Title: Unifying adaptive stress and adaptive habitat selection hypotheses through movement ecology

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Abstract

Habitat selection is adaptive, but habitats where animals exhibit elevated glucocorticoid production are often associated with lower fitness. The perception of fitness costs persists in part because glucocorticoid samples rarely represent the internal state of individuals at the time of sampling, making it difficult to demonstrate a causal link between glucocorticoid production and habitat selection at fine temporal and spatial scales. We show how to test this fine-scale causal link by integrating movement ecology with stress physiology. We first synthesize physiological evidence that effect of glucocorticoid production on movement aligns with habitat selection theory. We distinguish whether habitat selection exposes individuals to stressors that elevate their glucocorticoid levels, or whether elevated glucocorticoids stimulate selection for better habitat. We then test the directionality of these two pathways using movement-based habitat selection analysis at the individual level. We capitalize on a combination of individual movement data and the predictable time required for glucocorticoids to peak in samples like hair, feathers, and feces from individuals. Our proof-of-concept shows how to distinguish when glucocorticoids stimulate adaptive habitat selection. Finally, we suggest a potential evolutionary role for glucocorticoids in habitat specialization, offering a mechanism by which populations might adapt to their environments over generations.

Keywords: Glucocorticoids, stress physiology, optimal foraging theory, integrated step selection analysis, environmental challenges, non-invasive sampling, individual differences

Introduction

Animals are adapted to their environments, both physiologically through glucocorticoid production and behaviourally through habitat selection. Habitat selection is the process by which animals use some habitats disproportionately to their availability. For example, animals might relocate to sparser foraging patches that maximize energetic intake [1] while mitigating the stress of competition for forage [2]. Movement between foraging patches is driven in part by glucocorticoid hormone production. Glucocorticoids stimulate appetite when energy reserves are low [3] and make energy available for movement to find foraging resources [4]. Their role in energy regulation makes glucocorticoids a key indicator for internal state, which is integral to movement and habitat selection [5]. But glucocorticoid production is also associated with exposure to stressors that can compromise fitness [6]. Glucocorticoids have pleiotropic effects under stress, for example reducing body mass when they stimulate catabolism of energy reserves [7], and lowering reproductive success when they stimulate behaviours that interfere with reproduction [8]. While these costs usually only arise when animals face chronic and inescapable stressors [9], studies considering possible adaptive benefits of glucocorticoids are the minority. The benefits of glucocorticoid production are least considered when their production coincides with selection of stressful habitats.

Glucocorticoids decrease fitness directly when their long-term production becomes physiologically harmful or otherwise compromises reproduction or survival. Reproductive output, for example, declines after long-term experimental exposure to stressors [10]. Oxidative stress can accumulate even after short-term glucocorticoid exposure [11]. Though the physiological and behavioural consequences of glucocorticoid production can compromise fitness, glucocorticoid production is not inherently pathological. In fact, its production is often an

adaptive response to stressors faced in the wild [12]. For example, depletion of body reserves is associated with elevated glucocorticoids [13,14], but breakdown of protein and lipid reserves are is not always harmful over longer time scales. For example, when food availability is low or energetic demand is high, mobilizing lipid and protein reserves prevents starvation [7]. Sacrificing body condition might eventually compromise survival, but lifetime reproductive success may also be higher for animals that select habitat favouring their own survival when conditions are not favourable for offspring survival [15]. Quantifying survival and reproductive success associated with glucocorticoid production can help distinguish when glucocorticoid-driven habitat selection is adaptive. However, the distinction requires knowing whether glucocorticoids caused habitat selection, or habitat selection caused glucocorticoid production.

One way to establish the causal direction between glucocorticoids and habitat selection is to identify the timing of habitat selection relative to associated glucocorticoid production. When glucocorticoids are already elevated before moving to a new habitat, stressors in the selected habitat could not have caused their production. Instead, glucocorticoid production might have driven selection of the habitat, motivated by stressors experienced elsewhere. For example, migratory animals elevate glucocorticoids in spring as stressors intensify on winter ranges [16], energetically preparing them for migration to summer ranges [17]. Thus, during migration the glucocorticoids causing habitat selection are adaptive. In contrast, when glucocorticoids become elevated after selecting habitat, their production might represent reactions to stressors. For example, sandgrouse (*Pterocles alchata*) inhabiting a recreational area had elevated glucocorticoids only after weekends with high tourist traffic, suggesting human disturbance triggered their stress responses [18].

The sequence from glucocorticoid production to habitat selection is clearer when glucocorticoids are elevated in response to measurable stressors like human disturbance. However, many natural stressors facing wild animals are not readily observable. For example, when a wild animal selects a risky habitat where it briefly encounters a predator, the hypothalamic-pituitary-adrenal axis might stimulate an acute stress response. Eventually, glucocorticoids produced during the stress response can be measured in non-invasive samples like hair and feces. However, non-invasive samples are difficult to age and could represent stressors that occurred hours, days, or even weeks in the past [19]. The temporal disconnect between stressor and sample makes the exact timing of the stressor, and therefore the relative order of habitat selection and glucocorticoid production, ambiguous.

The spatial scale at which non-invasive samples are collected also complicates the causal relationship between glucocorticoids and habitat selection. Glucocorticoids are primarily responsible for fine-scale habitat selection decisions by individuals at the so-called second and third orders of selection (*sensu* Johnson [20]). However, the difficult task of linking individuals to non-invasive samples in the field [21] limits inferences to the effects of population-average glucocorticoids on coarser-scale habitat selection (i.e., first order selection [20]). These coarse-scale population-level comparisons create uncertainty around which stressors cause habitat selection. For example, predator abundance can be compared between habitats, but assumed stress responses to predation risk may instead be caused by other associated stressors like food shortages [22]. Furthermore, individual differences in diet and glucocorticoid metabolism between populations, rather than stressors per se, may result in different glucocorticoid levels between them [23]. The consequence is that glucocorticoids and habitat selection may be either correlated or share a causal relationship. Still, the most common interpretation of any

relationship between them is that selection of a stressful habitat both caused glucocorticoid production and reduced fitness (Table 1).

We show how the movement ecology lens (*sensu* [5]) can help distinguish adaptive production of glucocorticoids from their purported harmful effects. To demonstrate, we first synthesize evidence that glucocorticoids are mechanistically responsible for adaptive habitat selection behaviour. Our examples show how habitat selection changes when animals in different life-history stages manipulate glucocorticoid levels to support either reproduction or survival. Next, we discuss the time lag between stressor and peak glucocorticoid concentrations in tissues like blood, feces, and hair. Normally considered a challenge for researchers aiming to connect glucocorticoids with movement and habitat selection, we demonstrate how the consistency of this time lag presents an opportunity to pinpoint the stressor responsible for habitat selection. Once the timing of the stressor and individual identity of the sample are known, movement-based analyses can test causal links between glucocorticoids and adaptive habitat selection responses to the stressor. We demonstrate this analysis with a proof-of-concept. Our proof-of-concept acts as a template for promising research questions to which the same approach could be applied in the future.

Physiology of glucocorticoids as a mechanism for movement

Patterns of glucocorticoid production correlate with movement activity over daily and seasonal time periods. Baseline glucocorticoid levels, regulated by the autonomic nervous system [24], coincide with energy needs for locomotion at predictable times. For example, circadian activity patterns in mice coincide with the rise and fall of circulating glucocorticoids [25], and in turn, glucocorticoid rhythms adapt to regular feeding periods when energy is available for movement

[26]. The interplay among energy availability, movement, and these baseline glucocorticoid levels is a mechanistic explanation for diel habitat selection patterns that help prey species access forage while avoiding their predators [27]. Seasonal activity patterns like migration track longer-term baseline glucocorticoid rhythms [28]. Because baseline glucocorticoid levels are seasonally elevated during migration [17], birds are primed to adjust movement quickly in response to cues like wind assistance [29] and stressors upon arrival at migration stopover sites [30]. Non-migratory birds also elevate baseline glucocorticoids during reproduction when offspring require the most active provisioning [6]. Thus, via extension of their effects on activity and locomotion, glucocorticoids form the physiological connection between environmental pressures and adaptive patterns of behaviour to overcome them.

Environmental pressures, like predation risk, induce stress responses that make prey species avoid risky foraging habitat. Predator cues stimulate a cascade of hormone release via the hypothalamic-pituitary-adrenal (HPA) axis, the main products of which include glucocorticoids and other faster-acting hormones such as catecholamines. While catecholamines are mainly involved in increasing blood pressure, heart rate, and driving other more immediate responses to stress [31], glucocorticoids have a primary role in helping to regulate blood glucose levels. Glucose is mobilized when glucocorticoids prevent its uptake by adipose and muscle tissue [32] and stimulate gluconeogenesis [4], thereby providing needed energy to forgo foraging while escaping predators. Continued effects of elevated predation risk on glucocorticoid production can have long-term effects on habitat selection patterns. For example, decadal shifts in predation pressure by grey seals in Atlantic Canada caused Atlantic cod (*Gadus morhua*) and white hake (*Urophycis tenuis*) to redistribute to safer areas with less food [33]. In such cases, the potential cost of predation outweighs the value of energy gain.

The continued need to replenish calories under long-term predation risk, however, can also encourage selection for risky habitats that offer more productive foraging. As risk attenuates after a stress response, glucocorticoid levels return to baseline levels through a combination of negative feedback on the HPA axis and reduced glucocorticoid production. When reduction in HPA axis activity follows a stress response, still-elevated baseline levels stimulate feeding behaviour [4] and a preference for high-calorie foods [3]. If lower feeding rates during the stress response eventually compromise energy storage, peripheral satiety signals also decline and animals seek to replace energy stores [4]. For wildlife, replacing energy stores means movement to foraging habitats. Consequent hunger encourages animals exposed to chronic risk to spend even more time foraging [34]. Preferences for richer foods are reinforced over time because their consumption gradually blunts mounting of stress responses [35], keeping baseline glucocorticoid levels in a slightly elevated state. Similarly, predation risk elevates respiration rate, changing C:N ratios of animal tissue and stimulating selection of forage with greater carbon content [36]. Over time, habitat selection patterns emerge that prioritize energy gain over avoidance of predation risk. For example, rather than moving to unproductive areas with less poaching, elephant groups in Kenya responded to intense ivory poaching by moving to ranges with higher primary productivity [37]. Thus, glucocorticoid-stimulated movement mediates trade-offs between energy-motivated and risk-avoidant habitat selection, which is ultimately adaptive.

Integrating glucocorticoids into individual habitat selection

Testing when glucocorticoids stimulate adaptive habitat selection is difficult in the wild because glucocorticoid samples are imprecise measures of stressors faced by individuals. Precision comes from placing movement appropriately along the timeline from individual glucocorticoid

production to sample collection. Glucocorticoids are incorporated into growing hair cells from the plasma, and depending on the rate of hair growth, represent circulating levels over periods from weeks to months [38]. Feathers also require weeks or months to incorporate glucocorticoids, and only do so during growth periods prior to moult [19]. Fecal samples represent circulating glucocorticoids processed by the liver and deposited as metabolites within several hours to days depending on species-specific gut transport times (reviewed in [39]). In contrast, the temporal resolution of biotelemetry data is often hourly or finer, making glucocorticoid samples a relatively coarse representation of stressors faced during habitat selection. Moreover, samples are often collected without knowledge of individual identity. The mismatch between habitat selection and glucocorticoid production makes it necessary to pool population movement data over weeks, months, or even years. The more data are pooled, however, the more challenging it becomes to assess whether glucocorticoid production is a cause or consequence of habitat selection behaviour. Addressing the problem of causation requires validating the time required for glucocorticoids to peak in samples after stressors, then determining whether individual behaviour occurred before or after the hormone peak.

Determining whether behaviours caused or were caused by elevated glucocorticoid levels is especially important in the wild where stress responses are often inferred instead of observed. The general expectation is that perceived stressor severity determines the quantity of glucocorticoids produced in response [40]. However, the bioactive component of glucocorticoids, and not the overall amount produced in response to stressors, triggers behavioural responses. Habitat selection behaviours driven by baseline glucocorticoid levels, like foraging and migration, arise when glucocorticoids bind to low-affinity mineralocorticoid (MR) receptors [28]. For immediate behavioural effects during a stress response, glucocorticoids bind

high-affinity glucocorticoid (GR) receptors [41]. Receptor binding is controlled in part by corticosterone binding globulins (CBGs) which bind glucocorticoids in the plasma and prevent them binding receptors [42]. As a result, the effects of glucocorticoids on behaviour saturate when either high CBG concentrations, downregulated receptor transcription [43], or negative feedback on the stress response slow receptor binding rates downstream [44]. Estimating the amount of circulating glucocorticoids available to bind to receptors, however, requires either blood samples (e.g., [42]) or experimental hormone challenges (e.g., [45]). The controlled conditions required for these protocols make it difficult to link CBGs, receptors, and negative feedback to habitat selection under natural conditions. Alternatively, modelling individual glucocorticoid-habitat selection relationships could reveal when these downstream mediators adjust how the hormones affect behaviour. This is because when downstream mediators interact with glucocorticoids at high concentrations, their relationship with behaviour becomes nonlinear. For example, Adélie penguin (*Pygoscelis adeliae*) locomotory behaviour had a U-shaped relationship with glucocorticoid supplementation, where glucocorticoid effects on behaviour at high circulating concentrations were buffered by high CBG concentrations [46].

Adjusting behavioural responses to glucocorticoids downstream is beneficial for some individuals and during some seasons. For example, as day lengths shorten in winter, the effect of cortisol supplementation on white-crowned sparrow (*Zonotrichia leucophrys gambelii*) activity saturates [47]. Saturating effects of glucocorticoids on behaviour are particularly common during reproduction [43], when habitat selection must adjust to meet the needs of breeding individuals and those provisioning offspring [48]. Individuals having more experience with a stressor sometimes develop stronger negative feedback to prevent pathology from chronic stress [49]. The result is that the same stressor might produce different peak glucocorticoid levels in two

individuals or differences in how glucocorticoid effects are mediated downstream [50]. These differences might manifest in different adaptive habitat selection patterns by individual or by season in response to the environment.

Differences in how individuals respond to the environment pose a problem for disentangling cause and effect because they introduce variation into the glucocorticoid-habitat selection relationship. To control for this variation, individual movement must be linked to glucocorticoid samples collected from individual animals at known times. While repeated sampling of individual animals is often infeasible in the wild, biotelemetry data can help link individuals with opportunistically collected field samples. Feathers, hair, and feces contain genetic material in addition to glucocorticoids, allowing anonymous samples from populations to be matched to individuals with genetic data using genetic capture-mark-recapture techniques [39]. Conspicuous species like ungulates can be observed defecating or shedding hair [51]. When individuals also carry biotelemetry tags, areas like trails and locations of kill or rest sites can be targeted for sampling specific individuals (e.g., [52]), especially when fine-scale timestamped location data link individual movements to their circulating glucocorticoids during the same period [21].

When the timing of sample collection, estimated age of the sample, and the time required for glucocorticoids to peak in hair, feces, or feathers are known, the approximate timing of stressors can also be pinpointed. Movement-based models can then be used to interpret whether the stressor caused or was a product of habitat selection. One possible interpretation of elevated glucocorticoids is that the habitat selected by an animal exposed it to a stressor that affected its circulating glucocorticoid levels. This interpretation might be tested with double hierarchical models that include a coefficient for movement characteristics describing behaviours related to

foraging (e.g., tortuous movements) over the “steps” preceding the glucocorticoid peak (outlined in [53]). Another interpretation is that glucocorticoid levels became elevated by the stressor, which then stimulated habitat selection. Habitat selection analysis might be used to test this interpretation. Integrated step selection analysis [54], a specialized class of habitat selection analysis, can be used to test whether glucocorticoids produced before the beginning of a step affect the relative probability of habitat selection at its end (Box 1). In the following section, we provide a proof-of-concept demonstrating this approach.

Proof-of-concept: Glucocorticoid-mediated fine-scale habitat selection by elk

Background

For species that provide parental care, the fitness consequences of habitat selection manifest around parturition as an energetically demanding life stage. Female ungulates must both maintain sufficient body condition prior to calving and compensate for the demanding energetic costs of spring lactation via summer nutrition [55]. In part, these nutritional needs are met by shifting habitat use toward better forage. Increased used of forage rich habitat also coincides with elevated baseline glucocorticoid levels typical of ungulate calving periods [56]. However, calves are vulnerable to predation and in some species of ungulate are typically hidden for a few weeks following birth. To ensure reproductive success, mothers must compromise some of their nutritional needs for safety needs of the calf [57]. Safety typically requires use of cover habitat like forests and shrubland despite better foraging opportunities elsewhere [58].

Methods

We used integrated step selection analysis (Box 1; [54]) to test whether selection for cover habitat by 13 female elk (*Cervus canadensis*) shifted in response to fecal glucocorticoid metabolite levels, and whether the response changed during the approximate 16-d window when their calves are most vulnerable (see Supplementary 1 for detailed methods). We collected fecal glucocorticoid samples, which for elk correspond to circulating levels approximately 20 hours prior to defecation [59,60]. Our global positioning system-collared study population in southeastern Manitoba, Canada (49.134, -96.557) uses forest as cover habitat [61] to safely avoid humans in the largely agricultural landscape that also acts as a source of calving season forage [48]. Because animals forage to recover from stressors [4], we predicted that prior to calving and during the approximately 20 hours following glucocorticoid peaks, elk would select locations farther from cover. Foraging to recover energy after stress responses risks calf safety by exposing them to potential encounters with predators. However, lactating elk also require more energy. Thus, during the 16-d window of calf vulnerability we expected elk to use locations either farther from or closer to cover habitat depending on their priority for energy versus safety. If elk prioritized safety, those with young calves should remain closer to cover habitat even after glucocorticoid production increased. Alternatively, if they prioritized energy, they might have an even stronger preference for areas further from cover when glucocorticoid production increased. Importantly, a change in behaviour after calving might indicate either CBG concentration, receptor number, negative feedback, or all three are adjusted downstream to support calf survival.

Results & Discussion

Elk habitat selection differed with glucocorticoid levels and presence of young calves. Before calving and when calves were able to escape predators, their mothers were 50% more likely to select locations farther from cover habitat for each unit increase in glucocorticoids (Fig. 2, “pre” period). Elevated glucocorticoids increased the likelihood that elk selected locations further from cover even more strongly within the 16-d window when calves were most vulnerable (Fig. 2, “post” period). These results suggest the non-linear effects of glucocorticoids on habitat selection may be mediated post-partum either by CBG concentration, receptor availability, or negative feedback on glucocorticoid production. Thus, changes in glucocorticoid-mediated habitat selection by our elk population characterize life-history transitions, making glucocorticoid production and its downstream mediation an adaptive process that supports lifetime reproductive success. See Supplementary 2 for comprehensive results.

Conclusions and future directions

One of the most pervasive ideas about glucocorticoid overproduction is that it is an indicator of exposure to stressors that decrease fitness [6]. We interpreted the fitness implications of glucocorticoid production through its relationship to adaptive behaviours like habitat selection. In our proof-of-concept, elk with elevated glucocorticoid levels selected locations further from cover habitat, suggesting a preference for forage-rich agriculture. Selection for forage aligns with physiological evidence that glucocorticoids help animals recover energetically after a stress response [4]. When they had young calves, elk with elevated glucocorticoids exhibited even stronger selection for locations further from cover, suggesting they prioritized energy over remaining close to cover habitat for their calves (Fig. 1b). We suggest the change in behaviour

associated with care for young calves might have been brought about by physiological changes that altered the downstream effects of glucocorticoids. We demonstrated that glucocorticoid production and downstream mediation are adaptive with a modelling approach that could demonstrate a causal relationship between glucocorticoid production and habitat selection. Key to our inference was knowing the time required for glucocorticoids to peak in fecal pellet samples and which samples belonged to which individuals. These two pieces of information, already available or collected in many studies, hold promise for testing existing hypotheses in habitat selection and stress physiology and for exploring new links between individuals and their environments (Box 2).

One promising extension of our approach is to test whether adjusting habitat selection through glucocorticoid production helps individuals and populations respond to stressors over multiple generations. Individuals respond to stressors by adjusting glucocorticoid production over their lifetimes to support either survival or reproduction. For example, as they age and fewer reproductive opportunities remain, individual terns (*Sterna hirundo*) produce fewer glucocorticoids to support reproductive behaviours [62]. These age-dependent changes in reproduction are adaptive at the population level because they align with age-dependent reproductive investment as predicted by life-history theory. Individuals also differ in their glucocorticoid responses to the same stressors [63]. These stress-coping mechanisms are transmissible to offspring [64], meaning selection might act on habitat-specific glucocorticoid production as individuals are exposed to different stressors in the habitats they occupy [65]. Disruptive selection on both stress-coping mechanisms and habitat selection could reinforce differences among individuals, resulting in both the physiological and spatial separation of populations over time [66]. For example, frogs adapted to habitats with long-term exposure to

anthropogenic noise both produced fewer glucocorticoids in response to noise and had developed physiological coping mechanisms to equalize fitness with their noise-free habitat counterparts [67]. The evolutionary effects of glucocorticoid-habitat selection feedback might be preceded by habitat specialization at smaller scales, likely testable using our movement-based approach (Box 2).

Our synthesis highlighted a limitation in the way we currently interpret associations between habitat selection and glucocorticoids: namely that we rarely consider glucocorticoids an agent of adaptive habitat selection. The negative association between habitat selection and glucocorticoid production persists because it is more challenging to test when glucocorticoids drive adaptive habitat selection. We showed the tools to test an adaptive relationship between the two already exist in study systems where ecologists collect biotelemetry data and non-invasive glucocorticoid samples. The challenges going forward will be to integrate glucocorticoids into movement ecology and to explore alternative interpretations about glucocorticoid production in the context of habitat selection.

Conflicts of interest: The authors have no conflicts of interest to report.

Author contributions: LN and EVW conceived of the ideas. LN designed the proof-of-concept methodology, analyzed the data, and wrote the original draft of the manuscript. GFM oversaw hormone extractions. All authors contributed to reviewing & editing and approved the final manuscript for publication.

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Box 1 – Primer on habitat selection analysis and glucocorticoids

Habitat selection analysis (HSA) quantifies how stressors influence which habitats — or points in space characterized by a unique set of resources, risks and conditions [68] — are used preferentially or avoided by animals:

$$w_i = \frac{u_i}{a_i}$$

Where w_i is the "selection ratio" for habitat i, u_i is the proportion of habitat i used by the animal, and a_i is the proportional representation of habitat i within a landscape [69]. Note that use is often quantified using locations from biotelemetry devices. Unused locations are usually unknown, rendering proportion of use indeterminable. In these cases, selection for habitats can be estimated using logistic regression:

$$w_x = exp(\beta_0 + \beta_1 k_i + \cdots + \beta_n k_j)$$

Where the used locations are compared to a larger sample of "available" locations. Instead of probability of selection as in the selection ratio, coefficients β_1 to β_n represent selection for habitat variables, k, *relative* to j others in the model [70].

Later adjustments to the HSF model stemmed from technological advances in biotelemetry data collection. Modern biotelemetry devices collect used location data at regular time intervals. Intervals are often frequent enough that not all available locations are necessarily accessible, making biological constraints on movement a likely constraint on habitat availability. For example, locations visited within a day may not be accessible between hourly locations because the distance an animal can travel within an hour is constrained. Step selection functions

(SSF) address this constraint by drawing available locations from a set of trajectories to which the animal could move from each location point (Fig. 1; [71]). Turn angle and step length between location points become additional variables in the model, drawn from empirical distributions of the animal's movements. Movement, however, is also influenced by stressors habitat-specific stressors encountered along a movement path. Integrated step selection functions (iSSF) extend the SSF approach by incorporating this interdependency between movement and habitat selection within the modelling process [54].

Aside from its statistical justification, constraining availability and integrating movement into the habitat selection process makes iSSF models convenient for testing hypotheses about glucocorticoids and movement. Just as movement depends on habitats encountered along a movement path, it also depends on the animal's glucocorticoid production. Glucocorticoid samples from known time points along the movement path can be integrated into the iSSF model as interactions between habitat and movement covariates (Fig. 1). Interaction coefficients test hypotheses about the effects of glucocorticoids on both capacity and motivation for movement. For example:

Step length and turn angle interaction with glucocorticoids at the start of a step: When animals produce more glucocorticoids, are their movements faster and more directed (Fig. 1 a)?

Interaction between glucocorticoids at the start of a step and habitat at the end of the step: Do animals that produce more glucocorticoids select safer habitat or habitats with resources to replenish energy reserves (Fig. 1 b)?

Box 2 – Future directions

Integrating glucocorticoid hormones and habitat selection can both test the assumptions of some foundational hypotheses in stress physiology and space use ecology and generate new testable hypotheses that integrate both.

Glucocorticoid production can cause oxidative stress and reduce reproductive success (e.g., [72]), but elevated baseline levels often increase lifetime reproductive success if they facilitate immediate investment in current offspring (*cort-adaptation hypothesis*; [6]). Some investment behaviours, such as foraging trips by nesting birds, are based in habitat selection. By integrating glucocorticoids and habitat selection with iSSF models, we can detect proximate effects of glucocorticoids on investment behaviours. Are these investment behaviours repeatable within individuals? If so, how much does investment strategy contribute to individual differences in lifetime reproductive success?

The *landscape of fear* is a popular space use ecology concept linking habitat selection to predation risk: animals facing predation are expected to avoid unsafe habitats or use antipredator strategies like vigilance or safety in numbers to mitigate risk [73]. However, predator cues rather than presence are often used in practice as a proxy for risk, making fear itself difficult to quantify [74]. Can we use glucocorticoids to quantify the landscape of fear, and iSSF models to measure its responsibility for antipredator habitat selection strategies?

Just as glucocorticoids drive adaptive habitat selection behaviour, the habitats individual animals select should also affect future changes in their hormone levels. This two-way relationship between habitat and hormones may drive a positive feedback loop that reinforces habitat

selection behaviour. Evidence is emerging that habitat selection behaviour is indeed repeatable [75] and that adults continuously exposed to stressors can epigenetically alter offspring hormonal responses to the same stressors [76]. Do intergenerational glucocorticoid-habitat selection feedback loops underlie specialization of populations on habitats?

Looking beyond populations, an integrated approach to studying glucocorticoids and habitat selection may also provide insights into ecosystem functioning and the ability of species to respond to environmental change.

The *carbon stress hypothesis* [36] purports animals facing predation stress have higher cellular respiration rates, making them carbon limited. To maintain C:N ratios, stressed animals should seek forage with more of digestible carbon. Animals also select foraging habitat when they produce more glucocorticoids under stress physiology theory [77]. Can we use iSSF and spatial data of elemental landscape composition [78] to reconcile expectations of stress physiology with the carbon-stress hypothesis? Preferences for carbon-rich forage and resulting spatial variation in carbon-rich additions to detrital pools could fundamentally alter ecosystem structure [36].

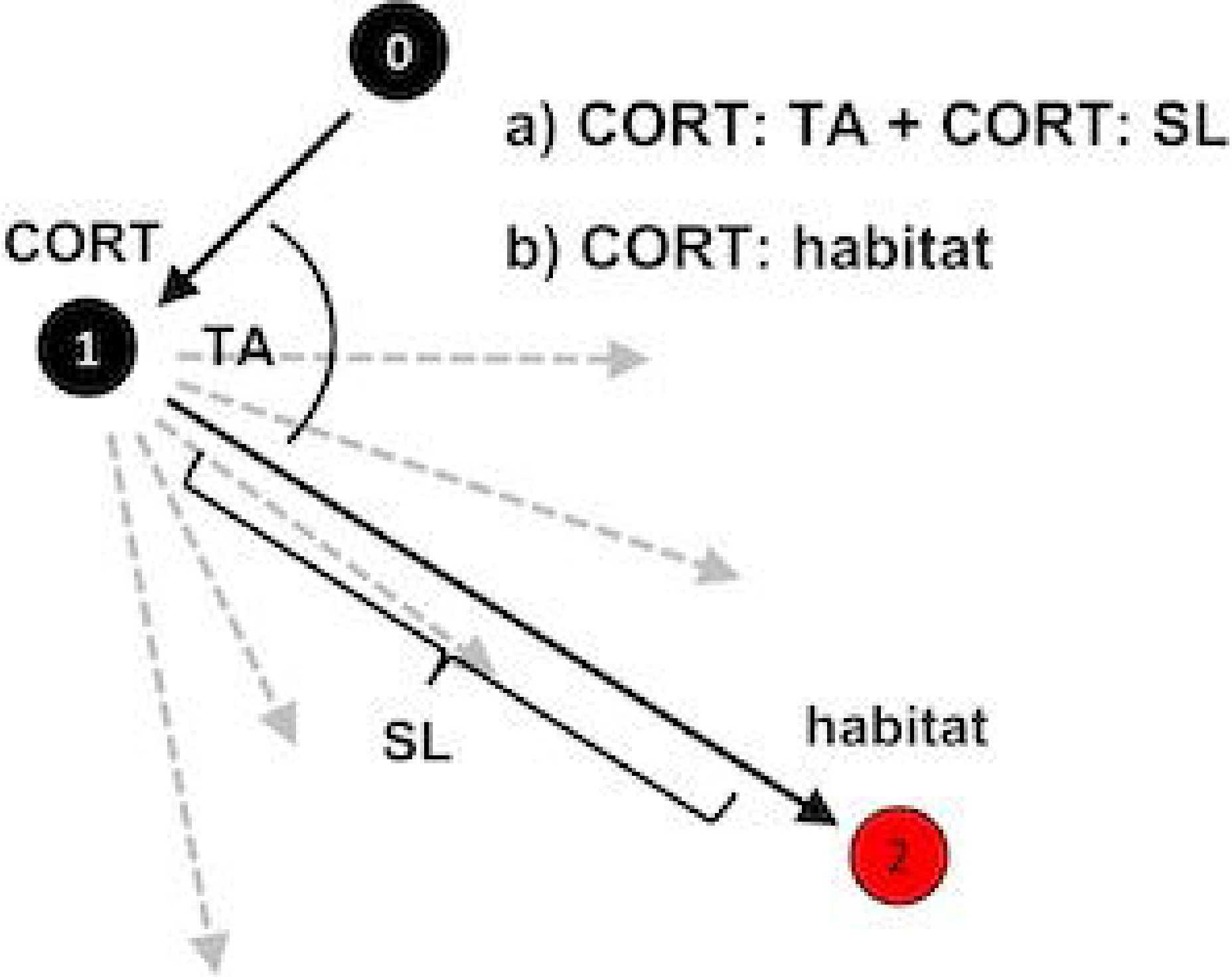
Even if glucocorticoids drive adaptive habitat selection, animals unable to access habitats that counteract stressors may instead behave in a manner that compromises fitness. Fitness costs may be particularly high in human-modified landscapes. For example, anthropogenic structures present physical barriers to movement and limit assessment of habitat selection costs (i.e., ecological traps; e.g., [79]). Can we use the null expectation that both habitat selection and glucocorticoid production are adaptive to identify cases where animals are not free to move? Glucocorticoids in these cases may measure need for management intervention.

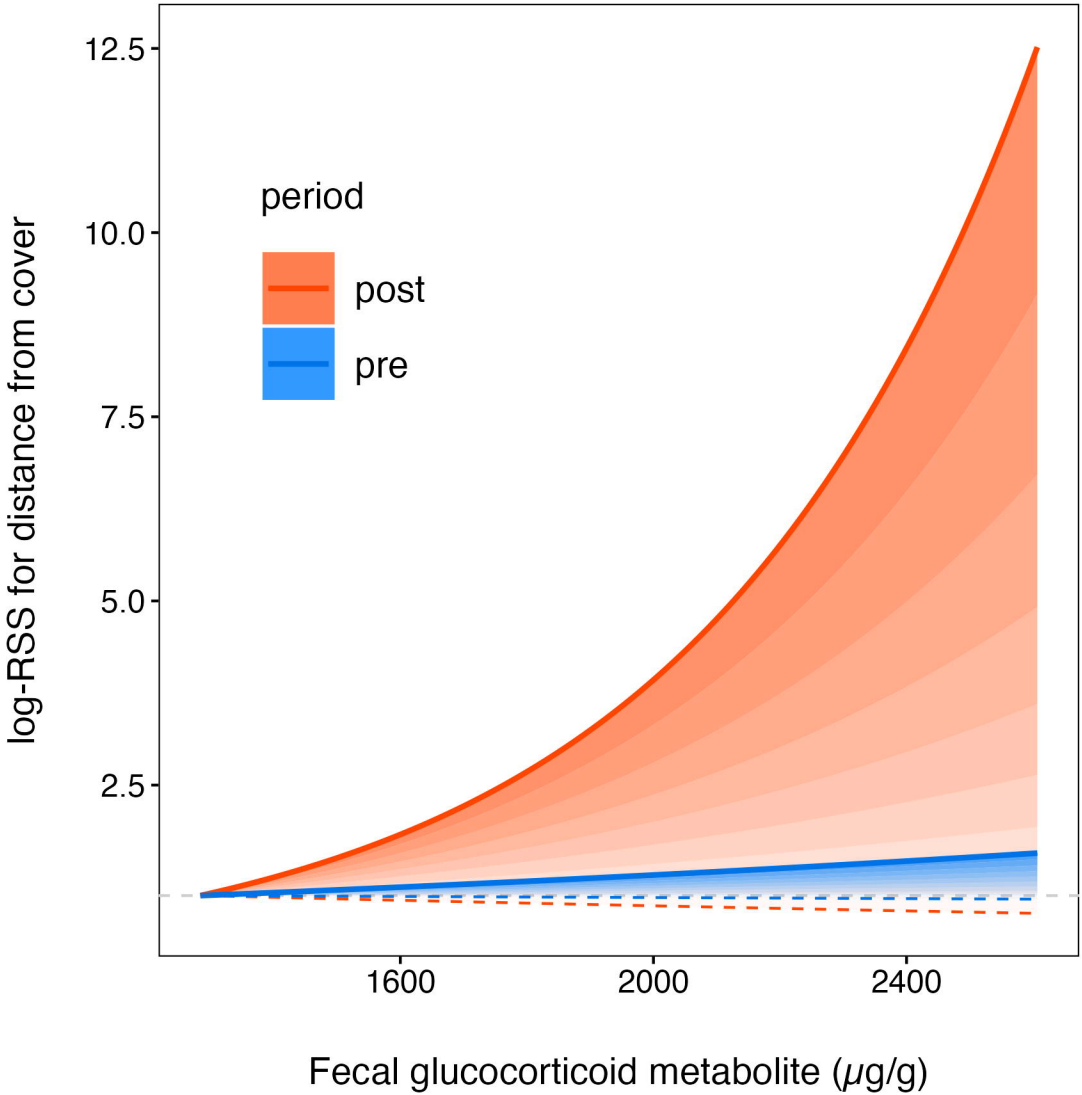
Table 1. Examples of recent field studies reporting negative relationships among habitat-associated stressors, glucocorticoid production, and fitness, without testing for causal relationships among the three. We searched for articles on Web of Science in June 2021 using the search query *habitat* AND *glucocorticoid** OR *corticosterone* OR *cortisol* AND *fitness* AND *chronic stress*.

Topic	Findings	Interpretation	Citation
Effect of human footprint on feather glucocorticoids and survival rate of Eurasian griffon vultures (*Gyps fulvus*)	Glucocorticoids elevated among birds living in areas with higher human footprint. Previous studies found lower survival rates in areas with greater footprint.	Chronic stress is associated with living in areas with higher human footprint	[80]
Effect of habitat quality on fecal glucocorticoids and population growth rate in Cape mountain zebra (*Equus zebra zebra*)	Glucocorticoids elevated in lower-quality habitat. Glucocorticoids also highest among populations with lower female fecundity and growth rates.	Populations in lower-quality habitat suffering from chronic stress due to poorer nutrition	[81]
Effect of invasive species on urinary glucocorticoids on reproduction and survival in Fijian ground frog (*Platymantis vitiana*)	Glucocorticoids elevated in enclosures with invader. Body condition and reproduction, but not survival, also lower with invader.	Competition with invader causes chronic stress that suppresses reproduction.	[82]

Figure 1. Schematic representation of used locations (circles) along a movement path from $t = 0$ through $t = 2$. Solid arrows represent the movement path between location points, and dashed arrows represent five available trajectories an animal could have taken at $t = 1$, drawn from assumed distributions of the animal's step lengths (SL) and turn angles (TA) between movement paths with parameters based on its movement characteristics. Effects of glucocorticoids (CORT) at $t = 1$ on movement between $t = 1$ and $t = 2$ can be tested using its interaction with TA (between $t = 0$ and $t = 1$) and SL (between $t = 1$ and $t = 2$) as variables in an iSSF model (a). Its interaction with habitat at $t = 2$ tests how glucocorticoids at $t = 1$ affect habitat selection.

Figure 2. Log relative selection strength (RSS) for a location within cover (dashed lines) versus 450 m from cover (solid lines) with increasing fecal glucocorticoid metabolite (FGM) levels. Blue ribbons show the change in RSS between these locations in the 16-d window ("pre" period) when young calves are most vulnerable, and orange ribbons outside of this period ("post" period). Distance between the solid and dashed lines indicate relative selection strength is stronger for locations closer to cover as FGM levels increase. Higher elevation of the orange ribbon indicates a stronger effect during the 16-d window of calf vulnerability, suggesting lower corticosterone binding globulin concentration, fewer receptors, or reduced negative feedback may be driving selection for foraging habitat when energy needs are greatest.


0
a) CORT: TA + CORT: SL
b) CORT: habitat
CORT
1
TA
SL
habitat
2


period
post
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12.5
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log-RSS for distance from cover
1600
2000
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Fecal glucocorticoid metabolite (μg/g)