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IN FOCUS

The adaptive potential of maternal stress exposure in regulating population dynamics

Adult root vole, *Microtus oeconomus*. Photo credit: Alice Kenney.

In Focus: Bian, J.-H., Du, S.-Y., Wu, Y., Cao, Y.-F., Nie, X.-H., He, H. & You, Z.-B. (2015) Maternal effects and population regulation: maternal density-induced reproduction suppression impairs offspring capacity in response to immediate environment in root voles *Microtus oeconomus*. *Journal of Animal Ecology*, **84**, 326–336.

Ecologists, evolutionary biologists and biomedical researchers are investing great effort in understanding the impact maternal stress may have on offspring phenotypes. *Bian et al.* advance this field by providing evidence that density-induced maternal stress programs offspring phenotypes, resulting in direct consequences on their fitness and population dynamics, but doing so in a context-dependent manner. They suggest that intrinsic state alterations induced by maternal stress may be one ecological factor generating delayed density-dependent effects. This research highlights the connection between maternal stress and population dynamics, and the importance of understanding the adaptive potential of such effects in a context-dependent manner.

The stress axis (hypothalamic–pituitary–adrenal axis) and subsequent release of glucocorticoid hormones (GCs) play a central role in mediating the link between environmental variability and variation in the behaviour, life-history strategies and fitness of vertebrates (Wingfield & Sapolsky 2003; Boonstra *et al.* 2014). The stress axis allows animals to cope with and respond to change and challenges in the face of both certain and uncertain environments. The stress axis also plays a central role in evolutionary adaptations to ecological pressures, through maternal stress effects. Maternal stress effects occur when maternal stress hormones alter offspring phenotype (Love, McGowan & Sheriff 2013). Many studies have shown that a wide variety of ecological stressors, acting via maternal stress during reproduction, can alter offspring phenotypes; quality of the rearing environment (Pravosudov & Kitaysky 2006), environment–maternal state interactions (Meylan *et al.* 2002; Love & Williams 2008), predation risk (Sheriff, Krebs & Boonstra 2009, 2010) and social environment (Landys, Goymann & Slagvold 2011). Further, it has been hypothesized that maternal stress-induced phenotypic responses in offspring are not simply unavoidable negative side effects, but rather may be adaptive responses that prepare individuals to cope, reproduce and survive in environments where ecological stressors are frequently encountered (Meylan & Clobert 2005; Love *et al.* 2013; Sheriff & Love 2013). However, if the offspring environment is mismatched with that of their mother, the offspring's phenotypic response may prove to be maladaptive. *Bian et al.* test this hypothesis and demonstrate that density-induced maternal stress in root voles (*Microtus oeconomus*) alters offspring stress axis, fitness and demography, and the adaptive potential of these effects is influenced by the density of the offspring population. These findings provide evidence that maternal stress not only alters

*Correspondence author. E-mail: mjs72@psu.edu

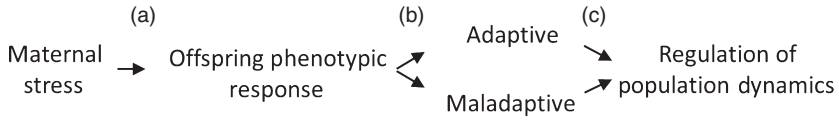

Fig. 1. Maternal stress may play a critical role in regulating population dynamics of free-living animals. (a) Maternal stress can alter offspring phenotypes, which (b) may be adaptive or maladaptive depending upon the environmental context in which they occur (matched or mismatched with that creating their mother's stress) and (c) thus can alter offspring fitness in ways which may have population-level consequences.

offspring phenotype, but may be a mechanistic link translating the maternal environment into adaptive (and possibly maladaptive) offspring phenotypes dependent upon the environment experienced by the offspring (Fig. 1).

Bian et al.'s findings emerged from an experimental manipulation in which they established low-density (80 ind./ha) or high-density (400 ind./ha) parental populations within the range of naturally occurring low phase and peak phase densities of this cyclic mammal. Offspring from these parental populations were then used to establish low- or high-density offspring populations resulting in four treatment groups; LH, HH, LL, HL (the first letter indicates the parental density, and the second letter indicates the offspring density, i.e. LH – low parental and high offspring density). In addition to measuring maternal and offspring faecal glucocorticoid metabolite levels (FCM – an integrative measure of stress), they also quantified offspring reproduction, overwinter survival, recruitment and, ultimately, how this translated into population density changes. Overall, they found that mothers from high-density populations had greater FCM levels and produced offspring that had altered phenotypes; the adaptive potential of these offspring depended upon the environment they then experienced. In offspring living in high-density populations (LH and HH), FCM levels increased throughout the first year of the experiment and remained high during the second; interestingly, HL offspring also displayed a similar pattern of increasing and high FCM levels, and there was no difference between the three groups. In contrast, FCM levels in LL offspring remained low throughout the experiment. Thus, *offspring born to high-density, stressed, mothers seem programmed to have greater stress levels regardless of their own experience, whereas offspring born to low-density, unstressed mothers seem to be able to respond to their environment*. Offspring fitness (as adults) was also affected by maternal stress. The proportion of reproductively active offspring living in high-density populations was 17 and 24% for LH females and males, but only 2 and 4% for HH offspring. In low-density populations, 69 and 66% of LL females and males, but only 23 and 4% of HL offspring were reproductively active. Conversely, the overwinter survival rate of offspring living in high-density populations was only 64% for LH offspring, but 88% for HH offspring. In low-density populations, the overwinter survival rate was 26% for LL offspring, but 80% for HL offspring. Thus, *offspring born to high-density, stressed mothers seem to trade-off reproduction for survival, whereas offspring born to low-density, unstressed mothers attempt to maximize reproductive output*. These individual fitness differences translated to differences in recruitment rates, further influenced by offspring density; offspring born to low-density mothers had significantly greater recruitment than those born to high-density mothers, and offspring living in low-density population had greater recruitment than those living in high-density populations. *Ultimately, these cumulative effects resulted in both LH and HH offspring maintaining high-density populations, LL offspring population numbers growing to a high density similar to the high-density treatments, and the HL population unable to increase remaining very low throughout the study*.

The findings of *Bian et al.* provide the first direct evidence that maternal stress effects may play a critical role in regulating population dynamics in vertebrates (Fig. 1). The hypothesis that maternal stress may be a driver of population dynamics in free-living animals stems from work by Boonstra and colleagues on cyclic populations of voles (e.g. Boonstra 1985; Boonstra & Boag 1987; Mihok & Boonstra 1992) and snowshoe hares (e.g. Boonstra & Singleton 1993; Boonstra, Krebs & Stenseth 1998b; Boonstra *et al.* 1998a; Sheriff, Krebs & Boonstra 2009, 2010, 2011). In voles, Boonstra *et al.*'s work supported the hypothesis of Dennis Chitty that some aspect of maternal condition was reflected in their progeny (Chitty 1952). In snowshoe hares, they have shown that snowshoe hares are highly sensitive to the risk of predation, and during a population decline phase, when risk is the greatest, mothers have the highest stress levels. Elevated maternal stress decreases reproduction, resulting in fewer, smaller, lighter babies that have greater stress levels themselves. After a population collapse, reproduction does not recover until the generational inheritance of predator-induced maternal stress declines. Sheriff & Love (2013) and Love, McGowan and Sheriff (2013) furthered the maternal stress hypothesis and suggested that maternal stress exposure may result in adaptive offspring phenotypes in a context-dependent manner. Recently, evidence for this has been shown in a number of taxa. In free-living red squirrels, Dantzer *et al.* (2013) showed that density-induced maternal stress increased offspring growth, potentially adaptively preparing offspring for surviving in a high-density environment. In common lizards, Bestion *et al.* (2014) found that predator-induced maternal stress increase offspring dispersal distance, an adaptive antipredator behaviour that likely would increase offspring

survival when facing a high-risk environment. *Bian et al.* further this field in its entirety, as the first study directly linking maternal stress-induced phenotypic responses in offspring with actual changes in fitness and population dynamics in a context-dependent manner. For example, they show that root vole offspring born to high-density, stressed mothers had high stress levels and low reproduction, but high overwinter survival. This phenotype was adaptive at high densities, and HH offspring maintained high-density populations throughout the study. Conversely, offspring born to high-density mothers were maladapted to living in low-density populations (HL offspring) and were unable to sufficiently reproduce and increase their population density. Thus, this study provides strong evidence supporting the hypothesis that maternal stress acts in a context-dependent manner, (mal)adaptively preparing offspring for their future environment.

Studies determining the adaptive potential of maternal stress often lack rigorous testing and assign significance following only short-term examinations of the proximate effects of offspring phenotype, without understanding these changing in a life history or environmental context (*sensu* Sheriff & Love 2013). This study of *Bian et al.* provides a complete picture of maternal stress effects; examining offspring phenotypic response to maternal stress, how these changes alter offspring fitness, and doing so in an environmentally relevant context. A key next step will be to determine how these experimental findings translate into real-world demography. As ecologists, we must continue to take a broad and integrated life-history perspective to fully understand the impact of maternal stress exposure. Within this framework, we must recognize the context in which maternal stress occurs, use biologically and species-relevant experimental manipulations in free-living systems, appreciate that variation in life histories will influence the adaptive potential of maternal stress exposure and appreciate that to fully understand the (mal)adaptive potential of maternal stress exposure it must translate into population-level changes.

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Michael J. Sheriff*

Department of Ecosystem Science and Management, Pennsylvania State University, University Park, PA 16802, USA

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