Biological sensitivity to context: I. An evolutionary–developmental theory of the origins and functions of stress reactivity

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Abstract

Biological reactivity to psychological stressors comprises a complex, integrated, and highly conserved repertoire of central neural and peripheral neuroendocrine responses designed to prepare the organism for challenge or threat. Developmental experience plays a role, along with heritable, polygenic variation, in calibrating the response dynamics of these systems, with early adversity biasing their combined effects toward a profile of heightened or prolonged reactivity. Conventional views of such high reactivity suggest that it is an atavistic and pathogenic legacy of an evolutionary past in which threats to survival were more prevalent and severe. Recent evidence, however, indicates that stress reactivity is not a unitary process, but rather incorporates counterregulatory circuits serving to modify or temper physiological arousal, and that the effects of high reactivity phenotypes on psychiatric and biomedical outcomes are bivalent, rather than univalent, in character, exerting both risk-augmenting and risk-protective effects in a context-dependent manner. These observations suggest that heightened stress reactivity may reflect, not simply exaggerated arousal under challenge, but rather an increased biological sensitivity to context, with potential for negative health effects under conditions of adversity and positive effects under conditions of support and protection. From an evolutionary perspective, the developmental plasticity of the stress response systems, along with their structured, context-dependent effects, suggests that these systems may constitute conditional adaptations: evolved psychobiological mechanisms that monitor specific features of childhood environments as a basis for calibrating the development of stress response systems to adaptively match those 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.

Biological reactivity to environmental stressors is now widely implicated in the processes linking psychological adversity to psychiatric and biomedical disorder. The neuroendocrine changes that reliably accompany stressful events, in humans and other species, are the physiological, homeostatic means by which survival under threat is protected, but are also among the dysregulatory pathways by which psychological trauma is transmuted into pathogenic biological processes. Individual differences in such “stress reactivity” are thought to underlie the broad variability in stress–illness associations and to reflect constitutional variation in susceptibility to stressful challenge. Highly reactive phenotypes, in which affected individuals mount vigorous and/or persistent