Received: 31 January 2017 | Accepted: 1 August 2017

DOI: 10.1111/1365-2656.12743

SYNTHESIS

Journal of Animal Ecology BRITISH ECOLOGICAL SOCIETY

Wildlife disease ecology from the individual to the population: Insights from a long-term study of a naturally infected European badger population

Jenni L. McDonald[1,*] | Andrew Robertson[2,3,*] | Matthew J. Silk[2,*]

[1]Centre for Ecology and Conservation, University of Exeter, Penryn, UK

[2]Environment and Sustainability Institute, University of Exeter, Penryn, UK

[3]National Wildlife Management Centre, Animal and Plant Health Agency, Gloucestershire, UK

Correspondence
Jenni L. McDonald
Email: j.mcdonald@exeter.ac.uk
Andrew Robertson
Email: a.robertson@exeter.ac.uk
Matthew J. Silk
Email: matthewsilk@outlook.com

Funding information
Natural Environment Research Council, Grant/Award Number: NE/L007770/1, NE/M004546/1 and NE/M010260/1; Department for Environment, Food and Rural Affairs

Handling Editor: Isabella Cattadori

Abstract

1. Long-term individual-based datasets on host–pathogen systems are a rare and valuable resource for understanding the infectious disease dynamics in wildlife. A study of European badgers (*Meles meles*) naturally infected with bovine tuberculosis (bTB) at Woodchester Park in Gloucestershire (UK) has produced a unique dataset, facilitating investigation of a diverse range of epidemiological and ecological questions with implications for disease management.
2. Since the 1970s, this badger population has been monitored with a systematic mark–recapture regime yielding a dataset of >15,000 captures of >3,000 individuals, providing detailed individual life-history, morphometric, genetic, reproductive and disease data.
3. The annual prevalence of bTB in the Woodchester Park badger population exhibits no straightforward relationship with population density, and both the incidence and prevalence of *Mycobacterium bovis* show marked variation in space. The study has revealed phenotypic traits that are critical for understanding the social structure of badger populations along with mechanisms vital for understanding disease spread at different spatial resolutions.
4. Woodchester-based studies have provided key insights into how host ecology can influence infection at different spatial and temporal scales. Specifically, it has revealed heterogeneity in epidemiological parameters; intrinsic and extrinsic factors affecting population dynamics; provided insights into senescence and individual life histories; and revealed consistent individual variation in foraging patterns, refuge use and social interactions.
5. An improved understanding of ecological and epidemiological processes is imperative for effective disease management. Woodchester Park research has provided information of direct relevance to bTB management, and a better appreciation of the role of individual heterogeneity in disease transmission can contribute further in this regard.

*Authors contributed equally to this work.

6. The Woodchester Park study system now offers a rare opportunity to seek a dynamic understanding of how individual-, group- and population-level processes interact. The wealth of existing data makes it possible to take a more integrative approach to examining how the consequences of individual heterogeneity scale to determine population-level pathogen dynamics and help advance our understanding of the ecological drivers of host–pathogen systems.

KEYWORDS

disease dynamics, disease ecology, long-term study, *Mycobacterium bovis*, pathogen transmission, superspreader, wildlife epidemiology

1 | INTRODUCTION

Studying disease ecology in wildlife is challenging (Delahay, Smith, & Hutchings, 2009; Restif et al., 2012). However, improving our understanding of wildlife epidemiology is important for the benefit of human health, animal welfare and productivity in agricultural systems, and global biodiversity (Daszak, Cunningham, & Hyatt, 2000; Delahay et al., 2009; Wiethoelter, Beltrán-Alcrudo, Kock, & Mor, 2015). Many emerging infectious diseases in humans arise from wild animals (Daszak et al., 2000; Jones et al., 2008), often as a result of human encroachment (Hassell, Begon, Ward, & Fèvre, 2017), and wildlife populations can comprise important reservoirs for economically significant livestock diseases (Craft, 2015; Wiethoelter et al., 2015). Additionally, infectious diseases have increasingly been recognized as a major threat to species of conservation concern (Daszak et al., 2000; Smith, Sax, & Lafferty, 2006).

Understanding the long-term consequences of infection and disease within a population requires more than just snapshots of prevalence. A huge diversity of factors influence the susceptibility of individuals to pathogens within populations and the resulting demographic consequences of infection (Hudson, Rizzoli, Grenfell, Heesterbeek, & Dobson, 2002). This is especially true when infection is not an isolated outbreak event, but is endemic in a population, as is often the case when wildlife populations are reservoirs of diseases transmitted to humans or domestic animals (Hassell et al., 2017). To add further complexity, individual heterogeneity in the acquisition, progression and transmission of infection is increasingly recognized as a pervasive feature of host–pathogen systems (Lloyd-Smith, Schreiber, Kopp, & Getz, 2005; VanderWaal & Ezenwa, 2016). Such heterogeneity can arise from differences in physiological traits such as condition, stress or immune competence, and behaviour (VanderWaal & Ezenwa, 2016). Consequently, long-term, individual-based ecological studies allow us to move towards a deeper mechanistic understanding of epidemiological processes at multiple scales, from individual to population (Restif et al., 2012; Rohani & King, 2010), and we discuss the contribution and prospects of one such study in this synthesis.

A long-term study of badgers naturally infected with *Mycobacterium bovis* (the causative agent of bovine tuberculosis [bTB]) at Woodchester Park (UK) has advanced the understanding of host–pathogen interactions, while providing valuable information applicable to management of a substantial socio-economic problem in Britain (Broughan et al., 2016; Donnelly et al., 2006; Godfray et al., 2013). In this synthesis, we first introduce bTB and the role of badgers in its transmission. We then describe the Woodchester Park study, discussing the population-level disease patterns in this system, before focussing on the importance of heterogeneity by detailing the role that social systems and among- and within-individual heterogeneity play in disease transmission. We end by taking stock of the current understanding of the system and discuss opportunities to derive deeper understanding of how individual heterogeneity relates to bTB dynamics.

2 | BOVINE TUBERCULOSIS IN BADGERS

bTB is a globally important disease of cattle that can infect a range of wild mammal species, which can act as reservoir or spillover populations (Palmer, Thacker, Waters, Gortázar, & Corner, 2012). In the UK, bTB infection has been identified in many wild mammals including deer, small rodents and carnivores (Delahay, De Leeuw, Barlow, Clifton-Hadley, & Cheeseman, 2002). However, it is European badgers, in which the disease can persist independently within populations (Delahay et al., 2013), which is believed to be the wildlife host of most significance in the UK and Ireland and involved in the transmission of infection to cattle (Donnelly et al., 2006; Griffin et al., 2005). Despite efforts to control the disease in cattle and badgers, the incidence of bTB in cattle has increased significantly in the UK in recent decades (Broughan et al., 2015), with substantial costs to the Government and farming industry.

In badgers bTB is primarily a disease of the pulmonary system, especially the lungs and associated lymph nodes, although virtually all organs can be affected. Badgers undergo a spectrum of responses to infection ranging from latency to generalized disease (Corner, Murphy, & Gormley, 2011; Corner, O'Meara, Costello, Lesellier, & Gormley, 2012). Latent infection occurs when the host is infected, but the bacteria are effectively contained resulting in no lesions (Parrish, Dick, & Bishai, 1998). Disease progression may involve spread to other organs, and it is likely that badgers in this disseminated disease state are most infectious, potentially shedding *M. bovis* through their sputum, faeces, urine, from bite wounds and/or open abscesses. Once infectious, onward transmission of *M. bovis* to other badgers is believed to occur

by aerosol transmission among individuals in close contact, via bite wounding (Jenkins, Cox, & Delahay, 2012), and indirectly from contamination of the environment (Corner et al., 2012; Courtenay et al., 2006).

3 | THE WOODCHESTER PARK STUDY

The Woodchester Park study was established in 1976 to investigate bTB epidemiology in a wild, naturally infected badger population. The study is situated on the Cotswold escarpment in Gloucestershire, South West England (2°16′E, 51°43′N,) and comprises a wooded valley surrounded by pastoral and arable farmland (Delahay, Carter, Forrester, Mitchell, & Cheeseman, 2006). Badger social structure is highly variable across their geographic range and in different habitat types, but when population density is high, individuals tend to live in social groups (Roper, 2010). The resident badger population at Woodchester Park occurs in a landscape typical of many high-density UK badger populations, and is organized into territorial social groups centred on a communal main sett. The population density at Woodchester Park is particularly high; however, the social structure is relatively typical of the moderate- to high-density badger populations found across the bTB-affected areas of Great Britain (Roper, 2010). The original study area was 11 km^2 in size and contained 36 badger social groups, although this has contracted slightly over time (Figure 1). While relatively small, the core area, covering *c*. 7 km^2 and containing a population of 21–23 social groups, has been studied continuously for 41 years and represents the longest temporal data source on bTB in wild badgers.

Early research at Woodchester Park established many techniques for studying badgers, including methods for capturing, marking and tracking individuals (Table S1). Each spring, social group territories are mapped using a bait marking technique (Delahay, Brown et al., 2000). Trapping of each group is conducted four times per year from May to January (February–April is a closed season to avoid trapping breeding females). Badgers are trapped in steel mesh box traps baited with peanuts. Under anaesthetic, each individual is permanently marked with a unique ID tattoo on the abdomen (Cheeseman & Harris, 1982), and variables recorded include age class, sex, weight, body condition, body length, reproductive status and tooth wear (Figure 2). Samples of sputum, faeces, urine and swabs of abscesses and wounds are taken for *M. bovis* culture (Gallagher & Horwill, 1977, Figure 2). Blood sampling of captured animals has facilitated the development, evaluation and application of several diagnostic tests for bTB. Currently blood samples are tested using the interferon-gamma immunoassay (IFN-γ; Dalley et al., 2008), used from 2006 to detect a cell-mediated immune response, and the Dual Path Platform test (DPP® Chembio. inc), used from 2015, to test for antibodies. Previous serological tests have included the Brock Elisa (Goodger et al., 1994; used from 1982 to 2006) and the BrockTB Stat-Pak test (Chambers et al., 2008; used from 2006 to 2015). Each test has imperfect performance, but combining them can improve overall diagnostic accuracy (Buzdugan, Chambers, Delahay, & Drewe, 2016; Drewe, Tomlinson, Walker, & Delahay, 2010). Consequently, while living badgers can be identified as test positive and test negative through the results of single tests, there is some uncertainty whether this equates to an individual being truly infected. For clarity, we refer to individuals being infected or uninfected when they are test positive or test negative, but acknowledge that some error is inherent here.

To date, the study has involved >15,000 capture events of >3,000 individuals. More than 85% of individuals are caught as cubs (in their first year), such that their natal social group and age are known. Regular trapping means that a wealth of life history and disease data are available for each individual to provide a powerful dataset for investigating

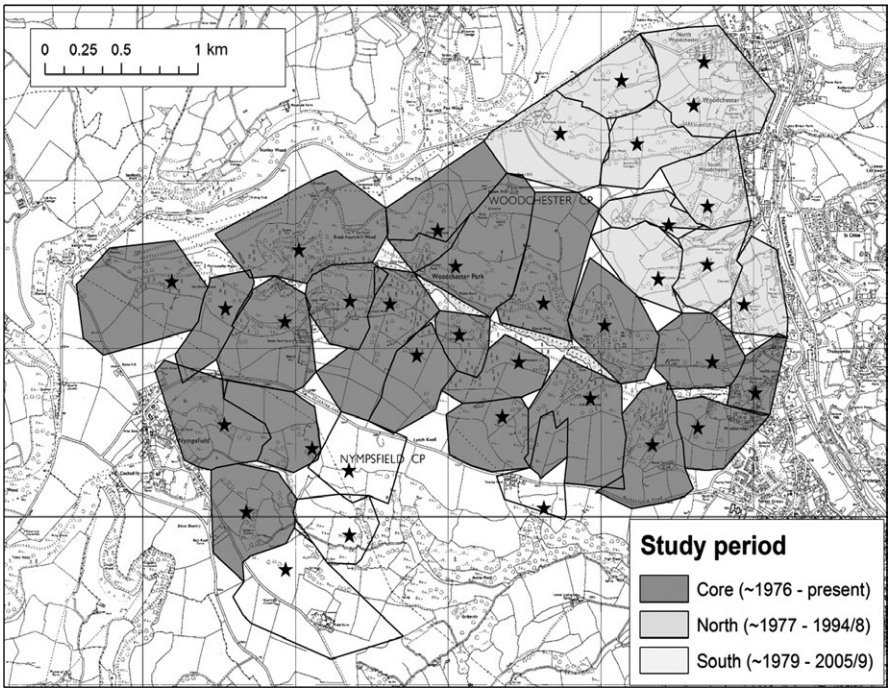

FIGURE 1 Badger main setts (black stars) and territory boundaries (polygons) at Woodchester Park in 1993. Territory boundaries vary annually and main setts have become active/inactive over the study period. The different shading reflects the period over which the study was active in that location

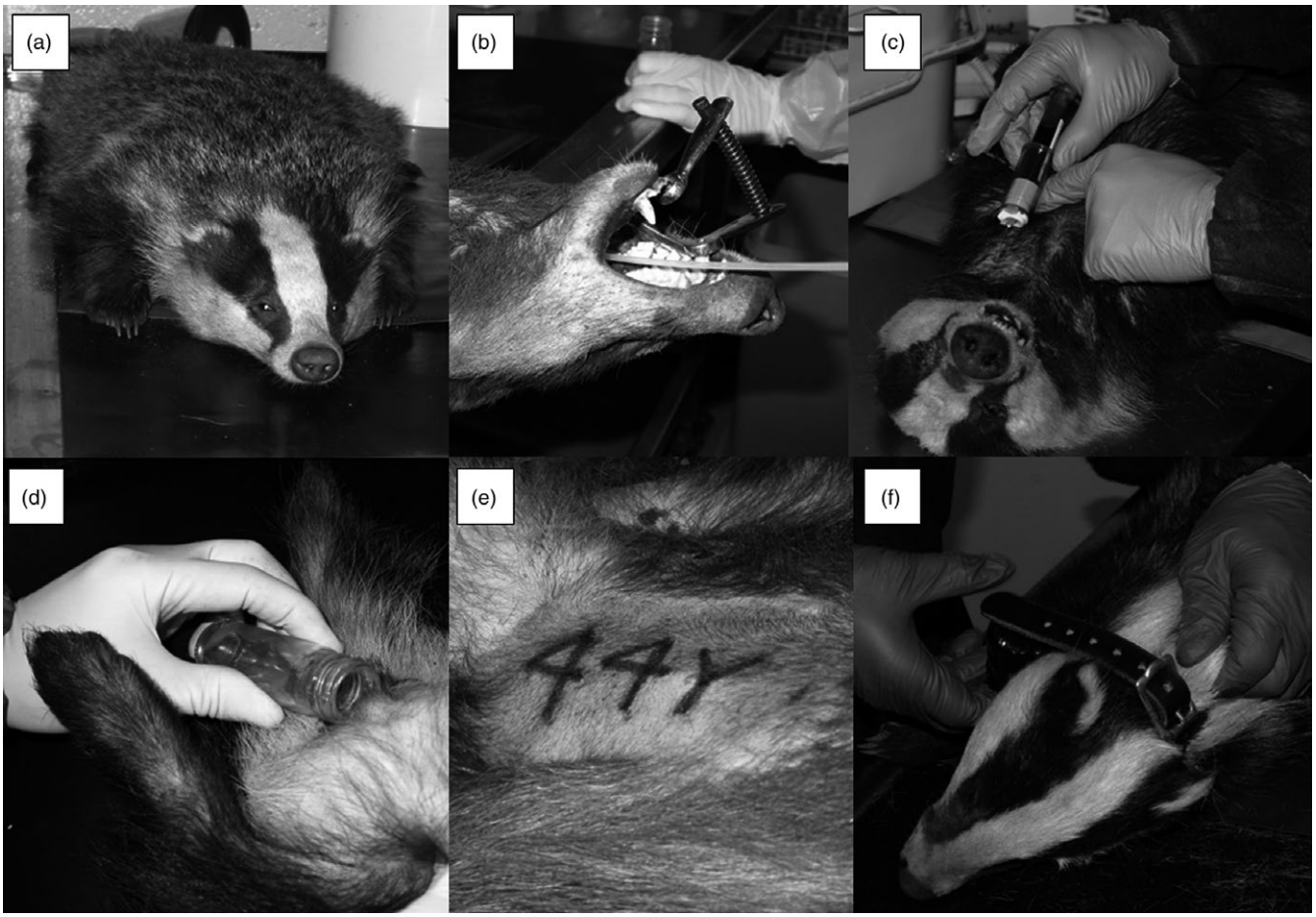

FIGURE 2 Photographs illustrating badgers being sampled at Woodchester park: (a) anaesthetised badger ready for examination; (b) inspection of dentition and collection of oesophageal aspirate using a catheter; (c) blood sampling from the jugular vein; (d) urine sample collection; (e) unique identifying tattoo on the abdomen; (f) proximity collar being fitted

ecological and epidemiological questions ranging from population-level epidemiology, through the role of social structure to individual differences in epidemiological importance. These are summarized in Table S2, and many are discussed throughout this synthesis.

4 | HOST AND PATHOGEN ECOLOGY AT THE POPULATION LEVEL

Throughout the study, there have been substantial fluctuations in the badger population (McDonald et al., 2016). The population increased from 7.8 badgers/km^2 in 1978 to 47 badgers/km^2 in 1999 (Delahay et al., 2013). After 1999, the population levelled out and gradually declined (Delahay et al., 2013), and numbers trapped have continued to decline in recent years (Figure 3), with the causes of this unclear. Variation in cub recruitment positively correlates with these population changes. Density-dependent regulation is considered the main driver of this (McDonald et al., 2016), although weather effects are also found to be important in this and other study systems (Macdonald, Newman, Buesching, & Nouvellet, 2010; McDonald et al., 2016).

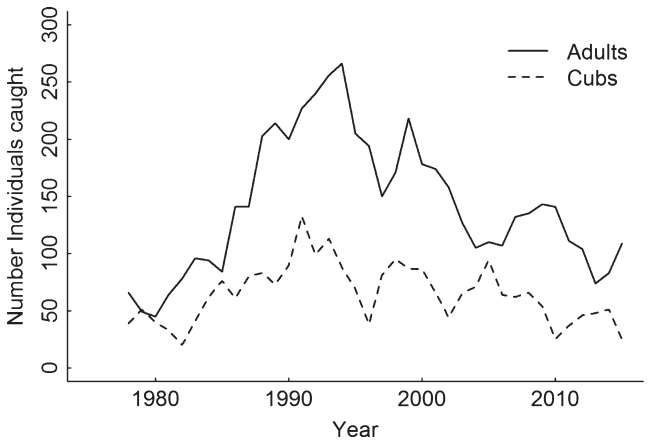

FIGURE 3 The number of individual badgers cage-trapped in core groups of the Woodchester Park study from 1977 to 2015. Cubs are classed as any badger born that year, while adults are all older age classes (≥1 years old)

Disease transmission is often described to be either density dependent, in which contact rates and therefore transmission increases with population density, or frequency dependent, in which case it does

not (Begon et al., 2002). Density-dependent transmission can influence disease dynamics and affect how wildlife diseases are managed. *M. bovis* prevalence and incidence at Woodchester Park have been highly variable through time, with an overall increase observed during the first *c*. 25 years of the study (Delahay et al., 2013). There is mixed evidence for density-dependent transmission. No significant positive association between population size and bTB incidence in the same (or preceding) year has been detected, although prevalence is positively correlated with population size in the previous year (Delahay et al., 2013). Within groups, there is stronger evidence that transmission is not density dependent; for example, cubs have a lower chance of being infected in their first year of life in larger groups (Benton et al., 2016). This result corroborates findings from other populations where prevalence is higher in smaller social groups (Woodroffe et al., 2009).

Despite the mixed evidence for density-dependent *M. bovis* transmission, there is evidence of interactions between disease and demographic processes in Woodchester Park. Adult survival in the population is lower with a high prevalence of diseased individuals (McDonald et al., 2016). Additionally, a density effect on disease progression has been suggested to explain a positive relationship between population size and the incidence of infectious badgers (positive culture result) in the following year (Delahay et al., 2013). Badgers with evidence of infection (positive serological tests) can live for many years before showing evidence of being infectious (positive culture or advanced pathology), and therefore, it seems unlikely that a rise in the number of infectious individuals is simply a function of increased transmission risk. Density-dependent decreases in body weight (Rogers, Cheeseman, & Langton, 1997) have been documented, suggesting that competition-induced physiological stressors (of which body weight is a likely proxy) may help explain increased disease progression at higher population densities.

Environmental factors independent of population density may also influence the progression of disease. For example, McDonald et al. (2016) reported that high autumn temperatures increased bTB-induced mortality, while during more favourable cooler conditions, the impact of disease on survival was negligible. If changing climate results in warmer autumns, this has the potential to alter disease dynamics in badger populations by increasing disease-induced mortality. However, population-level analyses disregard the diverse phenotypic variation among individuals, assuming that all individuals play an equivalent role in disease dynamics. Understanding the complex nature of interactions between density, disease and extrinsic factors requires exploring the role of social structure and individual heterogeneity within demographic processes (Figure 4).

5 | GROUP-LEVEL PATTERNS IN DISEASE AND THE ROLE OF SOCIAL STRUCTURE

Population social structure results in considerable variation in bTB prevalence across the Woodchester Park population. Annual measures of prevalence show marked variation among social groups,

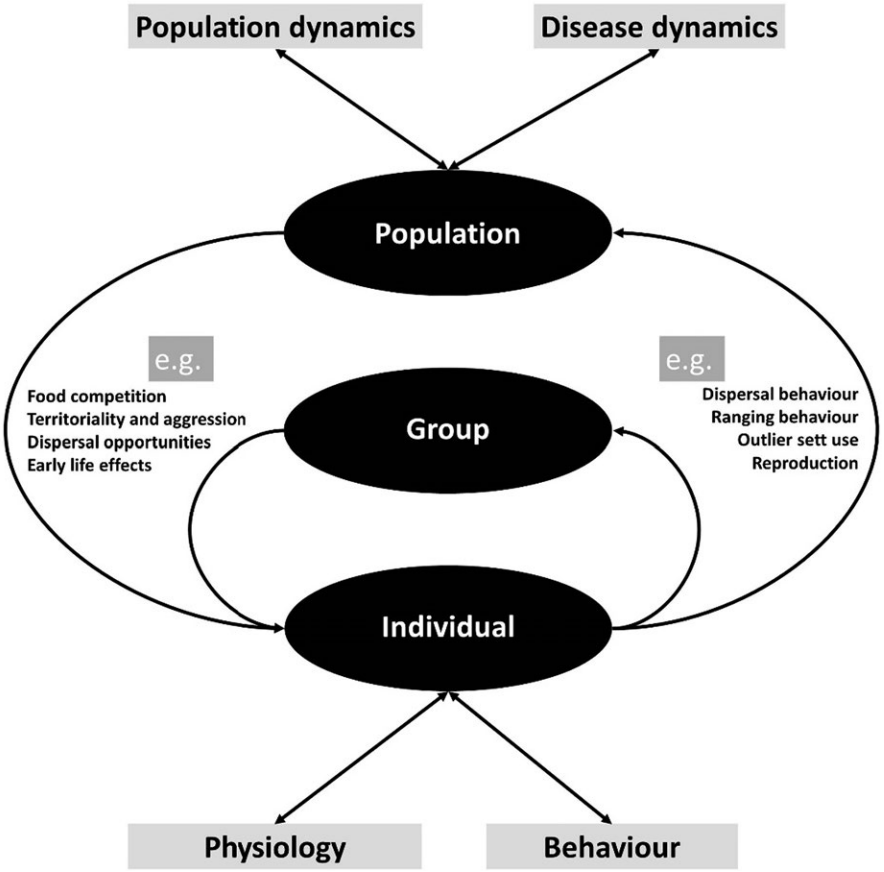

FIGURE 4 The links between population-level, group-level and individual-level processes and their role in determining disease dynamics in the Woodchester Park badger population

with strong evidence for spatial aggregation and persistence of infection within groups (Delahay, Langton, Smith, Clifton-Hadley, & Cheeseman, 2000; Rogers et al., 1998; Vicente, Delahay, Walker, & Cheeseman, 2007). Furthermore, a lack of temporal synchrony in bTB incidence and prevalence between nearby infected groups suggests low rates of transmission among them (Delahay, Langton, et al., 2000). The importance of group association in determining disease risk was highlighted by Vicente et al. (2007), who found that individuals from groups containing infected individuals were far more likely to become infected themselves.

Given the importance of social structure in constraining bTB spread (Carter et al., 2007; Delahay, Langton, et al., 2000; Weber, Carter, et al., 2013), dispersal might be expected to be a key mechanism mediating disease risk. Dispersal in high-density badger populations is typically limited (Frantz, San, Pope, & Burke, 2010). In Woodchester Park, 37% of males and 70% of females were classified as non-movers (only ever caught in one social group) (Rogers et al., 1998). Even badgers caught in multiple groups were most often (73%) only "occasional movers," defined as being captured at one or two social groups other than their main resident group. As few as 22% of badgers were defined as "permanent movers," whose dispersal resulted in a lasting change in their resident social group. Rogers et al. (1998) linked local movement and bTB incidence at a population-level, reporting the latter to be higher in years following higher movement rates. Similarly, Vicente et al. (2007) found that individuals in groups that experienced more movements (whether in or out) were more likely to become infected. These findings have been linked to potential epidemiological consequences of badger culling operations. Culling alters badger social organization and results in increased rates of movement, termed "social perturbation" (Carter et al., 2007; Woodroffe et al., 2006). This is hypothesized to explain increased bTB prevalence in badgers (Woodroffe et al., 2006) and cattle herd bTB incidence observed following badger culling (Donnelly et al., 2006), and continues to contribute to the debate around badger management and bTB control (Broughan et al., 2016; Donnelly et al., 2006; Godfray et al., 2013).

6 | INDIVIDUAL DIFFERENCES AND THEIR ROLE IN DISEASE TRANSMISSION

Within social groups, additional variation will arise among individual badgers in their risk of acquiring infection, disease progression and subsequent onward transmission (Figure 5). Such differences may be related to variation in social behaviour and/or physiological state (Figure 5). Higher infectiousness and/or high numbers of disease-transmitting contacts are characteristics of "superspreaders," individuals that contribute disproportionately to disease transmission and play a pivotal role in disease spread through the population (Lloyd-Smith et al., 2005). Both behaviour and disease progression can generate a "superspreader." For example, at Woodchester Park, a subset of infected badgers labelled "super-excretors" exhibit signs of relatively higher levels of bacterial shedding and elevated mortality rates, consistent with advanced disease (Graham et al., 2013; Wilkinson et al., 2000). However, individuals can also be "superspreaders" by interacting with many individuals or individuals in different groups (a "supercontactor"). The behavioural and physiological differences (Figure 5), or genetic predispositions, which may influence the emergence of "superspreader" individuals still require further investigation, although existing research has revealed a range of traits that correlate with bTB infection.

Given the importance of population structure in influencing bTB dynamics in badgers, individual variation in socio-spatial behaviour may be expected to affect heterogeneity in infection and transmission risk (Figure 5 right-hand side). The deployment of proximity collars at Woodchester Park allowed an early quantification of badger social interactions. Weber, Carter, et al. (2013) found that bTB-infected individuals tended to form more among-group and fewer within-group interactions than uninfected individuals. This effect varied seasonally; infected individuals had lower within-group degree and closeness (two measures of how central an individual is to its social group) in autumn and winter and higher between-group flow betweenness (a measure of the importance of an individual for flow through the network of between-group interactions) in summer and winter. Interestingly, there was also corresponding evidence of consistent individual variation in sett use (Weber, Bearhop, et al., 2013), with increased use of outlier setts being positively correlated with both bTB infection and the tendency to form among-group interactions (Weber, Bearhop, et al., 2013; Weber, Carter, et al., 2013). Individual variation in dispersal might also be expected to contribute to differences in social network position (and potentially also outlier sett use). Vicente et al. (2007) found that individuals with higher movement indices were more likely to become incident bTB cases. It is important to consider that social behaviour can have indirect as well as direct effects on the infection acquisition (Figure 5). For example, badgers with weaker connections within social groups, or those more likely to disperse or use outlier setts, may be more susceptible to infection because they are in poorer condition or more stressed. However, links between socio-spatial behaviour and physiological state remain unknown (Figure 5), although increased physiological stress has been found to be correlated with bTB infection in another badger population (George, Smith, Mac Cana, Coleman, & Montgomery, 2014).

Studies using radio-tracking to explore the relationship between bTB infection and ranging behaviour (Cheeseman & Mallinson, 1981; Garnett, Delahay, & Roper, 2005) have found evidence that badgers with more advanced disease tended to occupy larger home ranges than uninfected individuals. However, this focus on individuals with advanced infection, while aiding our understanding of transmission risks (both to other badgers and cattle), is uninformative in assessing how heterogeneity in ranging behaviour can influence acquisition of infection. Further, none of the studies relating bTB to socio-spatial behaviour have yet investigated causality within these relationships.

Individual variation in other traits could also help explain differences in ranging and social behaviour. One intriguing possibility is that foraging preferences could influence spatial and social behaviour, and hence both exposure and transmission risk, among badgers and between badgers and cattle (Garnett et al., 2005; Tolhurst, Delahay,

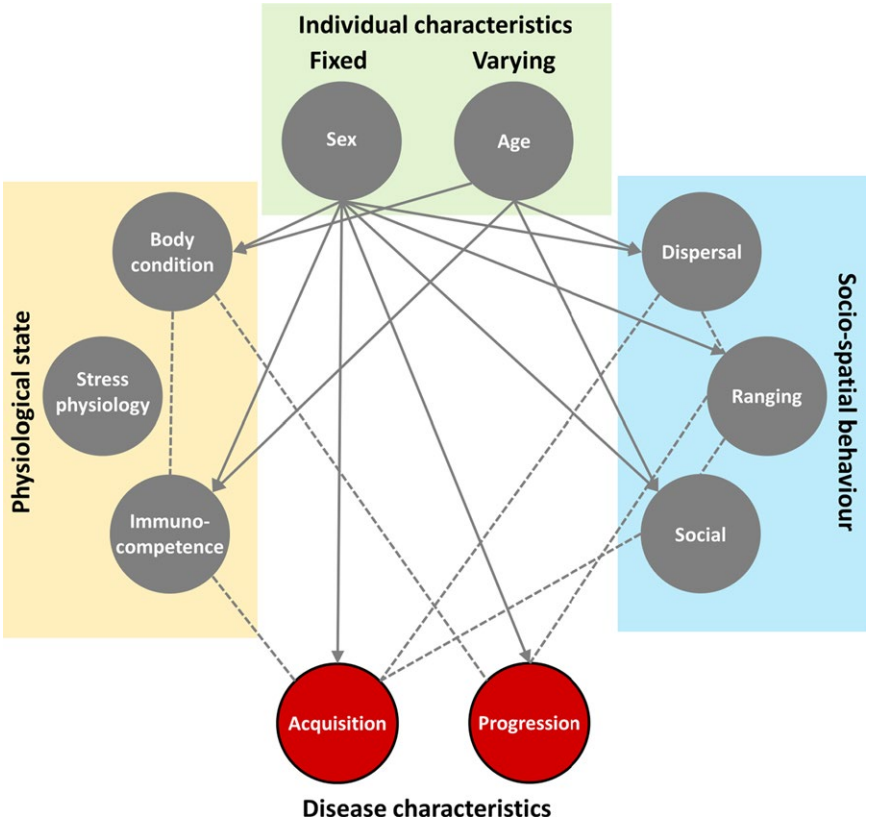

FIGURE 5 The role of individual characteristics, physiological state and socio-spatial behaviour in influencing the acquisition and progression of bovine tuberculosis in Woodchester Park badgers. We use sex and age as well-studied examples of fixed (among-individual) and varying (within-individual) individual characteristics, respectively. Solid arrows represent established directional effects, and dashed lines represent established correlations from Woodchester Park studies. Note the absence of links equates to an absence of evidence and not an absence of presence in the Woodchester Park population [Colour figure can be viewed at wileyonlinelibrary.com]

Walker, Ward, & Roper, 2009). Using stable isotope analysis, Robertson, McDonald, Delahay, Kelly, and Bearhop (2014) documented significant individual variation in foraging niches within badger social groups, which was largely unrelated to characteristics such as age and sex, suggesting individual specialization. Further, levels of individual specialization within groups depended on territory size and the amount of farmland available (Robertson, McDonald, Delahay, Kelly, & Bearhop, 2015). In groups with reduced availability of farmland (a key foraging habitat), increased specialization was correlated with improved body condition. Assuming body condition is related to the probability of infection (Figure 5), the relationship between dietary specialization and body condition could indicate an alternative role for foraging behaviour in influencing the acquisition and subsequent progression of infection. Individual differences in foraging niche could also directly influence the likelihood of acquiring infection, if certain prey sources, habitats or foraging behaviours differ in infection risk (e.g. Johnson et al., 2009).

Little is known about how among-individual variation in physiological traits such as immuno-competence and physiology contributes to heterogeneity in the acquisition, progression and transmission of bTB infection in Woodchester Park badgers (Figure 5 left-hand side). Various factors may underpin the magnitude of immune responses in vertebrates (Schmid-Hempel, 2003), including age and sex alongside the route and dose of infection. Among-individual variation in the magnitude of the cell-mediated response to bTB infection has been documented in Woodchester Park badgers and found to be a positive predictor of progression to seropositive (Stat-Pak positive) or excreting (culture positive) states (Tomlinson, Chambers, McDonald, & Delahay, 2015). Genetically acquired resistance or tolerance to bTB is suspected to also contribute to observed patterns of infection within groups (Benton et al., 2016), although the role of genetics in affecting disease acquisition or progression has not yet been studied directly. Previously, it has been suggested that vertical antibody transfer (from mother to young), as detected by transience of a serological response (a positive test result followed by negative results at subsequent captures), may provide transient resistance to infection (Newell, Clifton-Hadley, & Cheeseman, 1997). However, results from a further investigation (Tomlinson, Chambers, & Delahay, 2012) suggested that transient serological responses were not a valid proxy for maternal antibody transfer.

6.1 | An example of among-individual heterogeneity: Differences between the sexes

Multistate modelling has revealed that male badgers are more likely to acquire bTB than females and that infection in males is more likely to progress to seropositive and excreting states (Graham et al., 2013). In addition, later stages of infection are associated with a greater increase in mortality in males than females (Graham et al., 2013; McDonald, Smith, McDonald, Delahay, & Hodgson, 2014). Nevertheless, for both sexes, mortality increases substantially as bTB infection progresses to a super-excretor state (Graham et al., 2013; Wilkinson et al., 2000).

Individual-based studies provide a variety of mechanisms that might explain sex differences in infection risk and disease progression (Figure 5). Beirne, Waring, McDonald, Delahay, and Young (2016)

found that cell-mediated immune responses were consistently lower in males than females regardless of age. Male badgers are also more likely to use outlier setts than females during the mating season in winter and spring (Weber, Bearhop, et al., 2013), with time spent in outliers being positively correlated with between-group contacts and bTB infection as discussed previously. Similarly, while the evidence for sex-biased dispersal in badgers is mixed, and in high-density populations dispersal is typically very limited (Frantz et al., 2010), two observational studies using mark–recapture data from Woodchester Park have found a tendency for dispersal to be male biased (Cheeseman, Cresswell, Harris, & Mallinson, 1988; Rogers et al., 1998). Given the strong links between movement and bTB infection, even small differences in dispersal behaviour could substantially alter infection risk.

7 | CHANGES IN INDIVIDUAL DISEASE RISK OVER TIME

Over the course of an individual's life, the risks of pathogen acquisition, disease progression and onward pathogen transmission are likely to vary in relation to changes in intrinsic physiological state and social behaviour (Figure 5). Such within-individual variation could also occur on a less predictable short-term basis, reflecting seasonality for example.

One key component of individual heterogeneity in disease is early-life risk. Many individuals within the Woodchester Park population acquire bTB infection in their first year of life (annual estimates range from 5% to 37%; Benton et al., 2016; Tomlinson, Chambers, Carter, et al., 2013), and this is known to be related to characteristics of the natal group. For example, the presence of infectious breeding females has been related to a fourfold increase in the risk of cubs becoming test positive in their first year of life (Tomlinson, Chambers, Carter, et al., 2013). Similarly, the presence of infectious related males and females has been found to increase the risk of cubs becoming infected (Benton et al., 2016). While not all cubs are fathered by within-group males, it may be that a tendency to interact more with fathers or related males could explain this. However, the principal source of risk, based on these studies, is likely to be "pseudo-vertical" transmission from mothers to cubs (Tomlinson, Chambers, Carter, et al., 2013).

A second important age-related process influencing disease characteristics is senescence. Recent work at Woodchester Park has documented senescence in badger body mass (Beirne, Delahay, & Young, 2015), telomere length (Beirne, Delahay, Hares, & Young, 2014) and immune function (Beirne et al., 2016). Body mass senescence is greater in male than female badgers and related to high levels of intrasexual competition (Beirne et al., 2015). In contrast, rates of immune senescence do not differ between the sexes (Beirne et al., 2016). The relationship between body condition and the acquisition or progression of infection is less clear. A number of studies in Woodchester Park have found that badgers with advanced infection are in poor condition (e.g. Clifton-Hadley, Wilesmith, & Stuart, 1993; Tomlinson, Chambers, Wilson, McDonald, & Delahay, 2013; Figure 5). It may be that poor condition (low body mass) is associated with physiological stress or reduced immuno-competence. However, Beirne et al. (2016) found only a very weak relationship between IFN-γ response and body condition. Further, existing studies do not yet reveal whether poor body condition is a cause or a consequence of disease progression.

Finally, the socio-spatial behaviour of badgers is also likely to change during their lifetime, as dispersal behaviour, dominance ranking and mating behaviour may change. Age-related differences in the likelihood of changing social group (Rogers et al., 1998), and in seasonal patterns of outlier use (Weber, Bearhop, et al., 2013), have been documented. There is also evidence for changes in rates of parentage with age (Carpenter et al., 2005), although how this is reflective of age-related variation in mating behaviour is unclear.

8 | SCALING INDIVIDUAL HETEROGENEITY TO POPULATION-LEVEL PROCESSES

Forty years of research at Woodchester have revealed the importance of individual heterogeneity in the risks of infection and disease progression (Figure 5) and that variation in many traits can contribute to population-level epidemiological patterns. This has contributed considerably to an in-depth knowledge of badger ecology alongside many other important studies (see Roper, 2010 for a review), some of which have also made important contributions to our understanding of individual variation in disease state (e.g. George et al., 2014). Variability in individual infectiousness is a key determinant of disease persistence in wild populations (VanderWaal & Ezenwa, 2016) and incorporation in predictive models can have a profound impact on model outcomes (Lloyd-Smith et al., 2005). For the Woodchester Park research programme, the advent of a more individual-focussed set of studies has provided invaluable links between individual variation in behaviour and the group-level patterns that were well captured by previous studies (e.g. Delahay, Langton, et al., 2000; Rogers et al., 1998; Vicente et al., 2007). In particular, the Woodchester study has revealed phenotypic traits vital for understanding the social structure of badger populations, mechanisms related to disease spread at different spatial resolutions (Figure 4) and processes which provide insights into the potential impact of management-induced perturbation (albeit with the caveat the Woodchester Park study population is of higher than average density). However, the lack of continual bio-logging data on particular individuals has somewhat restricted a temporally dynamic understanding of the system. Additionally, it is only recently that sophisticated demographic approaches, which account for detection bias and uncertainty in disease state, have enabled a more in-depth understanding of demographic processes.

Future studies should build on this strong knowledge base, as well as that provided by other long-term studies of badgers, to try

and address some of the limitations highlighted above, to better understand how individual heterogeneity relates to bTB dynamics. We propose three strands of research that are ideally suited to investigation within the Woodchester system: (1) exploring how individual traits interact to determine heterogeneity in disease parameters; (2) taking a more dynamic approach to determine disease–behaviour co-dynamics; and (3) accounting for among-individual variation within demographic and epidemiological models.

8.1 | How do individual traits interact to determine disease risk and progression?

Understanding how individual traits interact to determine risks of infectious disease progression is central to teasing apart the underlying mechanisms driving variation in bTB epidemiology. Individual differences that result in substantially increased transmission opportunities ("supercontactors") and rapid transition to a super-excretor state represent an important component of the "superspreader" phenotype (individuals responsible for a high number of secondary infections). Describing a set of phenotypic characteristics that are associated with this "superspreader" status could have important practical implications for disease management and be of substantial interest from an epidemiological and evolutionary perspective (VanderWaal & Ezenwa, 2016). The wealth of genetic, physiological and behavioural data available on individually marked badgers at Woodchester Park offers a rare opportunity to address this issue.

One area of current research interest is the indirect role of social behaviour on disease acquisition and progression (Nunn, Craft, Gillespie, Schaller, & Kappeler, 2015). It has been suggested that social buffering against infection may be an important process for group-living species through its effects on condition, stress and immunocompetence (Ezenwa, Ghai, McKay, & Williams, 2016; Nunn et al., 2015). One explanation for a correlation between bTB infection risk and increased between-group movement and contacts is that these behaviours increase exposure to infection. However, an alternative explanation may be that these behaviours result in deteriorating condition, stress and/or reduced immuno-competence, making individuals more susceptible to being infected. An intriguing possibility, based on our existing knowledge, is that the interaction between behavioural and physiological processes might be expected to covary with sex or age. This could be important in generating predictable individual heterogeneity that would be beneficial to include in epidemiological models or management strategies.

Another component of among-individual variation that has received comparatively little attention within the Woodchester study has been the genetics of infection. The construction of a pedigree and its integration with existing phenotypic data will enable hypotheses about the role of variation in susceptibility to infection and resilience to disease to be addressed. Such effects on bTB in badgers may be expected given that genetic variation has been linked to immune function and infection with other pathogens in another high-density badger population (Sin et al., 2014). Genotypic variation may also determine some of the characteristics of the "superspreader" phenotype. Using a pedigree to better identify the role of genetic variation in such phenomena will be integral to providing a deeper understanding of host–pathogen dynamics in this system.

8.2 | What are the co-dynamics of bTB infection and behaviour?

A further area of current research interest in disease ecology is network dynamics; changes in host behaviour in response to infection can have an important influence on epidemiological patterns and vice versa (Ezenwa, Archie, et al., 2016). There is extensive empirical evidence from Woodchester Park relating behaviour to bTB infection. However, the directionality of the relationships between socio-spatial behaviour and the acquisition and progression of infection remain unclear (Figure 5). bTB in badgers is typically a chronic, slow-progressing disease, which means that behaviour–infection co-dynamics could play an important role and make the study system ideally suited to test these ideas. One feature of the badger–bTB system that has made studying behaviour–infection co-dynamics difficult has been uncertainty in diagnostic test results. However, the application of new Bayesian methods is providing a more sophisticated probabilistic interpretation of infection status (Buzdugan et al., 2016; Drewe et al., 2010), which will improve the ability to track lifetime disease status and enable uncertainty in disease status to be incorporated into any subsequent analyses. Studies of behaviour–infection co-dynamics will additionally require longitudinal and temporally dynamic approaches to network analysis that have previously not been used in this population. These approaches can exploit either: (1) technological developments in bio-logging approaches to increase the resolution of data on space-use and social interactions or (2) the incorporation of mark-recapture data, to enable longitudinal analysis of individual socio-spatial behaviour and dispersal throughout the lifespan of marked individuals.

8.3 | What is the role of individual heterogeneity in population-level epidemiological patterns?

Throughout this review, we have highlighted how individual heterogeneity could be important for host–pathogen dynamics (Figure 4). However, understanding the extent to which this is true and how this information could be used in an applied context is challenging. Bayesian hierarchical modelling enables individual heterogeneity to be incorporated within demographic models (e.g. McDonald et al., 2016). Recent developments in these modelling approaches facilitate the incorporation of individual covariates and now provide the potential to build upon these integrated approaches. For example, including information about foraging specialization, ranging behaviour and contact networks into demographic modelling approaches could identify underlying mechanisms driving variation in infection risk and mortality. Similarly, developing a comprehensive epidemiological model that incorporates empirically derived behavioural or physiological data (e.g. building upon Shirley, Rushton, Smith, South,

& Lurz, 2003; Wilkinson, Smith, Delahay, & Cheeseman, 2004) could provide valuable insights into how individual heterogeneity contributes to population-level disease dynamics. While the Woodchester Park population is not managed directly, the improved understanding gained through integrated approaches such as these can be incorporated into modelling approaches currently used to evaluate management interventions.

9 | CONCLUSIONS

The long-term study of badger–bTB dynamics at Woodchester Park has applied traditional behavioural and epidemiological approaches to great effect in studying the role of population structure and individual heterogeneity in disease dynamics and providing valuable information of direct relevance to disease management interventions. Woodchester Park represents just a single high-density badger population, and further comparison to other populations will be valuable. However, while we still lack a complete understanding of the role of individual variation in this population, the level of existing knowledge provides the foundation for future investigations to generate a deeper understanding of the role of individual heterogeneity in influencing population-level host–pathogen dynamics, especially when combined with a proliferation of new technological and analytical approaches. By further developing these new approaches, and integrating the empirical data they provide, the Woodchester Park study system has the potential to provide many more valuable insights into wildlife disease epidemiology and management.

ACKNOWLEDGEMENTS

We thank the Woodchester Park team (past and present) for conducting the research. The study is supported by the UK Department of Environment, Food and Rural Affairs. M.J.S. was supported by NE/M004546/1. J.L.M. research was motivated by NE/M010260/1 and currently supported by NE/L007770/1. We thank Dez Delahay and three anonymous reviewers for helpful comments on the manuscript.

AUTHORS' CONTRIBUTIONS

All authors conceived the ideas, made substantial contributions to writing the manuscript and gave final approval for publication.

DATA ACCESSIBILITY

This study does not contain any data.

ORCID

Jenni L. McDonald http://orcid.org/0000-0002-5050-750X
Matthew J. Silk http://orcid.org/0000-0002-8318-5383

REFERENCES

Begon, M., Bennett, M., Bowers, R. G., French, N. P., Hazel, S., & Turner, J. (2002). A clarification of transmission terms in host-microparasite models: Numbers, densities and areas. *Epidemiology and Infection*, *129*, 147–153.

Beirne, C., Delahay, R., Hares, M., & Young, A. (2014). Age-related declines and disease-associated variation in immune cell telomere length in a wild mammal. *PLoS ONE*, *9*, e108964.

Beirne, C., Delahay, R., & Young, A. (2015). Sex differences in senescence: The role of intra-sexual competition in early adulthood. *Proceedings of the Royal Society of London B*, *282*, 20151086.

Beirne, C., Waring, L., McDonald, R. A., Delahay, R., & Young, A. (2016). Age-related declines in immune response in a wild mammal are unrelated to immune cell telomere length. *Proceedings of the Royal Society of London B*, *283*, 20152949.

Benton, C. H., Delahay, R. J., Robertson, A., McDonald, R. A., Wilson, A. J., Burke, T. A., & Hodgson, D. (2016). Blood thicker than water: Kinship, disease prevalence and group size drive divergent patterns of infection risk in a social mammal. *Proceedings of the Royal Society of London B*, *283*, 20160798.

Broughan, J., Harris, K., Brouwer, A., Downs, S., Goodchild, A., Upton, P., & Smith, N. (2015). Bovine TB infection status in cattle in Great Britain in 2013. *The Veterinary Record*, *176*, 326–330.

Broughan, J., Judge, J., Ely, E., Delahay, R., Wilson, G., Clifton-Hadley, R., … Downs, S. (2016). A review of risk factors for bovine tuberculosis infection in cattle in the UK and Ireland. *Epidemiology and Infection*, *144*, 1–28.

Buzdugan, S., Chambers, M., Delahay, R., & Drewe, J. (2016). Diagnosis of tuberculosis in groups of badgers: An exploration of the impact of trapping efficiency, infection prevalence and the use of multiple tests. *Epidemiology and Infection*, *144*, 1717–1727.

Carpenter, P. J., Pope, L. C., Greig, C., Dawson, D. A., Rogers, L. M., Erven, K., … Burke, T. (2005). Mating system of the Eurasian badger, *Meles meles*, in a high density population. *Molecular Ecology*, *14*, 273–284.

Carter, S. P., Delahay, R. J., Smith, G. C., Macdonald, D. W., Riordan, P., Etherington, T. R., … Cheeseman, C. L. (2007). Culling-induced social perturbation in Eurasian badgers *Meles meles* and the management of TB in cattle: An analysis of a critical problem in applied ecology. *Proceedings of the Royal Society of London B: Biological Sciences*, *274*, 2769–2777.

Chambers, M. A., Crawshaw, T., Waterhouse, S., Delahay, R., Hewinson, R. G., & Lyashchenko, K. P. (2008). Validation of the BrockTB Stat-Pak assay for detection of tuberculosis in Eurasian badgers (*Meles meles*) and influence of disease severity on diagnostic accuracy. *Journal of Clinical Microbiology*, *46*, 1498–1500.

Cheeseman, C., Cresswell, W., Harris, S., & Mallinson, P. (1988). Comparison of dispersal and other movements in two badger (*Meles meles*) populations. *Mammal Review*, *18*, 51–59.

Cheeseman, C., & Harris, S. (1982). Methods of marking badgers (*Meles meles*). *Journal of Zoology, London*, *197*, 289–292.

Cheeseman, C., & Mallinson, P. (1981). Behaviour of badgers (*Meles meles*) infected with bovine tuberculosis. *Journal of Zoology*, *194*, 284–289.

Clifton-Hadley, R., Wilesmith, J., & Stuart, F. (1993). *Mycobacterium bovis* in the European badger (*Meles meles*): Epidemiological findings in tuberculous badgers from a naturally infected population. *Epidemiology and Infection*, *111*, 9–19.

Corner, L., Murphy, D., & Gormley, E. (2011). *Mycobacterium bovis* infection in the Eurasian badger (*Meles meles*): The disease, pathogenesis, epidemiology and control. *Journal of Comparative Pathology*, *144*, 1–24.

Corner, L. A., O'Meara, D., Costello, E., Lesellier, S., & Gormley, E. (2012). The distribution of *Mycobacterium bovis* infection in naturally infected badgers. *The Veterinary Journal*, *194*, 166–172.

Courtenay, O., Reilly, L., Sweeney, F., Hibberd, V., Bryan, S., Ul-Hassan, A., … Wilson, G. (2006). Is *Mycobacterium bovis* in the environment

important for the persistence of bovine tuberculosis? *Biology Letters*, *2*, 460–462.

Craft, M. E. (2015). Infectious disease transmission and contact networks in wildlife and livestock. *Philosophical Transactions of the Royal Society of London B*, *370*, 20140107.

Dalley, D., Davé, D., Lesellier, S., Palmer, S., Crawshaw, T., Hewinson, R. G., & Chambers, M. (2008). Development and evaluation of a gamma-interferon assay for tuberculosis in badgers (*Meles meles*). *Tuberculosis (Edinburgh)*, *88*, 235–243.

Daszak, P., Cunningham, A. A., & Hyatt, A. D. (2000). Emerging infectious diseases of wildlife – threats to biodiversity and human health. *Science*, *287*, 443.

Delahay, R., Brown, J., Mallinson, P., Spyvee, P., Handoll, D., Rogers, L., & Cheeseman, C. (2000). The use of marked bait in studies of the territorial organization of the European badger (*Meles meles*). *Mammal Review*, *30*, 73–87.

Delahay, R., Carter, S., Forrester, G., Mitchell, A., & Cheeseman, C. (2006). Habitat correlates of group size, bodyweight and reproductive performance in a high-density Eurasian badger (*Meles meles*) population. *Journal of Zoology*, *270*, 437–447.

Delahay, R., De Leeuw, A., Barlow, A., Clifton-Hadley, R., & Cheeseman, C. (2002). The status of *Mycobacterium bovis* infection in UK wild mammals: A review. *The Veterinary Journal*, *164*, 90–105.

Delahay, R., Langton, S., Smith, G., Clifton-Hadley, R., & Cheeseman, C. (2000). The spatio-temporal distribution of *Mycobacterium bovis* (bovine tuberculosis) infection in a high-density badger population. *Journal of Animal Ecology*, *69*, 428–441.

Delahay, R. J., Smith, G. C., & Hutchings, M. R. (2009). The science of wildlife disease management. *Management of Disease in Wild Mammals* (pp. 1–8). Tokyo, Japan: Springer.

Delahay, R., Walker, N., Smith, G., Wilkinson, D., Clifton-Hadley, R., Cheeseman, C., … Chambers, M. (2013). Long-term temporal trends and estimated transmission rates for *Mycobacterium bovis* infection in an undisturbed high-density badger (*Meles meles*) population. *Epidemiology and Infection*, *141*, 1445–1456.

Donnelly, C. A., Woodroffe, R., Cox, D., Bourne, F. J., Cheeseman, C., Clifton-Hadley, R. S., … Jenkins, H. (2006). Positive and negative effects of widespread badger culling on tuberculosis in cattle. *Nature*, *439*, 843–846.

Drewe, J. A., Tomlinson, A. J., Walker, N. J., & Delahay, R. J. (2010). Diagnostic accuracy and optimal use of three tests for tuberculosis in live badgers. *PLoS ONE*, *5*, e11196.

Ezenwa, V. O., Archie, E. A., Craft, M. E., Hawley, D. M., Martin, L. B., Moore, J., & White, L. (2016). Host behaviour–parasite feedback: An essential link between animal behaviour and disease ecology. *Proceedings of the Royal Society of London B*, *283*, 20153078.

Ezenwa, V. O., Ghai, R. R., McKay, A. F., & Williams, A. E. (2016). Group living and pathogen infection revisited. *Current Opinion in Behavioral Sciences*, *12*, 66–72.

Frantz, A., San, E. D. L., Pope, L., & Burke, T. (2010). Using genetic methods to investigate dispersal in two badger (*Meles meles*) populations with different ecological characteristics. *Heredity*, *104*, 493–501.

Gallagher, J., & Horwill, D. (1977). A selective oleic acid albumin agar medium for the cultivation of *Mycobacterium bovis*. *Journal of Hygiene*, *79*, 155–160.

Garnett, B., Delahay, R., & Roper, T. (2005). Ranging behaviour of European badgers (*Meles meles*) in relation to bovine tuberculosis (*Mycobacterium bovis*) infection. *Applied Animal Behaviour Science*, *94*, 331–340.

George, S. C., Smith, T. E., Mac Cana, P. S., Coleman, R., & Montgomery, W. I. (2014). Physiological stress in the Eurasian badger (*Meles meles*): Effects of host, disease and environment. *General and Comparative Endocrinology*, *200*, 54–60.

Godfray, H. C. J., Donnelly, C. A., Kao, R. R., Macdonald, D. W., McDonald, R. A., Petrokofsky, G., … McLean, A. R. (2013). A restatement of the natural science evidence base relevant to the control of bovine tuberculosis in Great Britain. *Proceedings of the Royal Society of London B*, *280*, 20131634.

Goodger, J., Nolan, A., Russell, W., Dalley, D., Thorns, C., Stuart, F., … Newell, D. (1994). Serodiagnosis of *Mycobacterium bovis* infection in badgers: Development of an indirect ELISA using a 25 kDa antigen. *The Veterinary Record*, *135*, 82–85.

Graham, J., Smith, G., Delahay, R., Bailey, T., McDonald, R., & Hodgson, D. (2013). Multi-state modelling reveals sex-dependent transmission, progression and severity of tuberculosis in wild badgers. *Epidemiology and Infection*, *141*, 1429–1436.

Griffin, J., Williams, D., Kelly, G., Clegg, T., O'Boyle, I., Collins, J., & More, S. (2005). The impact of badger removal on the control of tuberculosis in cattle herds in Ireland. *Preventive Veterinary Medicine*, *67*, 237–266.

Hassell, J. M., Begon, M., Ward, M. J., & Fèvre, E. M. (2017). Urbanization and disease emergence: Dynamics at the wildlife–livestock–human interface. *Trends in Ecology & Evolution*, *32*, 55–67.

Hudson, P. J., Rizzoli, A., Grenfell, B. T., Heesterbeek, H., & Dobson, A. P. (2002). *The ecology of wildlife diseases*. Oxford, UK: Oxford University Press.

Jenkins, H. E., Cox, D., & Delahay, R. J. (2012). Direction of association between bite wounds and *Mycobacterium bovis* infection in badgers: Implications for transmission. *PLoS ONE*, *7*, e45584.

Johnson, C. K., Tinker, M. T., Estes, J. A., Conrad, P. A., Staedler, M., Miller, M. A., … Mazet, J. A. (2009). Prey choice and habitat use drive sea otter pathogen exposure in a resource-limited coastal system. *Proceedings of the National Academy of Sciences of the United States of America*, *106*, 2242–2247.

Jones, K. E., Patel, N. G., Levy, M. A., Storeygard, A., Balk, D., Gittleman, J. L., & Daszak, P. (2008). Global trends in emerging infectious diseases. *Nature*, *451*, 990–993.

Lloyd-Smith, J. O., Schreiber, S. J., Kopp, P. E., & Getz, W. M. (2005). Superspreading and the effect of individual variation on disease emergence. *Nature*, *438*, 355–359.

Macdonald, D. W., Newman, C., Buesching, C. D., & Nouvellet, P. (2010). Are badgers 'Under The Weather'? Direct and indirect impacts of climate variation on European badger (*Meles meles*) population dynamics. *Global Change Biology*, *16*, 2913–2922.

McDonald, J. L., Bailey, T., Delahay, R. J., McDonald, R. A., Smith, G. C., & Hodgson, D. J. (2016). Demographic buffering and compensatory recruitment promotes the persistence of disease in a wildlife population. *Ecology Letters*, *19*, 443–449.

McDonald, J. L., Smith, G. C., McDonald, R. A., Delahay, R. J., & Hodgson, D. (2014). Mortality trajectory analysis reveals the drivers of sex-specific epidemiology in natural wildlife–disease interactions. *Proceedings of the Royal Society of London B*, *281*, 20140526.

Newell, D., Clifton-Hadley, R., & Cheeseman, C. (1997). The kinetics of serum antibody responses to natural infections with *Mycobacterium bovis* in one badger social group. *Epidemiology and Infection*, *118*, 173–180.

Nunn, C. L., Craft, M. E., Gillespie, T. R., Schaller, M., & Kappeler, P. M. (2015). The sociality–health–fitness nexus: Synthesis, conclusions and future directions. *Philosophical Transactions of the Royal Society of London B*, *370*, 20140115.

Palmer, M. V., Thacker, T. C., Waters, W., Gortázar, C., & Corner, L. A. (2012). *Mycobacterium bovis*: A model pathogen at the interface of livestock, wildlife, and humans. *Veterinary Medicine International*, *2012*, 1–17.

Parrish, N. M., Dick, J. D., & Bishai, W. R. (1998). Mechanisms of latency in *Mycobacterium tuberculosis*. *Trends in Microbiology*, *6*, 107–112.

Restif, O., Hayman, D. T., Pulliam, J. R., Plowright, R. K., George, D. B., Luis, A. D., … O'Shea, T. J. (2012). Model-guided fieldwork: Practical guidelines for multidisciplinary research on wildlife ecological and epidemiological dynamics. *Ecology Letters*, *15*, 1083–1094.

Robertson, A., McDonald, R. A., Delahay, R. J., Kelly, S. D., & Bearhop, S. (2014). Individual foraging specialisation in a social mammal: The European badger (*Meles meles*). *Oecologia*, *176*, 409–421.

Robertson, A., McDonald, R. A., Delahay, R. J., Kelly, S. D., & Bearhop, S. (2015). Resource availability affects individual niche variation and its consequences in group-living European badgers *Meles meles*. *Oecologia*, *178*, 31–43.

Rogers, L., Cheeseman, C., & Langton, S. (1997). Body weight as an indication of density-dependent population regulation in badgers (*Meles meles*) at Woodchester Park, Gloucestershire. *Journal of Zoology*, *242*, 597–604.

Rogers, L., Delahay, R., Cheeseman, C., Langton, S., Smith, G., & Clifton-Hadley, R. (1998). Movement of badgers (*Meles meles*) in a high–density population: Individual, population and disease effects. *Proceedings of the Royal Society of London B: Biological Sciences*, *265*, 1269–1276.

Rohani, P., & King, A. A. (2010). Never mind the length, feel the quality: The impact of long-term epidemiological data sets on theory, application and policy. *Trends in Ecology & Evolution*, *25*, 611–618.

Roper, T. (2010). *Badger (Collins New Naturalist Library, Book 114)*. London, UK: HarperCollins.

Schmid-Hempel, P. (2003). Variation in immune defence as a question of evolutionary ecology. *Proceedings of the Royal Society of London B: Biological Sciences*, *270*, 357–366.

Shirley, M. D., Rushton, S. P., Smith, G. C., South, A. B., & Lurz, P. W. (2003). Investigating the spatial dynamics of bovine tuberculosis in badger populations: Evaluating an individual-based simulation model. *Ecological Modelling*, *167*, 139–157.

Sin, Y. W., Annavi, G., Dugdale, H. L., Newman, C., Burke, T., & MacDonald, D. W. (2014). Pathogen burden, co-infection and major histocompatibility complex variability in the European badger (*Meles meles*). *Molecular Ecology*, *23*, 5072–5088.

Smith, K. F., Sax, D. F., & Lafferty, K. D. (2006). Evidence for the role of infectious disease in species extinction and endangerment. *Conservation Biology*, *20*, 1349–1357.

Tolhurst, B. A., Delahay, R. J., Walker, N. J., Ward, A. I., & Roper, T. J. (2009). Behaviour of badgers (*Meles meles*) in farm buildings: Opportunities for the transmission of *Mycobacterium bovis* to cattle? *Applied Animal Behaviour Science*, *117*, 103–113.

Tomlinson, A., Chambers, M., Carter, S., Wilson, G., Smith, G., McDonald, R., & Delahay, R. (2013). Heterogeneity in the risk of *Mycobacterium bovis* infection in European badger (*Meles meles*) cubs. *Epidemiology and Infection*, *141*, 1458–1466.

Tomlinson, A., Chambers, M., & Delahay, R. (2012). *Mycobacterium bovis* infection in badger cubs: Re-assessing the evidence for maternally derived immunological protection from advanced disease. *Veterinary Immunology and Immunopathology*, *148*, 326–330.

Tomlinson, A. J., Chambers, M. A., McDonald, R. A., & Delahay, R. J. (2015). Association of quantitative interferon-γ responses with the progression of naturally acquired *Mycobacterium bovis* infection in wild European badgers (*Meles meles*). *Immunology*, *144*, 263–270.

Tomlinson, A., Chambers, M., Wilson, G., McDonald, R., & Delahay, R. (2013). Sex-related heterogeneity in the life-history correlates of *Mycobacterium bovis* infection in European badgers (*Meles meles*). *Transboundary and Emerging Diseases*, *60*, 37–45.

VanderWaal, K. L., & Ezenwa, V. O. (2016). Heterogeneity in pathogen transmission: Mechanisms and methodology. *Functional Ecology*, *30*, 1606–1622.

Vicente, J., Delahay, R., Walker, N., & Cheeseman, C. (2007). Social organization and movement influence the incidence of bovine tuberculosis in an undisturbed high-density badger *Meles meles* population. *Journal of Animal Ecology*, *76*, 348–360.

Weber, N., Bearhop, S., Dall, S. R., Delahay, R. J., McDonald, R. A., & Carter, S. P. (2013). Denning behaviour of the European badger (*Meles meles*) correlates with bovine tuberculosis infection status. *Behavioral Ecology and Sociobiology*, *67*, 471–479.

Weber, N., Carter, S. P., Dall, S. R., Delahay, R. J., McDonald, J. L., Bearhop, S., & McDonald, R. A. (2013). Badger social networks correlate with tuberculosis infection. *Current Biology*, *23*, R915–R916.

Wiethoelter, A. K., Beltrán-Alcrudo, D., Kock, R., & Mor, S. M. (2015). Global trends in infectious diseases at the wildlife–livestock interface. *Proceedings of the National Academy of Sciences of the United States of America*, *112*, 9662–9667.

Wilkinson, D., Smith, G., Delahay, R., & Cheeseman, C. (2004). A model of bovine tuberculosis in the badger *Meles meles*: An evaluation of different vaccination strategies. *Journal of Applied Ecology*, *41*, 492–501.

Wilkinson, D., Smith, G., Delahay, R., Rogers, L., Cheeseman, C., & Clifton-Hadley, R. (2000). The effects of bovine tuberculosis (*Mycobacterium bovis*) on mortality in a badger (*Meles meles*) population in England. *Journal of Zoology*, *250*, 389–395.

Woodroffe, R., Donnelly, C. A., Cox, D., Bourne, F., Cheeseman, C., Delahay, R., … Morrison, W. (2006). Effects of culling on badger *Meles meles* spatial organization: Implications for the control of bovine tuberculosis. *Journal of Applied Ecology*, *43*, 1–10.

Woodroffe, R., Donnelly, C. A., Wei, G., Cox, D., Bourne, F. J., Burke, T., … Gilks, P. (2009). Social group size affects *Mycobacterium bovis* infection in European badgers (*Meles meles*). *Journal of Animal Ecology*, *78*, 818–827.

SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article.

How to cite this article: McDonald JL, Robertson A, Silk MJ. Wildlife disease ecology from the individual to the population: Insights from a long-term study of a naturally infected European badger population. *J Anim Ecol*. 2017;00:1–12. https://doi.org/10.1111/1365-2656.12743