2011

Assessing the developmental stress hypothesis in the context of a reaction norm

John P. Swaddle
William & Mary, jpswad@wm.edu

Recommended Citation

Swaddle, J. P. (2011). Assessing the developmental stress hypothesis in the context of a reaction norm. Behavioral Ecology, 22(1), 13-14.

Assessing the developmental stress hypothesis in the context of a reaction norm

John P. Swaddle
Institute for Integrative Bird Behavior Studies, Biology Department, College of William and Mary, Landrum Drive, Williamsburg, VA, USA

Spencer and MacDougall-Shackleton (S&M hereafter) comprehensively reviewed published evidence to suggest that early life developmental conditions can play important roles in later life selection events. My intention here is to provide a constructive critique of how this "developmental stress hypothesis" is framed and studied, which may help guide further research.

First, there is a lack of clarity as to the developmental origins of the phenotypic variation under consideration. In the majority of their review, S&M refer to differences in song phenotype among individuals (i.e., different birds in separate experimental treatment groups) that develop in different environmental conditions (e.g., "stressed" and "control" groups). The resulting phenotypic variation, therefore, originates from genotypic (G) and environmental (E) differences, and potentially any G × E interaction. S&M also consider fluctuating asymmetry (FA) of morphological traits to reveal equivalent developmental processes due to a breakdown in developmental stability (DS). However, the phenotypic variation produced by DS originates within genotypes and within environments; hence, FA has fundamentally different developmental origins than among-individual and among-treatment comparisons of bird song.

It is useful to visualize these traits and sources of variation in the context of a reaction norm. Each function depicts the production of a phenotype by a particular genotype across an environmental gradient (Figure 1). Studies that examine different birds in separate environmental treatment groups compare the phenotypes of one set of genotypes in one environment with the phenotypes of another set of genotypes in another environment (shaded areas of Figure 1). The recorded differences result from a complex of genetic and environmental developmental factors. DS is the production of a particular phenotype by a particular genotype in a particular environmental condition (Zakharov 1981) and, therefore, developmental instability (estimated by FA) can be thought of as phenotypic noise around a single reaction norm; indicated as error bars in Figure 1.

With these developmental differences highlighted, it is relevant to ask what type of variation should we measure when assessing the developmental stress hypothesis. As the hypothesis posits that early developmental conditions may have significant later life effects for an individual, it is the among-environment and within-genotype variation that appears most important; it seems we need to reduce among-genotype effects in tests of the developmental stress hypothesis. Therefore, I propose the need for repeated-measures methods.

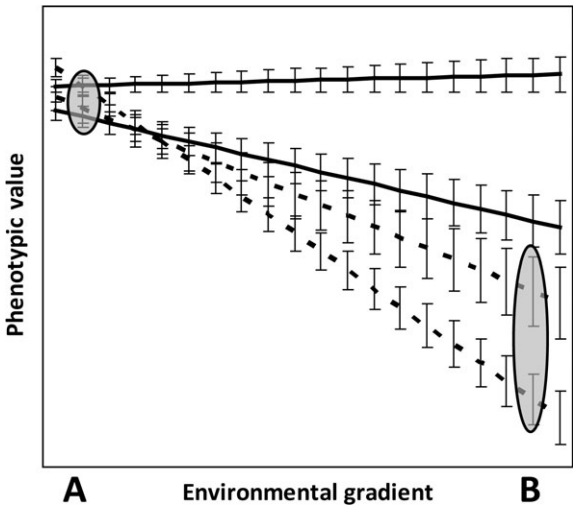

Figure 1
Hypothetical bundle of reaction norms, where each line represents the expression of a phenotype by a genotype across an environmental gradient. Only 4 lines are shown for simplicity; with 2 genotypes (solid lines) in a hypothetical "control" group environment A (indicated by shaded area on left) and another 2 genotypes (dotted lines) in a hypothetical "stressed" group environment B (indicated by shaded area on right). This example is intended to demonstrate the phenotypes produced by a typical experimental design reviewed by S&M and illustrate how the among-treatment differences in phenotype can be influenced by unhelpful among-genotype differences. The error bars depict developmental instability. In this example, developmental instability increases from environment A to B, as indicated by the generally increasing magnitude of error bars from A to B.

Fundamentally, we need to assess the production of a phenotype by the same genotype in different environments. As early development happens just once, this means the developmental stress hypothesis becomes rather intractable in many situations, especially in terms of studying bird song. However, in organisms that regrow structures (such as birds' feathers), it is feasible to assess the development of a phenotype in one environmental condition and then in another. In such studies, it may be beneficial to have a strong hypothesis about what the developmentally intended phenotype is, hence giving some directionality to the effects of stressors; therefore, I would recommend assessing FA, as we know that larger asymmetries are accidents of development (Møller and Swaddle 1998). For these collective reasons, studying the later life selection consequences of FA differences within individuals but across environments would be a fruitful way forward in assessing the applicability of the developmental stress hypothesis.

The reaction norm depiction of DS is also helpful in assessing whether DS is heritable and "visible" to selection. Fundamentally, the DS error bars must vary in magnitude among genotypes for DS to respond to selection pressures (Figure 1). It is also helpful to consider whether the size of the DS error bars for a signaling trait are large enough that receivers could reliably detect this variation (Swaddle 2003). As signal detection is seldom error-proof, the likelihood that DS can be an effective cue/signal will increase in proportion to the ratio of the phenotypic variation due to DS (size of the error bars) to the phenotypic variation that cannot be discerned due to detection error.

I am not aware that all the elements (measuring within-individual but among-environment phenotypic variation, showing that the variation is heritable and visible to selection and, of course, showing that there is later life selection) have been put together for any system; hence, there is much to be done in assessing the developmental stress hypothesis.

Address correspondence to J.P. Swaddle. E-mail: jpswad@wm.edu.

Received 2 February 2010; revised 2 February 2010; accepted 3 May 2010.

doi: 10.1093/beheco/arq089

REFERENCES

Møller AP, Swaddle JP. 1998. Asymmetry, developmental stability, and evolution. Oxford: Oxford University Press.

Swaddle JP. 2003. Fluctuating asymmetry, animal behavior, and evolution. Adv Study Behav. 32:169–205.

Zakharov VM. 1981. Fluctuating asymmetry as an index of developmental homeostasis. Genetika. 13:241–256.