Social network structure and parasite infection patterns in a territorial reptile, the tuatara (*Sphenodon punctatus*)

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ABSTRACT

We investigated whether the parasite load of an individual could be predicted by its position in a social network. Specifically, we derived social networks in a solitary, territorial reptile (the tuatara, *Sphenodon punctatus*), with links based on the sharing of space, not necessarily synchronously, in overlapping territories. Tuatara are infected by ectoparasitic ticks (*Amblyomma sphenodonti*), mites (*Neotrombicula* spp.) and a blood parasite (*Hepatozoon tuatarae*) which is transmitted by the tick. We recorded the location of individual tuatara in two study plots twice daily during the mating season (March) in 2 years (2006 and 2007) on Stephens Island, New Zealand. We constructed weighted, directed networks to represent pathways for parasite transmission, where nodes represented individual tuatara and edges connecting the nodes represented the extent of territory overlap among each pair of individuals. We considered a network-based hypothesis which predicted that the in-strength of individuals (the sum of edge weights directed towards a node) in the derived network would be positively related to their parasite load. Alternatively, if the derived social network did not reflect actual parasite transmission, we predicted other factors such as host sex, size or territory size may better explain variation in parasite infection patterns. We found clear positive relationships between the in-strength of tuatara and their tick loads, and infection patterns with tick-borne blood parasites. In particular, the extent that individuals were connected to males in the network consistently predicted tick loads of tuatara. However, mite loads of tuatara were significantly related to host sex, body size and territory size, and showed little association with network measures. The results suggest that the pathway of transmission of parasites through a population will depend on the transmission mechanism of the parasite, but that social networks provide a powerful predictive tool for some parasites.

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1. Introduction

Understanding the transmission of parasites in wildlife populations is a fundamental issue in ecology, epidemiology and wildlife conservation. Traditional epidemiological models assumed transmission through a homogenous host population with random infection risk (Anderson and May, 1979; May and Anderson, 1979). Later discussion acknowledged the importance of host behaviour and contact patterns among hosts in the transmission of parasites (Gudelj and White, 2004; Bansal et al., 2007). Both host behaviour and the degree of contact among individuals in a population will be influenced by the social organisation of the population. Thus social organisation is likely to play a central role in the transmission of parasites.

However, the importance of this role depends both on the form of social organisation and on the mode of parasite transmission. Animals arrange their social structure in a diversity of ways, including solitary-territorial systems, pair-living and gregarious groups (Alexander, 1974; Whitehead and Dufault, 1999). The impact of social organisation on parasite transmission has been explored most in hosts that aggregate in groups (Moller et al., 1993; Altizer et al., 2003). For parasites with direct transmission, group-living hosts should have high rates of intra-group transmission and lower rates of inter-group transmission (Loehle, 1995; Altizer et al., 2003), leading to an aggregated distribution of infection among groups (Arnold and Lichtenstein, 1993; Porteous and Pankhurst, 1998; Godfrey et al., 2006). Parasites that are transmitted by a vector, an intermediate host or that can persist in an environmental reservoir become less dependent on social contacts among group-living hosts as their off-host mobility increases (Poulin, 1999; Godfrey et al., 2006). We know less about the dynamics of parasite transmission in host species where populations do not form stable social groups. One challenge has been to quantify the

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complex and dynamic nature of different forms of social contacts in these species. Network models quantify pair-wise associations among host individuals within a population as a network of nodes (individuals) and edges (associations). These models provide a framework to explore how any form of social structure within a population can generate pathways for parasite transmission.

Network models have been used to explore the transmission of sexually transmitted diseases (particularly HIV/AIDs) in humans (Klovdahl, 1985; Friedman et al., 1997; Potterat et al., 2002), foot and mouth disease transmission in livestock among farms (Shirley and Rushton, 2005; Kiss et al., 2006), and various parasitic infections in wildlife populations (Cross et al., 2004; Porphyre et al., 2008; Godfrey et al., 2009). The extent that an individual is connected to other individuals in a network can influence both its risk of becoming infected and its propensity to spread the infection through a population (Bell et al., 1999; Christley et al., 2005). Thus, the composition of connections in the network affects how diseases spread through the population (Keeling, 2005; Shirley and Rushton, 2005). Although these concepts have been modelled extensively for theoretical networks (Keeling and Eames, 2005; Eames et al., 2009; Moslonka-Lefebvre et al., 2009) and for networks empirically derived from real populations (Cross et al., 2004; Kiss et al., 2006), relatively few studies have compared model predictions with empirical patterns of infection in natural wildlife populations and most of those have involved species with some stable group structure (Craft et al., 2009; Godfrey et al., 2009; Drewe, 2010). To understand the role of social networks in parasite transmission, it is important to empirically test the model predictions in a variety of forms of social organisation.

In solitary-territorial social systems, an individual typically defends an exclusive area that contains essential resources such as food and refuges (Burt, 1943; Stamps, 1994; Maher and Lott, 2000). Direct contact between individuals is usually limited to aggressive encounters to enforce territory boundaries and mating encounters (Stamps, 1994; Stamps and Krishnan, 1998), reducing opportunities for the direct transmission of parasites. However, territory boundaries can overlap to varying extents because even dominant territory holders cannot defend all of their boundaries at the same time (Mitani and Rodman, 1979). Asynchronous sharing of territory space may allow the indirect transmission of parasites by infectious, free living stages or by sedentary vector species that persist in the environment where the territory boundaries intersect. Thus, the spatial structure and overlap of territories may enable pathways for the indirect transmission of parasites. In this study we explored whether network structures in a territorial and solitary reptile can predict its patterns of individual parasite load.

We developed social network models for a territorial reptile, the tuatara (*Sphenodon punctatus*) on Stephens Island, New Zealand. Tuatara use burrows that have been constructed by nesting sea birds as refuges, and they bask outside burrow entrances where they can monitor their territory area for potential intruders (Newman, 1987; Walls, 1983). Individual male tuatara maintain and defend stable territories containing multiple burrows but allow some spatial overlap with the territories of neighbours of either sex (Gillingham et al., 1995; Moore et al., 2009a). Female tuatara have smaller territories, usually centred around a single burrow, which are not significantly clustered or dispersed from other females (Moore et al., 2009a). In the mating season (March), both social interactions (Moore et al., 2009a) and male testosterone levels (Cree et al., 1992) peak. Tuatara are seasonally monogamous within a year but show polygyny and polyandry across years (Moore et al., 2009b). Male and female partners have extensive territory overlap, while large, dominant males overlap with more female territories than do small males, and large males more effectively exclude other males from their territories through aggressive

encounters (Moore et al., 2009a). This mosaic of varying levels of contact and space sharing provides the background for our exploration of parasite transmission in this species.

Tuatara are host to ixodid ticks (*Amblyomma sphenodonti*), trombiculid mites (*Neotrombicula* spp.) and an apicomplexan blood parasite (*Hepatozoon tuatarae*). The tuatara tick is a three-host tick, specific to tuatara (Dumbleton, 1943; Klompen et al., 2002; Heath, 2006). It develops from egg to larva, nymph and then adult, and feeds on tuatara blood during each stage (Heath, 2006). After engorgement, each stage detaches from the host into the off-host environment, where they moult into the next infective stage or if a female, to lay eggs (Heath, 2006). Newly moulted or hatched ticks then locate another host, or relocate the same host. In-vitro experiments have shown that tuatara ticks prefer dark, moist environments and coarse substrates; reflecting the conditions within host burrows and leaf litter on the forest floor (Godfrey et al., 2011). Although the distance moved by tuatara ticks within the environment is unknown, other related reptile ticks (*Amblyomma limbatum* and *Bothriocroton hydrosauri*) adopt a "sit and wait" host seeking strategy, remaining in host refuges and moving less than 50 cm to locate a new host (Petney et al., 1983). Thus, the transmission of ticks among tuatara in the population predominantly relies on individual tuatara encountering ticks in the environment. Ticks are found attached to tuatara at all times of the year but infestation rates are highest in late autumn (March–May) (Godfrey et al., 2008).

Mites (*Neotrombicula* spp.) are not host-specific to tuatara and infest skinks and geckos on Stephens Island (Goff et al., 1987). They have a similar lifecycle to ticks but are only parasitic in the larval stage. Free-living nymphs and adults are probably predatory, feeding on small invertebrates in the leaf litter and soil (Wharton, 1952; Sasa, 1961). Transmission to tuatara depends on the location where female mites deposit eggs that hatch into infective larvae. The prevalence and intensity of mite infestations are more seasonal than ticks, with larval mites appearing on host tuatara only in summer and early autumn, with peak infestations in March (Godfrey et al., 2008).

Hepatozoon tuatarae (previously described as *Haemogregarina tuatarae*, Laird 1950) is an apicomplexan parasite that is specific to tuatara (Laird, 1950). Although the lifecycle of *H. tuatarae* is unconfirmed, developmental stages of the blood parasite have been found in the tuatara, suggesting transmission occurs through the ingestion of infected ticks (Herbert et al., 2010). Populations of tuatara on Stephens Island have a low prevalence (<30% infected) and intensity (1–10 infected cells/10^4 red blood cells) of blood parasite infection, but infections can last 18 months (Godfrey et al., in press).

The transmission of each of the three parasites between host individuals relies on them using common space. We asked whether we could predict patterns of parasite infection from the number and weight of connections to individuals in an empirically-derived network that represented possible pathways for parasite transmission. The weighted, directed network was based on the extent of non-synchronous overlap among territories of individual tuatara. Our hypothesis, that territory structure and overlap influenced parasite transmission, predicted positive relationships between the in-strength (sum of edge weights directed towards each individual) of tuatara in the social network and their parasite load. Alternatively, if parasite transmission was less influenced by our derived social network, we expected other factors, such as host sex, body size and territory size, to predict patterns of infestation more strongly. These two alternatives are not entirely exclusive, but from the relative strength of the effects of network properties on parasite infection patterns, we can gain an insight into the role of social structure on transmission for each of the parasites we considered.

Table 1
Summary of behavioural observations of tuatara (*Sphenodon punctatus*) in each study plot and year.

Study plot	Year	Network ID	Area (ha)	N	$N_{\geqslant 5}$	Obs_{total}	Obs_{freq} (±SE)	Obs_{max}
Keepers plot 1 (K1)	2006	K1/06	0.032	29	21	333	15.8 ± 1.9	35
	2007	K1/07	0.048	24	18	296	16.4 ± 1.7	30
Keepers plot 3 (K3)	2006	K3/06	0.029	27	21	379	18.0 ± 1.7	35
	2007	K3/07	0.025	24	17	293	17.2 ± 1.7	31

Year, the year the behavioural observations were collected; network ID, the identification code of the network representing each study plot and year; area (ha), the total area of study plots; N, the total number of individual tuatara that were observed; $N_{\geqslant 5}$, the number of marked individuals that were observed five or more times; Obs_{total}, the total number of observations; Obs_{freq}, the mean observation frequency of individuals; Obs_{max}, the maximum number of observations recorded for any individual. Obs_{total} and Obs_{freq} are based on the individuals incorporated into the networks (those with five or more observations). SE, standard error.

2. Materials and methods

2.1. Field study

We used two study plots (K1 and K3) located 40 m apart in closed canopy forest habitat on Stephens Island (Cook Strait, New Zealand, 40° 40′ S, 174° 0′ E). The area surveyed in each study plot varied between years (Table 1), but they were each within a larger, continuous tuatara population. In this population tuatara achieve a maximum density of 2700 per ha, and they have small, stable territories (males: 30.6 m², females: 13.9 m²) (Moore et al., 2009a). Thus, although the plots were close to each other, we considered individuals in each to be independent of those in the other. Individuals captured in each plot were never captured in the other plot. Our choice of study plots was restricted by permit conditions and accessibility within the forest habitat, and by a requirement to be able to collect behavioural observations from a central access track with minimal disturbance.

March is the mating season of tuatara, when territorial and mating interactions are at a peak. We observed each plot over 23 days in 2006 (28 February–22 March 2006) and over 24 days in 2007 (27 February–22 March 2007). Within the plots, individuals were permanently marked with a sub-dermal passive integrated transponder (PIT) tag (Allflex, NZ), and marked for observation with a unique coloured bead tag inserted into the nuchal crest (Fisher and Muth, 1989), or with a black number written on each flank. In each plot we used a central access track to avoid damage to tuatara burrows. We surveyed each plot twice daily between 1130 and 2000 h, when social activity of tuatara was highest. In each survey, we walked slowly along the track for 60–90 min, until the locations of all visible tuatara were recorded. The accumulated location records were used to define the territory of each individual.

2.2. Network construction

We developed networks for each study plot in each year (2006 and 2007), to model the possible pathways for parasite transmission. The networks were based on the extent of non-synchronous overlap among tuatara territories. Individuals observed less than five times were excluded from the territory estimation and network construction to reduce the bias caused by a low observation frequency (Table 1). First, we estimated the territory area of each individual using the 95% minimum convex polygon (MCP) method in Ranges 6 (Kenward et al., 2003. Ranges 6. Anatrack Ltd., Wareham, UK. For the analysis of tracking and location data), as has previously been applied to tuatara (Moore et al., 2009a). Then we estimated the extent of territory overlap among individuals, using reciprocal indices of territory overlap between each pair of individuals in each study plot and year. For each pair of individuals (*a* and *b*), we calculated territory overlap index 1 as the proportion of observations of individual *a* that occurred within the territory of individual *b*, and territory overlap index 2 as the proportion of locations of individual *b* that occurred within the territory of individual *a* (Fig. 1A). Unlike measures of percent of overlap area, this method does not assume
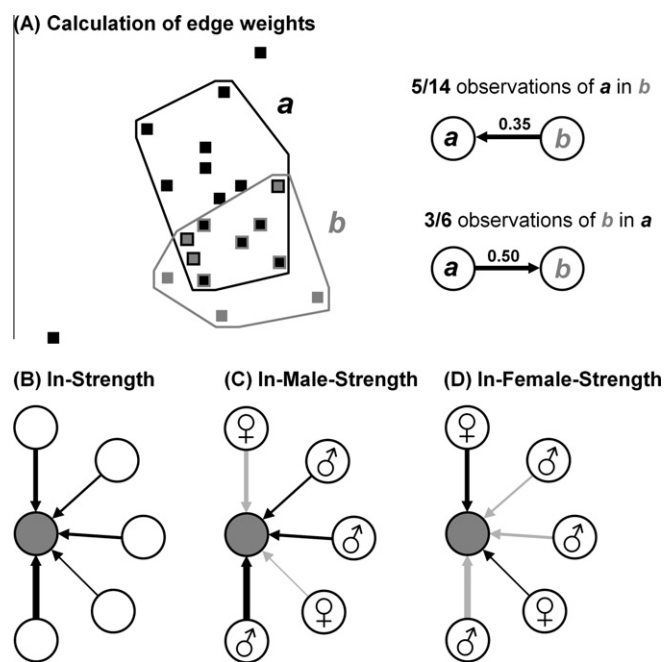

Fig. 1. Diagram demonstrating the calculation of edge weights (A), and node-based measures (B–D) in tuatara (*Sphenodon punctatus*) networks. (A) Polygons represent the 95% minimum convex polygons (MCP) of individuals *a* and *b*; black squares are observations for individual *a* and grey squares are observations for individual *b*. Edge weights (arrows) were calculated as the proportion of observations of individual *a* that occurred within the territory area of individual *b* and vice versa. Arrows indicate the direction of the edge. (B–D) Circles are nodes (individuals) in the network, lines are edges in the network, with arrows indicating the direction of edges and the width of lines indicating edge weights. Symbols indicate the sex of individuals. The grey node is the individual that node-based measures are calculated for. (B) In-strength is the sum of all edge weights (arrow widths) directed towards an individual. (C) In-male-strength is the sum of edge weights directed towards an individual from males (black arrows only) and (D) in-female-strength is the sum of edge weights directed towards an individual from females (black arrows only).

uniform use of the entire territory but assumes that our observations represented the patterns of territory use. We used individual tuatara as the nodes, and the overlap indices as weighted edges, to construct directed, weighted networks for tuatara in each study plot in each year. Our transmission model predicted that the extent to which individual *a* uses *b*'s territory area will influence the risk of *a* picking up parasites from *b*. Thus we used territory overlap index (1) as the edge directed towards individual *a*, and territory overlap index (2) as the edge directed towards individual *b* (Fig. 1A). We constructed separate networks from observations in each year.

2.3. Parasite surveys

We captured a sub-set of individual tuatara in the study plots in February–March 2006 (the first week of behavioural observations), September 2006 and March 2007 (after the behavioural

observations). Individuals were only sampled once within each survey period. We measured the snout to vent length (SVL) and weighed each individual, identified sub-adults (SVL < 170 mm), determined the sex of adults and counted the ticks, distinguishing between the different tick life cycle stages (larvae, nymphs and adults) and mites attached. Mite loads above 100 were estimated by extrapolation from the area of the host that contained 100 mites (Godfrey et al., 2008). Sub-adults were infrequently captured ($n = 2$) and insufficiently observed (<5 observations), so they were not included in the final network analyses.

We produced thin blood smears on microscope slides with blood collected from the caudal vein of hand-captured individuals in the study plots in February and September 2006, and in three previous surveys from 2004–2005. Slides were air dried, stained with a Modified Wright-Giemsa Stain in a Hematek slide-stainer, and examined under $1000\times$ oil immersion at a cell density of approximately 50 cells per microscope field. We scored the individual as uninfected if no blood parasites were detected after viewing 200 fields (about 10^4 red blood cells) in any of the samples collected from that individual over the period of the study. An individual was considered infected if blood parasites were detected in at least one of the surveys.

2.4. Analysis

We examined the factors that influenced parasite infection patterns of tuatara, considering our alternate hypotheses. If parasite transmission depended on network structure, we expected that network measures would predict patterns of parasite infection. Alternatively, if network structure was less important for parasite transmission, we expected that infection patterns would be more strongly explained by other factors such as host sex, body size and territory size. Network parameters derived for individual members of the network are not independent of each other, so they must be analysed using randomisation tests (Croft et al., 2008; James et al., 2009). This makes it difficult to compare, in a single analysis, the relative impact of network structural parameters and host-related parameters on patterns of infestation. Instead we considered each separately and simply determined whether they did or did not have a significant effect. We first tested the effects of network structure on parasite infection patterns in a series of randomisation tests. We then tested the effects of host properties on parasite infection patterns using a linear mixed effects model (lme) in R (R Core Development Team, 2009. R: a language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria).

2.5. Network position and parasite infection patterns

If parasites are transmitted along network edges, then individuals that have a greater number and weight of connections to other individuals in the network should have higher parasite loads. We
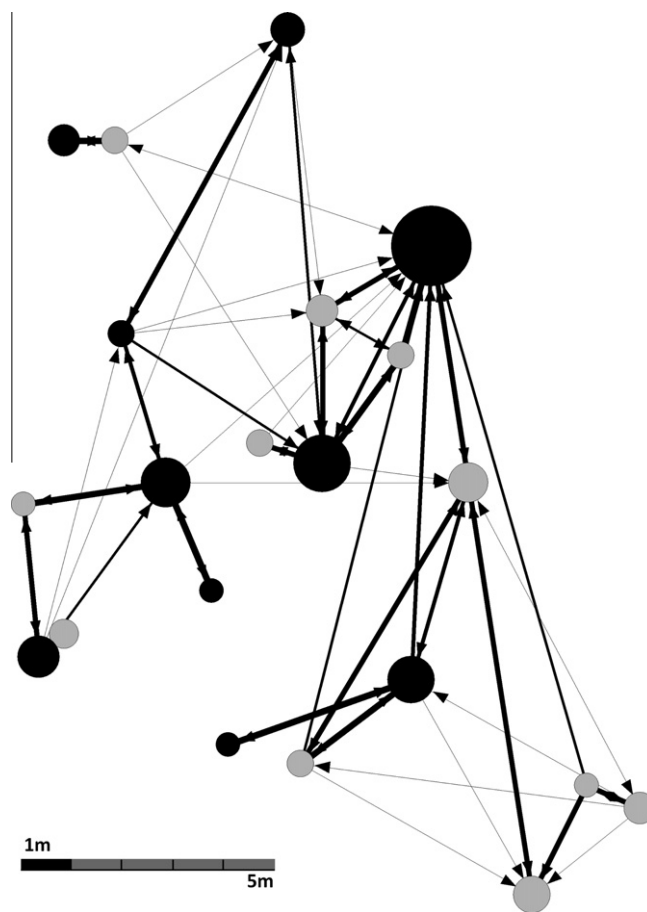

Fig. 2. Network diagram of territory overlap among tuatara (*Sphenodon punctatus*) for study plot K1 in 2006. Circles represent nodes (individuals) and the lines connecting them represent edges. Edge thickness represents the extent of territory overlap between each pair of nodes and arrows indicate the direction of the overlap. Node colour represents the sex of the individual (black = males, grey = females), and node size is scaled by the measured value of in-strength (sum of edge weights directed towards that individual) for the node. The two-dimensional placement of nodes corresponds to the geographical location of individuals within the study site.

calculated three node-based measures to quantify the extent that an individual was connected to other individuals in the network and the role of males and females in transmission. First, we measured the "in-strength" of each individual in the network, which is the sum of all edge weights that are directed towards that individual (Fig. 1B). We considered this a measure of the total risk of infection for an individual and predicted that individuals with higher in-strength would have higher parasite loads.

Second, we explored the role of each sex in parasite transmission. In tuatara populations, males have larger territories and might be more important in the spread of parasites. In that case,

Table 2
Summary of territory and network structure of tuatara (*Sphenodon punctatus*) in each study plot and year.

Network	T_{area} (± SE)	T_N (± SE)	T_{ove} (± SE)	N_{edges}	Density	IS_{mean} (±SE)	IS_{range}
K1/06	18.66 ± 3.84	3.38 ± 0.60	0.35 ± 0.04	71	0.161	1.19 ± 0.29	0.09–5.50
K1/07	28.19 ± 5.82	3.33 ± 0.51	0.34 ± 0.04	60	0.185	1.14 ± 0.25	0.04–3.95
K3/06	18.24 ± 2.91	5.04 ± 0.78	0.36 ± 0.03	106	0.240	1.83 ± 0.36	0–4.83
K3/07	16.83 ± 3.29	4.53 ± 0.72	0.39 ± 0.03	77	0.266	1.76 ± 0.35	0–5.45

T_{area}, the mean territory (95% minimum convex polygons) size (m^2) of tuatara in each study plot and year; T_N, the number of individuals that overlapped with an individual's territory; T_{ove}, the mean proportion of territory overlap (calculated as the proportion of observations of a that occurred in the territory of b between pairs of individuals that overlapped; N_{edges} is the number of edges in each network (edges counted twice if reciprocal and once if directed one-way); density, the density of the network (proportion of edges existing of total possible edges), IS_{mean}, the mean in-strength (sum of weighted edges directed towards an individual) of individuals in each study plot/year; and IS_{range}, the range of in-strength values. SE, standard error.

individuals that are connected to more males in the network should have a higher parasite load. We calculated "in-male-strength" as the total sum of edge weights directed towards an individual that were from a male (Fig. 1C), and "in-female-strength" as the total sum of edge weights directed towards an individual that were from a female (Fig. 1D). We compared relationships between parasite loads and in-male-strength and in-female-strength.

We tested these hypotheses for each of the three parasite groups using a series of randomisation tests. The randomisation tests involved calculating a statistic that quantified the relationship between two variables and determining whether this statistic was more extreme than values derived from randomized versions of the data (see Sections 2.5.1 and 2.5.2). Parasite loads were measured for a sub-set of individuals in each network and survey period (Section 2.3). Thus, only individuals with measured parasite loads were included in the randomisation tests.

2.5.1. Ectoparasites (ticks and mites)

We calculated Spearman rank correlation coefficients (r_{sp}) to quantify relationships between ectoparasite load and measures of in-strength. We calculated separate measures of r_{sp} for each of the in-strength measures for larval tick load, nymphal tick load, total tick load (the sum of all tick life stages present on the host) and total mite load of tuatara at the time the network data were collected, for each study plot in each year (2006 and 2007). We did not include adult ticks in analyses because they were infrequently recorded in our surveys.

Tick attachment rates are low in early March, increasing towards winter when ticks over-winter on the host (Godfrey et al., 2008). Ticks then begin to engorge and detach in early spring (Godfrey et al., 2008). Thus tick loads measured at the onset of spring (September) probably represent the cumulative exposure of hosts to ticks across the mating and subsequent post-mating season. Therefore, for the networks derived in March 2006 we also calculated r_{sp} between measures of in-strength, and ectoparasite load in the following September 2006.

2.5.2. Blood parasites

We used the Mann–Whitney u statistic, a scaled version of the Mann–Whitney U statistic that lies between 0 and 1 (Croft et al., 2008), to quantify the difference in measures of in-strength between individuals infected with *H. tuatarae* and uninfected individuals. Values of u greater than 0.5 indicated infected individuals had a higher mean in-strength than uninfected individuals.

2.6. Randomisation procedure

We resampled the original data for each comparison without replacement in a Monte Carlo randomisation test (Manly, 1997) with 1000 permutations, using a constrained resampling procedure to control for sampling bias (described in Section 2.6.1). We recalculated the statistic (u or r_{sp}) for each randomized version of the data and compared the resultant distribution of values with the observed value of the statistic. We derived P-values from the number of randomized values that were greater than the observed value. For each hypothesis tested, separate randomisation tests were conducted within each study plot and year. The randomisation procedures were conducted in PopTools 2.7 for Excel (Hood, 2006. PopTools, 2.7.5 ed. http://www.cse.csiro.au/poptools/).

2.6.1. Constrained resampling

Since our study plots were sub-groups within a continuous population, individuals at the edge of the study plot were likely to have unrecorded associations with adjacent individuals outside the study plots. Thus, nodal parameters for "edge individuals" might be underestimated and lower than for individuals in the centre of the study plot. We constructed an internal buffer zone around the perimeter of each study plot, with a width equal to half the median territory span of tuatara in that plot. Any individual whose territory overlapped this buffer by more than 10% was considered an "edge individual". In our randomisation tests, we constrained resampling to within either edge or central zones. By constricting resampling of edge individuals with other edge individuals, and of central individuals with other central individuals, we avoided making substitutions between individuals with substantial differences in estimated values of nodal parameters that were the result of the sampling procedure.

2.7. Effects of sex, size and territory area on ectoparasite infestation

If parasite transmission was less dependent on pathways created by the social network, we expected that parasite infection patterns would be more strongly explained by host properties, such as host sex, body size or territory size. Godfrey et al. (in press) reported that smaller male host individuals had higher prevalence and intensity of infection by blood parasites, but detected no other impact of host sex or territory size on infection patterns. In this paper we examined the factors influencing tick and mite loads of tuatara using a linear mixed effects model. We constructed separate models for larval ticks, nymphal ticks, total ticks and for mites. Log ($x + 1$) parasite load was the dependent variable, sex and month (except for mite models; mites were only found on hosts in March) were fixed factors and host size (SVL) was a covariate. We also included territory span (maximum diameter of the 95% MCP territory area (m)) as a linear measure of territory size as a covariate in the models. Individuals were nested within study plots as random effects. Since tuatara were sampled repeatedly, we used a continuous autoregressive covariance matrix to model variation among sampling periods. We began with a maximal model including all main effects, two-way and three-way interactions, and reduced the model by removing non-significant effects until further model reductions resulted in significant changes in deviance. Changes in deviance between models were tested with an ANOVA F-test.

3. Results

3.1. Territory overlap and network structure

A summary of the territory structure and the derived network properties in each study plot and year is presented in Table 2. Although each territory was overlapped by more other territories in K3 than K1, the proportion of territory overlap among individuals was similar among study plots and years (Table 2). An example of one of the networks (K1/06) is shown in Fig. 2. Networks were relatively sparse, with between 16.1% and 26.6% of possible edges existing (Table 2). The density and average in-strength levels were higher in K3 than in K1 in both years (Table 2).

Within each study plot, only a sub-set of individuals were recorded in each network for both years (K1, n = 14 and K3, n = 15). The positions of these individuals relative to each other in the networks were significantly correlated between years when compared in a Mantel test that tested the similarity of the association matrices (K1: ***r_{obs} = 0.418, r_{rand} = −0.009 (−0.142–0.221 (95% confidence interval (CI))), P < 0.001; K3: r_{obs} = 0.639, r_{rand} = 0.001 (−0.164–0.232 (95% CI)), P < 0.001).

We explored whether the role of individuals in networks varied between males and females by comparing in-strength, in-male-strength and in-female-strength between males and females in a series of Mann Whitney u randomisation tests (Table 3). Lower val-

Table 3

Effects of sex on the network structure of tuatara (*Sphenodon punctatus*) in each study plot and year. The results of Mann–Whitney *u* randomisation tests are shown for testing sex differences in in-strength, in-male-strength and in-female-strength.

	N	u_{obs}	u_{rand}	95% CI	P
In-strength					
K1/06	21	0.277	0.466	0.227–0.695	0.059
K1/07	18	0.138	0.408	0.167–0.653	**0.013**
K3/06	21	0.509	0.534	0.305–0.782	0.412
K3/07	17	0.111	0.515	0.250–0.806	**<0.001**
In-male-strength					
K1/06	21	0.372	0.507	0.250–0.768	0.150
K1/07	18	0.208	0.507	0.292–0.736	**0.003**
K3/06	21	0.722	0.544	0.305–0.768	0.939
K3/07	17	0.416	0.524	0.278–0.757	0.194
In-female-strength					
K1/06	21	0.313	0.460	0.236–0.668	0.095
K1/07	18	0.277	0.360	0.146–0.597	0.261
K3/06	21	0.213	0.521	0.291–0.759	**0.003**
K3/07	17	0.097	0.503	0.201–0.778	**0.005**

u_{obs}, the observed *u* value; u_{rand}, the mean random *u* value with 95% confidence intervals (CI) calculated from 1000 permutations and *P* is the one-tailed probability ($u_{obs} < u_{rand}$). Values of *u* less than 0.5 indicate males have a higher in-strength than females. *P*-values in bold denote significant values ($P < 0.05$).

ues of *u* (<0.5) indicated males had a higher measure of in-strength than females. Overall, males tended to be better connected within networks than females but this was not significant in all study plots and years (Table 3). Males were significantly more connected to other males than females were in K1 in 2007 (Table 3). Sex differences in in-female strength were only detected in K3, with males having a higher in-female-strength than females in both years (Table 3).

3.2. Parasite infection patterns

3.2.1. In-strength

The prevalence and intensity of parasite infection within each study plot and year are shown in Table 4. In-strength was positively correlated with total tick load (Table 5; Fig. 3A), and marginally positively correlated with larval tick load (Supplementary Table S1) of tuatara in K3 in March 2006. In-strength (measured in March 2006) was also positively correlated with total tick load (Table 5) and nymphal tick load (Supplementary Table S2; Fig. 3B) in September 2006 in K1. There were no associations between in-strength and mite load (Table 5). In-strength was significantly higher in individuals infected with blood parasites than uninfected individuals in K1 in both years (Table 5; Fig. 3C).

3.2.2. In-male-strength and in-female-strength

In-male-strength (measured in March 2006) was positively correlated with total tick load (Table 6, Fig. 4), and larval tick load (Supplementary Tables S1 and S2) of tuatara in both study plots

Table 5

The results of Spearman rank correlation (*r*) randomisation tests comparing in-strength of tuatara (*Sphenodon punctatus*) in transmission networks, with total tick (*Amblyomma sphenodonti*) loads in March, total tick loads in September, mite (*Neotrombicula* spp.) loads in March and the results of Mann–Whitney (*u*) randomisation tests, comparing in-strength between uninfected individuals and individuals infected with blood parasites (*Hepatozoon tuatarae*).

	N	r/u_{obs}	r/u_{rand}	95% CI	P
Ticks: March					
K1/06	11	0.275	0.091	−0.499–0.632	0.304
K1/07	15	−0.211	0.005	−0.540–0.517	0.788
K3/06	14	0.300	−0.037	−0.430–0.324	**0.040**
K3/07	12	−0.326	0.003	−0.620–0.585	0.848
Ticks: September					
K1/06	11	0.514	−0.009	−0.688–0.587	**0.050**
K1/07	–	–	–	–	–
K3/06	16	0.391	−0.017	−0.520–0.511	0.068
K3/07	–	–	–	–	–
Blood parasites					
K1/06	17	0.952	0.526	0.190–0.881	**0.009**
K1/07	17	0.928	0.529	0.143–0.881	**0.011**
K3/06	19	0.538	0.553	0.282–0.808	0.568
K3/07	16	0.654	0.580	0.291–0.855	0.321
Mites: March					
K1/06	11	0.264	−0.023	−0.655–0.582	0.213
K1/07	15	0.304	0.080	−0.368–0.568	0.185
K3/06	14	0.178	−0.030	−0.569–0.495	0.241
K3/07	12	0.315	−0.016	−0.503–0.469	0.103

r/u_{obs}, the observed correlation coefficient (*r*) or *u* value; r/u_{rand}, the mean random *r* or *u* value with 95% confidence intervals (CI) calculated from 1000 permutations; and *P*, the one-tailed probability ($r/u_{obs} > r/u_{rand}$). *P*-values in bold denote significant values ($P < 0.05$).

in March 2006 and September 2006. In-male-strength in March 2006 was also positively correlated with nymphal tick load in K1 in September 2006 (Supplementary Table S2; Fig. 4). We found no significant relationships between mite load or blood parasite infection and in-male-strength in any study plot or year (Table 6; Supplementary Table S1).

In-female-strength was positively correlated with nymphal tick load in K3 in March 2007 (Supplementary Table S1; Fig. 5A), and with mite load in K3 in March 2006 (Table 6; Fig. 5B). In-female-strength was significantly higher in individuals infected with blood parasites than uninfected individuals in K1 in both years (Table 6; Fig. 5C).

3.3. Host sex, size and territory area predict parasite infection

Sex had a significant effect on larval tick loads ($F_{1,38} = 4.10$, $P = 0.049$), total tick loads ($F_{1,38} = 5.56$, $P = 0.023$) and mite loads of tuatara ($F_{1,30} = 9.28$, $P = 0.004$). Males had significantly higher mean larval tick, total tick and mite loads than females (Fig. 6A). There was also a significant interaction between sex and size (SVL) on mite load ($F_{1,30} = 5.33$, $P = 0.027$). Host size (SVL) had no

Table 4

Summary of the prevalence (%) and mean load of ticks (*Amblyomma sphenodonti*), mites (*Neotrombicula* spp.) and blood parasites (*Hepatozoon tuatarae*) of tuatara (*Sphenodon punctatus*) within each study plot and year, for individuals included in networks. Blood parasite load is not shown since this was not included in our analyses, and overall prevalence for each study plot is given since infection status was derived from detecting an infected sample from individuals repeatedly sampled during the period of the study.

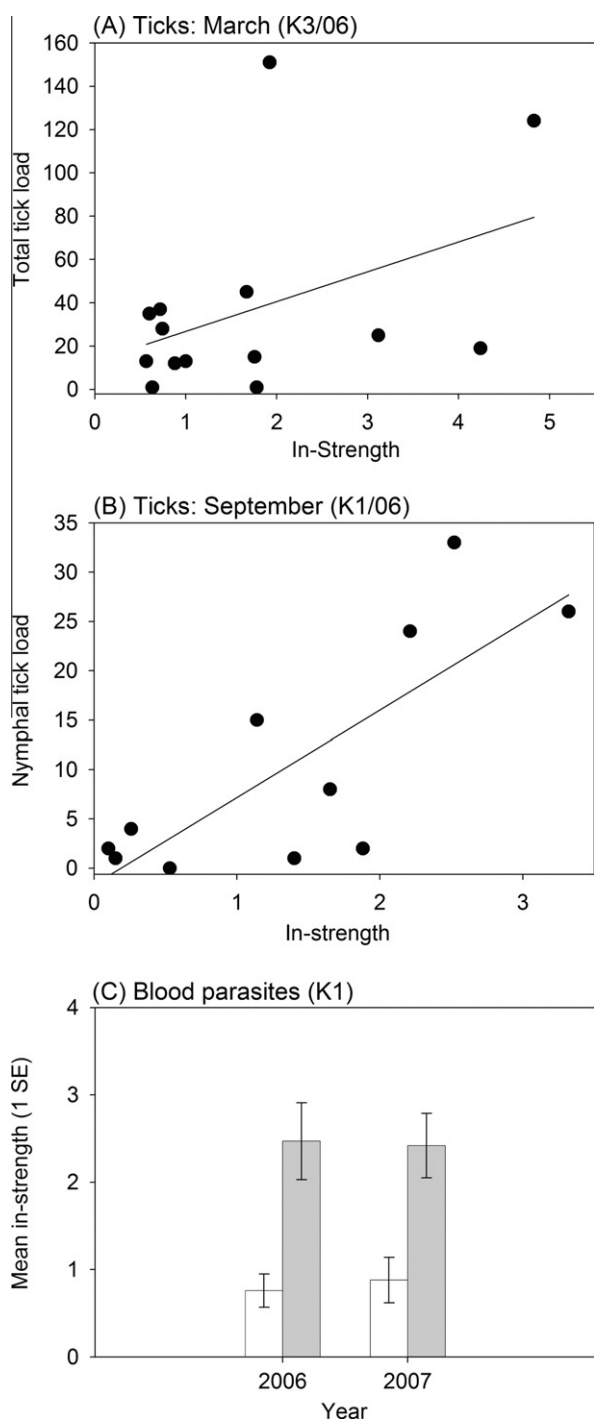| Network	Ticks: March		Ticks: September		Mites: March		Blood parasites
	%	Load ± SE	%	Load ± SE	%	Load + SE	%
K1/06	81.8	11.0 ± 5.9	100	26.4 ± 10.7	100	71.1 ± 10.5	17.6
K1/07	93.3	19.3 ± 5.4	–	–	93.3	300 ± 165.3	
K3/06	100	37.1 ± 12.0	100	78.5 ± 15.1	100	102.5 ± 23.3	31.6
K3/07	100	42.7 ± 10.1	–	–	100	199.7 ± 50.0	

SE, standard error.

Fig. 3. Correlations between in-strength of tuatara (*Sphenodon punctatus*) in networks and (A) total tick (*Amblyomma sphenodonti*) loads in study plot K3 in March 2006, (B) nymphal tick loads in K1 in September 2006 and (C) the mean in-strength of uninfected (white bars) and hosts infected with blood parasites (*Hepatozoon tuatarae*) (grey bars) in K1 in 2006 and 2007.

effect on mite load of males (0.805 + 0.005 (SVL), $F_{1,33}$ = 2.13, P = 0.153), but there was a marginally negative relationship between SVL and mite load of females (5.63–0.02 (SVL), $F_{1,15}$ = 4.26, P = 0.056, R^2 = 0.169). Host size (SVL) had a significant positive effect on nymphal tick load ($F_{1,38}$ = 7.37, P = 0.009, −0.344 + 0.004(SVL), R^2 = 0.046). Territory span (maximum diameter of the 95% MCP territory area (m)) had a significant positive effect on mite load of tuatara ($F_{1,16}$ = 7.44, P = 0.014, R^2 = 0.181, Fig. 6B). However, when an outlying individual that had almost twice the

territory span of any other tuatara was removed from the analysis, this relationship became non-significant (P = 0.1). Territory span had no effect on larval, nymphal or total tick loads of tuatara.

4. Discussion

In this study, we tested whether parasite infection patterns of tuatara were predicted by the extent that individuals were connected to other individuals in empirically-derived networks. Those networks were based on the extent of non-synchronous territory overlap among hosts. For networks derived in 2006, we found positive relationships between measures of in-strength of tuatara and their tick load. In both 2006 and 2007 there were positive relationships between in-strength and infection with tick-borne blood parasites. Many other factors might contribute to the level of infestation and we detected some of those in this study. However, we recognised the consistent, if incomplete, pattern of association between network properties and infestation as a signal that networks played a significant role in the transmission of these two parasites. Only one relationship was detected between mite load of tuatara and a measure of network in-strength. Instead, host sex, body size and territory size were stronger predictors of mite load. Our results suggest a role of network structure in parasite transmission that depends on the parasite species.

The overall number and weight of connections to individuals in the network (in-strength) was a positive predictor of tick load but not in all study plots or months. This shows that at least in some situations, the extent of overlap among territory boundaries may influence the cumulative exposure of host individuals to tick infestation. Connections to males (in-male-strength) were strong and consistent predictors of tick load, in both March and (more strongly) in September. Connections to females (in-female-strength) were significantly related to fewer measures of tick load. Our results suggest that network topology predicts tick load and that relationships between in-strength and tick load are stronger for connections to males than to females.

Male tuatara have larger territories than females (Moore et al., 2009a) and higher in-strength in networks, suggesting they occupy more central positions in the network. Males also have higher tick loads than females, meaning that areas overlapping with male territories may be more likely to become infectious. That is, individuals with high in-male-strength will have a greater risk of tick infestation than individuals with equivalent in-female-strength or total in-strength.

There were stronger (more positive) associations between network measures and tick load in September than in March when the behavioural data were collected. Because the tick attachment rate is low in early March (Godfrey et al., 2008), tick loads measured then may represent early stages of infestation when the network has had less time to influence the exposure of hosts to ticks. Ticks over-winter on tuatara and begin to engorge and detach in early spring (Godfrey et al., 2008). Thus, tick loads measured in September (spring) probably represent the cumulative exposure of hosts to ticks across the mating and subsequent post-mating season, leading to the stronger associations at that time.

Tick loads of tuatara were also influenced by host sex. Males had a higher average tick load than females, in both March and September. Male-biased parasitism is common among host-parasite systems, with several possible explanations. Testosterone can have an immunosuppressive effect on hosts and is usually higher in males, increasing male susceptibility to parasite infection (Folstad and Karter, 1992; Zuk and McKean, 1996). Also, if males move about more or have larger territory areas, they will have a higher exposure to infection sources (Krasnov et al., 2005). However, in this study we detected no relationship between territory size and

Table 6
The results of Spearman rank correlation (r) randomisation tests comparing in-male-strength and in-female-strength of tuatara (*Sphenodon punctatus*) in transmission networks, with total tick (*Amblyomma sphenodonti*) loads in March, total tick loads in September, mite (*Neotrombicula* spp.) loads in March, and the results of Mann–Whitney (u) randomisation tests, comparing in-strength between uninfected individuals and individuals infected with blood parasites (*Hepatozoon tuatarae*).

	N	In-male-strength				In-female-strength			
		r_{obs}	r_{rand}	95% CI	P	r_{obs}	r_{rand}	95% CI	P
Ticks: March									
K1/06	11	0.633	−0.001	−0.601–0.599	**0.018**	−0.134	0.050	−0.587–0.633	0.710
K1/07	15	−0.088	−0.005	−0.524–0.508	0.613	−0.282	0.010	−0.443–0.434	0.877
K3/06	14	0.261	−0.143	−0.514–0.189	**0.006**	0.117	0.003	−0.364–0.354	0.282
K3/07	12	−0.238	−0.125	−0.676–0.396	0.648	−0.301	0.024	−0.539–0.609	0.851
Ticks: September									
K1/06	11	0.708	−0.013	−0.629–0.597	**0.010**	0.454	0.059	−0.537–0.657	0.116
K1/07	–	–	–	–	–	–	–	–	–
K3/06	16	0.499	−0.046	−0.516–0.451	**0.014**	0.062	−0.054	−0.546–0.451	0.322
K3/07	–	–	–	–	–	–	–	–	–
Mites: March									
K1/06	11	0.128	−0.006	−0.706–0.647	0.372	0.156	−0.032	−0.633–0.569	0.286
K1/07	15	0.256	0.004	−0.481–0.492	0.190	0.120	0.152	−0.262–0.556	0.565
K3/06	14	−0.334	−0.069	−0.517–0.367	0.865	0.598	−0.023	−0.508–0.486	**0.003**
K3/07	12	−0.217	−0.229	−0.706–0.252	0.483	0.420	0.033	−0.483–0.538	0.084
Blood parasites									
K1/06	17	0.571	0.498	0.095–0.905	0.374	1.000	0.527	0.155–0.881	**<0.001**
K1/07	17	0.809	0.509	0.095–0.881	0.085	0.940	0.534	0.333–0.857	**0.010**
K3/06	19	0.641	0.575	0.333–0.821	0.309	0.455	0.548	0.256–0.821	0.766
K3/07	16	0.781	0.645	0.400–0.891	0.182	0.563	0.568	0.309–0.855	0.533

r/u_{obs}, the observed correlation coefficient (r) or u value; r/u_{rand}, the mean random r or u value with 95% confidence intervals (CI) calculated from 1000 permutations; and P, the one-tailed probability ($r/u_{obs} > r/u_{rand}$). P-values in bold denote significant values ($P < 0.05$).
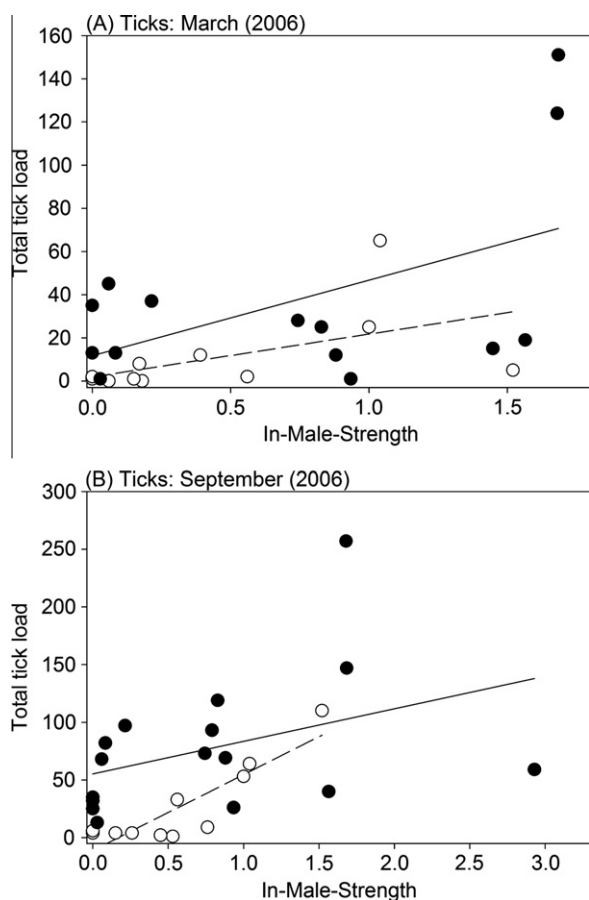

Fig. 4. Correlations between in-male-strength of tuatara (*Sphenodon punctatus*) in networks and total tick (*Amblyomma sphenodonti*) load in study plot K1 (hollow circle ○, dashed line –) and K3 (filled circle ●, solid line –) in (A) March 2006 and (B) September 2006.

tick load. Another explanation is that the sex difference in tick loads arises from network positions. Males may be exposed to more ticks than females if they overlap with more individuals, and this is supported by the higher in-strength values for males than females in some of the networks. In this explanation it is the position in the network rather than any specific physiological or behavioural characteristic of male tuatara that explains their higher tick loads. Alternatively, it is possible that male physiology and behaviour interact to enhance the parasite loads of males through network structure. Recently, Grear et al. (2009) found networks of wild rodents (*Peromyscus leucopus*) where males had experimentally elevated testosterone levels were more strongly connected and had a higher transmission potential than control networks. Thus, it is possible that an interaction between testosterone levels and host behaviour could also contribute to the higher parasite loads of males.

Most of the significant associations between network structure and tick load of tuatara occurred in 2006. Network structures were significantly correlated between years, so differences in network structure between years is unlikely to explain the lack of clear associations between tick loads and network properties in 2007. However, tick loads were higher in 2007 than 2006 (Table 4; Godfrey et al., 2008). In some epidemiological networks, as a disease percolates through a network, there is a point where the disease begins to saturate the network. It is at this point that network properties such as in-strength may no longer accurately predict infection patterns. Thus, it is possible that a similar phenomenon may occur for ectoparasite infestation. The signal of parasites on the network may become diluted as more parasites are added to the system, particularly where the data are observational. Alternatively, the lack of clear associations between networks and in-strength in 2007 could suggest that our observational data were insufficient to capture transmission under different ecological conditions.

Both in-strength and in-female-strength were significantly related to blood parasite infection status in one study plot in both years. More highly connected hosts are likely to have higher expo-
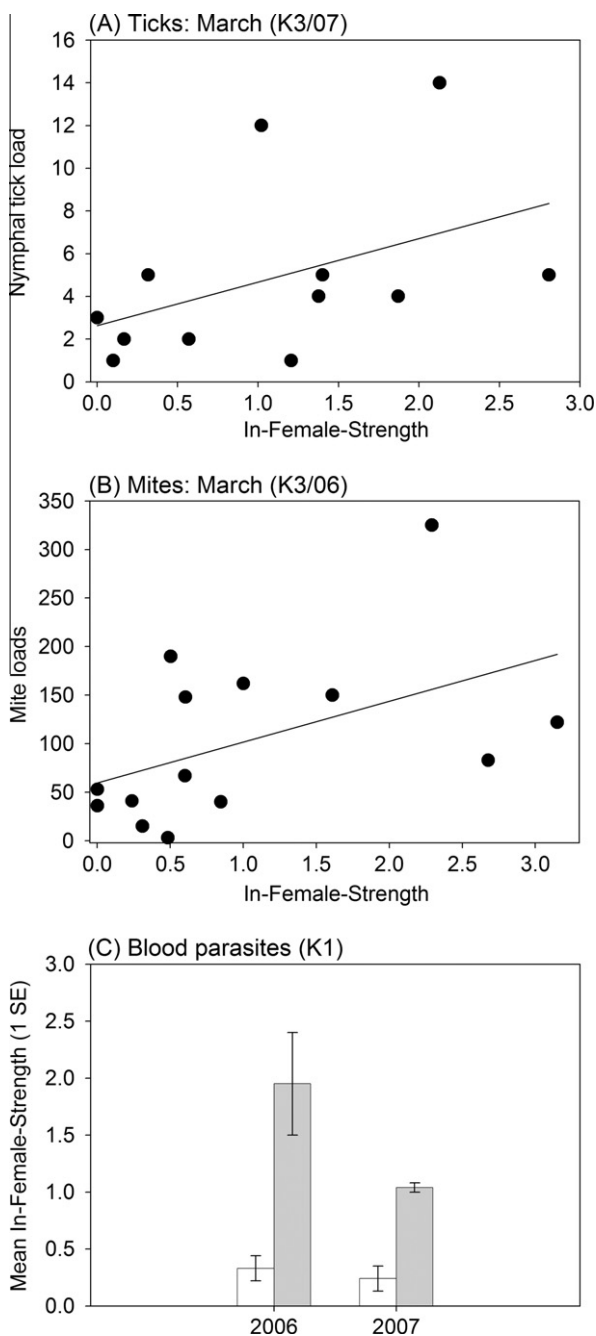
Fig. 5. Correlations between in-female-strength of tuatara (*Sphenodon punctatus*) in networks and (A) nymphal tick (*Amblyomma sphenodonti*) load in study plot K3 in 2007, (B) mite (*Neotrombicula* spp.) load in K3 in 2006 and (C) mean in-female-strength of uninfected (white bars) and hosts infected with blood parasites (*Hepatozoon tuatarae*) (grey bars) in K1 in 2006 and 2007.

sure to infection. Thus, like ticks, it appears that network structure influences blood parasite transmission, although unlike ticks, females may be more important than males in the transmission of the blood parasite.

We previously showed (Godfrey et al., in press) that in the forest where this study was conducted, the prevalence of blood parasite infection is influenced by tick loads, but this effect is dependent on host size. Small to moderately sized tuatara (<230 mm SVL) were more likely to be infected by blood parasites if they had high tick loads, whereas blood parasite infection of larger hosts was independent of tick load (Godfrey et al., in press). This could explain associations between blood parasite infection
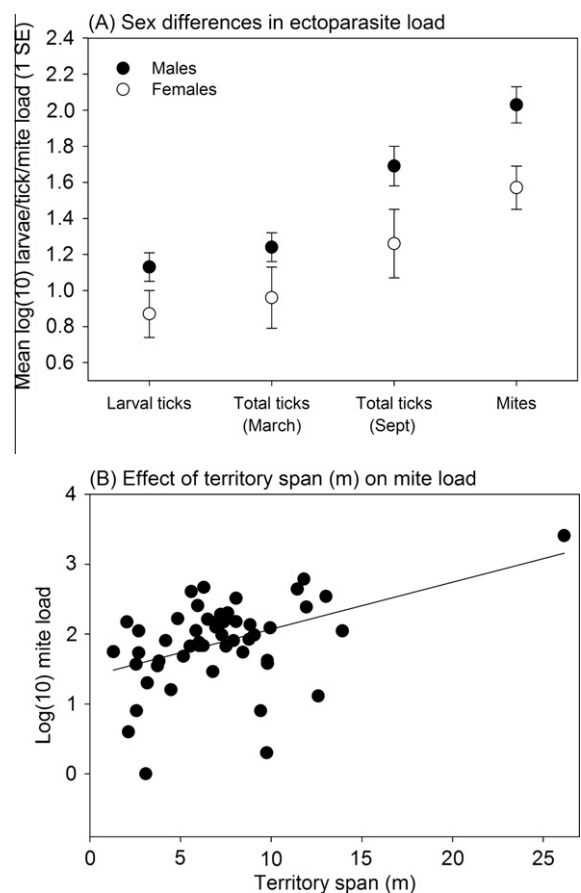

Fig. 6. Relationships between host properties and parasite infection patterns of tuatara (*Sphenodon punctatus*), showing (A) sex differences in larval tick (*Amblyomma sphenodonti*) load, total tick load and mite (*Neotrombicula* spp.) load of tuatara, and (B) relationship between territory span (maximum diameter of the 95% minimum convex polygons of territory area (m)) and mite loads of tuatara in March (2006 + 2007). SE, standard error.

and network properties, since highly connected individuals are more likely to have a high tick load. High tick loads can increase exposure to potential blood parasite vectors, and could also compromise the immune system of these individuals, increasing their susceptibility to blood parasite infection. The non-significant relationship between network measures and blood parasite infection in the second site could again be due to higher prevalence and infection saturation obscuring network processes.

We found no relationships between in-strength and mite loads of tuatara, although there was a positive relationship between in-female-strength and mites in one plot in 1 year. Host factors had a more consistent effect on mite loads. Male hosts had higher mite loads than females, a similar but much stronger trend than that detected for ticks. Mite load was negatively related to host size in female tuatara. Mite load was also positively related to host territory size, which could have contributed to the sex difference since male hosts have larger territories.

The single significant relationship between in-female-strength and mite load, and the strong effects of several host-related factors, suggest that the transmission of this parasite is less influenced by host network structure. This reflects the free-living status of nymphal and adult mites (Wharton, 1952; Sasa, 1961). After engorged larval mites detach from their host, nymphs and adults can move relatively freely through their environment, reducing the dependence of host spatial arrangements on mite transmission. If mites become randomly dispersed through the environment, a host that moves around a larger area will increase its cumulative exposure

to mites, leading to the positive relationship we detected between territory span and mite load. At the same time this would result in a reduced role for network structure in the transmission of these parasites.

Comparatively, the networks were more successful in predicting patterns of infection for less mobile parasites (ticks and tick-borne parasites) that were more dependent on host movement for their transmission. For those parasites, the cumulative exposure of hosts to infection increases with the extent that individuals overlap in territory area with other individuals, particularly if they are males.

Our observations are correlative and an alternative interpretation is that infection influences the position of individual tuatara in networks. That is, ticks and blood parasites could alter host behaviour, perhaps reducing the vigour of territory defence, such that infected hosts suffered higher levels of territory overlap with neighbours. Infection is known to reduce aggression and success in territorial contests (Maksimowich and Mathis, 2000; Marden and Cobb, 2004). However, in our study system larger male tuatara are consistently dominant (Moore et al., 2009a), and it seems unlikely that the relatively low levels of tick and blood parasite infections we recorded could affect male vigour and override the effect of male size on defence of territories. Furthermore, tick loads in September could not have altered host behaviour in the previous March, when we derived the networks. We consider a more likely explanation of the correlations is that network structure predicts parasite infestation.

Our findings suggest that territory structure in solitary-territorial systems can define pathways of parasite transmission and this in turn could adjust parasite loads of hosts that differ in their social dominance. Other studies have reported higher parasite loads in less dominant individuals in a population (Maksimowich and Mathis, 2000; Ezenwa, 2004; Marden and Cobb, 2004; Whiteman and Parker, 2004). Usually, this is explained by more subordinate individuals being impaired by high parasite loads in territorial contests (Freeland, 1981; Zuk et al., 1998; Hoodless et al., 2002; Mougeot et al., 2005). However, we suggest an alternate mechanism; that the more subordinate individuals, who are unable to maintain exclusive territories, may be exposed to more parasites through increased territory overlap.

More generally, parasite load may both influence, and be influenced by, host social networks. Thus, parasites could have either a regulatory or synergistic effect on territory structure and social dominance hierarchies. Although ticks are unlikely to affect tuatara behaviour in the short-term, high tick loads of tuatara can cause declines in host body condition (Godfrey et al., 2010). Tuatara have indeterminate growth and body size is influenced by resource availability rather than age (Nelson et al., 2002). Highly connected individuals with consistently high tick loads and reduced body condition may suffer from suppressed growth rates. This could generate a long term synergistic relationship between social rank, territory structure, tick loads and growth rates; where small males stay small and subordinate, while larger males become larger and remain dominant. Tuatara have an extremely long generation time (approximately 50 years (Allendorf and Luikart, 2006)) and an extremely slow growth rate, so this would be difficult to test in the duration of a normal study.

Our study established positive relationships between the network structure of a solitary-territorial host species and parasite loads of the hosts. We inferred that being more connected in the network leads to a higher exposure to infection sources through overlapping territory areas. Our findings are consistent with studies that examined the network structure and patterns of parasite infection in group-living lizards (Godfrey et al., 2009) and pair-living lizards (Leu et al., 2010). Although traditionally, solitary territoriality is thought to have little consequence for parasite transmission, the findings from this study indicate that similar selective pressures may operate on very different forms of social organisation. The connectivity of individuals within social networks is the underlying mechanism influencing transmission, but the importance of the network varies among parasite species with different transmission properties.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.ijpara.2010.06.002.

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