Landscape structure influences avian malaria ecology in the Western Cape, South Africa

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Abstract A central theme in landscape ecology is that of understanding the consequences of landscape heterogeneity for ecological processes. The effects of landscape heterogeneity on parasite communities are poorly understood, although it has been shown that anthropogenic impacts may contribute to outbreaks of both parasites and pathogens. We tested for effects of landcover type, composition, configuration, and urbanisation on avian diversity and avian malaria prevalence in 26 communities of wetland-associated passerines in the Western Cape of South Africa. We predicted that avian malaria prevalence would be influenced by the pattern of farmland and urban areas in the surrounding landscapes and the sizes of the wetlands in which birds were sampled. We quantified landscape pattern using a six-class simplification of the National Landcover data set at 35×35 m resolution and five extents of between 1 and 20 km from each wetland. The bird community was sampled using

point counts and we collected blood samples from birds at each site. We screened these for malaria using PCR and molecular techniques. Passerine species richness and infection prevalence varied significantly between different landcover types. Host richness and parasite prevalence were highest in viticultural and cropping sites respectively and lowest in urban sites. Wetlands located in indigenous vegetation had intermediate numbers of bird species and intermediate parasite prevalence. Landscape composition and habitat type surrounding wetlands emerged as useful correlates of infection prevalence. Anthropogenic landscape modification appears to have both direct and indirect effects on avian communities and their associated parasite assemblages, with attendant consequences for avian health.

Keywords Landscape composition · Heterogeneity · Species richness · Avian malaria · Urbanization

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Introduction

The consequences of landscape heterogeneity for avian populations and communities have been extensively studied. Far less is known about the consequences of landscape heterogeneity, and of changes in landscape composition and configuration, for parasites and pathogens. As humans alter landscape pattern, a number of impacts on parasites and their hosts and

vectors are likely to occur. Of particular relevance are changes in the abundance and species richness of host and vector species. These occur in response to changes in such variables as habitat structure, resource availability, predation rates, and ease of dispersal (With and Crist 1995; Flather and Sauer 1996). Unless all hosts and all vectors are equally competent, changes in host and/or vector communities can be expected to alter the transmission rates of parasites (and their overall prevalence within the host community) by altering the numbers of suitable and available hosts (Kruess and Tscharnkte 1994; Brisson et al. 2011), the number of successful transmission events that occur by either direct contact or via vectors, and/or the prevalence of infectious individuals among susceptible hosts (Roche et al. 2012).

The problem of deriving general principles from which to predict impacts of landscape change on parasite communities is complicated by a set of indirect and idiosyncratic effects. For example, more diverse host communities may exhibit lower infection rates because more ‘wasted bites’ on incompetent vector species occur (Dobson et al. 2006). Conversely, communities that are more diverse to begin with will be more likely to retain at least some highly competent hosts and vectors following anthropogenic habitat modification. Changes in landscape composition and configuration can influence dispersal rates and bring domestic and wild species (and people) into contact with one another, further altering parasite and pathogen dynamics. For instance, deforestation has been blamed for outbreaks of human malaria in the Amazon Basin (Vittor et al. 2006), cutaneous leishmaniasis in Costa Rica (Chaves et al. 2008), and severe acute respiratory syndrome (SARS) in South East Asia (Field 2009). Urbanization has similarly been linked to the proliferation of zoonotics such as Lyme disease (LoGiudice et al. 2003) and rabies (Singh et al. 2001).

Although birds are highly mobile, they may have highly specific resource requirements. In southern Africa, Child et al. (2009) identified a decline in species richness at upper trophic levels between protected (and mostly intact) and non-protected (fragmented) landscapes. Similarly, urbanization can have a negative effect on habitat quality and correspondingly on avian species richness in greater Cape Town (Dures and Cumming 2010). These changes would be expected to both directly and indirectly affect parasite and pathogen prevalence. In the USA, research on West Nile Virus

(WNV) showed that an increased prevalence of antibodies for WNV in songbirds corresponded to an increasing scale of urbanization (Bradley et al. 2008). In American wetlands, WNV prevalence decreased with increasing wetland size (Ezenwa et al. 2007). Similarly, wetland size has been linked to resource availability (Paracuellos and Tellria 2004), and may subsequently affect local avian species richness as a result.

We tested three predictions about the ecology of avian haemosporidia in the Western Cape, South Africa, with a focus on wetland passerine communities. First, we predicted that infection prevalence would be influenced by the composition of the surrounding landscape, and especially of urban areas, since these adversely impact bird species diversity and richness (Fair child et al. 2009; Dures and Cumming 2010). Species diversity and species richness have both been linked to infection prevalence (Keesing et al. 2006; Swaddle and Calos 2008). Second, we predicted that infection prevalence would be affected by the perimeter–area ratio and connectivity of habitats, as these potentially influence encounter rates between pathogens and hosts (as described above from other disease systems; viz Singh et al. 2001; Vittor et al. 2006; Chaves et al. 2008). Third, we predicted that wetland size would affect infection prevalence, because of its impacts on resource availability and species richness.

Methods

Study area and site selection

Twenty-six wetlands were selected as study sites from within the historical boundaries of the fynbos biome in the Western Cape. The area has strong gradients of human population density, ranging from a large city (Cape Town) to sparsely settled agricultural and rural areas; a diversity of land uses, ranging from intense crop agriculture, viticulture and forestry to virgin protected areas; strong gradients of rainfall, temperature and altitude; and indigenous vegetation, the fynbos, that is mostly either shrubland or dwarf shrubland. Sites were situated along an urban to rural gradient and restricted to altitudes of 0–300 m above sea level (Fig. 1). Each site was visited twice over the course of 2 years (2010 and 2011), with samples collected once during the summer (January to March) and once during the winter (July to September). Wetland size was determined by mapping

Fig. 1 Map of the Western Cape showing study site locations, landcover types, and urban centres closest to each wetland site

the perimeter of the main water body at each site using a handheld GPS unit (Garmin GPS map 76CSx).

Sampling

Birds were trapped using mist nets, with efforts focused on wetland passerines. Blood samples were taken by pricking the brachial vein with a 26-gauge needle (26G $\times$ ½ inch) and capturing 30–50 μ l of blood in a capillary tube (Dawson 2004). The blood was transferred into a vial containing lysis buffer. The vial was sealed and stored and all birds were released after sampling. Five birds were recaptured during the sampling period; these individuals were counted once during analysis.

Data analysis

Blood samples were analysed following a PCR protocol, as detailed by Cumming et al. (2013). Two PCR trials were conducted for each sample under identical conditions. PCR products were sequenced using a 10 μ l reaction using the Big Dye Terminator v.3.1 Cycle Sequencing Kit (Applied Biosystems), following manufacturer protocols. The final products were sent for automated sequencing at the DNA Sequencing Facility on Science Hill, Yale University. This PCR routine only detected parasites within the *Plasmodium* and *Haemoproteus* genera (Waldenström et al. 2004).

Spatial analysis was conducted using ArcGIS (9.3.1_2008) and Fragstats (version 4) (McGarigal et al. 2012). Additional statistical analysis was

Table 1 Landscape structural attributes calculated at each extent (all measurements are expressed as means)

Variable	Units	Derivation	Description
Patch area	Ha	TCA/10,000	Patch size of each land cover type converted to hectares (divided by 10,000)
Patch perimeter–area ratio (PARA)	Ratio	P/A	Measure of edge density of patch
Connectivity	%	$(N_c/T_c)*100$	The number of connections between patches of the same landcover type as a proportion of the total number of possible connections. Specified over a set area (i.e. at each extent)
Relative patch richness (RPR)	%	N_p/T_p	The number of landcover types at each extent as a proportion of the total number of land cover types
Wetland size	Ha	wa/10,000	Wetland area (m ²) converted to hectares
Urban density	–	nb/ws	Estimated by the number of buildings (rooftops and structures) within a 1 km radius of sites (following Dures and Cumming 2010), divided by wetland size, to correct for varying wetland sizes.

TCA total core area (m²) of each landcover type, total core area, *P* total perimeter, *A* area, *N_c* number of joins with patch of same landcover type, *T_c* total connections possible (of same landcover type), *N_p* number of landcover types, *T_p* total number of landcover types, *wa* wetland area (m²), *nb* number of buildings within a 1 km radius of wetland, *ws* wetland size

conducted in R (2011_12_22). Analysis of the landscape was approached with two objectives: (1) to determine the influence of landscape composition (areas of different landcover types) on bird communities and prevalence of avian haemosporidia; and (2) to determine the influence of landscape configuration (spatial arrangement, including perimeter to area ratios, edges, and connectivity) at various extents.

To classify landcover we used the South African National Landcover Data set (NLCD), which is derived from interpretation of Landsat 7 TM imagery at 35 m resolution (Fairbanks et al. 2000). We reclassified the NLCD to yield a simpler subset of the most relevant land cover types: fynbos, agriculture, urban, and viticulture. Sites were then grouped according to the dominant surrounding land cover class: fynbos (3 sites), agricultural (9 sites), urban (7 sites) and viticultural (7 sites). Other nearby landcover types (mining, forest, etc.) were reclassified into a single category labelled ‘other’ (Fig. 1).

Buffer zones of 1, 5, 10, 15 and 20 km in extent were created around each wetland in ArcGIS and extracted to give corresponding landscape patches. These extents were chosen to correspond to the range of reported flight distances (scales of landscape use) of birds from the family Ploceidae (bishops, weavers, and allies), which can be up to 20 km in weaver birds (Hockey et al. 2005). We based the analysis on the Ploceidae because our data show that they were the family that was most prevalently infected with avian

malaria and we expected that their incidence and movements would relate closely to infection patterns.

Variables specific to landscape area, edge, and connectivity were quantified using Fragstats (McGarigal et al. 2012), at each of our five extents of analysis, to determine which structural aspects were influential to infection prevalence (see Table 1).

Prevalence data for *Plasmodium* and *Haemoproteus* were combined and referred to as infection prevalence. Bird species richness (site species richness, infected species richness and passerine species richness) was taken into account due to its response to landscape structure (Child et al. 2009).

We used one-way ANOVA and parametric regression analysis to determine the nature of the relationships between landscape type and pattern; wetland size; urban density; infection prevalence and bird species richness (site and passerine). Due to the large number of comparisons being made, a Bonferroni correction was made for critical *p* values at the 0.05 and 0.01 level, to reduce the probability of type I errors (Dunn 1961). Critical *p* values were set as

$$\text{Bfc}_{0.05} = 1 - (1 - 0.05)/26 \quad (1)$$

$$\text{Bfc}_{0.01} = 1 - (1 - 0.01)/26 \quad (2)$$

where Bfc is the Bonferroni correction factor, the subscript indicates the probability value to which the Bfc applies, and 26 was the number of comparisons made.

Generalized linear mixed models (GLMMs) were used to explore which landscape parameters best explained infection prevalence patterns. Models were fitted with canonical links (corresponding to the nature of the prevalence distribution), with data arranged by extent. Area, connectivity, perimeter–area ratio (PARA) and landcover type featured as fixed effects in models, and passerine species richness and site featured as factors. We ranked candidate models according to their Akaike's Information Criterion (AIC) value and retained or excluded variables according to their effect on the AIC value (Burnham and Anderson 2002), with lower AIC values indicating a better fit of a model to the data.

Results

Landscape, infection prevalence and avian species richness

A total of 200 out of 974 samples were both positive for avian malaria and successfully sequenced, with the majority of infected species belonging to the family Ploceidae ($n = 165$). The majority of birds were sampled from wetlands within agricultural landscapes (344), followed by wetlands within viticultural landscapes (308); then fynbos and urban landscapes (98 and 224 birds respectively). Sites within a viticultural landscape had the highest mean species richness (all birds) whereas agricultural sites had the highest mean passerine species richness. Fynbos sites had the lowest mean site species richness (Fig. 2). There was little difference in mean infected species richness, which ranged from 9 to 12 between landscapes. Mean infected species was highest in viticultural landscapes (12) and lowest in urban landscapes (9). Urban landscapes also had the lowest mean passerine species richness (16). Significant variation in infection prevalence within different landscapes did not occur with either site species richness ($F_{3, 22} = 0.96$; $p = 0.43$) or infected species richness ($F_{3, 22} = 1.16$; $p = 0.35$); however, passerine species richness was associated with landscape type ($F_{3, 22} = 3.55$; $p = 0.03$).

There was a clear disparity in the mean infection prevalence of birds sampled from wetlands within different landscapes ($F_{3, 22}$; $p = 0.002$). Birds sampled from agricultural landscapes had the highest infection prevalence, whereas birds sampled from

Fig. 2 Patterns of variation in bird species richness across wetlands within contrasting landscapes

Fig. 3 Variation in mean infection prevalence between birds sampled around wetland sites in contrasting landscapes ($n = 26$). Birds sampled per landscape is indicated on top of each bar

urban landscapes had the lowest overall infection prevalence (Fig. 3). This pattern was also apparent when the regional landscape was viewed as a whole (Fig. 4).

After Bonferroni correction, urban landcover stood out as the most influential landcover type on passerine species richness (Table 2). Increasing the area of urban landcover had a consistently negative influence on passerine species richness, with the largest effects seen at the 15–20 km extents. Passerine species richness declined with increasing urban density ($r^2 = -0.40$; $p = 0.01$). Edge and connectivity of urban cover was also significantly related to passerine

Fig. 4 Variation in infection prevalence across Western Cape landscapes; pie chart radii correspond to infection prevalence (the most prominently infected sites are encircled)

species richness, although only at the 1 km extent. Area and connectivity of agricultural land was similarly influential on infection prevalence and species richness at the more local extents (1–5 km). Also notable was the influence of edge of other landcover types on infection prevalence at the 20 km extent.

Variation in species richness and prevalence with landcover structure

Urban proximity was also influential on infection prevalence, with increased proximity causing a decline in prevalence ($r^2 = 0.40$; $p = 0.04$). Other species richness measures did not show any notable association with either building density or urban proximity; site species richness was, however, significantly associated with wetland size ($r^2 = 0.43$; $p \leq 0.001$).

Using generalized linear mixed modelling, the best fitting models featured site as a highly influential factor with landcover type or passerine species richness as mixed effects (Table 3). Site, landcover type and passerine species richness were the most influential factors to infection prevalence, with negligible variation between models featuring only these variables ($\Delta AIC \leq 2$). Structural parameters (area, perimeter–area ratio and connectivity) produced a poorer model fit and were comparable to each other in

terms of their influence, with no discernible difference in fit seen when one parameter was substituted for another.

Discussion

In general, wetlands within agricultural and viticultural landscapes ranked highest for passerine and site species richness respectively. By contrast, infected species and passerine species richness were lowest in fynbos and urban landscapes (Fig. 2). This outcome seemed to reflect habitat preferences of wetland passerines. The heaviest malarial infections and highest passerine species richness were both found in agricultural landscapes, whereas the lowest values occurred in urban landscapes (Figs. 2, 3). Birds from the Ploceidae family also exhibited some of the heaviest infection prevalences. Many sampled passerine species were seed-eaters (Hockey et al. 2005), occurring mostly in the Ploceidae and Passeridae (sparrows and allies). It is natural that seed-eaters would be commoner in agricultural landscapes, as these landscapes are presumably richer in available resources for seed-eating birds. The finding also concurs with previous studies, where bird species richness mirrored resource availability (Paracuellos and Tellria 2004; Child et al. 2009).

Table 2 Pearson's correlation (r^2) values for relationships between land cover; infection prevalence (IP); and passerine species richness (PSR) at all extents

	Mean area (m ²)		Mean perimeter–area ratio		Mean connectivity (%)	
	IP	PSR	IP	PSR	IP	PSR
Fynbos						
1	−0.10	−0.02	0.11	−0.07	0.25	0.20
5	−0.12	−0.16	0.18	0.23	−0.06	−0.06
10	−0.10	−0.22	0.24	0.22	0.09	0.26
15	−0.12	−0.18	0.29	0.22	−0.03	0.23
20	−0.18	−0.15	0.14	0.04	−0.15	0.20
Agriculture						
1	0.65**	0.20	−0.17	0.10	−0.16	0.15
5	0.34	0.02	0.29	0.45	0.43	0.58*
10	0.30	−0.02	0.20	0.22	0.49	0.47
15	0.33	0.11	0.20	−0.06	−0.12	0.09
20	0.21	0.12	0.27	−0.08	−0.01	0.26
Urban						
1	−0.45	−0.44	−0.36	−0.59*	−0.40	−0.66**
5	−0.36	−0.55	−0.17	0.03	−0.50	−0.40
10	−0.43	−0.54	−0.39	−0.48	−0.26	−0.38
15	−0.44	−0.58*	−0.26	−0.19	−0.08	−0.03
20	−0.48	−0.60*	−0.55	−0.46	−0.18	−0.13
Viticulture						
1	0.03	0.04	0.01	0.36	−0.04	0.25
5	−0.06	0.04	−0.28	0.0008	−0.12	0.22
10	−0.11	0.02	−0.07	0.14	−0.50	−0.003
15	−0.09	0.006	−0.08	−0.08	−0.33	−0.13
20	−0.07	0.04	0.14	−0.14	0.02	0.06
Water						
1	−0.21	−0.03	−0.01	0.49	−0.13	0.12
5	−0.33	0.08	0.009	0.30	−0.04	0.11
10	−0.30	−0.20	−0.09	0.17	0.13	0.15
15	−0.29	−0.32	−0.05	0.37	0.26	0.25
20	−0.29	−0.29	−0.10	0.39	−0.26	−0.21
Other						
1	−0.26	0.11	0.005	0.15	−0.15	−0.02
5	−0.12	0.19	0.28	−0.17	−0.12	−0.02
10	0.12	0.30	0.13	−0.20	0.05	0.01
15	0.20	0.39	0.41	0.09	−0.14	−0.10
20	0.16	0.30	0.58*	0.45	0.09	−0.05

Significant relationships (Bonferroni corrected) are denoted by * ($p \leq 0.002$) and ** ($p \leq 0.0004$)

Landcover type and structural attributes vs infection

GLMMs showed that landcover type, site and passerine species richness were the variables best describing infection prevalence (Table 3). Model fit changed only minimally when configurational variables were

added, indicating that landscape configuration was not as influential as landcover type. This result is comparable to the outcomes from related studies on disease prevalence in various landscapes, where infection prevalence varied significantly between different vegetation types (Boone et al. 2000), and to more general studies that have indicated a larger effect of

Table 3 Mixed effects models describing variation in infection prevalence with landscape structural features and cover type ($n = 26$)

Model	Log likelihood	Deviance	AIC	Δ AIC
$Site_{factor} + LCT_{effect}$	1.13^{-09}	2.26^{-09}	54	–
$PSR_{effect} + site_{factor}$	1.86^{-08}	3.38^{-08}	54	–
Connect + $site_{factor} + LCT_{effect}$	7.104^{-09}	1.42^{-09}	56	2.00
$PSR_{effect} + site_{factor} + LCT_{effect}$	5.46^{-08}	1.09^{-07}	56	2.00
$Area + site_{factor} + LCT_{effect}$	3.22^{-14}	1.79^{-07}	56	2.00
$PARA + PSR_{effect} + site_{factor}$	8.99^{-10}	$1.80i^{-09}$	56	2.00
$Area + PSR_{effect} + site_{factor}$	2.97^{-09}	5.95^{-09}	56	2.00
Connect + $PSR_{effect} + site_{factor}$	4.05^{-08}	8.08^{-09}	56	2.00
$Area + Connect + site_{factor} + LCT_{effect}$	2.45^{-08}	4.90^{-08}	58	4.00
$PSR_{effect} + Area + PARA + Connect + site_{factor}$	2.50^{-08}	5.00^{-08}	60	4.00
$PARA + site_{factor} + LCT_{effect}$	5.40	10.8	66.8	12.8

Models are ordered according to deviance, as several models shared the same AIC values. Bold rows indicates the best fit models; site and land cover type featured as both random effects and factors

PSR passerine species richness, *Area* area of landscape, *PARA* landscape perimeter–area ratio, *Connect* landscape connectivity, *LCT* land cover type

habitat amount than of habitat configuration (e.g., Flather and Bevers 2002). Other studies on vector-borne disease prevalence and landscape composition and configuration have, however, indicated that both land use and connectivity can also play primary roles in influencing disease prevalence (Linard et al. 2007; Vanwambeke et al. 2010). These outcomes suggest that host and vector ecology not only contribute to the influence of landscape factors in vector-borne disease, but also affect the magnitude of their influence.

Wetlands in agricultural landscapes had the highest infection prevalences among those sampled (Fig. 3). While this could be solely a consequence of the fact that passerine (host) species richness was highest in agricultural areas, vector-related factors may also have played a part in this outcome. Previous studies in Kenya and Ethiopia have demonstrated a positive link between *Anopheles gambiae* larval development and the agricultural modification of landscapes, which often enhances conditions for mosquito breeding (Kebede et al. 2005; Munga et al. 2009). It is feasible that the same mechanisms may be in effect in the Western Cape of South Africa, where crops can provide a rich nutrient source for the larvae of avian malaria vectors. Additionally, irrigation practices in agricultural landscapes in the Western Cape (Venter 2005) facilitate vector breeding and development, as virtually all avian malaria vectors have a water-dependent stage in their lifecycle (Valkiūnas 2005).

Wetland size, landscape configuration and composition were not associated with each other, as wetlands of assorted sizes occurred in a variety of landscapes. Previously, wetland area has been shown to affect both pathogen prevalence and avian species richness (Paracuellos and Telléria 2004; Ezenwa et al. 2007). In our study, wetland size did not exhibit any apparent association with infection prevalence. Wetland size, however, was significantly associated with site species richness, suggesting that it exerts an indirect influence on passerine species richness.

The urban landscape, bird species richness and infection

Urban landcover was consistently influential on passerine species richness; urban edge and connectivity in particular both exerted a direct influence on passerine species richness, with the greatest effect observed at the most local extents. The largest influence of landscape structural factors was at local extents (1–5 km) and restricted to specific landscape types (agricultural and urban). This was also an indication that factors pertaining to species richness with the most influence on prevalence patterns were most intense at close proximity (Table 2). The low passerine species richness and correspondingly low infection prevalence in urban areas (Figs. 2, 3) suggested that most sampled passerine species either do not adapt

well to urban environments; or that predominantly infected birds (from the Ploceidae) are not the dominant bird species in urban wetlands. Wetlands in urban landscapes had similarly lowest values for passerine species richness—further indicating that many wetland passerines avoid urban landscapes (as do many other species; e.g., see McKinney 2002; Bradley et al. 2008). An alternative explanation is that the encounter rate in urban landscapes between passerine hosts and malarial pathogens is much lower than in other landscapes due to a lack of infectious vectors and/or susceptible hosts (Roche et al. 2012). Host adaptability to the landscape can have similarly significant implications for host viability and disease prevalence. Piersma (1997) postulated that some avian species are restricted to parasite-poor habitats as a trade-off with investing in immunocompetence, resulting in some species spending much more energy migrating to parasite-poor habitats in order to avoid infection. This hypothesis prompts several considerations: for instance, are migratory birds less prevalently infected than resident species? Do urban landscapes qualify as ‘parasite-poor’ habitats in the avian malaria model? There is also the possibility that birds act as sources of disease, as was the case in Chicago, USA, where migratory birds were the main hosts promoting the invasion of ticks (including non-endemic species) into the city (Hamer et al. 2012).

Patterns of disease across a landscape are often the consequence of spatially explicit and interlinked factors that respond similarly to changes in landscape structure (Patz et al. 2000). The response of pathogens and hosts to changes in the landscape may vary widely. Our findings contrast with those from a West African study on avian malaria, which reported a higher prevalence of avian malaria in undisturbed habitats compared to modified (deforested) habitats (Chasar et al. 2009). In our study, undisturbed habitats (fynbos) had lower infection prevalence than agriculturally modified habitats (agricultural and viticultural). Our findings also contrast with several other related studies, where the prevalence of a variety of diseases in wildlife populations increased with, or was promoted by, urban land development (Singh et al. 2001; Farnsworth et al. 2005). On the other hand, a human malaria study found a lower prevalence in urban areas, which concurred with outcomes seen in this study (Omumbo et al. 2005). Our overall impression is therefore that landcover can act as either a

positive and negative influence on disease, with its exact role being co-dependent upon a diversity of mechanisms in host and vector ecology (Patz et al. 2000).

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