Received: 27 February 2017 | Accepted: 4 July 2017
DOI: 10.1111/1365-2664.12968

RESEARCH ARTICLE

Journal of Applied Ecology BRITISH ECOLOGICAL SOCIETY

Invasive pathogen drives host population collapse: Effects of a travelling wave of sarcoptic mange on bare-nosed wombats

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Funding information
Holsworth Wildlife Research Endowment; Cradle Coastal Authority

Handling Editor: Luke Flory

Abstract

1. Emerging and invasive pathogens can have long-lasting impacts on susceptible wildlife populations, including localized collapse and extirpation. Management of threatening disease is of widespread interest and requires knowledge of spatiotemporal patterns of pathogen spread.
2. Theory suggests disease spread often occurs via two patterns: homogenous mixing and travelling waves. However, high-resolution empirical data demonstrating localized (within population) disease spread patterns are rare.
3. This study examined the spread of sarcoptic mange (aetiological agent *Sarcoptes scabiei*) in a population of bare-nosed wombats (*Vombatus ursinus*), and investigated whether pathogen spread occurred by homogenous mixing or a travelling wave.
4. Using 7 years of population surveys and 4 years of disease severity surveys, we show that mange was first detected in the east of a wombat population in northern Tasmania, and progressed westward as a travelling wave. Wombat mortality rates reached 100% behind the wave, with a 94% decline in overall wombat abundance within the park.
5. *Synthesis and applications*. Globally distributed pathogens may have severe impacts on susceptible host species. This is the first study to quantify population-level impacts of sarcoptic mange upon bare-nosed wombats, showing a wave of mange disease which resulted in a dramatic population decline. Successful management of the spread of this and similar pathogens may hinge on the capacity to establish transmission barriers at local or between-population scales.

KEYWORDS
bare-nosed wombat, disease invasion, disease spread, disease transmission, disease wave, homogenous mixing, invasive pathogens, sarcoptic mange, travelling wave

1 | INTRODUCTION

Invasive pathogens impose a major threat to wildlife conservation globally (Smith, Acevedo-Whitehouse, & Pedersen, 2009; Thompson, Lymbery, & Smith, 2010; Tompkins, Carver, Jones, Krkošek, & Skerratt, 2015). The arrival of virulent pathogens into naïve host populations can result in dramatic population declines and localized host extinctions (Smith et al., 2009; Thompson et al., 2010). Notable examples include white-nose syndrome in north American hibernating bat species (Blehert et al., 2009) and chytrid fungus in amphibian species (Briggs, Knapp, & Vredenburg, 2010). However, few empirical studies exist documenting the spatiotemporal pattern of disease spread and host population decline during epizootics. Understanding how pathogens spread through naïve and susceptible host populations is important as it can inform strategies for disease management, such as targeted treatment or vaccination campaigns (Lange, Kramer-Schadt, & Thulke, 2012).

Travelling waves and homogenous mixing represent two alternative patterns by which pathogens are observed to invade host populations. For virulent pathogens, these patterns of pathogen spread may also manifest as patterns of host decline (e.g. Frick et al., 2010; Skerratt et al. 2007). Under homogenous mixing, the pathogen rapidly spreads following arrival with little detectable spatial or temporal structure in the pattern of population decline. In contrast, with travelling waves, the pathogen spreads as a disease front, with a spatiotemporal rate of decline. The type of pathogen or disease spread pattern observed may vary with scale, even within the same system, owing to differing host–pathogen contact rates. For example, disease spread may occur by homogenous mixing at a local scale (within a population of individuals capable of randomly mixing) where host–host or host–pathogen contact rates are high, with spread by travelling wave observable at regional scales (multiple groups of non-randomly mixing individuals scales) where contact rates are low. There are several well-documented regional scale examples for both homogenous mixing and travelling waves (Biek, Henderson, Waller, Rupprecht, & Real, 2007; Conner & Miller, 2004; Foley, Clifford, Castle, Cryan, & Ostfeld, 2011; LaDeau, Kilpatrick, & Marra, 2007; Lips et al., 2006; Lucey et al., 2002; Vredenburg, Knapp, Tunstall, & Briggs, 2010; Walsh, Biek, & Real, 2005), while a paucity of empirical disease spread data exists at the local scale. The lack of information regarding disease spread at local scales may be due to the challenges in acquiring the high-resolution data needed to reveal spread patterns. While regional patterns of disease spread are often well understood, these patterns may not be conserved, or applied, at local scales.

Research into the dynamics of pathogen spread is critical for disease management in wildlife populations particularly at local scales, because effective intervention strategies are reliant upon some understanding of the nature by which target pathogens spread (Wobeser, 2002). Effective management methods for pathogen spread by homogenous mixing may include widespread vaccination events (Killian, Fagerstone, Kreeger, Miller, & Rhyan, 2007). Conversely, successful intervention for pathogens that spread via travelling waves may require alternative measures, such as establishing barriers to pathogen movement (a tactic proposed to protect livestock from wildlife disease spill-over [Gortazar et al., 2015]). There is an urgent need to couple empirical studies of pathogen spread with advances in disease management, particularly due to the increased threat of invasive pathogens for the persistence of small, isolated wildlife populations (Smith et al., 2009; Tompkins et al., 2015).

Sarcoptes scabiei is among the most widespread of parasitic mites, infecting >100 mammal species globally (Bornstein, Mörner, & Samuel, 2001; Pence & Ueckermann, 2002). It is considered an emerging and invasive pathogen of many wildlife populations (Bornstein et al., 2001; Pence & Ueckermann, 2002; Tompkins et al., 2015). The *S. scabiei* mite burrows into the outer epidermal layers of its host, causing a range of symptoms including alopecia, pruritus, hyperkeratosis and emaciation (Pence & Ueckermann, 2002). The global dispersal of this pathogen is likely the result of human host movement, with spill-over into naïve domestic animals and wildlife populations where it can have dramatic population impacts (Fraser, Charleston, Martin, Polkinghorne, & Carver, 2016; Pence & Ueckermann, 2002). Notable examples of this pathogen impacting wildlife include wombats (Skerratt, 2005), coyotes (Murray, Edwards, Abercrombie, & St. Clair, 2015), arctic and red foxes (Forchhammer & Asferg, 2000; Mörner, 1992), lions and cheetahs (Gakuya et al., 2012), grey wolves (Jimenez, Bangs, Sime, & Asher, 2010) and mountain gorillas (Graczyk, Mudakikwa, Cranfield, & Eilenberger, 2001). Despite the array of host species affected, the spatial structure of disease spread is not well understood (Pence & Ueckermann, 2002), in part due to the complexity of transmission. Transmission can occur through both direct and indirect contact, as mites can survive in the environment for short periods (Arlian, Vyszenski-Moher, & Pole, 1989). Little empirical data exist describing the pattern of mange spread during outbreaks.

Sarcoptic mange is arguably the most important disease of wombats in Australia, affecting two of the three extant species (Hartley & English, 2005; Martin, Handasyde, & Skerratt, 1998; Skerratt, 2005; Thompson, Kutz, & Smith, 2009). Anecdotal reports of mange causing population declines and localized extirpation events exist (Martin et al., 1998), but empirical documentation of such events are currently lacking. This knowledge gap is increasingly relevant for attempts to manage sarcoptic mange in this iconic Australian marsupial. A greater understanding of pathogen spread will help implement intervention programs for this and similar host-invasive pathogen scenarios. Using 7 years of population surveys and 4 years of pathogen severity surveys from northern Tasmania, this study examines the spatiotemporal characteristics of pathogen spread and host population decline at a local (within population) scale. We investigate whether *S. scabiei* spread occurs via homogenous mixing or a travelling wave at this scale.

2 | MATERIALS AND METHODS

2.1 | Ecology and social organization of bare-nosed wombats and mange transmission

Bare-nosed wombats are the largest burrowing herbivorous mammals, and utilize a network of core and peripheral burrows to take shelter during mostly diurnal conditions (Johnson, 1998; Triggs, 2009). They are solitary (having limited direct physical interaction among adult individuals) and non-territorial (Evans, 2008; Favreau et al., 2010). Sarcoptic mange infections in wombats cause severe alopecia, skin lesions and parakeratosis (Skerratt, 2003). Wombats may transmit mange between one another by direct contact or via the environment, with environmental transmission being the more widely supported hypothesis (Skerratt, Martin, & Handasyde, 1998). Potential environmental transmission may occur through burrow sharing.

2.2 | Data collection

2.2.1 | Field location and transect surveys

This study took place at Narawntapu National Park (NNP), located on the central-north coast of Tasmania, Australia (−41°N, 146°W, see Figure 1). The park contains a variety of habitats, including costal heathlands, eucalypt and she oak forest, grassland, schlerophyll woodland, bracken/scrubland and ex-agricultural land. The portion of the park that was historically converted to grassland for pasture (3.29 km^2) and used as farmland is now utilized by macropods and wombats for grazing. These areas are surrounded on three sides: the east by a small mountain range (Briggs Regional Reserve), the west by Rubicon Estuary, and the north by the Bass Strait. These natural features create a semi-isolated wombat population, with the only potential avenues for immigration/emigration being through the south and east (Figure 1). Open landscapes and the increased diurnal behaviour of Tasmanian wombats (relative to mainland wombats, see "Discussion" section) allow for extensive monitoring of the population at NNP, and thus a unique opportunity to document spatial and temporal changes in wombat abundance and disease spread.

Wombat abundance surveys were conducted annually from 2010, which coincided with the year a mange outbreak was first detected in this population. These abundance surveys were later supplemented by disease severity surveys, conducted simultaneously from 2013. Combined, these surveys document the spatiotemporal impact of *S. scabiei* on the wombat population, and spread of the pathogen, as assessed by visual signs of mange. For wombat abundance, 12–15 transects were walked each September within the grassland habitat of the park. All transects were walked at dawn, simultaneously, and repeated once per day for three to four consecutive days (effort 2010–2012, 2016 = 3 days; 2013–2015 = 4 days). Observations of each wombat included the distance from the transect (using a Nikon Forestry Pro Range Finder) and the transect number. Natural geographic features within NNP result in five logical areas for which transects and observations of wombats occur within, but not between (Figure 2). These features are predominately tree lines not only along drainage ditches but also include buildings between Areas 3 and 4. Within each area, there was some minor variation in the number and placement of transects each year (see Table S1). Thus, wombat abundance was estimated for each park area, based on total kilometres of transect surveyed within a given area, and averaged across survey days (3–4 days) to produce

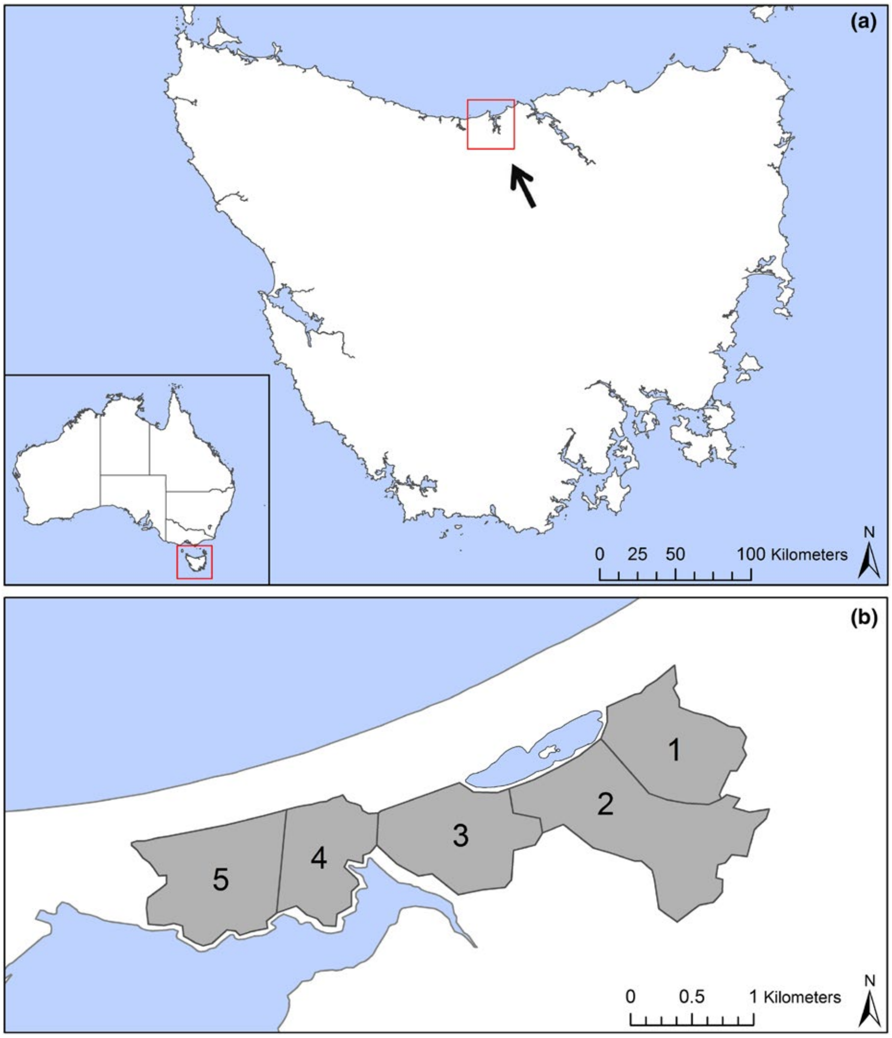

FIGURE 1 (a) Narawntapu National Park, located on the central-north coast of Tasmania, Australia (−41.150, 146.600). (b) The five surveying areas within Narawntapu National Park (grey shading). The total surveying area is 3.29 km^2. Areas are numbered from east to west, corresponding with the first observation of mange (originated in Area 1). The areas are pasture, which are surrounded by forest, waterways and hills [Colour figure can be viewed at wileyonlinelibrary.com]

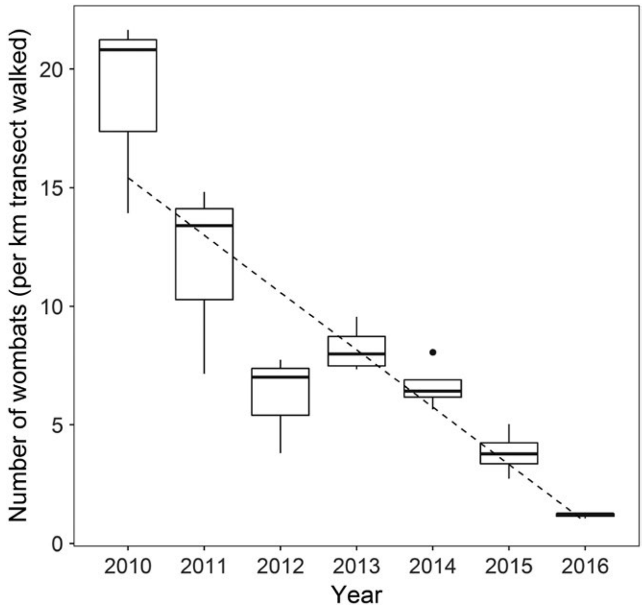

FIGURE 2 The change in wombat abundance at Narawntapu National Park from 2010 to 2016 ($R^2 = 0.7215$, $F_{1,22} = 60.57$, $p < .001$). Wombat abundance was derived from all transects surveyed in all areas, and was averaged across three to four sampling days for each year. The median number of wombats is presented by the black line within each box and the linear regression is the dotted line. Over 6 years, wombat abundance declined by 93.6%

the average number of wombats per kilometre. These surveys enabled documentation of spatiotemporal wombat decline during the mange outbreak.

Targeted mange severity surveys were performed from 2013 to 2016. Two groups of observers started in opposite ends of the park (one in the east, one in the west), surveying the entirety of each area by walking systematic north–south sweeps, and converging at approximately the central area of the park. As surveying groups came into visual contact, communication via hand-held radio was used to ensure all wombats were only recorded once. For each wombat spotted, a GPS location and mange severity score was taken. These surveys started at noon and continued until all wombats in the grasslands were scored. Wombats were observed using a Leica TELEVID 77 spotting scope (20–60× zoom) and Nikon binoculars (10 × 24), and infections were visually diagnosed.

Mange scores were assigned following Simpson, Johnson, and Carver (2016), by which the wombat is divided into 14 body segments and each is assigned a mange score from 0 to 10. Clinical signs of mange that are visually obvious develop around 14 days after infection (Skerratt, 2003). Our surveys were based on visual observation of clinical mange, but the actual disease front is likely ahead of the visual front. Thus, the front reported here is indicative of symptomatic disease. We ranked mange severity by the highest body segment score assigned to an individual wombat, as follows: highest segment score of 0–2 healthy, 3 early mange, 4–6 moderate mange, 7–8 severe mange, 9–10 late stage (see Figure S1). Individuals with body segment scores ≥9 are less common due to mortality from poor body condition, emaciation and infection severity that accompany this state. We believe, based on longitudinal observations, that this is a slightly more robust approach than our previous methodology (Simpson et al., 2016) where a segment score of two was indicative of mange. Analyses performed with the Simpson et al. (2016) classification deviated only slightly from the results presented here (see Figure S2).

2.3 | Analyses

2.3.1 | Wombat abundance and spatial decline

We used the multi-year wombat abundance surveys to test the spatial and temporal pattern of population decline in the park. To understand the park-wide change in wombat abundance from 2010 to 2016 (mean number of wombats per kilometre, across all transects), a linear regression was performed. To explore temporal patterns in changing wombat abundance within each area, piece-wise linear models were used. Each area was fitted with two linear segments (segmented R package, Muggeo, 2008) to identify (1) when wombat abundance began to decline, and (2) the intensity of the decline (slopes of the segments). To understand the spatiotemporal component of declining wombat abundance, the relationship between the distance of each area from the eastern grazing limit and the mid-point (year) of respective area decline was examined using a linear regression. The outbreak is confirmed to have begun in the east, but the exact location of the origin in the eastern area is not certain. Therefore, the distance to each area was quantified as the distance (m) from the eastern grazing limit of the park (longitude 146.6253°W) to the longitudinal mid-point of each area.

2.3.2 | Mange front progression and connecting disease to wombat abundance

To understand shifts in the geographic distribution of mange and estimate the rate of pathogen movement within NNP 2013–2014, 2014–2015 and 2015–2016, wombat locations and mange severity scores were mapped using ArcMap 10.2. The surface between wombat locations was smoothed using inverse distance weighting interpolation based on the mange score assigned. The mange front progression was defined by the longitudinal change in the western-most location of an individual with one or more body segments exhibiting a score ≥3.

To assess the population effect of sarcoptic mange on wombats, we evaluated the relationship between mange prevalence and wombat abundance. This relationship was quantified for NNP Areas 3–5, although Area 3 was excluded from the statistical analyses owing to only having a single year of mange and wombat abundance data before this area was extirpated. Mange prevalence calculations were based on the number of wombats in the area with any body segment scoring ≥3, with 95% confidence intervals calculated using the 1-sample proportions test with continuity correction (R, Newcombe, 1998). We used a Bayesian mixed effects regression model, with park area as a random effect, to test how mange prevalence predicted wombat abundance (MCMCglmm R package, Hadfield, 2010). We also demonstrated that the relationship obtained was not due to park area differences by conducting linear regression analyses for each area, independently. Analyses for each area were conducted from the year before mange was observed to year 2016.

3 | RESULTS

3.1 | Wombat abundance and spatial decline

Wombat abundance at NNP declined significantly from 2010 to 2016, dropping 93.6% from 18.6 wombats per km in 2010 to 1.2 wombats per km in 2016 (linear regression, $R^2 = 0.7215$, $F_{1,22} = 60.57$, $p < .001$; Figure 2). The decline in wombat abundance was observed in all five park areas, and complete collapse was observed in three of five areas by 2016 (abundance = 0, Figure 3). The mange outbreak started in the eastern end of the park with significant declines initiating in 2010 for Area 1 ($p < .001$), Area 2 ($p < .001$), and Area 3 ($p = .002$), based on the piece-wise linear regressions (Table 1; piece-wise linear regression *p*-values represent slope coefficients, significant values correspond to slopes that are significantly different from zero). These areas experienced complete population collapse in 2014, 2016 and 2014 respectively. Rates of decline varied within the eastern and central areas, with the steepest declines occurring in Area 2, followed by Area 1, and more gradual decline in Area 3 (Table 1). Continued progression of mange through the western areas of the park resulted in a wave-like decline, with Area 4 experiencing significant declines starting in July of 2013 (breakpoint estimate = 2013.56; decline $p = .005$), and Area 5 declines beginning in April of 2014 (breakpoint estimate = 2014.38; decline $p = .184$). There was a notable relationship between the distance of the areas from the eastern end of the park and the year that abundance declines began within areas ($R^2 = 0.76$, $F_{1,3} = 13.83$, $p = .03$; Figure 4), showing a spatiotemporal wave of decline.

3.2 | Mange front progression and prevalence-abundance relationships

A total of 105 wombats were scored for mange across 4 years. Each received either a full score (all 14 segments, $N = 98$), a half score (entire right or left side, $N = 6$), or a partial score (<7 segments of one side, $N = 1$). Half and partial scores were the result of wombats fleeing to burrows before score completion. The mange front progressed westward annually from 2013 to 2016 (Figure 5). The western limit of

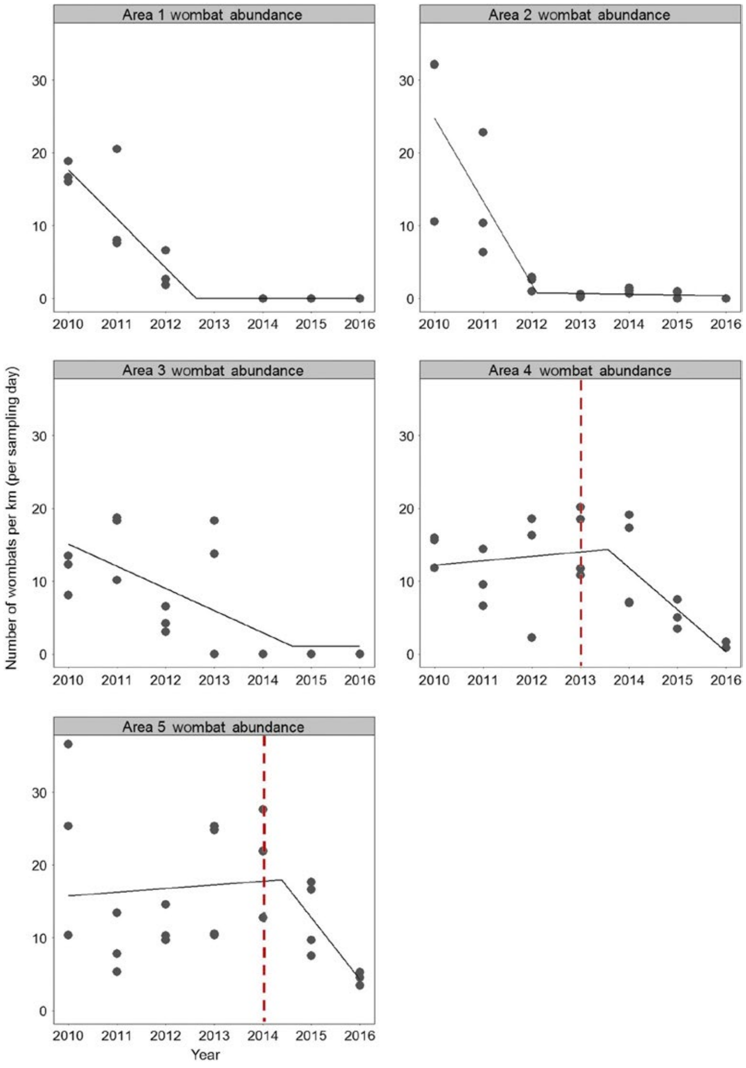

FIGURE 3 Temporal and spatial decline of wombats within Narawntapu National Park. Each area of the park (Figure 1b) was surveyed from 2010 to 2016 (effort 2010–2012, 2016 = 3 days, 2013–2015 = 4 days). Piece-wise linear models were used to quantify the relationship between wombat abundance and time. Each linear model was fitted with two segmented relationships. The eastern end of the park was the location where the outbreak was first observed in 2010, and Areas 1, 2 and 3 exhibited declines from 2010. Consecutive declines were observed as the front progressed into the west: Area 4 (2013) and Area 5 (2014). The vertical, red dashed line in Areas 4 and 5 represent the arrival of the mange front [Colour figure can be viewed at wileyonlinelibrary.com]

TABLE 1 Patterns of wombat abundance over time for each area of NNP. Patterns assessed by piece-wise linear regressions, fitted with segmented relationships (two per area)

	Regression piece 1			Breakpoint		Regression piece 2			
Area	Coefficient	*SE*	*p*-value	Estimate	*SE*	Coefficient	*SE*	*p*-value	R^2 (overall)
1	−6.73	±1.61	<.001	2012.63	±0.50	<0.01	±1.08	1.00	0.85
2	−11.38	±1.96	<.001	2012.10	±0.35	−0.16	±1.15	.893	0.76
3	−3.04	±0.86	.002	2014.62	±1.45	<0.01	±3.86	1.00	0.47
4	0.60	±1.61	.611	2013.56	±0.61	−5.74	±1.83	.005	0.44
5	0.51	±1.37	.715	2014.38	±0.89	−8.45	±6.15	.184	0.13

mange infections varied between years, moving 733 m west between 2013 and 2014, an additional 276 m west between 2014 and 2015, and only 6 m between 2015 and 2016 as a consequence of reaching the western-most limit of the parks observable wombat foraging area. The net progression was 1,015 m over four consecutive survey years (average per year 338.3 m ± 367.5 *SD*).

Decline in wombat abundance within areas correlated with the movement of the mange front into these areas. As mange prevalence within a given area increased, wombat abundance decreased (Figure 6). Mange entered Area 4 in 2013 (S. Carver, pers. observ.) and Area 5 in 2014. A significant negative relationship between mange prevalence and wombat abundance was observed (coefficient = −14.977; 95% CI −26.53 to −3.87; *p* = .018; Figure 7), where wombat abundance decreased as mange prevalence increased. Linear regressions revealed this relationship was consistent within areas (Area 4 abundance, 2013–2016, $\beta = -0.19 \pm 0.03$ *SE*, $R^2 = 0.946$, $F_{1,2} = 53.93$, $p = .02$; Area 5 abundance, 2014–2016, $\beta = -0.12 \pm 0.001$ *SE*, $R^2 = 0.999$, $F_{1,2} = 9159$, $p < .01$), showing western wombat population declines occurring concomitantly with the westward spread of sarcoptic mange.

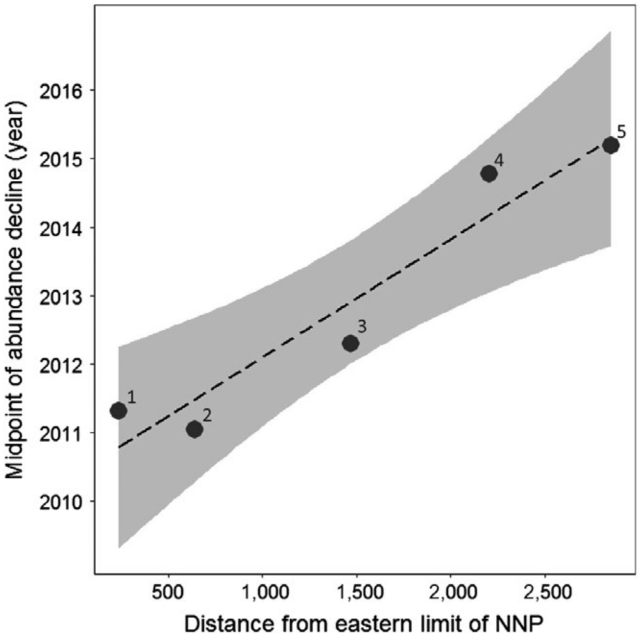

FIGURE 4 The spatiotemporal pattern of wombat decline within Narawntapu National Park as derived from piece-wise linear regression estimates of the mid-point of the decline slope within each area. There is a strong correlation between the distance an area (numbered 1–5 on graph) is from eastern limit of the park, and the mid-point (year) of the decline in that respective area ($R^2 = 0.89$, $F_{1,3} = 34.39$, $p < .01$)

4 | DISCUSSION

Through high spatial resolution surveys, we show that sarcoptic mange disease spreads as a travelling wave through a population of bare-nosed wombats. Invasive pathogens have the capacity to significantly reduce the population size of naïve hosts, and potentially drive localized extinction (Daszak, Cunningham, & Hyatt, 2000; Lafferty & Kuris, 2005; Pedersen, Jones, Nunn, & Altizer, 2007). This pathogen caused a substantive population impact, resulting in a 100% decline of wombats in the eastern and central areas of the park, with a 94% decline overall and may lead to population collapse in the near future. This study is the first to empirically demonstrate the impact this pathogen can have on a wombat population. Furthermore, this study contributes empirical information on pathogen spread at the local (within population) scale, which, to the best of our knowledge, is rare in the literature.

Disease spread information is pivotal for developing management and intervention strategies. There remains much to be learned about how mange impacts wombats at a national level. Nevertheless, our study has some clear implications for mange disease management in threatened wombat populations. Our research suggests that effective management of mange may be achieved by establishing barriers (either physical or immunological) to pathogen spread across populations, or where feasible, by administration of a population scale treatment. Research testing management strategies of *S. scabiei* in wombat populations is currently underway (Martin, Burridge, Polkinghorne, Fraser, & Carver, in preparation). Owing to the global distribution of this pathogen, these findings may also apply to other impacted wildlife species, and more broadly to other similarly transmitted pathogens (Cunningham et al., 2008; Foley et al., 2016).

This study is the first to empirically document mange impacts upon a bare-nosed wombat population. There have been anecdotal reports of wombat population collapse in response to the disease previously (Martin et al., 1998), but empirical documentation has until now remained elusive. Reports of sarcoptic mange outbreaks date back to the 1930s (Gray, 1937; Skerratt et al., 1998). The often nocturnal behaviour of wombats has perhaps led to a dearth of population and pathogen studies for these marsupials. The combination of our results and anecdotal studies may indicate a significant disease burden upon bare-nosed wombats (possibly also southern hairy-nosed wombats) at a national scale, which should be investigated further. However, few reports have historically considered mange to be a mechanism for the extirpation of stable populations, as the disease spread is assumed to be self-limiting, based on host density (Pence & Ueckermann, 2002). Our study of an ongoing epizootic that is projected to result in localized extinction challenges this assumption. Mange may persist and impact wombats across a range of dynamic scenarios, from epizootic to stable chronic infectivity (Martin et al., 1998; Ruykys, Breed, Schultz, & Taggart, 2013; Ruykys, Taggart, Breed, & Schultz, 2009; Skerratt, Skerratt, Martin, & Handasyde, 2004), and we suggest that more critical quantitative assessments of the impacts of sarcoptic mange are necessary.

More broadly, this would not be the first instance of sarcoptic mange driving isolated and/or small host populations to extinction. Two examples include the Bornholm island red fox population in Denmark (Henriksen, Dietz, Henriksen, & Gjelstrup, 1993) and the isolated Las Rasos Spanish ibex population in Spain (León-Vizcaíno et al., 1999). In the first example, a mange epizootic extirpated the naïve fox population from Bornholm. In the latter example, a prior mange epizootic isolated the Las Rasos ibex population within Cazorla National Park. The disease persisted in the ibex population, only to flare again, driving the isolated population to extinction within 5 years. These examples, as well as our study, showcase the vulnerability of isolated and/or small populations to mange outbreaks. Two studies of mange spread at regional scales show a wave of spread (Soulsbury et al., 2007; Turchetto et al., 2014); however, ours is the first study to document the pattern of pathogen spread (including rates of advancement) at a within population scale, providing information which may be vital for disease mitigation. Mitigation of sarcoptic mange is particularly important in populations that are subjected to additional threats, such as the critically endangered northern hairy-nosed wombat (Hartley & English, 2005).

Predictions about the spatiotemporal spread of pathogens and associated management efforts are likely heavily influenced by host social behaviour. This is particularly the case when infection impacts host behaviour, which can change disease spread pattern. Sarcoptic mange infections result in a range of behavioural changes in the host which potentially impact the pattern of disease spread (Chronert, Jenks, Roddy, Wild, & Powers, 2007; Overskaug, 1993; Soulsbury et al., 2007). Specifically, behavioural changes for wombats affected by mange include travelling further than healthy wombats (seasonally, Skerratt et al., 2004), increased diurnal activity (Borchard, Eldridge, & Wright, 2012; Ruykys et al., 2009), spending more time scratching and drinking (Simpson et al., 2016), and spending more time being active outside of the burrow (Simpson et al., 2016). These changes may be the result of host manipulation by the pathogen to enhance

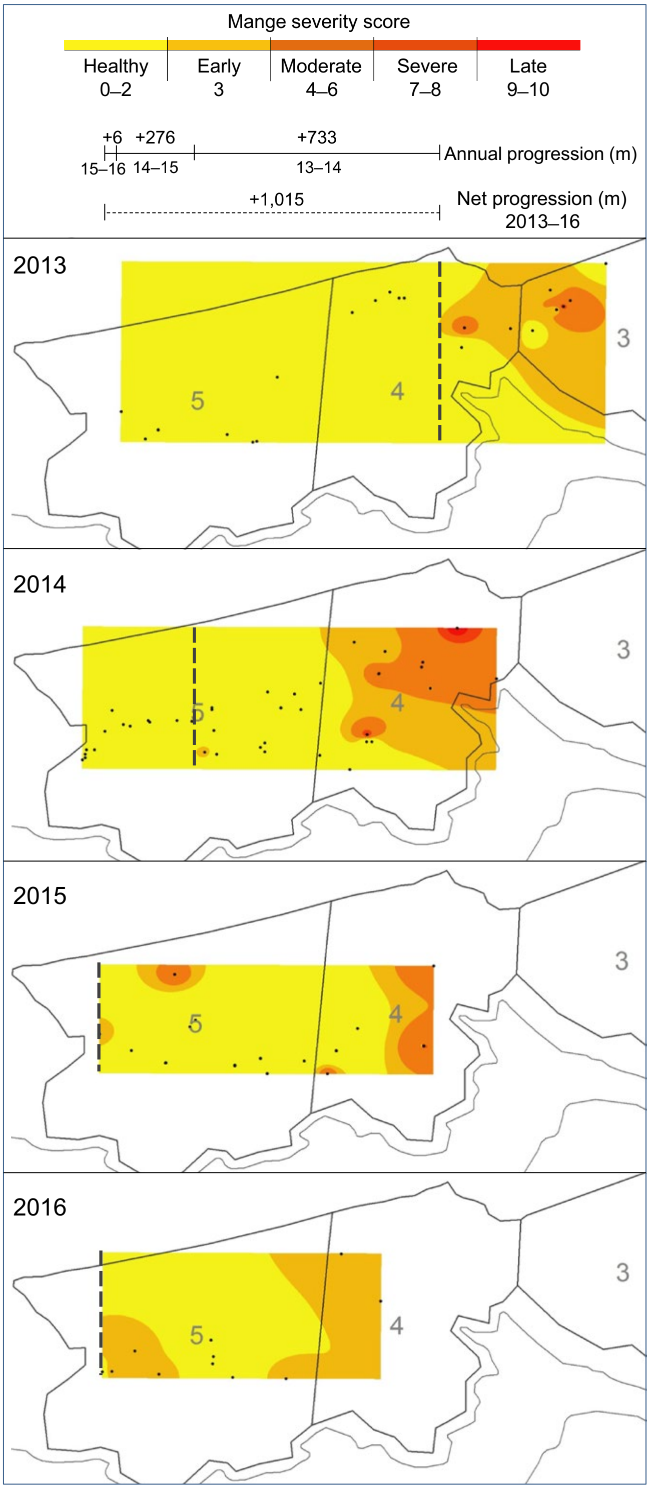

FIGURE 5 Progression of mange across Narawntapu National Park. Four consecutive years (2013–2016) of wombat locations with accompanying mange scores plotted in ArcMap, smoothed with inverse distance weighting (IDW) interpolation. Each black point represents an individual wombat observed. The mange front (dotted black line) progressed westward each year, moving a total of 1,015 m from 2013 to 2016. The size of the mange severity box represents the extent of wombats observed (black dots). Areas of NNP (Figure 1) are denoted by grey numbers [Colour figure can be viewed at wileyonlinelibrary.com]

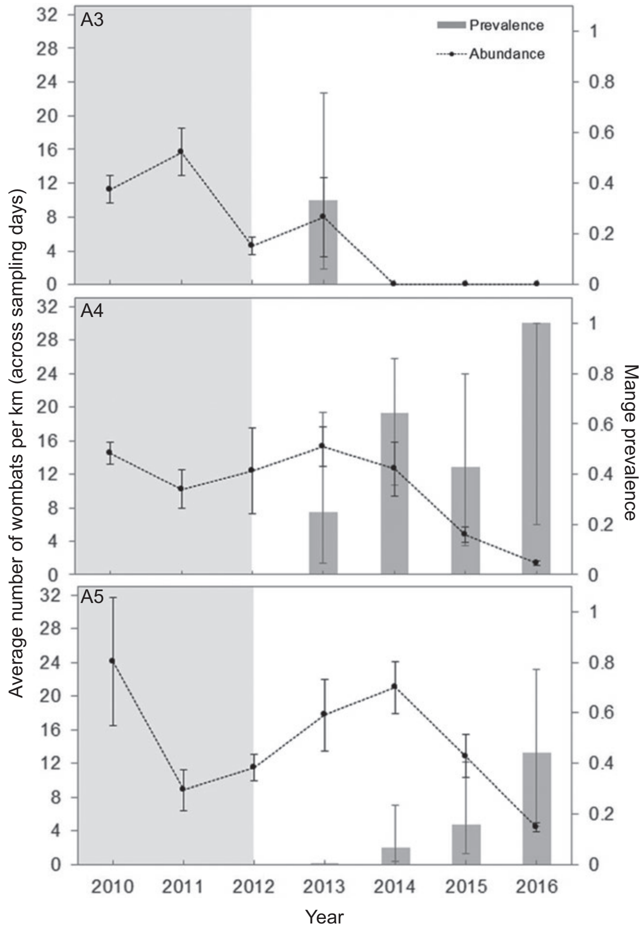

FIGURE 6 Mean (±*SE*) change in wombat abundance and mange prevalence (±95% CI) over time. Graphs are based on the four consecutive years (2013–2016) in which combined abundance and mange data were collected for Areas 3, 4 and 5 (A3, A4, and A5). Mange prevalence is quantified as the proportion of wombats exhibiting visual signs of mange. Collection of prevalence data began in 2013 (non-shaded part of graph), and thus there is uncertainty as to when mange entered Area 3. Mange entered Area 4 in 2013 and Area 5 in 2014. See Figure 7 for the relationship between wombat abundance and mange prevalence

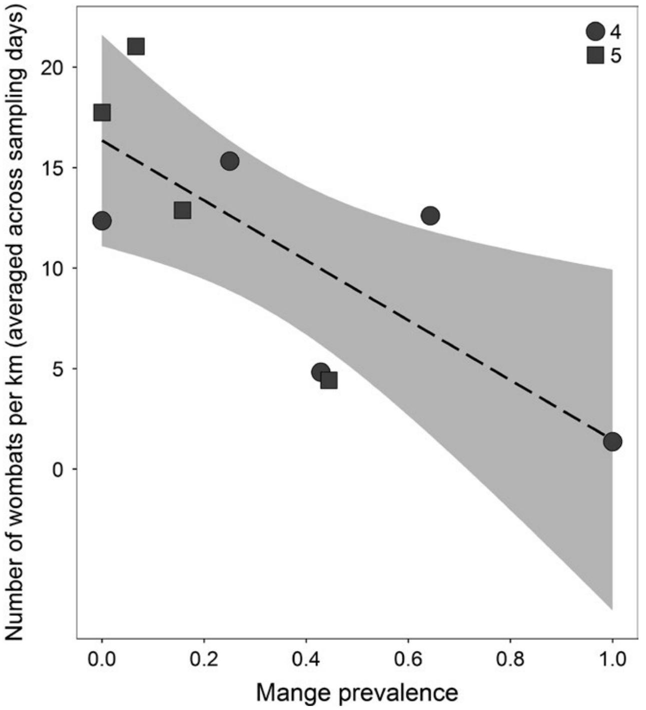

FIGURE 7 The relationship between wombat abundance and mange prevalence within NNP Areas 4 and 5. The relationship was assessed using abundance data from Areas 4 and 5, including the year prior to mange arrival (prevalence = 0) through to the last year of data collection (2016). As mange prevalence increased, wombat abundance decreased (Bayesian mixed effects regression with park area as the random effect—coefficient = −14.977; 95% CI −26.53 to −3.87; $p = .018$). Linear regressions were run for both Areas 4 and 5, using only years with both prevalence and abundance data (Area 4 2013–2016, Area 5 2014–2016). Regressions revealed significant abundance declines for both areas after the arrival of mange (Area 4, $R^2 = 0.946$, $F_{1,2} = 53.93$, $p = .02$; Area 5, $R^2 = 0.999$, $F_{1,2} = 9159$, $p < .01$)

transmission opportunities. This is particularly plausible when behavioural changes result in visitation to a greater number of burrows, given the hypothesized role that mite deposition in the burrow plays in transmission (Skerratt et al., 1998, 2004). However, the specific contribution of these behavioural changes in mange transmission dynamics is yet to be fully explored. Understanding behavioural changes and the projected progression of disease provides an opportunity for management to act ahead of the disease front, protecting uninfected and susceptible individuals.

It is possible that diurnal activity of mangy wombats, compared to more nocturnal behaviour of healthy wombats (Borchard et al., 2012), influenced detection of wombats in the eastern and central areas, confounding our conclusions of host population collapse in these areas. False signals of collapse could have resulted from the die-out of diurnally active, mangy wombats while nocturnally active, healthy wombats went undetected. However, two lines of evidence suggest this is not the case. (1) We informally undertook visual inspections of these areas each year, intensively searching for wombat scat, diggings and burrow activity, all of which could indicate false-negative conclusions of host collapse. These inspections re-affirmed our conclusions and suggest wombats are not colonizing these eastern most areas of NNP from the west or through immigration. (2) Our surveys from within the same population show Tasmanian wombats are more likely to be diurnal, regardless of health status (Simpson et al., 2016), as a result of less restrictive thermal conditions (Hogan et al., 2011; Triggs, 2009).

Disease is one of several threatening processes acting on bare-nosed wombat populations. Other major threats include: a reduced and fragmented range since European settlement (Buchan & Goldney, 1998; McIlroy, 1995); increased negative human interactions (Ramp, Caldwell, Edwards, Warton, & Croft, 2005); predation by feral species (Banks, 1997); and other disease threats (Donahoe et al., 2015; Skerratt, 1998). Here, we exemplify the need to understand the impacts of mange, especially on a formerly stable host population. While mange itself impacts host populations, the combination of threatening processes may exacerbate disease driven effects. For example, increased fragmentation creates a risk for isolated populations affected by mange, such as in this study, to be locally extirpated. A highly infectious disease, such as mange, has a high likelihood of affecting spatially separated, fragmented or distinct populations due to its longevity within populations (Cross, Lloyd-Smith, Johnson, & Getz, 2005). Furthermore, the loss of isolated populations throughout the bare-nosed wombat range may deplete genetic diversity and reduce gene exchange among metapopulations (Frankham, 2005).

Continued research into variables affecting mange transmission pattern is required to understand the complexity of mange epizootics. Specifically, there is a critical knowledge gap regarding the role that indirect (environmental) transmission plays in disease spread. Not only can mange be spread through direct contact but it also has an environmental component whereby under certain abiotic conditions, the *S. scabiei* mite can survive off a host for several weeks (Arlian et al., 1989; Pence & Ueckermann, 2002). Burrows provide a suitable microclimate for the *S. scabiei* mite, and likely act as a transmission pathway through burrow sharing by wombats (Skerratt et al., 1998). This environmental pathway hypothesis currently lacks scientific support, but remains a plausible hypothesis for more formal investigation. Further research into the frequency and mechanisms driving burrow sharing among wombats, the role of environmental pathogen spread and persistence, and disease dynamics of sarcoptic mange in other wombat populations are needed to fully appreciate the impacts of this pathogen across Australia. Filling these critical knowledge gaps will allow for more focused management and mitigation efforts.

5 | CONCLUSIONS

Here, we utilized empirical data to explore two hypotheses of pathogen spread within host populations, showing that a travelling wave model was supported for the invasion of sarcoptic mange into a bare-nosed wombat population. This pathogen had profound impacts upon the host population causing 100% mortality as the wave progressed. Our study contributes empirical evidence of the rate sarcoptic mange has spread in a bare-nosed wombat population. The evidence of spatiotemporal patterns for disease progression may also apply to other host populations, depending on host behaviour and density.

While this study helps to fill the knowledge gap regarding the effect and management of mange in Australian wildlife, there is still a need for further research. Critical questions remaining include: (1) what role do other Australian fauna (native and non-native) play in the spread of the mange mite? (2) What role does the burrow play in transmission and in mite survival? (3) What are the full effects of mange on host behaviour and physiology, and can these be managed (food supplementation, barrier to movement, etc.)? (4) Are similar declines/spread patterns occurring in other wombat populations at a national scale? Sarcoptic mange is among the most globally widespread of wildlife emerging infectious diseases (Tompkins et al., 2015), but is nonetheless a treatable infection. Through advances in understanding pathogen spread (such as in the present study), disease dynamic modelling, and future experiments into field treatment strategies, there are opportunities to establish evidence-based strategies of disease control for this and similarly transmitted pathogens in threatened wildlife populations.

ACKNOWLEDGEMENTS

We thank the Tasmanian Department of Primary Industries, Parks, and Environment (DPIPWE) and the Tasmanian Parks and Wildlife Services for their continued support and cooperation throughout this research. We also thank Sebastien Comte, Meggan Craft, Christopher Johnson, Amy Edwards, Rodrigo Hamede and Barry Brook for their suggestions and contributions during the drafting process. Lastly, we would like thank the Holsworth Wildlife Research Endowment and the Cradle Coastal Authority for their support of this work.

CONFLICT OF INTEREST

The authors declare no competing interests.

AUTHORS' CONTRIBUTIONS

The specific contributions are as follows: S.C., J.I. and C.P.B. conceived and designed the research; A.M.M., S.C., J.I. and C.P.B. collected all data; A.M.M. analysed the data; A.M.M., S.C., C.P.B. and T.A.F. interpreted results; A.M.M., S.C. and C.P.B. drafted the manuscript, and all authors participated in manuscript modifications.

DATA ACCESSIBILITY

All data available through the Dryad Digital Repository https://doi.org/10.5061/dryad.gd489 (Martin, Burridge, Ingram, Fraser, & Carver, 2017).

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How to cite this article: Martin AM, Burridge CP, Ingram J, Fraser TA, Carver S. Invasive pathogen drives host population collapse: Effects of a travelling wave of sarcoptic mange on bare-nosed wombats. *J Appl Ecol*. 2018;55:331–341. https://doi.org/10.1111/1365-2664.12968