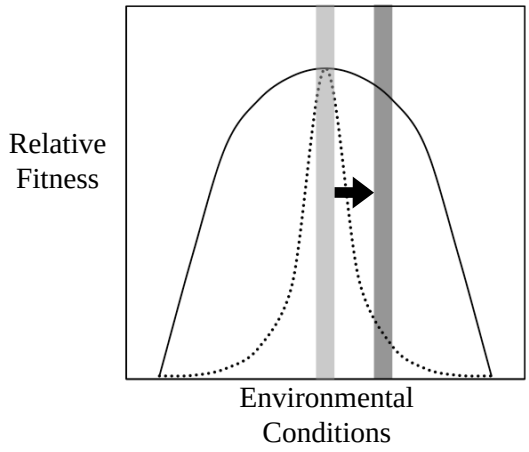
Coping mechanisms in a changing world

Environmental challenges abound in nature. They include sudden storms, predation attempts, disease, and food limitation, and also human-induced challenges such as urbanization and climate change. **How do animals cope with environmental challenges?** This question forms the basis for most of my research. I am interested in short-term behavioral, physiological, and life history responses, as well as longer-term demographic and evolutionary responses. I rely on question-driven comparative, field, and laboratory studies that draw on an array of research tools, including behavioral, endocrine, population genetic, and immune ecological methods. Below, I outline ongoing and proposed work in two distinct areas of my research program.

Competitive dominance or environmental tolerance: How do organisms persist in cities?

The globe is becoming increasingly urbanized, with the majority of the human population now living in cities. As an area becomes urbanized, the biotic community changes dramatically. Most organisms do not persist in cities, yet other species not only persist, but thrive. *How do organisms cope with the challenges of urbanization?*

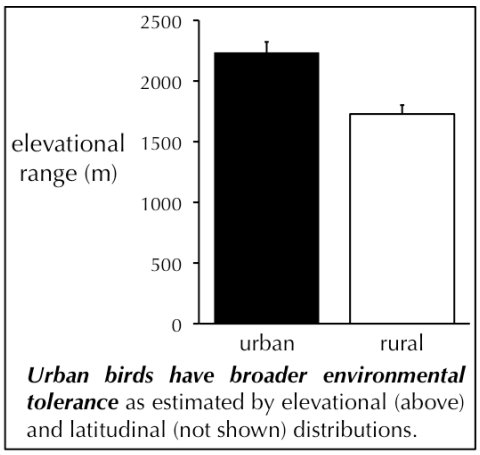


The Environmental Tolerance Hypothesis. Fitness of an organism with narrow tolerance (dotted curve) drops off steeply with changes in the environment. In contrast, an organism with broad tolerance (solid curve) has maximum fitness across a wide range of environmental conditions. As an area where the two species coexist becomes urbanized (arrow), environmental conditions (vertical lines) shift, and are more likely to fall within the range of tolerance of an organism with broad environmental tolerance than that of an organism with narrow tolerance. Adapted from Figure 1, Bonier et al. Biology Letters 2007.

I am currently testing two hypotheses to explain mechanisms of response to urbanization. The *Environmental Tolerance Hypothesis* suggests that diversity is reduced in cities because only species with broad environmental tolerance can persist there (figure, at right). In contrast, the *Behavioral Exclusion Hypothesis* asserts that diversity is reduced in cities because dominant species thrive there and exclude subordinate species. These two hypotheses are not mutually exclusive, but these distinct mechanisms likely contribute differentially to species loss in cities.

In previous work, we provided the first global, phylogenetically controlled comparison of species that do and do not occur in urban environments, and found support for both the Environmental Tolerance Hypothesis and the Behavioral Exclusion Hypothesis. First, in a comparison of species from 100 avian genera, *urban birds had dramatically broader environmental tolerance than non-urban congeners* (figure below, Bonier et al. 2007 Biol Lett). Second, *widespread urban bird species were heavier than non-urban congeners*. Using size as a predictor of behavioral dominance, these findings suggest that both competitive interactions and environmental tolerance might underlie reduced diversity in cities.

In ongoing work, we are compiling observations of behavioral interactions between phylogenetically paired urban and non-urban birds, and summarizing cases where one species is consistently dominant to the other. To date, *the urban species is behaviorally dominant to its non-urban congener* in 23 of 26 avian species pairs (binomial exact test, $P < 0.001$). These exciting findings suggest that competitive interactions result in the exclusion of the subordinate species from cities, and/or that traits associated with behavioral dominance, such as boldness or behavioral flexibility, contribute to the dominant species' ability to persist in urban habitat.



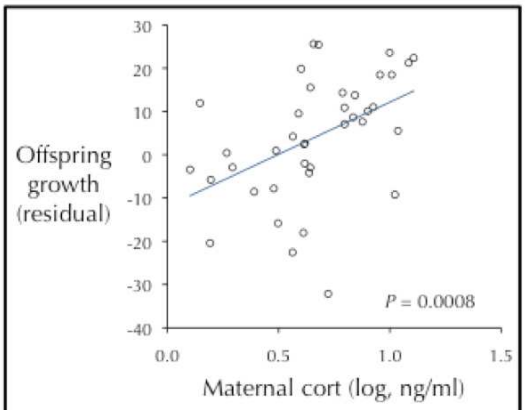
Urban birds have broader environmental tolerance as estimated by elevational (above) and latitudinal (not shown) distributions.

In a position at UC Riverside, I would use field and aviary studies to further address these and other hypotheses. This work would be aimed at identifying [1] traits that broaden environmental tolerance and provide adaptations to urban environments, [2] competitive relationships among species that might limit diversity in cities, [3] associations between competitive dominance and traits important in urban adaptation, and [4] the importance of plastic versus heritable traits in urban adaptation.

Proximate mechanisms underlying variation in life history traits.

Why do some individuals produce more offspring than others? Why do some individuals work harder to provision their offspring? Why do some individuals produce more sons than others? Such variation in life history traits has fascinated biologists for decades, yet answers to these questions remain elusive. In answering these "why" questions, I seek multiple levels of explanation, from fitness costs and benefits, to environmental causes, to physiological mechanisms underlying among individual, among population, and among species variation in life history traits. A central focus of my research program involves investigating how the endocrine system regulates variation in life history traits and mediates responses to challenges in the environment. Below, I illustrate this research with two examples.

The role of stress hormones in regulating resource allocation. Various hormones have been proposed as candidate mechanisms for the regulation of life history traits, in part because they mediate physiological and behavioral responses to changes in the internal and external environment. In previous work, I have shown that glucocorticoids, or stress hormones, covary with fecundity, offspring growth, offspring sex ratio, parental behavior, and apparent survival (figure at right, Bonier et al. 2007 Behav Ecol a & b; 2009 Gen Comp Endocrinol, 2009 Trends Ecol Evol, 2011 Biol Lett). Based on these findings, I propose several alternative hypotheses to explain the influence of glucocorticoid hormones on life history traits: [1] the *Cort Adaptation Hypothesis* (Bonier et al. 2009 Trends Ecol Evol), which asserts that glucocorticoids facilitate allocation of resources towards all energetic demands confronting an animal; [2] the *Context Dependent Hypothesis* (Bonier et al. 2011 Biol Lett), which asserts that glucocorticoids facilitate allocation of resources towards energetic demands in a manner that varies with an individual's current reproductive value; and [3] the *null hypothesis*, which asserts that while glucocorticoid concentrations might covary with life history traits and energetic demands, these hormones do not directly respond to energetic demands or influence allocation of resources towards specific functions.

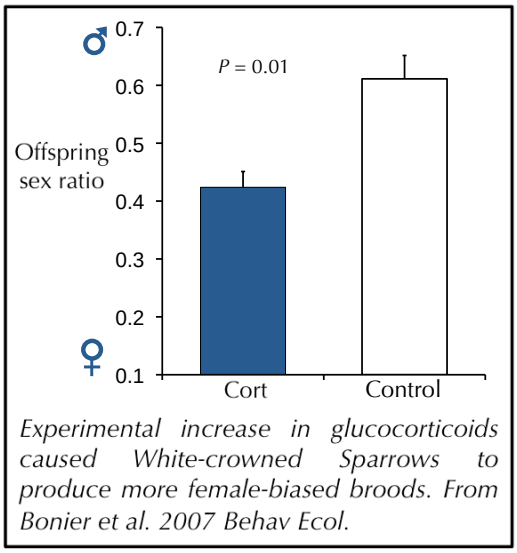


In Tree Swallows, maternal glucocorticoid levels correlate positively with offspring growth. From Bonier et al. 2009 Gen Comp Endocrinol and unpublished data.

To test these hypotheses, I have designed a series of critical experiments. First, I have experimentally manipulated the cost of reproduction in large-scale experiments with free-ranging Tree Swallows. An experimental increase in the energetic demands of reproduction resulted in coincident increases in circulating glucocorticoid levels, supporting both the Cort Adaptation and the Context Dependent Hypotheses, but allowing rejection of the null hypothesis (Bonier et al. 2011 Biol Lett). Next, with the support of our recently funded NSF grant, I plan to experimentally manipulate circulating glucocorticoids in this same population of Tree Swallows, in individuals with differing values of current reproduction. Under the Cort Adaptation Hypothesis, the influence of augmented glucocorticoids on

allocation to life history traits should not differ among individuals with differing reproductive values, whereas we expect an effect of reproductive value under the Context Dependent Hypothesis.

The potential impacts of differentiating between the Cort Adaptation and Context Dependent Hypotheses are numerous and significant. For example, if glucocorticoids can flexibly influence allocation to life history functions in a manner that varies with reproductive value, the constraints on the evolution of life history strategies might be reduced relative to those expected if glucocorticoids have inflexible effects on resource allocation. Additionally, our interpretations of the significance of among- and within-individual variation in circulating glucocorticoid concentrations will be impacted by tests of these hypotheses. What might be a detrimentally elevated glucocorticoid level in one individual or during one life history stage could be an adaptively elevated hormone level in another.



Experimental increase in glucocorticoids caused White-crowned Sparrows to produce more female-biased broods. From Bonier et al. 2007 Behav Ecol.

Intergenerational effects of stress hormones on life history.

A central life history decision confronting individuals is the manner in which to invest resources in offspring. Parents can influence offspring phenotype through differential allocation of resources, and also through transmission of hormonal signals. A considerable body of literature has revealed important effects of maternal hormone levels and hormone transmission to the embryo. For example, after providing the first evidence of a natural correlation between offspring sex ratios and maternal glucocorticoid levels, we showed that a conservative experimental augmentation of circulating glucocorticoids directly influenced offspring sex ratio (figure at left, Bonier et al. 2007 *Behav Ecol*).

Less well known are the potential intergenerational effects of hormones during both development and non-reproductive adulthood on offspring phenotypes and life history traits. In a position at UC Riverside, I would seek to establish a captive-breeding population that will permit investigation of intergenerational effects that are not easily tracked in natural systems. One of the first experiments I would conduct with this captive system would test the following non-exclusive hypotheses: [1] the *Organizational Effects Hypothesis*, which asserts that variation in hormones during an individual's development influence the life history strategies of that individual's offspring; [2] the *Activational Effects Hypothesis*, which asserts that variation in hormones during non-reproductive adulthood (i.e., outside of direct maternal transmission to the embryo) influence life history strategies of an individual's offspring; [3] the null hypothesis, which asserts that variation in hormone levels during development and adulthood do not influence offspring life history strategies. I would test among these hypotheses by experimentally manipulating hormone levels in captive developing and non-breeding adult individuals, then breeding those individuals, and comparing the life history strategies of their offspring to those of offspring of sham-manipulated parents. As in the previous section, I would then seek to assess the generalizability of my findings by replicating the studies in taxonomically diverse systems.

The potential for hormone exposure in one generation to influence offspring life history strategies could have important impacts. For example, evolutionary responses to environmental change, such as climate change and urbanization, might be accelerated relative to the responses predicted without intergenerational effects, particularly in populations where the parental and offspring environment are similar. In contrast, the potential detrimental influence of exposure to compounds that mimic hormonal effects (such as estrogenic pollutants) could be amplified if those effects span generations. Depending on the findings of the above-described experiments, I would seek to address these potentially important implications of our findings with subsequent captive and field experiments.