Population regulation of territorial species: both site dependence and interference mechanisms matter

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Spatial patterns of site occupancy are commonly driven by habitat heterogeneity and are thought to shape population dynamics through a site-dependent regulatory mechanism. When examining this, however, most studies have only focused on a single vital rate (reproduction), and little is known about how space effectively contributes to the regulation of population dynamics. We investigated the underlying mechanisms driving density-dependent processes in vital rates in a Mauritius kestrel population where almost every individual was monitored. Different mechanisms acted on different vital rates, with breeding success regulated by site dependence (differential use of space) and juvenile survival by interference (density-dependent competition for resources). Although territorial species are frequently assumed to be regulated through site dependence, we show that interference was the key regulatory mechanism in this population. Our integrated approach demonstrates that the presence of spatial processes regarding one trait does not mean that they necessarily play an important role in regulating population growth, and demonstrates the complexity of the regulatory process.

Keywords: density dependence; interference hypothesis; matrix population model; Mauritius kestrel Falco punctatus; population regulation mechanism; site dependence hypothesis

1. INTRODUCTION

How populations are regulated is a fundamental theme in ecology as it contributes to the understanding of population dynamics and persistence in response to environmental conditions. A regulatory process involves some negative feedback mechanism that decreases vital rates as population size grows, and vice versa, leading to a long-term stationary probability distribution of population densities [1]. Density dependence is thought to be a key process controlling population dynamics [1–5], but the underlying mechanisms are poorly understood and difficult to assess in the wild [1,6].

Two main mechanisms driving density-dependent population regulation have been proposed: site dependence and interference. Rodenhouse et al. [7] proposed a site-dependent mechanism of regulation based on the pre-emptive selection of habitat that differs in suitability (i.e. quality) for reproduction and/or survival. To optimize their fitness, individuals should preferentially occupy sites of higher quality and prevent other individuals from settling at the same sites. As a result, any increase in population size will inevitably lead to the progressive use of lower-quality habitat. At the population level, this is predicted to lead to a decline in per capita vital rates as the population grows. At the site level, constant vital rates are expected under this hypothesis as density should not directly affect site or individual quality (i.e. reduce the per capita amount and quality of resources available for survival and reproduction [7]). These patterns have been identified in a number of studies, and site dependence, through differential use of space, is thought to be a widespread regulatory mechanism in territorial species [7–10]. Alternatively, any decrease in vital rates at the site level with increasing population size would be indicative of an ‘interference’ mechanism (interference hypothesis [1–3,8,11,12]). Here, interference does not refer to the direct physical exclusion by conspecifics, but to density-dependent competition for resources (e.g. increased aggressiveness between social group members [13] interfering with foraging, survival or reproduction; territory compression [14,15]), or adverse conditions induced by higher density (e.g. increased parasitic load [16]). Interference may either affect vital rates directly (e.g. through reduced resources) or indirectly (through a decrease in individual quality).

Spatial patterns of site occupancy in relation to habitat heterogeneity are widespread in vertebrate populations, but we have little evidence about the extent to which spatial processes, through a site-dependent mechanism, actually regulate population growth in the wild. This is because most studies have only focused on single vital rates, particularly reproductive parameters [8–17], and have typically not explored the role of spatial mechanisms.
in a population-dynamics context. Survival, however, may contribute profoundly to population growth rate as it frequently outweighs the contribution of reproduction, particularly in long-lived species [18,19], and variability in juvenile survival may play an important role in population dynamics [20,21]. Therefore, there is a need to consider both reproductive and survival parameters to obtain a full understanding of the mechanisms involved in population regulation and to assess the contribution of the mechanisms involving these vital rates to population growth.

Territorial species are typically tightly regulated, and thus we often only have data for populations around a demographic equilibrium, which makes density dependence particularly hard to detect [22]. In the Mauritius kestrel (*Falco punctatus*), detailed long-term monitoring of the population following a reintroduction programme allows us to study reproduction and survival over a large range of population sizes. The current population fluctuates between 40 and 44 pairs, with around half of the range of population sizes. The current fluctuation between 40 and 44 pairs, with around half of the available breeding sites unoccupied in a given year [23,24], which provides a good opportunity to investigate processes acting on population regulation [25]. Previous work has demonstrated some evidence of site dependence in recruitment in this population, although this is modified by dispersal [26]. However, recruitment is the product of two key vital rates—breeding success and juvenile survival—and in order to understand the impact of spatial processes on each trait, they must be examined separately.

In this study, we test for: (i) the presence or absence of density dependence in breeding success and juvenile survival; (ii) the regulatory mechanisms in each trait acting at the site level (i.e. site dependence hypothesis versus interference hypothesis); and (iii) the contribution of these mechanisms to the regulation of population dynamics. We demonstrate that the presence of a site-dependent mechanism does not necessarily imply that spatial processes play an important role in regulating population growth.

2. MATERIAL AND METHODS

(a) *Species, study area and data collection*

The Mauritius kestrel is a small falcon endemic to the Indian Ocean island of Mauritius. Mauritius kestrels are territorial, and the home range size during the breeding season is about 1 km² [27], with adults defending only the immediate area around the nest site. Kestrels typically form monogamous pairs and their breeding season spans the Southern Hemisphere summer. The earliest eggs (clutch size 2–5) are laid in early September and the latest fledglings (brood size 1–4) leave the nest in late February. Fledglings do not usually stay at their natal site after independence and radiotracking data has shown that they tend to disperse into specific areas of high-quality habitat [28].

The study was conducted on a population that was reintroduced into the Bambous Mountains (57°42′ E, 20°20′ S) on the east coast of Mauritius in the 1987/1988 breeding season [27]. The restoration programme involved the release of 46 captive-produced kestrels at six sites in the centre of the study area from 1987/1988 to 1989/1990, plus some additional management until 1994/1995 (see [27] for details). The population increased rapidly from one breeding pair in 1988/1989 up to a stable level of 40–44 breeding pairs since the early 2000s. The study area supports 80 known breeding sites (sites where at least one breeding pair has been recorded), including both nest-boxes (50 sites) and natural cliff and tree cavities (30 sites). The study area covers 163 km², encompassing a predominantly forested mountainous area buffered by agricultural land (almost exclusively sugar cane). The surrounding agricultural land coupled with the relatively short dispersal distance observed [27,28] ensures that at present this is a closed population remaining isolated from two other kestrel populations in Mauritius.

Since the initial reintroduction, the population has been intensively monitored (see [29] for details). Each breeding season territorial pairs were identified through a unique combination of colour rings and a numbered aluminium ring, and their breeding attempts were monitored to establish the number of young fledged. Individuals were sexed based on field observations of breeding pairs and in the nest based on biometrics [29]. All released individuals and over 93 per cent of the wild-bred fledglings were individually marked. For this study, we use data collected from 1988/1989 to 2007/2008.

(b) *Definition of variables*

Density dependence is defined by the relationship between a vital rate and population size. As the probability of detecting a breeding individual in the study area was high (see capture probability derived from the capture-recapture model in §3) we used the number of breeding pairs as a proxy of population size.

One prerequisite for the site-dependent regulation hypothesis is heterogeneity in habitat quality [7]. A previous study on our kestrel population found considerable variation in the production of recruits between sites, showing differences in site quality (SQ) across the study area [26]. Here, we describe habitat heterogeneity by an index of SQ that was estimated independently of vital rates to avoid circularity in our analysis (but which was linked to variation in breeding success; see §3). This index is based on site occupancy, which measures long-term site preference and is generally considered as a relevant indicator of food availability and predation risk [17,30]. Because kestrels have been released at six sites from which the population has expanded, and because of their restricted dispersal behaviour [26,27], we accounted for the location of the release sites in the estimation of the index. Residuals from the significant regression between the number of years a site has been occupied by a breeding pair and the distance to the nearest release site were used to describe the propensity of a site being occupied, given its distance to release sites. A positive value indicates that a site was more frequently occupied than the average at this distance from the release sites, which we assumed to be attributed to the high quality of this site, and *vice versa*. This index is constant over the whole study period.

(c) *Data analysis*

(i) *Population-level density dependence in vital rates*

We aimed to establish the presence and functional form of density dependence in breeding success and juvenile survival. As the reproductive trait, we analysed breeding success, which we defined as the annual number of fledglings produced per breeding pair. We only considered unmanaged clutches (*n* = 511 breeding events) to avoid a bias owing to manipulated costs of reproduction during the reintroduction

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Density-dependent juvenile survival was modelled as a linear relationship, or as a threshold with a plateau below and a linear trend in juvenile survival above a population size of 25 breeding pairs (according to Nicoll et al. [29], this particular threshold supported the best fit to the data over the period 1987/1988–2002/2003). The proportion of variability explained by a covariate is given by $R^2$, as described by Grosbois et al. [37]. Models were run with program Mark [38]. Model selection procedure was based on the AIC corrected for small sample size (QAICc). The model with the smallest QAICc value was selected as the best supported model if the difference of its QAICc to other models (ΔQAICc) was greater than two; otherwise, models with ΔQAICc < 2 were considered to be statistically equivalent [32].

(ii) Regulatory mechanisms

Here, we aimed to test the two hypotheses about the mechanisms driving population-level density dependence in both traits. It has been suggested that site-dependent and interference hypotheses can be distinguished based on a comparison of the mean and coefficient of variation of a trait [8,11]. This method, however, has been criticized as not being appropriate to formally distinguish between density-dependent hypotheses [39]. Therefore, and to provide a more direct assessment of the hypotheses, we chose to explicitly test site-level responses of vital rates to population size, using generalized linear mixed models, as advocated by Carrete et al. [12]. Another advantage of mixed models is the reduction of non-estimated parameters, in case of small sample sizes, because of the simultaneous treatment of all data in analysis.

We applied model selection to distinguish between different hypotheses described by a set of mixed models with site identity as a random effect and different combinations of fixed effects. By separating among- and within-site variation, mixed models provide a straightforward assessment of the response of a trait to an increase in population size. An increasing use of low-quality sites (site dependence) would result in differences in vital rates between sites, whereas a within-site decline in vital rates with increased population size suggests interference, affecting vital rates either directly or indirectly through a decrease in individual quality. As presented in figure 1, this involved five models: a null model M0 (trait ~ 1; which only includes an intercept), where the trait did not vary with SQ; model M1 (trait ~ SQ), where the trait varied according to SQ but also declined in response of a trait to an increase in population size. An over-dispersion coefficient c-hat was used to take into account the remaining lack of fit [34].
conforming to a mixture of site-dependent and interference mechanisms with similar effect of density at all sites; and model M4 (trait ~ SQ + N + SQ × N), where the trait varied according to SQ and declined more rapidly in high- or low-quality sites as population size increased, suggesting a mixture of site-dependent and interference mechanisms with different effects of density between sites.

For breeding success, the contribution of the fixed-effect variables was assessed with the AIC. Because mixed models for capture–recapture data are not yet routinely available in a frequentist framework (but see [40]), we analysed juvenile survival by implementing the five models in a Bayesian framework (see electronic supplementary material, S1). Model selection was done using the deviance information criterion, which is the Bayesian counterpart of the AIC [41].

(iii) Regulation of the population dynamics
We aimed to assess the relative importance of the density-dependent mechanisms in the regulation of our study population. We implemented a three-stage, pre-breeding census matrix population model, derived from Butler et al. [24], to model the influence of density dependence on population dynamics in the ULM program [42]. The first stage comprised 1-year-old pre-breeding individuals (pb), the second comprised 1-year-old breeders (b1) and the third was breeders aged 2 and older (b2). The population size at time \( t + 1 \) is defined by \( N(t + 1) = A \cdot N(t) \), with \( N(t) \) representing the population vector at time \( t \) and \( A \) the population matrix, such that:

\[
N(t) = \begin{bmatrix} n_{pb} \\ n_{b1} \\ n_{b2} \end{bmatrix},
\]

and

\[
A(t) = \begin{bmatrix} 0 & 1/2.F1.S_{a}(1 - R_{b1}) & 1/2.F2.S_{a}(1 - R_{b1}) \\ 0 & 1/2.F1.S_{a}R_{b1} & 1/2.F2.S_{a}R_{b1} \\ S_{a} & S_{a} & S_{a} \end{bmatrix},
\]

where \( n_{pb} \), \( n_{b1} \) and \( n_{b2} \) are the number of individuals in each stage, \( F1 \) and \( F2 \) the fecundity (defined as the breeding success) in stages \( b1 \) and \( b2 \), \( S_{a} \) and \( S_{a} \) the juvenile and adult survival, and \( R_{b1} \) the recruitment to the breeding population at age 1 (see electronic supplementary material, S2 for details).

3. RESULTS
(a) Population-level density dependence in vital rates
Both breeding success and juvenile survival declined with increasing population size. This decrease in breeding success was only visible after accounting for the age of breeders as a two-age-class factor. We detected a decline in the number of fledglings produced by a given age class as the population size increased (models breeding success ~ a versus breeding success ~ a + N: \( \Delta AIC = 2.90 \); figure 2) over the period 1991/1992–2007/2008, excluding the breeding seasons 1989/1990 and 1990/1991 when data for only two unmanaged pairs were available (population range: 17–44 breeding pairs). The number of fledglings produced per breeding attempt dropped by 26.1 per cent as the population size rose from 20 to 40 pairs.

For juvenile survival, the initial selection procedure supported \( p(a) S(a) \) as the best model, where both capture and survival probabilities were greater in adults than in juveniles but did not vary between sexes or over time (capture: 0.556, s.e. = 0.031 and 0.852, s.e. = 0.015; survival: 0.489, s.e. = 0.023 and 0.767, s.e. = 0.014; for juveniles and adults, respectively). This model constituted our starting point to investigate the influence of population size on juvenile survival. Including population size in the model, either as a linear relationship or a threshold response (see §2), improved the fit to the data \( p(a) S(a) \) versus \( p(a) S(a1 \times N + a2) : \Delta AIC = -1.96 \); \( p(a) S(a) \) versus \( p(a) S(a1 \times \text{plateau}_{<25}) N_{>25} + a2) : \Delta AIC = -3.87 \), supporting a significant decrease in juvenile survival as population size increased. The model with the threshold response explained a greater proportion of the variability in juvenile survival \( (R^2 = 0.29 \); figure 3) than the linear model \( (R^2 = 0.19 \); see electronic supplementary material, S3 for details).

(b) Regulatory mechanisms
Site-level breeding success differed between sites according to SQ, supporting the site-dependent hypothesis, which attributes a decline in breeding success at high density to the increasing use of poor sites. We found that the average quality index of the occupied sites declined as the population grew (GLM: \( z = -3.562, p < 0.001 \)), from an index of 5.415 (s.e. = 0.997, n = 16) at population sizes below 10 breeding pairs to an index of 2.592 (s.e. = 0.257, n = 276) when population size exceeded 40 breeding pairs. This effect occurred primarily owing to site-level differences in breeding success among 1-year-old individuals. For these, breeding success was independent of population size and increased with SQ from 0.499 (s.e. = 0.500) to 1.374 (s.e. = 0.213) fledglings produced at low- (index = 5) and high-quality sites (index = 5), respectively (table 1a, model M1; see also figure 1). In contrast, this effect was
Figure 3. Density-dependent decline of juvenile survival at the population level in the Mauritius kestrel. Inter-annual fluctuations in juvenile survival (filled dots) and estimated relationship between juvenile survival and population size (solid line) represented by the model with a plateau: $p(a) = S(a_1 \times (\text{plateau} < 25), N_{>25}) + a_2$ (model 10, electronic supplementary material S3) over the period 1988/1989–2006/2007.

Table 1. Modelling site-specific breeding success as a function of site quality and population size in the Mauritius kestrel: test for site dependence and interference hypotheses. All models include site identity as a random effect and a Poisson distribution of errors. AIC is the Akaike’s information criterion and $\Delta$AIC is the difference of the AIC between the best and the current model. Model numbers refer to the different regulatory models in figure 1 (see text for more details). BS, breeding success; SQ, site quality (which refers to an index based on long-term site occupancy; see §2 for details); $N_j$, population size.

<table>
<thead>
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<th>model</th>
<th>model structure</th>
<th>AIC</th>
<th>$\Delta$AIC</th>
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<td>(a) 1-year-old breeders</td>
<td></td>
<td></td>
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<tr>
<td>M0</td>
<td>$S_j \sim 1$</td>
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<tr>
<td>M2</td>
<td>$S_j \sim N$</td>
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</tr>
<tr>
<td>M3</td>
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<td>1.95</td>
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<tr>
<td>M4</td>
<td>$S_j \sim SQ + N + SQ \times N$</td>
<td>98.16</td>
<td>3.46</td>
</tr>
<tr>
<td>(b) 2-year-old breeders</td>
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<td></td>
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<tr>
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<tr>
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<td>1.25</td>
</tr>
<tr>
<td>M2</td>
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<td>1.08</td>
</tr>
<tr>
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<tr>
<td>M4</td>
<td>$S_j \sim SQ + N + SQ \times N$</td>
<td>482.97</td>
<td>4.25</td>
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Table 2. Modelling site-specific juvenile survival as a function of site quality and population size in the Mauritius kestrel: test for site-dependent and interference hypotheses. All models include site identity as a random effect. DIC is the deviance information criterion and $\Delta$DIC is the difference of the DIC between the best and the current model. Model numbers refer to the different regulatory models in figure 1 (see text for more details). $S_j$, juvenile survival; SQ, quality (which refers to an index based on long-term site occupancy; see §2 for details); $N_j$, population size.

<table>
<thead>
<tr>
<th>model</th>
<th>model structure</th>
<th>DIC</th>
<th>$\Delta$DIC</th>
</tr>
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<td>136.5</td>
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<tr>
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<td>$S_j \sim SQ + N + SQ \times N$</td>
<td>1929.5</td>
<td>61.55</td>
</tr>
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</table>

Figure 4. Density-dependent regulation mechanisms. M. Nevoux et al. 2177

Density-dependent regulation mechanisms

(c) Regulation of the population dynamics

Density-dependent juvenile survival was a more powerful regulatory process than density-dependent breeding success in this Mauritius kestrel population. The full density-dependent model (i.e. density dependence in breeding success and juvenile survival) produced a stable population size trajectory with 43.4 breeding pairs at equilibrium. This fell within the observed range of population size at stability of 40–44 pairs (figure 4). When density dependence was removed from breeding success, the population size increased slightly (by 4.7%) and stabilized at 45.4 breeding pairs, which was still very close to the observed data. When density dependence was instead removed from juvenile survival, population size increased by 32.4 per cent, stabilizing at 57.4 breeding pairs. In a density-independent model, the trajectory of the simulated population showed an exponential growth, as expected in theoretical situations without regulation.

4. DISCUSSION

In this study, we investigated the underlying mechanisms driving density-dependent processes in vital rates in a Mauritius kestrel population where almost every individual was monitored. Using an explicit site-level approach, we were able to identify differences in the mechanisms acting on different traits, with breeding success regulated by site dependence and juvenile survival by interference. Although territorial species are frequently assumed to...
be regulated through site dependence, we show that interference, associated with density-dependent juvenile survival, was the key regulatory mechanism in this population.

(a) Site heterogeneity and population-level density dependence in vital rates
The index selected to define SQ was a good predictor of the breeding success, reflecting significant heterogeneity between sites. Settlement was pre-emptive and individuals settled preferentially in the best available sites [26]; we can now deduce that the overlap has no impact on the breeding success.

(b) Regulatory mechanisms
Although population-level patterns conformed to the predictions of the site-dependent hypothesis, site-level investigation revealed that different density-dependent mechanisms acted on breeding success and juvenile survival, providing evidence for juvenile survival being regulated by interference. Regulation of breeding success conformed to a site-dependent mechanism as the number of fledglings produced at each site remained constant irrespective of population size. That is to say, the quality of sites or individuals breeding at these sites seemed not to be affected by an increase in the number of breeding individuals. Instead, breeding success differed according to SQ, and because the quality of each site was constant over the study period (see §2), the observed population-level decline is probably explained by an increase in the proportion of sites of low quality occupied at high population size. Site and individual quality may often be confounded as high-quality individuals are commonly found at the best-quality sites [7,43–46]. If individuals of low quality were to be produced at high population size, any new and low-quality sites, thus exacerbating heterogeneity in breeding success between sites. This means that a certain proportion of the observed difference between sites may be attributable to individual quality so that both site and individual quality would contribute to limit breeding success at high population size.

Our finding of site-dependent regulation of breeding success is in accordance with the results of most studies that explicitly compared site-dependent and interference hypotheses in reproductive parameters [7,9–11]. This mechanism seems to be widespread in numerous species conforming to a site-dependent distribution pattern, whereby individuals settle preferentially in sites with high food availability and low predation risk [10,43–45]. Good sites seem to provide enough resources to allow reduced territory size [46], which would explain why they can be occupied at high density and maintain successful reproduction without negative interference from the neighbourhood. In the Mauritius kestrel, territorality is restricted to a reduced area around the nest site, with foraging areas often shared by several individuals [27]. Our results suggest that this overlap has no impact on the breeding success.

In contrast to breeding success, site-level juvenile survival did not differ between sites of different quality but declined at high population size, suggesting that this trait was rather shaped by an interference mechanism. This result is in accordance with the impact of population size on the site-level production of recruits previously detected in this population [26]; we can now deduce that this effect was primarily mediated by post-fledgling survival. Although habitat conditions experienced at the natal site might influence survival of fledglings [47–49], juvenile survival appeared here to be strongly influenced by conditions encountered after juveniles left their natal site. In common with other territorial species [50,51], Mauritius kestrel fledglings leave their natal site after independence and fledglings appeared to gather in an area known as a high-quality patch of native forest [28]. Such behaviour may lead to increased pressure on good habitat at the end of each breeding season, where high local density and the associated interference (i.e. competition) may limit the resources available to juveniles.

It has also been shown in other territorial species that are generally thought to be regulated by site dependence that interference effects are important for population dynamics. In the Seychelles magpie robin (Copsychus sechellarum), it has been demonstrated that interference through territorial disputes increases with increasing density and has the potential to reduce fitness [13]. Similarly, interference effects owing to both territory shrinkage and territorial disputes on reproductive success were
crucial for the population dynamics of the Seychelles warbler \textit{(Acrocephalus sechellensis)} [15]. In the goshawk \textit{(Accipiter gentilis)}, density-dependent breeding success was attributed to the site-dependent mechanism; however, an interaction between weather conditions and density also explained a significant proportion of the population growth rate [9]. Although not formally tested, the authors suggest that weather conditions might relate to interference acting on survival of non-territorial juveniles. This study, therefore, provides another example of the duality between site dependence and interference effects on vital rates.

\textbf{(c) Regulation of the population dynamics}

Density dependence appeared to act through multiple mechanisms, and using a matrix population model we show that density dependence in juvenile survival via interference was the key regulatory process. Survival (juvenile or adult) is an important life-history parameter affecting population growth rate in many birds and mammals \cite{9,11,13,15,18}. Thus, our results emphasize the value of considering survival in addition to reproductive parameters when investigating population regulation. Given the high sensitivity of population growth to changes in juvenile survival in birds and mammals, mechanisms associated with density-dependent survival might be important in many populations.

Although our population presented a pattern of site occupancy characteristic of the site-dependent hypothesis \cite{7}, spatial processes were not the main driver of population dynamics. Interference-based density dependence also appears to be important in other territorial species \cite{above}, and may be more common than initially thought. In the Mauritius kestrel, the interference density-dependent mechanism seemed to act outside the breeding sites. Such a mechanism could therefore be easily overlooked by most studies that usually only monitor individuals at their breeding sites. Because many territorial species only retain and defend a territory for a certain period of the year, centred on the breeding season, interaction processes outside breeding sites may be more common than currently anticipated. More generally, in territorial species, both site dependence and interference mechanisms are probably involved simultaneously in the regulation of population dynamics, as indicated by this and other studies \cite{13,15}.

Misleading conclusions about the key driver of population dynamics may have non-trivial consequences in terms of habitat use and management guidance. In a system regulated by site dependence, improving the quality of breeding sites should allow population size recovery \cite{9,11}. In contrast, if interference mechanisms regulate population size, restoration measures should focus on improving poor habitat patches to reduce heterogeneity in habitat quality and decrease conflict pressure, as recommended for the Seychelles magpie robin \cite{13}. Although increasing the number of sites (nest-boxes) in the best habitat patches might increase the average breeding success of the Mauritius kestrel, this study suggests that such management would have little impact on the population growth rate if not coupled with action at a larger scale to expand high-quality habitat in order to reduce interference competition among juveniles.

In conclusion, we emphasize three main points. First, mechanisms shaping population dynamics are complex and can depend on the trait considered. Most studies investigating density-dependent regulation have focused on reproductive traits and, as in this study, found evidence for the site-dependent mechanism operating through spatial processes. However, by studying survival, we have provided evidence for the interference mechanism, which proved to be the more powerful of the two regulatory mechanisms identified in our study population. Thus, it seems crucial to study all vital rates that are likely to have an important impact on population growth before drawing conclusions about how a population is regulated. Second, understanding which mechanisms regulate a population is important for explaining species abundances, predicting the success of biological controls and designing management plans for species conservation. The relative contribution of site-dependent or interference mechanisms to population regulation will affect a population’s response to environmental conditions and the rate at which it returns to equilibrium after disturbance \cite{see also}. Third, this study also demonstrates that even though the assumptions underlying site-dependent patterns of site occupancy are met \cite{7}, this does not necessarily mean that the population is strongly regulated by spatial mechanisms. As a result, we suggest that the importance of interference for territorial species may in fact be underestimated.

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