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Toward an Evo-Devo Theory of Reproductive Strategy, Health, and Longevity: Commentary on Rickard et al. (2014)

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Abstract

Rickard and associates (2014, this issue) challenge the theoretical claim that early developmental experiences influence sexual development and behavior as a result of the continuity of early- and later-life environments over the course of human history (Belsky, Steinberg, & Draper, 1991). Instead, they contend that sexual development, health, and longevity are regulated by internal (bodily) state reflective of morbidity and mortality risk. By highlighting the importance of internal state—and thereby underscoring the value of focusing on it *and* on the external environment early in life—these theoreticians continue the tradition of extending a line of human evolutionary–developmental (“evo-devo”) theorizing in important ways. In fact, what they make clear is that what was originally conceived as an evolutionary theory of socialization by Belsky et al. (1991) can and should develop into an evolutionary–developmental life-course theory of reproductive strategy, health, and longevity.

Keywords

evolution, reproductive strategy, predictive adaptive response, psychosocial acceleration theory

Almost 25 years ago now, my colleagues and I advanced an evolutionary theory of socialization (Belsky, Steinberg, & Draper, 1991). We sought to explain not only *how* the broader ecological context of the family and intrafamilial dynamics regulated early and subsequent psychological and behavioral development, but also *why* development operated the way we theorized it did. Our theory of adaptive developmental plasticity (West-Eberhard, 2003) has come to be characterized in terms of *psychosocial acceleration* (Ellis, 2004), meaning that it addresses the contextual regulation of *rate of development* (i.e., speeding up, slowing down) in the service of (reproductive) fitness goals.

In the time since we endeavored to recast traditional socialization theory in an evolutionary perspective, numerous efforts have been made to expand, extend, revise, and enhance our theoretical model (for review, see Belsky, 2007). Thus, “fellow evolutionary-developmental travelers” have highlighted the need to distinguish (a) paternal and maternal influence (Ellis & Garber, 2000), (b) environmental harshness and unpredictability (Ellis, Figueredo, Brumbach, & Schlomer, 2009), and (c)

more and less consistent contextual cues (Frankenhuis & Panchanathan, 2011), while underscoring the importance of (d) extrinsic mortality and morbidity (Chisholm, 1999), as well as (e) future orientation (Chisholm, 1993); (f) the differential susceptibility of individuals to environmental influence (Belsky, 1997, 2000, 2005; Belsky & Pluess, 2009) (g) the differential development of boys and girls (James, Ellis, Schlomer, & Garber, 2012), perhaps especially in middle childhood (Del Giudice, 2009); and (h) the role of the stress-response system in the contextual regulation of reproductive strategy (Del Giudice, Ellis, & Shirtcliff, 2011). In continuing this tradition of theoretical development, Rickard and associates’ (2014, this issue) contribution makes clear that psychosocial acceleration theory can and should develop into a more general evolutionary–developmental (“evo-devo”) model

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of reproductive strategy, health, and longevity. Their thinking thus complements Ellis, Del Giudice, and Shirtcliff's (2013) recent evolutionary analysis of allostatic load (i.e., "live fast and die young").

In many respects, psychosocial acceleration theory has much in common with traditional socialization theories (e.g., attachment theory, social learning theory, life-course sociology theory), but it uniquely predicts that environmental factors, forces, and mechanisms long thought to shape psychological and behavioral development also influence somatic development, most notably pubertal timing, all in the service of reproductive-fitness goals. And as Rickard and associates make clear, much work has proved consistent with psychosocial acceleration theory, including its critical puberty prediction (for review, see Belsky, 2012).

Nevertheless, these authors raise questions about the evolutionary dynamics underlying the now well-established links between developmental experiences and environmental exposures early in life and subsequent sexual development and reproductive functioning. Most important, they challenge the notion that probabilistic connections between early- and later-life environments over the course of human evolution account for the evolution of what are today conceptualized as *predictive adaptive responses* (Gluckman, Hanson, & Spencer, 2005)—that is, that evolution equipped individuals to monitor the early environment in order to make "weather forecasts" (Bateson, 2008) of the future so as to guide development in the service of fitness goals. Instead, Rickard and associates argue that it is internal (bodily) state reflective of morbidity and mortality risk, which is often—but not always—a result of early experiences, that regulates sexual development, reproduction, health, and longevity.

Significantly, the authors hedge their bets as to whether they are advancing an alternative explanation of what have been conceptualized as predictive adaptive responses or amending and extending prevailing evo-devo ideas:

Our model makes the single *modification* [emphasis added] of the argument that one functional reason future prospects are poor where childhood environment is harsh may be the detrimental effects of harshness on the developing body. The process we propose involves individuals adapting not, *or not only* [emphasis added], to their future external environment but rather to their own bodies (or internal state). We thus uphold some of the contentions of Belsky et al. (1991) and others but provide an alternative, *or additional* [emphasis added], reason for why those contentions may hold. (Rickard et al., 2014, p. 11)

The ambivalence expressed here seems wise in light of the authors' own recent mathematical modeling of the likelihood that the developmental processes under consideration could have evolved through natural selection. Notably, Nettle, Frankenhuys, and Rickard (2013) compared two prediction models, one emphasizing only the external environment and the other focusing on both the external environment and internal state, and found clear support for the latter. If nothing else, such results underscore the utility of integrating theoretical ideas rather than presuming them to be incompatible alternatives.

In fact, one of the problems with pitting external-environment and internal-state models against each other, as Rickard et al. do at times, is that it risks misrepresenting the complexity of the developmental processes under consideration. Perhaps most important, although the issue of environmental stability in ancestral human environments is central to the plausibility of any predictive-adaptive-response framework, environmental stability, at least in the terms of psychosocial acceleration theory and its derivatives, does not only concern contextual features and factors beyond an individual's control, such as climate. Developmentalists have long appreciated that humans *select and create* their own environments—they are not just passive recipients of them—and this has surely been so ever since our species emerged on the African savanna. Environmental harshness, then, could prove stable—and thus developmentally informative in its own right—not just because the number of hostile competitors remains the same over time, but because the early experience of abuse and/or exposure to other adversities leads one to attribute bad intent to others, to hit first and ask questions later, and to thereby create contextual stability in environmental hostility even in a seemingly changing world. Moreover, such transactional person-environment processes could easily shape and be shaped by internal state.

More attention needs to be paid to such dynamics when questioning the bases of predictive-adaptive-response models of human development. In fact, given well-established causal links between external environment and internal state, any (nonexperimental) effort to evaluate their relative importance in regulating rate of development will need to control for the other when making such determinations. Further, given Belsky et al.'s (1991) speculation that the hypothalamic-pituitary-adrenal axis would play a role in mediating environmental effects on pubertal timing and the now extensive evidence linking cortisol regulation with a host of health-related phenomena (e.g., inflammation), there is no real theoretical basis for Rickard et al.'s claim that only their model would predict that childhood adversity precedes somatic damage, which itself precedes accelerated maturation.

Even if Rickard et al. have not convinced this reader that internal-state prediction represents an alternative to external-environment prediction—and they seem, as already noted, rather ambivalent about this notion themselves—these scholars should be applauded for highlighting the contribution of internal state in regulating rate of development while also endeavoring to integrate evolutionary thinking focused on the development of reproductive strategies with that concerned with health and longevity. In fact, what their efforts and those of Ellis et al. (2013) convincingly demonstrate is that my colleagues and I were not imaginative enough to think about physical health and longevity when it came to theorizing about how and why early developmental experiences shape human development. But should it turn out that we were correct in recasting thinking about socialization and human development in evolutionary, life-history, reproductive-strategy terms, even if for the wrong reasons (emphasizing the external environment rather than internal states), we can live with that!

Declaration of Conflicting Interests

The author declared no conflicts of interest with respect to the authorship or the publication of this article.

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