LANDSCAPE ECOLOGY IN REVIEW

Integrating landscape ecology and conservation physiology

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Abstract The need to understand how anthropogenic landscape alteration affects fauna populations has never been more pressing. The importance of developing an understanding of the processes behind local extinction is widely acknowledged, but inference from spatial patterns of fauna distribution continues to dominate. However, this approach is limited in its ability to generate strong predictions about future distributions and local extinctions, especially when population-level responses to landscape alteration are subject to long time lags. We review the potential for indices of physiological stress and condition to contribute to understanding of how landscape pattern affects species persistence. Such measures can indicate habitat quality from the perspective of the individual animal, and can reveal environmental stressors before their negative consequences begin to manifest at a population level. Spatial patterns of chronic stress may therefore yield valuable insight into how landscape alteration influences species. We propose that the emerging disciplines of conservation physiology and macrophysiology have much to offer spatial ecology, and have great potential to reveal the physiological pathways through which habitat alteration affects fauna populations and their persistence in fragmented landscapes.

Keywords Body condition · Chronic stress · Habitat fragmentation · Habitat quality · Macrophysiology · Physiological indicators · Spatial ecology

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Introduction

Understanding how faunal populations respond to landscape spatial pattern is an urgent problem in the face of anthropogenically-driven habitat fragmentation and degradation. Much current research relies on relating species distribution to landscape patterns (Mazerolle and Villard 1999; Elith and Leathwick 2009). More recently, there has been increasing focus on exploring the key processes behind distributional patterns—shifts in the strength of interspecific interactions such as predation, parasitism and interspecific competition (Chalfoun et al. 2002; Benson et al. 2010; Maron et al. 2011); impeded mobility at daily, generational and genetic timescales (Baguette and Van Dyck 2007; Shanahan et al. 2010) and changed patterns of availability of key resources (Watson

2011). Development of such understanding relies on shifting focus from patterns of species distribution towards attempting to characterise better the chain of cause and effect which links landscape change to population responses.

While information about species' distributions (i.e., presence/absence, abundance, species richness) is often straightforward to collect with minimal equipment or expertise and has contributed significantly to landscape ecological knowledge, there are limitations to the type of inferences that can be made from such data. Firstly, the deleterious effects of anthropogenic landscape alteration on a population can only be detected using this approach once the population has begun to decline. Secondly, the trajectory of faunal relaxation following fragmentation is uncertain, with predictions complicated by several factors such as a lack of pre-fragmentation data (MacHunter et al. 2006; Ford et al. 2009), and so examining a pattern of distribution cannot tell us whether extinction debts in a landscape have been fully paid.

The effects of fragmentation on populations start with effects on individuals. There are multiple processes through which fragmentation and other forms of landscape alteration might affect individuals. Reducing the area of contiguous habitat below a species' home-range size reduces resource availability, meaning individuals must either increase energy expenditure and employ a strategy of multiple patch use, or suffer consequences of resource limitation (Hinsley 2000). Dispersal between patches becomes more hazardous and infrequent as remnants become more isolated and the matrix more hostile, increasing the risks and costs to the individual (Baguette and Van Dyck 2007). Additionally, habitat quality declines, with increased edge to interior ratios mediating changes in resource density, increased exposure to extra-patch abiotic conditions and elevated risk of deleterious interspecific interactions such as predation (Burke and Nol 1998; Hinsley 2000). In most of these cases, the impact of these processes is manifest on individuals at a physiological level before negative consequences are expressed and observed as population-level declines or local extinctions.

We argue that developing an understanding of how vertebrates respond physiologically to stressors exacerbated by anthropogenic landscape change therefore holds great potential for a deeper understanding of how such change impacts fauna, both individually and at the population level. For example, while the occupancy of particular habitats (habitat selection) is an indication of habitat preference, it does not necessarily indicate true habitat quality (Van Horne 1983; Johnson 2007; Patten and Kelly 2010). Directly measuring an individual's physiological response to its environment can yield insights into true habitat quality. Yet although many studies outside the field of landscape ecology have investigated the physiological responses of vertebrates, mainly birds, to external stressors such as handling by humans and varied food availability (Kitaysky et al. 1999; Vleck et al. 2000; Mortimer and Lill 2007), relatively few have used the approach to evaluate effects of landscape change such as fragmentation on the health and condition of individuals (Mazerolle and Hobson 2002; Hinam and St. Clair 2008; Janin et al. 2011).

Systematic patterns in physiological parameters measured on individual animals may provide early indicators of populations in trouble before the stress of a disturbance significantly impacts reproduction or other measures of performance (Wingfield et al. 1997, 1998). These indicators can be used to establish baseline data on populations, determine effects of disturbances, understand mechanisms of disease, understand nutritional requirements, monitor changes to populations and guide habitat management and restoration efforts (Carey 2005; Wikelski and Cooke 2006; Cooke and Suski 2008). Consideration of these physiological indicators in a spatial context provides insight into the mechanisms by which habitat loss and fragmentation may result in negative outcomes for faunal populations. Indeed, such approaches are directly aligned with the emerging fields of conservation physiology (Stevenson et al. 2005; Wikelski and Cooke 2006, Cooke and Suski 2008, Cooke and O'Connor 2010) and macrophysiology (Chown et al. 2004; Chown and Gaston 2008; Gaston et al. 2009).

In this review, we aim to: (1) examine recent published studies in landscape ecology to compare the frequency of research into patterns of species' distributions with that of more proximate indicators of process, including physiological stress and body condition; (2) describe the generalised stress response in vertebrates; (3) introduce a conceptual framework outlining potential stressors linked to habitat fragmentation; and (4) explore the potential for indices of stress and condition to enhance our understanding of

how landscape pattern affects the persistence of species.

Landscape ecology research: distribution patterns versus ecological processes

In order to evaluate the prevalence of process-focussed research in landscape ecology, particularly research evaluating animal stress and condition in fragmented landscapes, we conducted a review of empirical studies using Web of Science. We searched for articles published between 2005 and 2011 using the search terms "habitat and fragmentation", "habitat and landscape pattern", and "habitat and spatial pattern", and included only articles and proceedings papers. This search yielded 4,683 results, 950 of which were empirical studies relating fauna (vertebrate and invertebrate) to some measure(s) of habitat. For each of these studies, we classified the response variable(s) as either a distribution-related measure (presence/absence, abundance, species richness) or a process-related measure including the broad categories predation, reproduction, movement, gene flow, and individual condition.

Of the 950 studies, 73% related some measure of vertebrate or invertebrate distribution to habitat and/or landscape factors, while 45% measured more proximate indicators of process. The most commonly investigated processes were gene flow with 136 studies (14%) and movement with 125 studies (13%), followed by reproduction with 71 studies (8%). Only 36 studies (4%) explored links between health, stress, body condition or physiological status of animals, and habitat or landscape factors. Of these 36 studies, most examined morphology or parasite load as an indicator of body condition. The small number of such studies suggests that physical and physiological indicators—particularly indicators of chronic stress—have yet to be widely embraced as indicators of faunal responses to habitat spatial pattern.

Stress in terrestrial vertebrates

Ecologists, drawing on a wealth of veterinary and biomedical literature, are increasingly recognising that the stress responses of animals are correlated with their overall health (Romero 2004; Davis et al. 2008). Measurements of physiological stress responses and/or chronic stress levels in individuals can thus be used to assess the effects of parameters such as habitat quality, anthropogenic disturbances, interspecific interactions, and energy availability on whole populations. The utility of physiological stress indices in revealing the long-term effects of an animal's environment on its health, survival and/or fitness depends on an understanding of the causes and consequences of chronic stress. In this section, we outline the generalised stress response in vertebrates, discuss the consequences of chronic stress, and describe the major sources of stress in wild vertebrates. We end this section by describing commonly measured indicators of chronic stress.

The stress response

A stressor is an external noxious stimulus that activates an acute 'stress response' of physiological and behavioural coping activities (Romero 2004). When an individual experiences a stressor, several neurological and endocrine actions follow in sequence (Fig. 1). In the first phase of the acute stress response, activation of the sympathetic nervous system leads to the release of epinephrine and norepinephrine (i.e., catecholamines) from the adrenal medulla. In the second phase of the acute stress response, neuroendocrine signals are sent from the brain via the hypothalamic–pituitary–adrenal (HPA) axis to the adrenal cortex which releases glucocorticoids (mainly corticosterone or cortisol, depending on the taxonomic group). Increased circulating levels of catecholamines and glucocorticoids in the body trigger a suite of behavioural, hormonal and physiological responses that reduce non-essential activities and help the individual escape from or cope with the stressor, as summarised in Fig. 1 (Wingfield et al. 1997, 1998; Wingfield and Ramenofsky 1999; Wingfield and Kitaysky 2002; McEwen and Wingfield 2003).

Glucocorticoids play several complementary roles in the chronology of a stress response. These roles include: (1) direct stimulation or inhibition of activities to help the individual to cope with the stressor (e.g., increase gluconeogenesis, promote alertness and escape behaviour, regulate immune system function, suppress reproductive behaviour), (2) permissive regulation of other body systems not directly involved

First phase of acute stress response
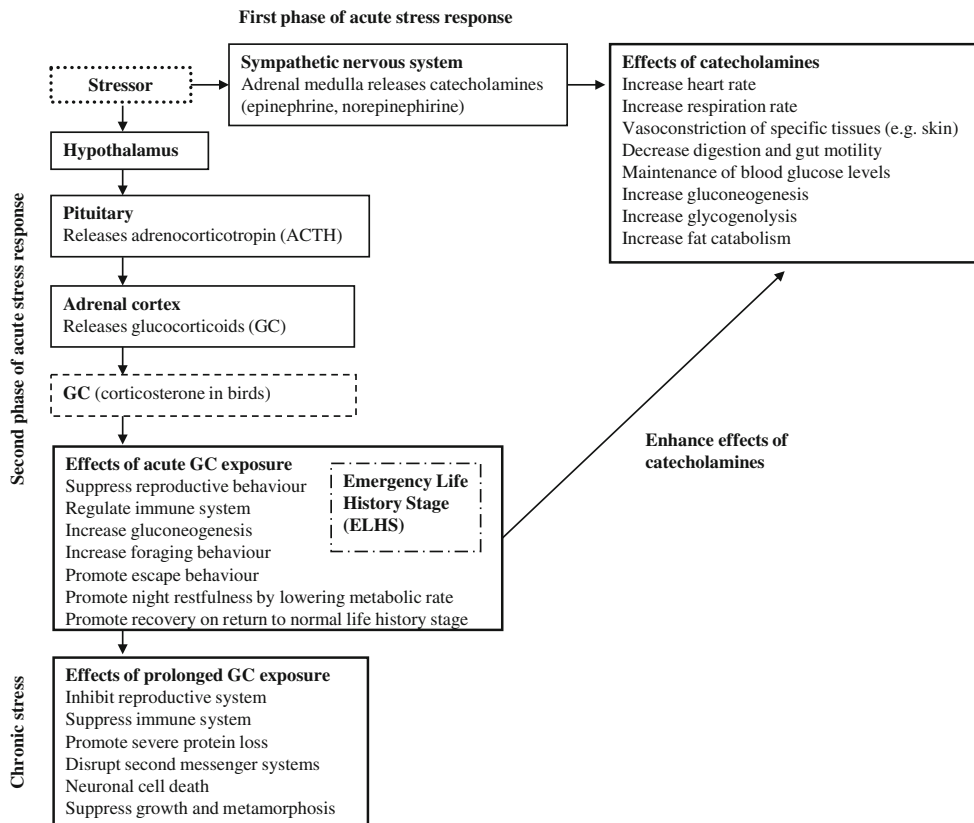
Fig. 1 Mechanisms of stress response and the effects of acute and chronic stress on animals (adapted from Wingfield et al. 1997, 1998; Romero 2004). In the first phase of the acute stress response, activation of the sympathetic nervous system leads to release of catecholamines by the adrenal medulla. In the second phase of the acute stress response, neuroendocrine signals travelling along the hypothalamic–pituitary–adrenal (HPA) axis lead to the release of stress hormones (glucocorticoids) by the adrenal cortex. The acute stress response induces an Emergency Life History Stage (ELHS), which allows the animal to cope with or escape from the stressor(s). Chronic exposure to high levels of glucocorticoids (i.e., chronic stress) can have detrimental effects on physiological function, reproduction and survival

in the acute stress response (e.g., sustain increased cardiovascular function), and (3) termination of the stress response via negative feedback inhibition, recovery and preparation for future stressors (Sapolsky et al. 2000; Romero 2004). This acute Emergency Life History Stage (ELHS), summarised in Fig. 1, helps the individual survive the disturbance, and then return to normal activities such as reproduction and territorial defence once the stressor is gone (Wingfield et al. 1997, 1998; Wingfield and Ramenofsky 1999; Wingfield and Kitaysky 2002; McEwen and Wingfield 2003). However, stressors that are chronically present in the environment may cause overstimulation of these coping activities and lead to detrimental 'chronic stress' (Romero 2004).

Consequences of chronic stress

Prolonged exposure to stressors (e.g., food limitation, anthropogenic disturbance) leading to chronically elevated levels of stress hormones, particularly glucocorticoids, can have substantially detrimental effects on an individual and ultimately lead to its demise (Wingfield et al. 1997, 1998; Wingfield and Ramenofsky 1999; Wingfield and Kitaysky 2002; McEwen and Wingfield 2003). This prolonged state of chronic stress (or 'allostatic overload'; sensu McEwen and Wingfield 2003) can result in depressed immune response, reduced reproductive success, reductions in muscle mass (i.e., protein loss), and suppressed growth (Wingfield et al. 1997, 1998; Wingfield and

Ramenofsky 1999; Wingfield and Kitaysky 2002; McEwen and Wingfield 2003).

For example, an important primary effect of elevated circulating glucocorticoid levels is to increase plasma glucose levels (i.e., the mobilization of energy from fat stores and muscle via gluconeogenesis). This provides the individual with the energy necessary to endure or escape from the stressor (Wingfield et al. 1997; 1998). This mobilization of energy is useful in the short term, but continued depletion of fat stores and protein loss reduces body condition and fitness and may lead to death (Wingfield et al. 1997; 1998; Romero and Wikelski 2001).

Elevated glucocorticoid levels can also suppress reproductive behaviour to allow the animal to survive a stressor (Wingfield et al. 1998; Romero 2004). Studies on pied flycatchers (*Ficedula hypoleuca*) and side-blotched lizards (*Uta stansburiana*) show that increased (implanted exogenous) corticosterone suppresses reproductive behaviour (Wingfield et al. 1998). By avoiding the additional time and energy demands of reproduction, such as egg production or feeding young, this response increases the probability of individual survival and allows the individual to be better prepared to commence reproductive effort when the stressor has passed if it is temporary, such as unpredictable bad weather (Wingfield et al. 1998). However, if a permanent stressor such as habitat loss and fragmentation caused the animal to abandon reproductive effort, it may not recommence reproduction, hence increasing the risk of population decline or local extinction (Wingfield et al. 1997).

Finally, while disease itself can act as a stressor (e.g., Lindström et al. 2005), chronically stressed individuals can also suffer suppressed immune responses and increased susceptibility to disease (Apanius 1998; Wingfield and Ramenofsky 1999; Buchanan 2000; Wingfield and Kitaysky 2002; Lindström et al. 2005; Martin 2009). Immune suppression due to chronic stress (especially when mediated by chronically elevated levels of glucocorticoids) seems to be mainly limited to the humoral or adaptive components, often leaving innate immunity intact or even enhanced (Wingfield and Ramenofsky 1999; Hangalapura et al. 2005; Bourgeon and Raclot 2006; Martin 2009; Stier et al. 2009). Martin (2009) provides an excellent recent review of the complex topic of stress and immunity in wild vertebrates, emphasizing the importance of time scale (i.e., duration of stressor)

in considering effects on immune function. Buchanan (2000) reviews the mechanisms by which glucocorticoids may suppress immunity, and discusses some of the complexities of and debate regarding interpretation of the effects of stress hormones on immune function.

Stressors

Both short- and long-term environmental perturbations can trigger a stress response in wild vertebrates. Variations and disturbances in the natural environment, such as inclement weather and inconsistent food supply, can invoke a stress response. Studies of white-throated sparrows (*Zonotrichia leucophrys*) and dark-eyed juncos (*Junco hyemalis hyemalis*) show that unpredictable snow storms can cause increased secretion of corticosterone (the main stress hormone in birds and reptiles) and steer the bird away from its normal behaviour (Rogers et al. 1993; Wingfield and Ramenofsky 1997). This increase in circulating corticosterone leads to increased foraging behaviour (Rogers et al. 1993). When unfavourable weather conditions cease, corticosterone levels return to normal, bringing the bird out of the ELHS and restoring its normal activities (Rogers et al. 1993; Wingfield et al. 1998).

However, stressors such as prolonged severe weather, interspecific competition, loss of mate, pollution, habitat change or loss, and other prolonged disturbances can all trigger a stress response in free-living vertebrates (Wingfield et al. 1998). These stressors directly harm the individual by reducing food availability, increasing energy demands or restricting access to resources and habitat (e.g., patch isolation) (Wingfield et al. 1998). For example, American redstarts (*Setophaga ruticilla*) wintering in Jamaica segregate into male and female areas, and females were found to have higher corticosterone levels and lower body mass (Marra and Holbertson 1998). Although the increase in corticosterone may have increased the foraging activity of the females, their lower body mass indicated that the increased foraging effort was not adequate to compensate for the stress of habitat segregation, probably because male dominance behaviour forced females to use less than ideal habitat (Marra and Holbertson 1998).

In some cases, reduced food supply can last longer and be more severe than can be endured. In the Galapagos Islands, the El Niño event of 1998 resulted

in a failure of the upwelling of nutrients that normally produces the algae food source for marine iguanas (*Amblyrhynchus cristatus*) (Romero and Wikelski 2001). Affected iguanas had high levels of corticosterone and low body condition, probably due to protein catabolism triggered by corticosterone in an effort to mobilize energy resources (Romero and Wikelski 2001). Iguana populations with elevated corticosterone levels had significantly reduced survivorship (Romero and Wikelski 2001). This example demonstrates the potential for measures of individual condition to predict population-level consequences of environmental perturbations.

Quantifying physiological stress

Common methods of measuring stress in vertebrates are through circulating levels of glucocorticoids in the blood, a count of the leukocyte profiles (ratios of heterophils or neutrophils to lymphocytes) (Gross and Siegel 1983; Wingfield et al. 1998; Davis et al. 2008) or levels of glucocorticoids in the feces (Wasser et al. 1997; Martinez-Mota et al. 2007). Animals experience stress upon capture that is almost immediately measurable through circulating glucocorticoids in the blood, and so direct measurements of glucocorticoid levels must be taken as soon as possible (i.e., within 3–5 min, depending on the species) after an individual is captured (Davis et al. 2008). However, chronically elevated levels of circulating glucocorticoids alter the leukocyte profiles, increasing the number of heterophils (birds and reptiles) or neutrophils (mammals and amphibians), and decreasing the number of lymphocytes. Thus, the ratio of heterophils or neutrophils to lymphocytes has increasingly been used as a measure of chronic stress (Gross and Siegel 1983; Vleck et al. 2000; Davis et al. 2008). This ratio appears to be a more reliable indicator of chronic stress because the ratio does not significantly increase immediately upon capture (Davis 2005), whereas circulating levels of glucocorticoids in the blood are a better indicator of short term stress responses (Gross and Siegel 1983; Vleck et al. 2000; Mortimer and Lill 2007). The H/L or N/L ratio has been found to be a useful predictor of both individual and population responses to long-term stressors in most vertebrate groups (Davis et al. 2008).

Heat-shock proteins (HSPs, also more recently known as "stress proteins") are produced by cells in response to a variety of stressors, and may act to prevent or reverse protein damage (reviewed by Buchanan 2000). HSP responses (and the regulation thereof) are complex and a subject of much current research, but they do appear to represent a reliable alternative for quantifying stress in wild vertebrates (Merino et al. 1998). Other measures linked to chronic stress include lowered hematocrit and haemoglobin concentrations, and poor body condition (Fowles et al. 1993; Wingfield and Kitaysky 2002; Lindström et al. 2005).

Habitat fragmentation and stress

In the context of landscape ecology, the measurements described above can be used to reveal stress responses in vertebrates linked to landscape change. These measurements can thus indicate variation in habitat quality within areas currently occupied by a population. Systematic spatial patterns of chronic stress may therefore allow prediction of the likelihood of species persistence and/or be used to guide conservation and habitat restoration efforts. For example, if stress is chronically elevated in populations occupying habitat fragments below a certain size threshold, then landscape restoration efforts can prioritise increasing patch areas beyond this threshold, potentially preventing the loss of local populations from such patches. Janin et al. (2011) and Maron et al. (unpublished data) found that physiological state indicators, but not patterns of occurrence, revealed aspects of landscape configuration which negatively affected their focal species (common toads *Bufo bufo* in France and eastern yellow robins *Eopsaltria australis* in Australia, respectively) and were thus able to provide recommendations for restoration before local populations declined. In this section we introduce a simple conceptual framework outlining the major ultimate and proximate stressors linked to habitat fragmentation, and how they may impact on animals living in a fragmented landscape.

Understanding the chain of cause and effect which links landscape alteration to population-level responses is crucial for identifying effective approaches for arresting population declines. Habitat loss (actual loss in amount of habitat), fragmentation (breaking up of habitat) and degradation (reduced habitat condition) refer to different and distinct processes, but also act concurrently and not independently of one another (Ford et al. 2001; Fahrig 2003). These processes

negatively affect multiple aspects of habitat quality, and individuals exposed to such reduced habitat quality are subject to several types of stressors (Fig. 2), which we describe below.

Food limitation

Two of the major negative consequences of fragmentation are reduced patch size and increased edge extent (Fahrig 2003). In both cases, a significant mediator of negative effects on animals can be reduced availability of resources which can be an important stressor. In particular, resources required on a daily basis, such as food, may become limiting for individuals restricted to a small area of habitat (Robinson 1998). Ovenbirds (*Seiurus aurocapillus*) and eastern yellow robins are sensitive to patch size because smaller habitat fragments have a lower abundance of insect prey (Burke and Nol 1998; Zanette et al. 2000). Similarly, abiotic and biotic changes at edges can alter habitat quality, reducing food availability. Insects may be more susceptible to desiccation or less productive at patch edges, creating further problems for insectivorous birds (Wenny et al. 1993; Zanette et al. 2000). Individuals exposed to reduced food availability over a long period, such as confronts individuals occupying small or poor-quality habitat patches, experience chronically elevated stress levels and reduced body condition. The availability of food is a factor which is often challenging to measure in a way that reflects the individual's perception (Hutto 1990), and so it is also difficult to identify patches with insufficient resources until a local extinction or population reduction is evident. Even then, it is hard to attribute population changes to food limitation rather than stochastic

events. Measurement of chronic stress and body condition of individuals in potentially resource-limited patches can, however, indicate whether their inhabitants are normal and healthy or energy-stressed.

Much understanding of the physiological consequences of habitat fragmentation comes from research on bird populations. Exploration of the body condition of ovenbirds on their breeding grounds identified that food supply is the mechanism behind their sensitivity to fragmentation (Burke and Nol 1998). Larger patches had higher food abundance and more suitable nest sites away from the patch edge (Burke and Nol 1998). Ovenbird body condition on their tropical wintering grounds has also been shown experimentally to be positively associated with food availability (Strong and Sherry 2000; Brown and Sherry 2006), and male ovenbirds in continuous forest are in better condition and have lower chronic stress than males in forest fragments (Mazerolle and Hobson 2002). Although this pattern may also emerge due to better-quality males dominating the best habitat, the fitness consequences of this pattern are potentially significant as male pairing success is reduced in poor-quality sites (Burke and Nol 1998; Mazerolle and Hobson 2002). The basis of the Eurasian treecreeper's (*Certhia familiaris*) sensitivity to habitat loss and fragmentation also appears to be decreased food abundance (spiders and other invertebrates) associated with lower forest cover and habitat quality (Suorsa et al. 2003). Suorsa et al. (2004) manipulated brood sizes of treecreepers in nest boxes by taking one nestling from a brood (reduced brood) and adding it to another (enlarged brood). Nestlings in both enlarged or control broods in smaller patches had higher H/L ratios and

Fig. 2 The main drivers of changes in habitat quality as a result of habitat fragmentation and the pathways through which they result in stressors affecting animals (adapted from Wingfield et al. 1998; Ford et al. 2001)
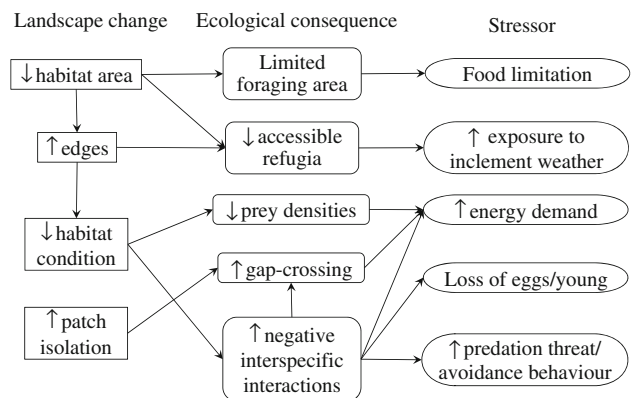
poorer body condition than nestlings in larger patches (Suorsa et al. 2004).

Male northern spotted owls (*Strix occidentalis caurina*) living in close proximity to logging roads or areas that had been recently logged have higher levels of fecal corticosterone than males living further away from such disturbances (Wasser et al. 1997). Northern saw-whet owls (*Aegolius acadicus*) in highly fragmented landscapes with low forest cover have smaller home ranges, higher levels of chronic stress as measured by H/L ratios, lower rates of provisioning to their nestlings and lower reproductive success (Hinam and St. Clair 2008). This lower overall success as landscape cover decreases may be an early warning signal that this owl population is in decline (Hinam and St. Clair 2008).

Physiological consequences of habitat loss and fragmentation are also evident in black howler monkeys (*Alouatta pigra*), with elevated levels of fecal cortisol (the main glucocorticosteroid in mammals) in monkeys living in forest fragments compared to those living in continuous forest (Martinez-Mota et al. 2007). Fragmentation of the forest reduces food quality and availability, and a less-extensive canopy also forces monkeys to forage on the ground, leaving them more susceptible to predation by dogs or disturbances by humans and presumably more stressed (Martinez-Mota et al. 2007). Similarly, spider monkeys (*Ateles geoffroyi yucatanensis*) in fragmented habitats have higher fecal cortisol than those in more continuous habitats (Rangel-Negrin et al. 2009).

Exposure to inclement weather

Poor weather conditions, while not necessarily associated with habitat condition, may also act as a stressor. Following a significant snowfall, dark-eyed juncos were found to have elevated levels of corticosterone and increased fat deposits (Rogers et al. 1993). The elevated corticosterone levels may have caused an increase in foraging behaviour, accounting for the greater fat deposits (Rogers et al. 1993), and serves as an example of how species can enter an Emergency Life History Stage to overcome temporary stressors (Wingfield and Ramenofsky 1997; Wingfield et al. 1998). However, if individuals are unable to access appropriate refugia due to habitat degradation or pervasive edge effects, exposure to inclement weather and weather extremes may act as persistent sources of environmental stress.

Increased energy demands

Living in a fragmented habitat is costly for animals because of the increased energy demands of movements on a daily or seasonal basis. More mobile taxa such as birds are often able to use more than one patch of habitat if they live in a fragmented area, but this generally requires crossing open gaps between patches, resulting in time lost to travel that could have been spent foraging and provisioning nestlings (Hinsley 2000). Increased travel distances for food may result in fewer trips, less provisioning, and reduced nestling success (Suorsa et al. 2004; Hinam and St. Clair 2008; Hinsley et al. 2008). Eurasian treecreepers generally forage within 200 m of the nest, but if nests are located in small patches, parents may need to visit other patches in order to acquire sufficient food (Suorsa et al. 2004). Male northern saw-whet owls in areas of low forest cover provisioned their nestlings less as the distance to the nearest patch increased (Hinam and St. Clair 2008). In addition, crossing gaps between patches increases exposure to factors such as predators, environmental extremes and reduced food supply that may be more prevalent at patch edges or in the matrix (Hinsley 2000). However, the extent to which the matrix is a hazard for an individual depends on the species and the nature of the matrix (Kupfer et al. 2006; Fischer and Lindenmayer 2007).

Limited research has used indicators of chronic stress to explore the costs of multiple patch use, although one study of male spotted salamanders (*Ambystoma maculatum*) living in a forest separated from breeding ponds by a parking lot showed an increase in corticosterone after crossing, suggesting that this fragmentation is a stressor for the salamanders (Newcomb Homan et al. 2003). A separate component of the same study also showed that female salamanders in undisturbed sites exhibited an increase in corticosterone after handling, but those in disturbed sites did not have the same increase, suggesting that individuals in the disturbed site are already chronically stressed and are thus unable to respond to the additional stress from being handled (Newcomb Homan et al. 2003).

Negative interspecific interactions: loss of eggs or young, predation threat and agonistic interactions

Habitat fragmentation increases the amount of edge habitat in a landscape (Fahrig 2003). An important

consequence of increased edge habitat resulting from fragmentation is an increase in the prevalence of negative interspecific interactions, including nest predation and parasitism (Chalfoun et al. 2002). Brood parasitism by the brown-headed cowbird (*Molothrus ater*) in North America and predation by pied currawongs (*Strepera graculina*) in Australia are significant causes of nest failure and are more prevalent at habitat edges (Paton 1994; Debus 2006). Aggressive interactions between species are also a significant source of stress to wild vertebrates. For example, not only do urban rufous-collared sparrows (*Zonotrichia capensis*) exhibit a higher stress response and lower body mass than their rural counterparts because of reduced food availability in urban areas, it has been suggested that increased interactions with aggressive house sparrows (*Passer domesticus*) contribute to increased acute and chronic stress in urban sparrows (Ruiz et al. 2002). Noisy miners (*Manorina melanocephala*) are often associated with fragmented, degraded habitat in Australia, and are known to aggressively exclude other species (Ford et al. 2001; Maron 2007). Their presence is often associated with the lack of other woodland bird species, and this interspecific competition is often suggested as the cause of declines in populations of other passerines (Ford et al. 2001; Maron 2007). Birds persisting in or near such areas are likely to exhibit elevated stress due to encounters with aggressive noisy miners.

Limitations

Despite the generally negative consequences of elevated chronic stress, elevated stress does not always indicate reduced fitness. Individuals may respond to stressors differently depending on their initial physiological condition, life history stage or genetic composition (Wingfield and Kitaysky 2002). Food seasonality and availability can induce a stress response independent of landscape change factors. Spider monkeys within a conserved habitat showed variations in stress responses explained not by landscape cover but by seasonality, with lower cortisol levels during the wet season when food for the frugivorous primate is more abundant (Rangel-Negrin et al. 2009).

Higher stress levels can be a consequence of higher reproductive success because of the increased energetic demands associated with reproduction. Though male northern saw-whet owls in areas of lower forest cover exhibited a higher stress response, males with larger broods also exhibited higher stress levels, presumably because of the additional costs of provisioning a large brood (Hinam and St. Clair 2008). Healthy female tree swallows (*Tachycineta bicolour*) might have initially low levels of corticosterone, and this condition promotes high reproductive effort as evidenced by large clutch sizes (Bonier et al. 2009). After this large clutch hatches, the female may experience increased levels of corticosterone because of the higher than usual demand of caring for a larger brood of chicks (Bonier et al. 2009). This higher level of corticosterone in the female could be misinterpreted as being the result of landscape changes or other stressors that reduce reproductive success, when in fact it is the result of being so successful (Bonier et al. 2009).

Other actions and events in an individual's life such as territorial defence result in costs to the individual. Although male ovenbirds in continuous forest were in better physiological condition than those in forest fragments, males in continuous forest had higher energy demands because of the need to defend territories and reproduce (Mazerolle and Hobson 2002). Therefore, to interpret correctly a finding of elevated stress in wild vertebrates it is important to evaluate and account for factors likely to mediate the effect of stress on fitness, such as reproductive effort, stage of breeding, and food seasonality and availability (Bonier et al. 2009; Rangel-Negrin et al. 2009).

Conclusion

The ubiquity of habitat loss, fragmentation and degradation drives the need for greater understanding of the mechanisms by which these processes affect fauna populations. Currently, relatively few landscape ecologists employ physiological approaches to evaluate habitat quality or identify attributes of landscapes that may act as stressors, which may lead to population decline. Techniques in conservation physiology (Wikelski and Cooke 2006) and macrophysiology (Chown et al. 2004, Chown and Gaston 2008, Gaston et al. 2009) have the potential to reveal populations on the road to extinction before declines occur—knowledge that cannot be gained from exploration of population distribution and abundance alone. Not only can physiology contribute to conservation

biology and landscape ecology, but there is also untapped potential for physiology to be integrated into restoration ecology (Cooke and Suski 2008). The development of 'landscape physiology' research will help reveal early warning signs of populations in trouble, and can play a major role in monitoring and evaluating the success of habitat restoration efforts.

References

Apanius V (1998) Stress and immune defense. Advances in the Study of Behavior 27:133–153

Baguette M, Van Dyck H (2007) Landscape connectivity and animal behavior: functional grain as a key determinant for dispersal. Landscape Ecol 22:1117–1129

Benson TJ, Anich NM, Brown JD, Bednarz JC (2010) Habitat and landscape effects on brood parasitism, nest survival, and fledgling production in Swainson's warblers. J Wildlife Manage 74:81–93

Bonier F, Moore IT, Martin PR, Robertson RJ (2009) The relationship between fitness and baseline glucocorticoids in a passerine bird. Gen Comp Endocrinol 163:208–213

Bourgeon S, Raclot T (2006) Corticosterone selectively decreases humoral immunity in female eiders during incubation. J Exp Biol 209:4957–4965

Brown DR, Sherry TW (2006) Food supply controls the body condition of a migrant bird wintering in the tropics. Oecologia 149:22–32

Buchanan KL (2000) Stress and the evolution of condition-dependent signals. Trends Ecol Evol 15:156–160

Burke DM, Nol E (1998) Influence of food abundance, nest-site habitat, and forest fragmentation on breeding ovenbirds. Auk 115:96–104

Carey C (2005) How physiological methods and concepts can be useful in conservation biology. Integr Comp Biol 45:4–11

Chalfoun AD, Thompson FR, Ratnaswamy JM (2002) Nest predators and fragmentation: a review and meta-analysis. Conserv Biol 16:206–318

Chown SL, Gaston KJ (2008) Macrophysiology for a changing world. Proc R Soc B 275:1469–1478

Chown SL, Gaston KJ, Robinson D (2004) Macrophysiology: large-scale patterns in physiological traits and their ecological implications. Funct Ecol 18:159–167

Cooke SJ, O'Connor CM (2010) Making conservation physiology relevant to policy makers and conservation practitioners. Conserv Lett 3:159–166

Cooke SJ, Suski CD (2008) Ecological restoration and physiology: an overdue integration. Bioscience 58:957–968

Davis AK (2005) Effect of handling time and repeated sampling on avian white blood cell counts. J Field Ornithol 76: 334–338

Davis AK, Maney DL, Maerz JC (2008) The use of leukocyte profiles to measure stress in vertebrates: a review for ecologists. Funct Ecol 22:760–772

Debus SJS (2006) The role of intense nest predation in the decline of scarlet robins and eastern yellow robins in remnant woodland near Armidale, New South Wales. Pacific Conserv Biol 12:279–287

Elith J, Leathwick JR (2009) Species distribution models: ecological explanation and prediction across space and time. Annu Rev Ecol Evol Syst 40:677–697

Fahrig L (2003) Effects of habitat fragmentation on biodiversity. Annu Rev Ecol Evol Syst 34:487–515

Fischer J, Lindenmayer DB (2007) Landscape modification and habitat fragmentation: a synthesis. Global Ecol Biogeogr 16:265–280

Ford HA, Barrett GW, Saunders DA, Recher HF (2001) Why have birds in the woodlands of Southern Australia declined? Biol Conserv 97:71–88

Ford HA, Walters JR, Cooper CB, Debus SJS, Doerr VAJ (2009) Extinction debt or habitat change?: Ongoing losses of woodland birds in north-eastern New South Wales, Australia. Biol Conserv 142:3182–3190

Fowles JR, Fairbrother A, Fix M, Schiller S, Kerkvliet NI (1993) Glucocorticoid effects on natural and humoral immunity in mallards. Dev Comp Immunol 17:165–177

Gaston KJ, Chown SL, Calosi P, Bernardo J, Bilton DT, Clarke A, Clusella-Trullas S, Ghalambor SK, Konarzewski M, Peck LS, Porter WP, Pörtner HO, Rezende EL, Schulte PM, Spicer JI, Stillman JH, Terblanche JS, van Kleunen M (2009) Macrophysiology: a conceptual reunification. Am Nat 174:595–612

Gross WB, Siegel HS (1983) Evaluation of the heterophil/ lymphocyte ratio as a measure of stress in chickens. Avian Dis 27:972–979

Hangalapura BN, Nieuwland MGB, Reilingh GD, Buyse J, van den Brand H, Kemp B, Parmentier HK (2005) Severe feed restriction enhances innate immunity but suppresses cellular immunity in chicken lines divergently selected for antibody response. Poult Sci 84:1520–1529

Hinam HL, St. Clair CC (2008) High levels of habitat loss and fragmentation limit reproductive success by reducing home range size and provisioning rates of Northern saw-whet owls. Biol Conserv 141:524–535

Hinsley S (2000) The costs of multiple patch use by birds. Landscape Ecol 15:765–775

Hinsley SA, Hill RA, Bellamy PA, Harrison NM, Speakman JR, Wilson AK, Ferns PN (2008) Effects of structural and functional habitat gaps on breeding woodland birds: working harder for less. Landscape Ecol 23:615–626

Hutto RL (1990) Measuring the availability of food resources. Studies in Avian Biol 13:20–28

Janin A, Léna J, Joly P (2011) Beyond occurrence: body condition and stress hormone as integrative indicators of habitat availability and fragmentation in the common toad. Biol Conserv 144:1008–1016

Johnson MD (2007) Measuring habitat quality: a review. Condor 109:489–509

Kitaysky AS, Piatt JF, Wingfield JC, Romano M (1999) The adrenocortical stress-response of black-legged kittiwake chicks in relation to dietary restrictions. J Comparative Physiol B 169:303–310

Kupfer JA, Malanson GP, Franklin SB (2006) Not seeing the ocean for the islands: the mediating influence of matrix-based processes on forest fragmentation effects. Global Ecol Biogeogr 15:8–20

Lindström KM, Hawley DM, Davis AK, Wikelski M (2005) Stress responses and disease in three wintering house finch (*Carpodacus mexicanus*) populations along a latitudinal gradient. Gen Comp Endocrinol 143:231–239

MacHunter J, Wright W, Loyn R, Rayment P (2006) Bird declines over 22 years in forest remnants in southeastern Australia: Evidence of faunal relaxation? Canadian J Forest Res 36:2756–2768

Maron M (2007) Threshold effect of eucalypt density on an aggressive avian competitor. Biol Conserv 136:100–107

Maron M, Main A, Bowen M, Howes A, Kath J, Pillette C, McAlpine CA (2011) Relative influence of habitat modification and interspecific competition on woodland bird assemblages in eastern Australia. Emu 111:40–51

Marra PP, Holbertson RL (1998) Corticosterone levels as indicators of habitat quality: effects of habitat segregation in a migratory bird during the non-breeding season. Oecologia 116:284–292

Martin LB (2009) Stress and immunity in wild vertebrates: timing is everything. Gen Comp Endocrinol 163:70–76

Martinez-Mota R, Valdespino C, Sanchez-Ramos MA, Serio-Silva JC (2007) Effects of forest fragmentation on the physiological stress response of black howler monkeys. Anim Conserv 10:374–379

Mazerolle DF, Hobson KA (2002) Physiological ramifications of habitat selection in territorial male ovenbirds: consequences of landscape fragmentation. Oecologia 130:356–363

Mazerolle DF, Villard M (1999) Patch characteristics and landscape context as predictors of species presence and abundance: a review. Ecoscience 6:117–124

McEwen BS, Wingfield JC (2003) The concept of allostasis in biology and biomedicine. Horm Behav 43:2–15

Merino S, Martínez J, Barbosa A, Møller AP, de Lope F, Pérez J, Rodríguez-Caabeiro F (1998) Increase in a heat-shock protein from blood cells in response of nestling house martins (*Delichon urbica*) to parasitism: an experimental approach. Oecologia 116:343–347

Mortimer L, Lill A (2007) Activity-related variation in blood parameters associated with oxygen transport and chronic stress in little penguins. Aust J Zool 55:249–256

Newcomb Homan R, Regosin JV, Rodrigues DM, Reed JM, Windmiller BS, Romero LM (2003) Impacts of varying habitat quality on the physiological stress of spotted salamanders (*Ambystoma maculatum*). Animal Conserv 6:11–18

Paton PWC (1994) The effect of edge on avian nest success: how strong is the evidence? Conserv Biol 8:17–26

Patten MA, Kelly JF (2010) Habitat selection and the perceptual trap. Ecol Appl 20:2148–2156

Rangel-Negrin A, Alfaro JL, Valdez RA, Romano MC, Serio-Silva JC (2009) Stress in Yucatan spider monkeys: effects of environmental conditions on fecal cortisol levels in wild and captive populations. Animal Conserv 12:496–502

Robinson SK (1998) Another threat posed by forest fragmentation: reduced food supply. Auk 115:1–3

Rogers CM, Ramenofsky M, Ketterson ED, Nolan V, Wingfield JC (1993) Plasma corticosterone, adrenal mass, winter weather, and season in nonbreeding populations of dark-eyed juncos (*Junco hyemalis hyemalis*). Auk 110:279–285

Romero LM (2004) Physiological stress in ecology: lessons from biomedical research. Trends Ecol Evol 19:249–255

Romero LM, Wikelski M (2001) Corticosterone levels predict survival probabilities of Galapagos marine iguanas during El Nino events. Proc Natl Acad Sci USA 98:7366–7370

Ruiz G, Rosenmann M, Novoa FF, Sabat P (2002) Hematological parameters and stress index in rufous-collared sparrows dwelling in urban environments. Condor 104:162–166

Sapolsky RM, Romero LM, Munck AU (2000) How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. Endocr Rev 21:55–89

Shanahan DF, Possingham HP, Riginos C (2010) Models based on individual level movement predict spatial patterns of genetic relatedness for two Australian forest birds. Landsacape Ecol 26:137–148

Stevenson RD, Tuberty SR, DeFur PL, Wingfield JC (2005) Ecophysiology and conservation: the contribution of endocrinology and immunology: introduction to the symposium. Integr Comp Bio 45:1–3

Stier KS, Almasi B, Gasparini J, Piault R, Roulin A, Jenni L (2009) Effects of corticosterone on innate and humoral immune functions and oxidative stress in barn owl nestlings. J Exp Biol 212:2085–2091

Strong AM, Sherry TW (2000) Habitat-specific effects of food abundance on the condition of ovenbirds wintering in Jamaica. J Anim Ecol 69:883–895

Suorsa P, Huhta E, Nikula A, Jantti A, Helle H, Hakkarainen H (2003) Forest management is associated with physiological stress in an old-growth forest passerine. Proc R Soc B-Biol Sci 270:963–969

Suorsa P, Helle H, Koivunen V, Huhta E, Nikula A, Hakkarainen H (2004) Effects of forest patch size on physiological stress and immunocompetence in an area-sensitive passerine, the Eurasian treecreepr (*Certhis familiaris*): an experiment. Proc R Soc B-Biol Sci 271:435–440

Van Horne B (1983) Density as a misleading indicator of habitat quality. J Wildl Manage 47:893–901

Vleck CM, Vertalino N, Vleck D, Bucher TL (2000) Stress, corticosterone, and heterophil to lymphocyte ratios in free-living adelie penguins. Condor 102:392–400

Wasser SK, Bevis K, Hanson E (1997) Noninvasive physiological measures of disturbance in the northern spotted owl. Conserv Biol 11:1019–1022

Watson DM (2011) A productivity-based explanation for woodland bird declines: poorer soils yield less food. Emu 111:10–18

Wenny DG, Clawson RL, Faaborg J, Sheriff SL (1993) Population density, habitat selection and minimum area requirements of three forest-interior warblers in central Missouri. Condor 95:968–979

Wikelski M, Cooke SJ (2006) Conservation physiology. Trends Ecol Evol 21:38–46

Wingfield JC, Kitaysky AS (2002) Endocrine response to unpredictable environmental events: stress or anti-stress hormones? Integ Comp Biol 42:600–609

Wingfield JC, Ramenofsky M (1997) Corticosterone and facultative dispersal in response to unpredictable events. Ardea 85:155–166

Wingfield JC, Ramenofsky M (1999) Hormones and the behaviorial ecology of stress. In: Balm PHM (ed) Stress physiology in animals. Sheffield Academic Press, Sheffield, pp 1–51

Wingfield JC, Hunt K, Breuner C, Dunlap K, Fowler GS, Freed L, Lepson J (1997) Environmental stress, field endocrinology, and conservation biology. In: Clemmons JR, Buchholz R (eds) Behavioral approaches to conservation in the wild. Cambridge University Press, Cambridge, pp 95–131

Wingfield JC, Maney DL, Breuner CW, Jacobs JD, Lynn S, Ramenofsky M, Richardson RD (1998) Ecological bases of hormone-behavior interactions: the "emergency life history stage". Am Zool 38:191–206

Zanette L, Doyle P, Tremont SM (2000) Food shortage in small fragments: evidence from an area-sensitive passerine. Ecol 81:1654–1666