

Biological Sensitivity to Context

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ABSTRACT—*Conventional views suggest that exaggerated biological reactivity to stress is a harmful vestige of an evolutionary past in which threats to survival were more prevalent and severe. Recent evidence, however, indicates that effects of high reactivity on behavior and health are bivalent rather than univalent in character, exerting both risk-augmenting and risk-protective effects depending on the context. These observations suggest that heightened stress reactivity may reflect increased biological sensitivity to context, with potential for negative health effects under conditions of adversity and for positive effects under conditions of support. From an evolutionary perspective, the developmental plasticity of the stress-response systems, along with their structured, context-dependent effects, suggests that variation in these systems has been adaptively patterned to increase the capacity of children to match their stress-response profiles to anticipated developmental environments. Taken together, these theoretical perspectives generate a novel hypothesis: that there is a curvilinear, U-shaped relation between early exposures to adversity and the development of stress-reactive profiles, with high-reactivity phenotypes disproportionately emerging within both highly stressful and highly protected early social environments.*

KEYWORDS—*stress reactivity; evolutionary psychology; developmental psychobiology*

Developmental psychologists frequently consider the effects of life experience on development but rarely consider how these effects have been structured by natural selection. Despite this oversight, the burgeoning field of evolutionary-developmental biology has exciting and profound implications for the study of human development (see especially West-Eberhard, 2003). Over the last two decades, theory and research in the field has come to acknowledge that, in most species, single “best” strategies for survival and reproduction are unlikely to evolve. This is

because the optimal strategy varies as a function of the physical, economic, and socioemotional parameters of one’s specific environment (Crawford & Anderson, 1989), and thus a strategy that promotes success in some environmental contexts may lead to failure in others. Selection pressures therefore tend to favor *adaptive phenotypic plasticity*, the capacity of a single genotype to support a range of phenotypes in response to particular ecological conditions that recurrently influenced fitness during a species’ evolutionary history. Importantly, the development of alternative phenotypes is a nonrandom process; that is, it is the outcome of structured transactions between genes and environments that were shaped by natural selection to increase the capacity and tendency of individuals to track their developmental environments and adjust their phenotypes accordingly.

We have recently proposed a developmental model of adaptive phenotypic plasticity in the human stress-response systems (see Boyce & Ellis, 2005). The model articulates the precepts and rationale for a new claim about the nature of relations between early life experience and stress reactivity, a claim that we have also explored empirically (Ellis, Essex, & Boyce, 2005). We contend that heightened stress reactivity may reflect not simply exaggerated arousal under challenge but, rather, a form of enhanced, neurobiologically mediated sensitivity to context, or *biological sensitivity to context* (BSC).

The logic of our argument can be summarized in the following way. Biological reactivity to psychological stressors comprises a complex, integrated system of central neural and peripheral neuroendocrine responses designed to prepare the organism for challenge or threat. Developmental experience plays a role, along with heritable variation, in calibrating the response dynamics of this system. Individual differences in such stress reactivity are thought to underlie broad variability in associations between stress and illness and to reflect constitutional variation in susceptibility to stressful challenge. Highly reactive phenotypes, in which affected individuals mount vigorous or sustained autonomic, adrenocortical (cortisol), or other biological responses to stressors, have been viewed as an atavistic health risk factor, a legacy of physiological responses more commensurate with the perils of prehistoric human environments. Often overlooked in such accounts is a body of anomalous observations revealing oppositional, counter-regulatory processes within the

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