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Abstract

Most developmental work regards adverse developmental experiences as forces that undermine well-being. Here, I present an alternative—or complementary—view, summarizing recent evidence on puberty, endocrinology, cellular aging, and brain connectivity that collectively reveals developmental acceleration in response to contextual adversity. Findings are cast in evolutionary-developmental terms, highlighting the trade-off between accelerated aging and (a) increased morbidity and (b) premature mortality.

Keywords

adversity, childhood, development, acceleration, evolution, plasticity

Most developmental thinking regards adverse developmental experiences (e.g., harsh parenting) and environmental exposures (e.g., poverty) as factors and forces that undermine developmental well-being. And this is so whether thinking in terms of attachment theory, social-learning theory, life-course sociological theory, and other developmental perspectives. Here, I consider an alternative—or complement—to this prevailing viewpoint, contending that adversity—or at least certain kinds—can accelerate development (e.g., D. W. Belsky et al., 2017) while viewing relevant evidence through an evolutionary-developmental lens. *Accelerated* here does not mean precocious in any positive—or negative—sense of the term; it simply refers to developmental phenomena that occur earlier than otherwise would be the case.

In the view of evolutionary-developmental thinkers such as myself, the developmental acceleration highlighted here reflects one common adaptation to a range of recurrent childhood adversities frequently encountered in human evolutionary history. *Adaptation* as used here, then, refers to responses to select environmental conditions that have evolved through the process of natural selection as a result of their once—and perhaps ongoing—beneficial effect on the dispersion of genes in future generations (i.e., reproductive fitness). As a consequence, evolutionary adaptations may or may not be considered psychologically or culturally

beneficial. Having said that, there is every reason to presume that many well-documented psychological and behavioral responses to adversity enable one to better cope—or at least once did—with the world in which one finds oneself (Ellis & Del Giudice, 2019). In other words, such proximate adaptations should not be considered at odds with evolutionary adaptations.

The idea that adversity (i.e., experiences and exposures that are stressful and known to undermine physical and mental health) accelerates development, especially during adolescence, is not new. After all, it is widely appreciated that youth growing up in high-risk environments engage in several adultlike behaviors earlier than their more advantaged age-mates (e.g., drinking, smoking, sex). But rather than being treated as evidence of accelerated development, these ways of functioning are typically regarded as problem behaviors, no doubt because they are considered antisocial in nature while posing significant health risks. Even if this way of thinking is understandable from a traditional health perspective, it risks mischaracterizing and thus obscuring the nature of development as much as illuminating it.

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Evolutionary life-history theory calls attention to the regulatory effects of both early-life experiences and concurrent life conditions (Ellis & Del Giudice, 2019). Where it differs from traditional developmental models—and informs us—is in how it regards the nature of environmental effects. Rather than guided by an implicit, if not explicit, health model emphasizing “optimal” or healthy development versus dysfunction, dysregulation, or disorder, it casts environmental effects in adaptive terms. Effects of varying early-life conditions, then, are not considered inherently better or worse, so much as facilitating the dispersion of genes in future generations. Accordingly, under some conditions, certain responses to particular adversities are functional in terms of promoting reproductive fitness, even if not health and well-being. However, it should be noted that there are costs associated with such reproductive benefits.

J. Belsky, Steinberg, and Draper (1991) drew on this evolutionary perspective when positing that early-life adversity (e.g., marital conflict, hostile parenting) would not just foster an opportunistic, advantage-taking social orientation, consistent with traditional theories, but, uniquely, that it would also accelerate pubertal—and thus reproductive—development. Why? Because in a high-risk world, this should increase the chance of reproduction, the ultimate goal of all living things, before dying or having one's mate quality seriously compromised. Accelerated pubertal development, then, was considered a conditional adaptive strategy, enhancing survival and reproduction within expectable environments—or at least would have in ancestral times (J. Belsky et al., 1991; Ellis & Del Giudice, 2019). In other words, and consistent with more proximate perspectives on the effects of adversity on human development, including behavioral, psychological, and neuroscientific ones (Callaghan & Tottenham, 2016; Ellis & Del Giudice, 2019; Gee et al., 2013), accelerated development was conceptualized as a means of fitting the developing individual to his or her current and likely future environment.

Here, I summarize mostly very recent evidence of adversity-induced accelerations across developmental domains. I begin by summarizing puberty-related work that has appeared since a prior review of this research in this journal (J. Belsky, 2012) before turning to what remain mostly independent lines of inquiry—dealing with the endocrinological coupling of hormones, cellular aging, and brain structure—all in hopes of calling attention to the common theme of developmental acceleration in response to contextual adversity. Well appreciated, of course, is that if developmental conditions are extreme, perhaps threatening survival itself (e.g., calorie restriction, starvation), development may not be accelerated, as energy and resources go into maintenance rather than growth and reproduction (J. Belsky

et al., 1991; Ellis & Del Giudice, 2019). Indeed, this may explain why Sumner, Colich, Uddin, Armstrong, and McLaughlin (2019) found that early-life deprivation (e.g., physical or emotional neglect, food insecurity), but not threat exposure (e.g., child abuse, domestic violence), forecasts delayed pubertal development.

What this contrast reveals is that not all contextual conditions currently considered adverse by developmental scholars—such as poverty, sexual abuse, homelessness, food insecurity, harsh parenting, and maternal depression—can be presumed to accelerate development. Indeed, a challenge going forward will be to identify conditions that do and do not exert such effects. What should be appreciated, however, is that many of the developmentally accelerating early-life adversities to be considered also undermine physical and mental health (e.g., inflammation, psychopathology). Once again, then, complementary perspectives on the effects of early-life adversity—accelerating development and undermining well-being—should not be considered mutually exclusive. In fact, evolutionary-developmental scholars contend that they are fundamentally related, as I make clear in my concluding comments.

Pubertal Timing

Recent work linking early-life adversity with earlier age of menarche or Tanner stage has highlighted a variety of developmental stressors. Consider evidence that exposure to trauma in the first decade of life (Lei, Beach, & Simons, 2018) and sexual abuse (but not other stressors; Magnus et al., 2018) predict earlier age of menarche, as do frequent residential moves, themselves associated with reduced feelings of family support (Clutterbuck, Adams, & Nettle, 2015). Consider next the aforementioned Sumner et al. (2019) report indicating that early-life exposure to threat forecasts earlier pubertal development (using Tanner-stage measurements). Also noteworthy is meta-analytic evidence that father absence does the same (Webster, Graber, Gesselman, Crosier, & Schember, 2014; but for an alternative, narrative reading, see Sohn, 2017). In fact, Gaydosh, Belsky, Domingue, Boardman, and Harris (2018) chronicled such a father-absence effect even with a polygenic index of menarche controlled, while also discounting the possibility of gene–environment correlation. Permitting even stronger causal inference are the results of a natural experiment. It revealed that greater geographic proximity to the 2008 earthquake in China forecasted earlier pubertal development and that this effect was most pronounced when exposure occurred within the first 7 years of a girl's life (Lian et al., 2018), thereby proving consistent with J. Belsky et al.'s (1991) theorizing about the timing of contextual regulation of

reproductive strategy. Somewhat surprising, though, was that even male pubertal development (i.e., first ejaculation) was accelerated after earthquake exposure, a finding similar to Australian research linking lower socioeconomic status with earlier pubertal development in males and females—and in a dose-response manner (Sun, Mensah, Azzopardi, Patton, & Wake, 2017; see also Sumner et al., 2019).

The fact that children who experienced early institutional care did not evince accelerated pubertal development (e.g., Johnson et al., 2018) would seem to be consistent with the earlier observation that the reproductively strategic thing to do in the face of extreme deprivation is to devote energy to maintenance rather than growth and reproduction. As I will show, however, there is evidence that even institutional care is related to other indicators of accelerated development. Clearly, it will take future research to clarify which developmental systems are accelerated by particular adversities and which are not—and why.

Endocrinology

Pubertal development is a highly complex physiological process, indeed, one still not fully understood. Suggestive evidence indicates that the coupling of two hormones—cortisol, a product of the hypothalamic-pituitary-adrenal (HPA) axis, and testosterone, a product of the hypothalamic-pituitary-gonadal (HPG) axis—not only changes with development but also can be affected by early-life conditions. Although it is well established that cortisol and testosterone are inversely correlated in adulthood because of the reciprocal gonadal-hormone suppression of the HPA and HPG axes, two new findings seem especially noteworthy. First, such hormonal coupling changes from childhood to adolescence, going from positively to negatively correlated, especially in girls (Matchock, Dorn, & Susman, 2007). Second, this switch to the adult coupling pattern occurs earlier—and is more pronounced—for girls exposed to early-life adversity (i.e., parental depression, family anger) than for nonexposed age-mates (Ruttle, Shirtcliff, Armstrong, Klein, & Essex, 2015).

Cellular Aging

DNA has recently become the focus of research on biological aging. Two different indicators of cellular aging provide evidence of the accelerating effects of adversity.

Telomeres

Telomeres play a critical role in the maintenance of chromosomal integrity. They consist of repeated DNA sequences that cap and protect eukaryotic chromosomes.

With increasing age, telomeres shorten substantially, thus making telomere length a biomarker of biological aging (J. Belsky & Shalev, 2016). Behavior-genetic evidence further indicates that environmental forces are the dominant influence on postnatal telomere length (Hjelmberg et al., 2015). Especially notable, then, are repeated findings that *prenatal* stress predicts shorter telomeres at birth (e.g., Send et al., 2017), as does growing up in an extremely deprived Romanian orphanage (e.g., Humphreys et al., 2016). Most compelling, though, are longitudinal findings from Shalev and associates (2013) that exposure to violence in middle childhood forecasts accelerated erosion of telomeres from the age of 5 to 10 years.

Epigenetic aging

Recent epigenetic research has identified a set of DNA methylation markers that can be used to estimate chronological age with great accuracy ($R = .96$; Hovath, 2013). Of interest, then, is evidence that exposure to violence is associated with accelerated epigenetic age among 6- to 13-year-old African American children (Jovanovic et al., 2017) and ethnically diverse 8- to 16-year-olds (Sumner et al., 2019), as is childhood sexual abuse when methylation is measured in middle age (Lawn et al., 2018). It is conceivable that Zannas and associates (2015) failed to document similar results in their study of African American adults because they relied on retrospective assessments of childhood experience, recollections that are demonstrably prone to error.

Brain Development

Neuroscience also provides evidence of accelerated development following early-life adversity, this time involving connections between the amygdala and prefrontal cortex (PFC), particularly medial regions. In three studies, youths with histories of early caregiving adversity (i.e., institutional care, traumatic experiences, harsh parenting) exhibited more adultlike profiles of amygdala-PFC connectivity than age-mates who did not have such histories (Colich et al., 2017; Gee et al., 2013; Thijssen et al., 2017). These profiles were themselves linked to adultlike behavior (e.g., less developmental anxiety; Gee et al., 2013).

Conclusion

Human development is a long and slow process. The biological plasticity afforded by this long period of immaturity allows us to respond to environmental cues. As documented here, there is repeated indication across a number of biological systems that maltreatment,

exposure to poverty, growing up in a conflicted family or a violent neighborhood, or perhaps worse, institutional caregiving can accelerate certain aspects of development. In light of such findings across different indicators of biological aging, it comes as somewhat of a surprise that many such biomarkers are themselves not strongly related to one another, at least in midlife (D. W. Belsky et al., 2018). It will be important to determine whether the same proves true in childhood and adolescence. Only investigations that measure a variety of potential indicators of accelerated development will afford insight into this issue.

Despite the evidence summarized linking early-life adversity and accelerated development, it should be appreciated that effect sizes under consideration are often small. This, along with recent theory regarding differential susceptibility to environmental influences (J. Belsky & Pluess, 2013), raises the possibility that some children are more susceptible to the accelerating effects of adversity than others. And, significantly, there is repeated evidence to this effect in the case of both pubertal timing (e.g., Hartman, Widaman, & Belsky, 2015) and telomere length (e.g., Mitchell et al., 2014). Such findings underscore the need to consider organismic factors (e.g., temperament, genetics, physiology) and environmental forces (e.g., parenting, early intervention) that might moderate accelerating effects of adversity—by amplifying or mitigating them.

Much research further indicates that accelerated biological development is itself associated with increased morbidity and mortality risk later in life, especially that which involves pubertal development (e.g., Day, Elks, Murray, Ong, & Perry, 2015) and cellular aging (e.g., Bojesen, 2013; Marioni et al., 2015). This is notable in view of extensive evidence that many of the developmentally accelerating contextual conditions highlighted here are themselves related to compromised health in adulthood (e.g., inflammation, cancer, cardiovascular disease; Rasmussen et al., 2019). These observations raise the question of why development would operate this way—being accelerated in childhood in the face of adverse conditions and associated with later-life health risks. Because of the central importance of reproduction, evolutionary-minded theorists interpret these apparent consequences of growing up under conditions of adversity as evidence of a trade-off, not simply as adversity-induced wear and tear on the developing individual. Compromised health and longevity, evolutionary-developmental thinkers contend, are a cost that natural selection has imposed—or at least accepted—in exchange for the benefit of increasing the chances of reproducing (e.g., J. Belsky & Shalev, 2016; Ellis & Del Giudice, 2019).

Seemingly consistent with this claim is Binder and associates' (2018) recent discovery that a “faster ticking rate of the epigenetic clock,” which we have seen can be induced by adversity, “is associated with faster pubertal development in girls,” which itself is associated with increased morbidity (e.g., reproductive cancers). Then there is the well-established fact that early pubertal development itself predicts earlier engagement in sex (e.g., J. Belsky, Steinberg, Houts, Halpern-Felsher, & the NICHD Early Child Care Research Network, 2010). A question that arises in light of these observations is whether any of the nonpubertal accelerated developments considered here causally influence reproductive strategy (i.e., pubertal timing, sexual debut, mating).

Given the preceding evolutionary-developmental analysis, the accelerated developments considered here should reinforce efforts to reduce children's exposure to adversity, while also encouraging us to think differently about human development. For too long we have viewed children through the lens of the Enlightenment rather than of evolution. Children have not evolved to be secure, curious, autonomous, and goal-oriented—unless contextual conditions or genetic makeup have inclined them to do so. Under other developmental and genetic conditions, children should—and do—develop differently. Often these alternative ways of functioning are evolutionarily, biologically, and even psychologically sensible and strategic, not disturbed or disordered, even if this is not widely appreciated or in line with more traditional ways of thinking. All of this is not to say that there are not true and difficult behavioral and mental-health struggles with which children may contend if exposed to early adversity. Nevertheless, treating accelerations as “natural” and evolved responses to certain adverse developmental experiences and exposures, just like many delays and other developmental difficulties, brings us closer to understanding the nature of development. So if we do not want this evolved developmental wisdom to manifest itself—in accelerated development and poor physical and mental health as a result of particular early-life adversities—then we need to change the contextual conditions that give rise to it.

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Action Editor

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Declaration of Conflicting Interests

The author(s) declared that there were no conflicts of interest with respect to the authorship or the publication of this article.

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