the enzootic phase, even though this may have been underestimated due to infected animals dying in inaccessible places (e.g. Wydeven *et al*., 2003). Juvenile animals appear to be more susceptible to mange (Pryor, 1956; Goltsman, Kruchenkova & Macdonald, 1996) and tend to have shorter survival times following infection (Todd, Gunson & Samuel, 1981; Pence *et al*., 1983; Newman *et al*., 2002), possibly because they prioritize nutrient intake for growth rather than immune function (Coop & Kyriazakis, 1999). In addition, reduced dispersal into the study site may also limit the rate of population recovery.

As with mange outbreaks in other countries, it is not clear why the recent epizootic in Britain arose, since areas of south-east Britain have had enzootic mange since at least the 1940s (Teagle, 1967; Lloyd, 1980; S. Harris, unpublished data) but the disease did not spread beyond this relatively restricted area for approximately 50 years. Some authors have suggested that epizootics of mange arise following the mutation of virulent strains of mites or the introduction of a new strain to a population lacking genetic resistance (Pence *et al*., 1983; Lindström, 1991; Pence & Windberg, 1994). As there is no acquired immunity following infection and individuals can become reinfected (Little *et al*., 1998), the ability of an indi-

vidual to endure infection and/or recover may depend on a range of factors such as age, genetic predisposition, nutrition or physiological status. Environmental conditions and habitat quality may also affect individual stress and have been suggested as potentially contributing to mange outbreaks (Pence *et al*., 1983; Gortázar *et al*., 1998; Skerratt *et al*., 1999).

In summary, many aspects of the epidemiology of sarcoptic mange outbreaks in Britain and elsewhere remain unclear. For example, the reasons for the sudden spread of the disease outside London and the south-east are not known, but could be related to a range of factors including changes in the virulence and transmissibility of the parasite, changes in fox density or movements of individual animals. Similarly, population modelling has suggested that population recovery may be linked to the evolution of genetic resistance, although no experimental studies have examined this hypothesis (Leung & Grenfell, 2003). In the present study, there was no significant difference in the age at death of adult animals in the pre-mange, epizootic or enzootic periods. Although the age at death of animals recovered during the epizootic period is attributable mainly to the age of those animals alive at the time of the outbreak and its short duration, the fact that animals in the enzootic period (which to date has lasted 8 years) are living as long as animals in the pre-mange period suggests that individuals are now more able to tolerate infection. The reasons for this are unclear and warrant further investigation. Possible mechanisms include selection for hosts with increased resistance, a reduction in the effects of host density on social stress and immune function and/or a reduction in parasite virulence. Focusing on these key aspects will aid our understanding of the evolutionary processes associated with wildlife diseases, and enable more effective management strategies to be developed where appropriate.

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