

LETTER

Social living mitigates the costs of a chronic illness in a cooperative carnivore

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Abstract

Infection risk is assumed to increase with social group size, and thus be a cost of group living. We assess infection risk and costs with respect to group size using data from an epidemic of sarcoptic mange (*Sarcoptes scabiei*) among grey wolves (*Canis lupus*). We demonstrate that group size does not predict infection risk and that individual costs of infection, in terms of reduced survival, can be entirely offset by having sufficient numbers of pack-mates. Infected individuals experience increased mortality hazards with increasing proportions of infected pack-mates, but healthy individuals remain unaffected. The social support of group hunting and territory defence are two possible mechanisms mediating infection costs. This is likely a common phenomenon among other social species and chronic infections, but difficult to detect in systems where infection status cannot be measured continuously over time.

Keywords

Grey wolf, infection costs, infection risk, parasite, sarcoptic mange, sociality, social immunity.

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INTRODUCTION

Sociality is seen as a risk factor for directly transmitted infections (Freeland 1976; Altizer *et al.* 2003; Møller *et al.* 1993). This increased risk of infection is thought to translate to a cost of group living: high within-group contact rates are assumed to lead to more transmission, which are assumed to result in more disease-induced morbidity and mortality.

If group living is associated with an increase in risk of infection, social species should be under selection for behavioural or immunological adaptations that mitigate this increased risk (Freeland 1976; Loehle 1995; Altizer *et al.* 2003). Adaptations that serve to reduce disease transmission may occur at the individual level, such as increased immune investment (Altizer *et al.* 2003; Nunn *et al.* 2003), or may be expressed at the group-level, including territoriality (Loehle 1995), social substructuring (Stroeymeyt *et al.* 2014) or other specific anti-parasite behaviours that benefit the group, termed ‘social immunity’ (Cremer *et al.* 2007; Cotter & Kilner 2010). Social species may also mitigate the consequences of increased infection risk by reducing infection costs. In some cases, this may take the form of anti-parasite behaviours that reduce pathogen load such as allogrooming or the application of antimicrobials by group-mates, as seen in leaf-cutting ants (Hughes *et al.* 2002). But, we postulate that sociality comes with additional benefits such as cooperative care, foraging and territory defence that may also offset the individual costs associated with an infection. This is expected to be particularly true for chronic or moderately pathogenic infections where the costs

of infection are realized over an extended period of time, and for heterogeneous infections that leave some proportion of group-mates healthy and functional.

Mitigating the individual costs of infection may be an underappreciated benefit of group living. Renwick *et al.* (2007) suggested that the individual impacts of a chronic infection with bovine tuberculosis might be less pronounced for social lions than solitary leopards in Kruger National Park. Several intriguing examples come from studies of human infections that have examined the impacts of critical care on survival outcomes. Large-scale human epidemics can be so devastating, in part because large numbers of diseased individuals strain public health infrastructure and can reduce individuals’ access to sufficient care (Sinuff *et al.* 2004; Rubinson & O’Toole 2005). By contrast, the level of care per infected individual may be comparatively high during non-epidemic conditions. ‘Care’ need not simply refer to medical care; it may also include access to nutritional resources, psychological benefits of having supportive conspecifics, and protection from predators or others during territorial conflicts, all of which may have an impact on individual survival outcomes.

Here, we explore the dynamics of a chronic infection for 7 years after its initial invasion into a susceptible population. We ask, does social group size covary positively with infection risk for groups and individuals, as generally predicted by theory, and can group size offset the individual survival costs of an infection? Conversely, do infected individuals constitute a burden on their group-mates in terms of survival? We address

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these questions using data from an outbreak of sarcoptic mange, caused by the mite, *Sarcoptes scabiei*, in a well-studied population of grey wolves (*Canis lupus*) in Yellowstone National Park, WY, USA. Given the extreme territoriality of wolves, we predict that infection risk of packs, and by extension individuals, is unlikely to exhibit a strong positive association with group size in our population. Furthermore, we predict that since wolves are cooperative in their foraging and territory defence, that the benefits of group living should offset the costs of individual infections, particularly for chronic or moderately pathogenic organisms such as *S. scabiei*. Specifically, we predict that infected individuals should disproportionately benefit from increases in group size. Although we would ideally evaluate potential mechanisms driving any effect of group size on infection costs, we currently lack sufficient data on food acquisition and territory defence, within the context of mange infections, to do so.

S. scabiei is primarily a directly transmitted infection; upon colonizing a new host, it burrows into the outer layers of its host's skin (epidermis) where it feeds and reproduces, triggering an allergic, inflammatory reaction that causes severe irritation in the host (Pence & Ueckermann 2002). This in turn causes the host to scratch and bite, resulting in thickening of the skin, hair loss and increased susceptibility to secondary skin infections. Infections are chronic, in some cases lasting many months to years, although recovery and short-term acquired immunity have been documented (Arlan *et al.* 1996; Pence & Ueckermann 2002; Jimenez *et al.* 2010). For unknown reasons, susceptibility and infection severity are highly variable among individuals, even within the same social group (Almberg *et al.* 2012). Mange is somewhat unique in that we are able to visually track an individual's infection status by monitoring the appearance and size of mange-induced hairless lesions. This has allowed us to assess the infection status of all radio-collared wolves within the population and to estimate the prevalence of mange within packs on a monthly basis since the mite invaded Yellowstone's wolf population in 2007.

To the best of our knowledge, this is the only study to date that demonstrates the benefits of social group living in mitigating the individual costs of infection in a large, wild mammal. Much of the discussion on optimal group sizes of social carnivores has focused on hunting and food consumption (Packer *et al.* 1990; Vucetich *et al.* 2004; MacNulty *et al.* 2012), territoriality (Mosser & Packer 2009), and reproductive success (Stahler *et al.* 2013). Here, we show the benefits of larger groups for mitigating some of the impacts of chronic diseases; we then quantify the relative costs of infected individuals on the survival of their group-mates.

MATERIALS AND METHODS

Study area

Yellowstone National Park encompasses 8991 km² of protected land in northwestern Wyoming and adjacent parts of Montana and Idaho in the western United States (448330 N, 1108300 W). Yellowstone National Park is surrounded by the Greater Yellowstone Ecosystem, a 60 000 km² area that includes Yellowstone and Grand Teton National Parks,

national forests, wildlife refuges and a mosaic of state and private lands. Yellowstone is mountainous (elevation range: 1500–3500 m), and contains varied land cover, including riparian vegetation, shrubland, grassland, alpine meadows and mixed coniferous forests. The 1000 km² northern region of the park, referred to as the 'Northern Range' is characterized by lower elevations, prime wintering habitat for ungulates, and traditionally higher densities of wolves than the rest of the park.

Population monitoring and disease status

Since grey wolf reintroduction in Yellowstone National Park in 1995–1996 (Smith & Bangs 2009), the National Park Service has captured and radio-collared an annual average of 25 wolves (range: 14–39) spanning all known packs in the park (mean packs sampled per year = 8; range = 6–12). Collaring efforts, which take place between November and March, generally target breeders and young of the year, with an emphasis on maintaining contact with each pack. At the time of collaring, researchers record sex, coat colour (black or grey), weight and body condition, estimate age based on tooth wear (Gipson *et al.* 2000), collect blood samples for genetic and serological analyses and examine the body for ectoparasites, including the clinical signs of infection with *S. scabiei*. The project team subsequently radio-tracks individuals on a daily to monthly basis with the goal of obtaining visual observations of entire packs. During each aerial or ground sighting, researchers record location, pack size, membership, behaviour and, since it was first recorded in January 2007, the infection status and severity of mange for each individual within each pack (Almberg *et al.* 2012). An individual was recorded as being positive for infection with *S. scabiei* based on the presence of visible, hairless lesions and scratching behaviour. The date of the first observation of a positive individual within a pack became known as the date of first infection for that pack. The severity of infection was categorically assessed based on the percentage of an individual's body that was affected by hairless lesions: 1–5%, 6–50% and > 50% were scored as class 1, 2 and 3 mange, respectively, following Pence *et al.* (1983). By categorizing infections, performing annual staff trainings and attempting repeated observations of individual wolves within a month, we aimed to minimize inter-observer variability in classifying mange status. We have attempted to record the infection status of all radio-collared individuals on a monthly basis. Missing data and data interpolation are described below, where relevant.

Risk of infection among packs and individuals

We evaluated the effect of group size on a pack's risk of infection using a Cox proportional hazards model [coxme package (Therneau 2012) in Program R (R Core Team 2012)] specified with a continuous-time study-based baseline hazard (Cox 1972; Fieberg & DelGiudice 2009). The risk set was defined as all uninfected packs within the park; once a pack became infected, they were permanently removed from the risk set. The full data set included data from January 1, 2007 to April 1, 2014, and 19 pack infection events among 27 at-risk packs.

We evaluated the effect of group size on an individual's risk of infection using a Cox proportional hazards model with a random pack effect specified with a continuous-time study-based baseline hazard. In addition to group size, we evaluated the effect of several other group and individual-level covariates on an individual's risk of infection. These pack-level covariates included the number of infected pack-mates and the prevalence of mange within the pack (these two were never included in the same model), which capture density and frequency-dependent transmission respectively (Begon *et al.* 2002) (Figure S1, for plots of pack size, number of infected wolves, and mange prevalence). Individual covariates included age, sex, coat colour (black/grey), whether the individual recovered from previous mange infection, and the severity of an individual's last infection or any previous infection (class 0, 1, or 2/3). Coat colour was considered as a risk factor for infection because the dominant allele that codes for black coat colour is the result of a 3-base pair deletion at the K locus (CBD103), a gene involved in immune function (Candille *et al.* 2007). Black wolves that carry this allele are thought to experience up-regulated production of this beta-defensin, which in turn may reduce susceptibility to some infections.

In the analysis of an individual's risk of infection, we defined the risk set as all uninfected radio-collared individuals within infected packs after the first detected infection within the pack (individuals within uninfected packs were not considered to be within the risk set, as we assumed that these packs had never been exposed). Individuals that recovered from infection and remained negative for ≥ 90 days returned to the risk set; the purpose of this definition of recovery was to eliminate cases that were either misclassified or those that were most likely attributable to recrudescence rather than actual clearance and re-infection. An infection event occurred when an individual moved from an uninfected status to class 1 (or 2 if they were not first detected at class 1). The full data set included data from February 1, 2007 to April 1, 2014, and 78 infection or re-infection events among 81 at-risk individuals (26 individuals never became infected and 18 individuals were infected more than once).

We were missing data on pack size and number of infected pack-mates for 180/1078 (17%) individual monthly records used in our Cox proportional hazard analysis. We performed some basic data interpolation whereby we used the pack size and number of infected pack-mates associated with the most recent observation of the pack within the previous 2 months. This approach reduced missing data to 86/1078 (8%) individual records, and as this did not substantially change parameter estimates or the best-supported models, we report on the original data set throughout the remainder of the manuscript.

Survival

We evaluated cost of mange in terms of survival by conducting a survival analysis using a generalized additive model specified with a complementary log-log link function. This approach was preferable to a Cox Proportional Hazards model in this case because it allowed us to explicitly examine the baseline hazard of mortality over time as well as evaluate time-related covariates such as temperature. Survival status was evaluated

on a monthly time-step. In this analysis the covariates of interest included an individual's mange status [infected (class 1–3) vs. uninfected (Table S1)], the number of pack-mates, and their interaction. We also assessed the burden of mange within the pack by analysing the effect of the number of infected pack-mates, as well as its interaction with mange status, on individual survival. As the number of infected pack-mates was always a subset of the total number of pack-mates, this allowed us to interpret the effect of prevalence on mortality hazards.

As in the previous analyses, we also evaluated individual and environmental covariates thought to influence survival probabilities including age, sex, coat colour (black/grey), a running 3 month average temperature and its interaction with mange status, and the ratio of Northern Range elk to Northern Range wolf counts as an index of resource abundance throughout the park (Houston 1982). We considered the impacts of temperature on survival because it is thought that infected individuals with hairless lesions face the greatest survival costs during cold temperatures.

Of 206 radio-collared individuals followed from January 1, 2005 to April 1, 2014, we were missing data on mange status and/or pack size for 32/101 (32%) mortalities and 1133/4410 (26%) monthly observation records. To evaluate the potential bias associated with missingness in our data, we performed some basic data interpolation. If an individual was not seen in a given month, we used the most recent mange sighting within the previous month or the most recent pack size and infected pack-mate count within the previous 2 months. This process reduced missing data in our data set to 18/101 (18%) mortalities and 522/4410 (12%) monthly observation records. We ran the analysis both ways, and as it did not substantially change parameter estimates or top models, we report on the original data set throughout the rest of the manuscript.

Model selection and parameter coefficients

For both analyses, we evaluated an *a priori* list of models which included various permutations of individual, group, and environmental covariates and evaluated model fit using Akaike's Information Criterion (AIC) (Burnham & Anderson 2002) (Tables S2 and S3). Correlated covariates of mange burden within the pack (e.g. 'number of infected pack-mates' and 'prevalence') and of exposure history ('recovered', 'previous mange', and 'max previous mange') were evaluated against one another and the best fitting covariate was used in subsequent models (Table S2). Unless otherwise noted, we report parameter coefficients from the best-supported models (< 2 AIC units from the top model).

RESULTS

Epidemic dynamics

Mange invaded Yellowstone's wolves in January 2007 and has remained within the population ever since (Fig. 1). The prevalence of mange has settled into a seasonal cycle with peaks during the winter months (mean date of peak infection at the population level is November 8, range September 3 – January 1; mean date of peak infection within infected packs is

December 8, range September 2 – February 2) and troughs during the summer months (mean date of lowest infection at the population level is May 30, range April 2-August 3; mean date of lowest infection within infected packs is April 10, range February 2-June 3) (Fig. 1a and c respectively). The prevalence of more severe infections ($\geq 6\%$ of body covered in hairless lesions) exhibited similar dynamics and on average, comprised 30% of all infections.

Risk of infection

Pack size did not significantly covary with a pack's risk of becoming infected ($\exp(\beta_{\text{packsize}}) = 1.02$, 95% CI: 0.93, 1.11, $P = 0.67$). Of 15 models (Table S2) that examined the risk of individual infection, we found very little model support for an effect of pack size (the sum of model weights for all models in which it appeared = 0.1), nor was pack size a significant predictor of infection risk in the best-ranked model in which it appeared [$\exp(\beta_{\text{packsize}}) = 1.00$, 95% CI: 0.92, 1.09, $P = 0.99$]. The model with the best support included only prevalence as a predictor of infection risk. An individual's risk of infection increased by 61% for every 10% point increase in prevalence within the pack [$\exp(\beta_{\text{prev10\%}}) = 1.61$, 95% CI: 1.43, 1.80, $P < 0.001$]. We found no model support for, or significant effects of individual covariates including age [$\exp(\beta_{\text{age}}) = 0.94$, 95% CI: 0.82, 1.06, $P = 0.32$], sex [$\exp(\beta_{\text{sex(male)}}) = 1.00$, 95% CI: 0.58, 1.73, $P = 0.98$], or coat colour [$\exp(\beta_{\text{color(grey)}}) = 1.58$, 95% CI: 0.88, 2.81, $P = 0.13$] on the risk of infection. Having recovered from a previous infection was not significantly associated with a reduced risk of re-infection [$\exp(\beta_{\text{recovered}}) = 0.77$, 95% CI: 0.39, 1.53, $P = 0.46$].

Survival

A solitary individual with a mange infection experienced five times the mortality hazard of a solitary healthy individual ($\beta_{\text{mange}} = 1.73$, 95% CI: 0.81, 2.65, $P < 0.001$), however, the mange effect declined with increasing pack sizes ($\beta_{\text{mange*packmates}} = -0.38$, 95% CI: -0.67 , -0.09 , $P = 0.01$) (Fig. 2a and 3). An uninfected individual's hazard of mortality also declined with increasing pack size, although to a lesser extent ($\beta_{\text{packmates}} = -0.15$, 95% CI: -0.21 , -0.09 , $P < 0.001$) (Fig. 3a). Individuals infected with mange experienced a marginally significant increase in their hazard of mortality with

increasing proportions of infected pack-mates ($\beta_{\text{mange*infected packmates}} = 0.55$, 95% CI: -0.04 , 1.14, $P = 0.07$), whereas healthy individuals appeared unaffected ($\beta_{\text{infected packmates}} = -0.21$, 95% CI: -0.68 , 0.26, $P = 0.39$) (Fig. 3). Mortality hazards declined with increases in the elk:wolf ratio ($\beta_{\text{elk/wolf}} = -0.02$, 95% CI: -0.04 , $-5e-4$, $P < 0.01$) and increased with the 3-month average temperature ($\beta_{\text{temp}} = 0.02$, 95% CI: $5e-4$, 0.04, $P = 0.02$). The interaction between temperature and mange status was neither among the top models nor significant ($\beta_{\text{temp*mange}} = 0.01$, 95% CI: -0.03 , 0.05, $P = 0.64$). Wolves with a grey coat colour, as opposed to black, experienced a higher hazard of mortality [$\beta_{\text{color(grey)}} = 0.55$, 95% CI: 0.06, 1.04, $P = 0.03$]. Age ($\beta_{\text{age}} = 0.01$, 95% CI: -0.10 , 0.11, $P = 0.91$) and sex [$\beta_{\text{sex(male)}} = -0.09$, 95% CI: -0.58 , 0.39, $P = 0.71$] were neither among the top models nor significant predictors of mortality risk.

To illustrate the relative importance of the variables described above, we plotted the standardized parameter estimates from our top model in Fig. 2b. Continuous predictors were standardized by subtracting the mean and dividing by 2 SD (Gelman & Hill 2006). The coefficients of all standardized variables are interpreted as the effect sizes associated with a shift for that variable from 1 SD below the mean to 1 SD above the mean while all other variables are at their mean value. Thus, under average conditions (i.e. within an average-sized pack, and with an average number of infected pack-mates, average temperature, and average elk:wolf ratio) mange has no significant effect on individual mortality (Fig. 2b). However, for below-average pack sizes (i.e. 1 SD below the mean) or above-average numbers of infected pack-mates (i.e. 1 SD above the mean), mange-infected individuals experience a significantly increased hazard of mortality (Fig. 2b).

DISCUSSION

Infection risk of directly transmitted pathogens is generally assumed to increase with social group size, and thus translate to a cost of group living. Using a long-term data set on the dynamics of sarcoptic mange in Yellowstone's wolves, we did not find strong evidence for a positive association between group size and infection risk, but we did find that increasing pack size could offset individual costs of infection with sarcoptic mange. We also demonstrate that for infected individuals,

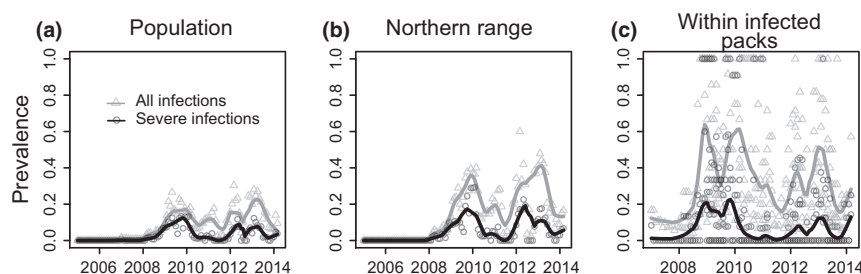


Figure 1 Prevalence of all mange infections (class 1–3; grey) and severe mange infections (class 2–3; black) within (a) Yellowstone National Park's (YNP) wolf population (b) the Northern Range of YNP, the region where we see the majority of infections, and (c) within actively infected packs. Lines represent lowest fits to the data.

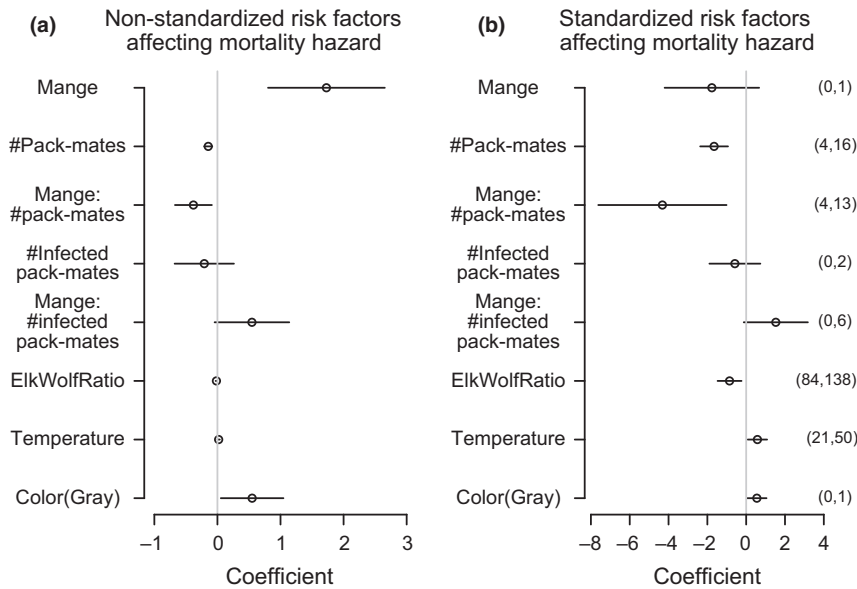


Figure 2 (a) Non-standardized and (b) standardized effect sizes of factors from the top-ranked model associated with an individual's hazard of mortality. The coefficients of all standardized variables are interpreted as the effect sizes associated with a shift for that variable from 1 SD below the mean to 1 SD above the mean (the boundaries listed in parentheses to the right) while all other variables are at their mean value. Negative and positive coefficients suggest reductions and increases, respectively, in mortality hazards. Error bars represent 95% confidence intervals. Mange is the effect of an infection (class 1/2/3) relative to no infection; Mange:#Pack-mates and Mange:#Infected Pack-mates refer to the interaction between the two variables; Temperature refers to a 3 month temperature average.

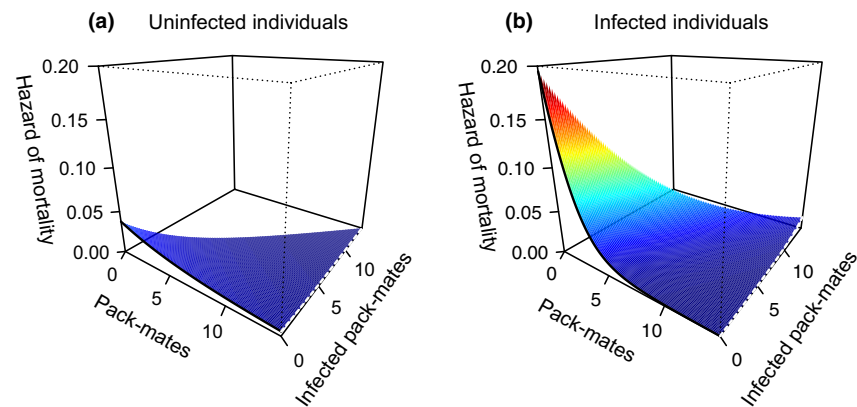


Figure 3 Predicted monthly mortality hazards for (a) uninfected and (b) mange-infected individuals given the number of total and infected pack-mates. The colour ramp reflects the relative hazard of mortality and is comparable across plots. The solid black lines, when compared across plots, highlight the interaction between mange status and pack size, whereas the dashed white lines highlight the interaction between infection status and number of infected pack-mates.

increasing proportions of infected pack-mates were associated with an increasing hazard of mortality, suggesting that infected pack-mates do not offer the same benefits to an infected individual as their healthy counterparts. To the best of our knowledge, our study provides the first evidence in a wild mammal of the benefits of group living for mitigating the impacts of a chronic disease and is one of the first non-human studies to quantify the costs of infected individuals to their group-mates. We suspect that reducing infection costs is a widespread benefit of group living, particularly among social carnivores, but that it remains underappreciated because of the difficulties in regularly measuring the infection status of wild animals.

Many studies have found evidence that risk of parasitism increases with social group size. Caillaud *et al.* (2006) found that social gorillas experienced higher risks of infection with Ebola virus than solitary males. Ezenwa (2004) found that group size was positively correlated with parasite prevalence in African bovids. Several meta-analyses (Cote & Poulin 1995; Rifkin *et al.* 2012; Patterson & Ruckstuhl 2013) have found wide support for a positive association between group size and parasite risk, although estimated effect sizes are often weak and accompanied by high levels of uncertainty (Rifkin *et al.* 2012). Although we failed to find a statistically significant relationship between group size and risk of mange in

Yellowstone's wolves, the uncertainty in our estimates suggest that infection risk for a pack may increase as much as 11%, or decrease by 7%, for each additional pack-mate based on the 95% confidence intervals. Thus, we cannot rule out the possibility that risk of infection with mange increases by a small amount with increasing group size, but there is no current support for an effect.

Despite the long-held notion that infection risk and social group size should positively co-vary, there is substantial theory and growing evidence suggesting this might not always be the case (Altizer *et al.* 2003; Cremer *et al.* 2007; Stroeymeyt *et al.* 2014). Social species, in particular, may be under strong selection for individual or collective anti-parasite defences that reduce infection risk. Social insects exhibit a range of behaviours, including territory defence, prophylactic use of antimicrobials within the nest, allogrooming, and complex social sub-structuring that generate 'social immunity' and reduce transmission risk despite high local densities of host (Cremer *et al.* 2007). Although direct analogies to our system may be limited, territoriality among wolves may be one such example of a social behaviour that slows or limits disease transmission across groups, muddling the measurable relationship between group size and infection risk. Within-group contact rates are generally assumed to be high, uniform, and independent of group size within wolf packs, and in accordance with this, we found that group size was a poor predictor of an individual's risk within an infected pack. Instead, individual risk within an infected pack was best explained by pack prevalence, a result consistent with the most basic predictions for frequency-dependent transmission where the probability of infection is proportional to the contact rate*prevalence of the infection within the group (Begon *et al.* 2002). Among larger packs, we sometimes observe additional within-group sub-structuring, which could theoretically slow pathogen transmission across large groups (Griffin & Nunn 2012), but for chronic infections, it's not clear whether this would ultimately reduce long-term probability of infection (Cross *et al.* 2005). Previous work in our system suggests that regional or pack density, driven by resource availability (Smith & Bangs 2009; Smith *et al.* 2011), may be a better predictor than group size of between-group contact rates and hence individual risk (Almberg *et al.* 2012).

Most of the support for the social immunity hypothesis comes in the form of evidence for socially mediated mechanisms that reduce transmission risk. Our study supports the smaller but growing body of evidence that group living actually mitigates infection costs (Hughes *et al.* 2002; Cremer *et al.* 2007; Cotter & Kilner 2010). Although an infection with mange can be quite costly in terms of reduced individual survival, this cost can be removed with increases in group size. An infected individual faces a hazard of mortality equivalent to that of an uninfected individual when it is surrounded by 5 additional pack-mates; but that same individual may face nearly four times the monthly mortality hazard of an uninfected individual when it only has one additional pack-mate (Fig. 3a). Although we currently lack the data to explicitly test for the mechanisms responsible this effect of pack size, we suspect that it is largely driven by food acquisition and territory defence. Wolves are cooperative hunters, and research

suggests that hunting success is maximized in groups of ≥ 4 wolves (MacNulty *et al.* 2012). Severely infected individuals may either be consciously abstaining from energy-intensive hunts to conserve energy (especially in winter when their costs of infection are expected to be highest), or may in some cases be in too poor of shape to effectively contribute to a hunt. Group size is also known to be important in territorial skirmishes (Mosser & Packer 2009; Cassidy 2013), and severely infected individuals may not be able to effectively contribute during such encounters.

We found no significant evidence that healthy individuals bore survival costs associated with supporting their infected group-mates, but we did find that infected individuals experienced increasing hazards of mortality associated with increasing proportions of infected pack-mates (Fig. 3b). Presumably, this increased mortality hazard among infected individuals is the result of paying a cost, in terms of reduced hunting success (MacNulty *et al.* 2012) or territory defence (Mosser & Packer 2009; Cassidy 2013), associated with having proportionally fewer fully functional pack-mates. Infected individuals may be particularly sensitive to reductions in resource acquisition or territory defence if their infections increase their energy demands (Lochmiller & Deerenberg 2000; Bonneaud *et al.* 2012), make them poorer within-group competitors for limited resources, or make them more vulnerable in the face of territorial conflicts. Despite these nuances, both infected and uninfected individuals still accrue a net survival benefit from living in a social group, even if all of their group-mates are infected. It is worth noting that our analyses do not exclude the possibility that healthy individuals bear a reproductive cost associated with supporting infected group-mates, but this has yet to be tested.

The monitoring of sarcoptic mange dynamics within Yellowstone's wolves is ongoing. Given what we have observed to date and that we were unable to detect any strong signal of acquired immunity, we predict that mange will remain endemic within our population. The extent to which mange affects overall wolf population dynamics will depend on the prevalence and ensuing costs of infection. Our work suggests that if natural and/or anthropogenic forces conspire to reduce pack size, or if the prevalence of mange within packs increases, that the relative costs of infection may increase.

Sarcoptic mange in Yellowstone's wolves is a relatively unique wildlife-disease study system in which we have been able to track individuals' infection status continuously throughout the population and across time. But mange may serve as a very useful analogy to a number of other chronic helminth, bacterial, viral and fungal infections or to physical injuries that are quite common but that are much more difficult to track. Like mange, the susceptibility to or intensity of many infections or injuries are heterogeneous within groups, often leaving a portion of the group relatively unaffected. Similarly, the physiological costs of many infections or injuries may be borne out over extended periods of time, during which the benefits of having socially cooperative group-mates may help offset individual costs. One of the challenges going forward, both within our system but also within the field at large, will be to weigh the relative importance of group size in mitigating infection costs against the more traditionally

acknowledged benefits of group size to resource acquisition and intake (Vucetich *et al.* 2004; MacNulty *et al.* 2012), territory defence (Mosser & Packer 2009; Cassidy 2013), and reproductive success (Stahler *et al.* 2013).

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AUTHORSHIP

ESA coordinated mange-related data collection, led analyses, and wrote the first draft of the manuscript. PCC, PJH, and APD contributed to analyses and write-up. DWS, MCM and DRS contributed additional data and intellectual input. All authors contributed substantially to manuscript revisions.

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