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Special Issue Article

The Future of Developmental Psychopathology: Honoring the Contributions of Dante Cicchetti

The nature of nurture: Darwinian and mendelian perspectives

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Abstract

Two perspectives on the nature of nurture are reviewed, one Mendelian and the other Darwinian, in an effort to draw links between the two and, thereby, integrate them in a developmental modern synthesis, mirroring the one that took place in biology early in the last century. Thus, the heritability of environmental measures and gene-X-environment interaction are discussed with respect to Mendelian nature before turning attention to Darwinian nature and thus the development of reproductive strategies and differential susceptibility to environmental influences. Conclusions are drawn with respect to both frameworks indicating that it is time to abandon the biology-is-destiny resistance to both approaches to studying and thinking about development, especially when it comes to the nature of nurture. Implications for the future development of the field of developmental psychopathology are highlighted.

Keywords: evolution; genetics; nature; nurture

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It is typically the case when the subject of nature and nurture are discussed that nature refers to Mendelian nature, having to do with the extent to which differences in individuals' genetic make-up systematically map on to differences in their development, cognition and behavior, what geneticists term penetrance. Darwinian nature, in contrast, is about adaptations over the course of human ancestral history that fostered, directly or indirectly, survival and reproduction, all in the service of passing genes onto subsequent generations, termed "reproductive fitness". Since it is genes that are selected in the process of such natural (and sexual) selection, they also figure prominently in Darwinian nature.

What is perhaps surprising given how non-overlapping these two streams of nature-of-nurture inquiry are in the developmental sciences is how such "separateness" flies in the face of the Modern Synthesis which emerged in biology and thus the life sciences quite some time ago now.[1] This development which occurred in the early to mid-20th century refers to the integration of what had been separate ways of looking at life, specifically, Darwinian natural selection and Mendelian genetics. It created a comprehensive framework for understanding, among other things, how evolution works at the molecular level via the selection of genes that contribute to adaptations that themselves foster, again, directly or indirectly, the dispersion of genes in future generations.

Despite this historic and fruitful integration of Darwinian and Mendelian nature, it never seems to have impacted, at least in any major way, developmental science. Especially when it comes to the study of the nature, origins and consequences of individual differences in human development, so central to much of developmental science today, it would not be misguided to observe that one of the two contributing elements of what has also been referred to as the Neo-Darwinian Synthesis has been treated as a step sister to the other (Belsky, in press). Even if the influence of genetics in shaping development remains subject to debate, there can be no question that Mendelian genetics has figured prominently in developmental science for many decades. This was true when the field relied exclusively on behavior-genetic designs (involving identical and fraternal twins or adopted and biological children) to estimate heritability and, more recently, as molecular-genetic inquiry became possible and attention turned first to genotype-phenotype associations based on candidate genes and subsequently to genome-wide association studies (GWAS).

To my reading of the developmental literature, the same level of attention has not been accorded evolution and thus Darwin's theory of natural (and sexual) selection, at least when it comes to accounting for individual differences in human development. This is not to say that the topic has been totally ignored. Introductory child development textbooks have long heralded the Bowlby (1969, 1973) claim that attachment evolved because it fostered survival of our long-ago ancestors. Never fully appreciated by Bowlby or even by many developmental scholars today is that survival alone does not get the evolutionary job done unless it furthers the

[1]As I have argued in these pages before (Belsky & Pluess, 2013), the study of how we humans develop should also be regarded as a life science, no longer (just) a social science. This would seem obvious given how much research dealing with development and psychopathology focuses on genetics, epigenetics, inflammation, biological aging, the brain, with, no doubt, so much more to come (e.g., the microbiome).

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fundamental goal of all living things, namely, the dispersion of genes to descendants. Also still misunderstood is that neither survival nor reproduction is, from a modern evolutionary perspective, about benefits to the species, as in "survival of the species". That turns out to be "just" the collective byproduct of the selection of genes that benefit the reproductive fitness of the *individuals* carrying them. Nor is modern evolutionary understanding just about genes inherited by one's children and linear descendants (i.e., grandchildren, great grandchildren . . . .). Inclusive-fitness thinking makes clear that genes shared by collateral kin (e.g., uncles, cousins) have also proved important to the process of evolution by natural selection (Dawkins, 1976; Hamilton, 1964).

In this paper I once again seek to underscore the importance of a modern evolutionary perspective for understanding human development and specifically the nature of nurture. I do this by highlighting the legacy of two papers, each now more than three decades old. One, by Plomin and Bergemen (1991), informatively titled "The Nature of Nurture," was based on Mendelian nature and the other, by myself and two colleagues, was based on Darwinian nature (Belsky et al., 1991). Here I consider the legacy of each, while endeavoring to integrate them, hopefully moving our field toward a developmental modern synthesis. In so doing, I draw implications of such a synthesis for the future of the study and understanding of developmental psychopathology.

Mendelian nature

It would not be misguided to claim that it almost doesn't matter what psychological, behavioral or developmental phenotype one is interested in if one wants to find evidence of Mendelian-genetic effects. They are simply that ubiquitous (Bourchard, 2004). In what follows, I first revisit evidence that nurture is also heritable before revisiting additional evidence that genetic influence is contextually variable, along with its implications for understanding the nature of nurture.

Heritability of nurture

Plomin and Bergemen (1991), in reviewing nowhere near as much evidence as exists today, made a compelling case that many if not most measures developmental scholars, including myself, routinely rely upon to index nurture and thus chronicle the role of the environment in shaping development prove strikingly heritable. That is, they tap into Mendelian-genetic nature, not just the (non-genetic) environment as so long and still assumed. This proved true when it came to adults recalling their childhood environments (Plomin et al., 1989); parental ratings of their own parenting (Loehlin & Nichols, 1976; Rowe, 1983); parental reports and observations carried out in the home (Plomin et al., 1988) using the HOME inventory (Caldwell & Bradley, 1978); videotaped observations of mother-child interaction (Dunn & Plomin, 1986); as well as the putative environmental indices of SES (Fulker & Eysenck, 1979), parental education (Taubman, 1976), television viewing (Plomin et al., 1990), peer characteristics (Daniels & Plomin, 1985), social support (Bergeman et al., 1990) and life events (Plomin et al., 1990). Recent work expanding on this foundation further indicates that adult social media use is also heritable (Ayorech et al., 2023). Whether that is so in the case of children and adolescents remains to be determined, though it seems likely that it will also prove to be heritable.

But it is not just behavior-genetic research, much of it quite old, that makes clear that environmental exposures are not randomly assigned but apparently reflect genetic selection, at least to a not insubstantial degree. So does recent molecular-genetic inquiry. Consider first evidence from the well-known Dunedin Multidisciplinary Health and Development Study being conducted in New Zealand. It indicates that a widely studied subset of genes on which individuals vary, one initially found to be related to educational attainment but since to many other aspects of health and behavior, predicted how warm, sensitive and stimulating both mothers and fathers were observed to be when videotaped interacting with their three-year olds in their homes (Wertz et al., 2019). Notably, the same polygenic index also predicted how cognitively stimulating was the home environment itself, in terms of the availability of things like books and toys, with similar results emerging in the E-Risk Study carried out in the United Kingdom (Wertz et al., 2020).

That consideration of such Mendelian nature of nurture is important when seeking to illuminate parenting effects is made especially clear in research that included four additional cohort studies from the UK and USA to complement those already mentioned, resulting in a focus on more than 36,000 parents (Wertz et al., 2023). That it also included data on children's as well as parents' genetic make-up enabled the investigatory team to discount direct transmission of genes from parents to children, thereby affording evaluation of the effect of parents' genetic make-up on their parenting that could not be attributed to any evocative effect of children's genetic make-up on the parenting which children experienced. Results indicated, among other things, that those parents carrying more gene variants associated with their own educational attainment, relative to those with fewer such variants, provided more supportive and stimulating parenting during their offspring's childhood and adolescence. Given the control for child genetic make-up, what this work further revealed was that genetic influence of parents on their children could be environmentally mediated.

Gene-X-environment interaction (GXE)

The illustrative evidence documenting the Mendelian nature of nurture just summarized should not be read to imply that any research documenting the heritability of some phenotype, including parenting, means that its findings can be glibly generalized across time, place and populations, or really just the samples studied. All too often in the developmental and psychological sciences this fundamental truth of heritability research is not made crystal clear, as it should be.

Perhaps the best evidence that genetic penetrance is not fixed in stone just because someone inherits the relevant DNA for a particular phenotype can be found in GXE research documenting the role of the environment in influencing or moderating the strength of genetic effects. Given the importance of reproduction when considering Darwinian nature below, let me start by calling attention to GXE evidence related to age of first sexual intercourse and age of first birth and the importance of time in moderating genetic effects. One early inquiry which involved questioning adults in 1993 about this subject while comparing those younger than 40 with those over 40 years of age, revealed that genetics influenced these reproductive phenotypes to a greater extent in the younger and thus more recent cohorts than in older ones (Dunne et al., 1997). The same trend of increasing heritability over time emerged when, in a more recent investigation, the focus of a molecular-genetic inquiry was age at first birth of women born before 1940, in 1940 and in 1965 (Mills et al., 2021). While the specific environmental features responsible for such temporal variation in heritability was not empirically addressed, one causal

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possibility is that as a more laissez-fair social climate took hold, genetic penetrance increased.

Let's turn now to family-related and lived experiences that also appear to moderate or differentially affect the magnitude of genetic and thus non-genetic effects. When it comes to the heritability of age of sexual debut, research indicates that genetic influence is greater and shared environmental effects smaller when sibling pairs have little social contact with one another than when highly involved in each other's lives (Hunt & Rowe, 2003). Turning to age of first consensual intercourse, other work reveals that the heritability of this phenotype in the case of sexual-abuse victims is reduced relative to their non-abused counterparts (Waldron et al., 2008). Sadly, this does not seem surprising, while implying that sex abuse does not reflect a genetically mediated and evocative child effect.

But it is not just features of reproductive strategy whose heritability varies by contextual conditions and thus nurture. This also appears to be so with respect to perhaps the most controversial topic in the study of genetic influence, the Mendelian genetics of intelligence. Being referred to here is the GXE work of Erik Turkheimer which underscores the moderating effect of population. In a series of related behavior-genetic investigations carried out in the USA, he and colleagues discovered that genetic penetrance varies as a function of family socioeconomic status. In one relevant inquiry, evidence indicated that whereas young-children's IQs appeared strongly influenced by their genotypes if they were growing up under conditions of socioeconomic advantage, that proved to be much less the case for children living in socioeconomically disadvantaged ones (Turkheimer et al., 2003). Similar results emerged in a second inquiry focused on the cognitive abilities of even younger children just two years of age (Tucker-Drob et al., 2011), as well as in a third study of adolescents (Harden et al., 2007).

Results such as these raise the question of whether the differential heritability of intelligence of children growing up in more and less socioeconomically advantaged families would emerge when place is the moderating focus. One team of investigators hypothesized that this would be less likely in countries in which there exists a greater social safety net than in the USA. And that is exactly what emerged when the heritability of IQ was evaluated in Sweden, Great Britain, the Netherlands and Australia, all so-called "socialist" nations when compared to the USA (Tucker-Drob & Bates, 2016). Thus, the GXE interaction evident in the USA did not replicate overseas. It would be a mistake, of course, to classify this situation as a "failure to replicate." And this is because it underscores the influence of context in shaping genetic penetrance.

Many developmentalists interpret the genetics-of-IQ research just summarized in terms of the Bronfenbrenner and Ceci's (1994) bioecological model of how nature and nurture interact, emphasizing the role of the environment in fostering an individual's *genetic potential* or failing to and thus whether a person can become all that she could be. But, as will become evident when Darwinian nature becomes the focus of attention, this reflects a rather romanticized view of human development. After all, would the same interpretation be applied if the phenotype to be explained was criminality, which evidence also indicates is heritable (e.g., Kendler et al., 2015)? So, for example, if research revealed that growing up under conditions of adversity led to greater penetrance of would-be criminality genes than growing up under conditions of safety and security would there be reference to realizing one's (criminal) potential? I doubt it.

But there is no reason to just take my word for it, at least in the case of children growing up in more and less socioeconomically advantaged families. A Norwegian study of more than 28,000 children made no reference to the idea of "realizing genetic potential" or of "becoming all that you can be" upon discovering that the heritability of behavioral problems such as aggression, disobedience and delinquency proved greater for those from more disadvantaged households than more advantaged ones (Badini et al., 2023).

The preceding consideration of Mendelian nature makes two things clear, while raising a provocative alternative interpretation of GXE findings. The first point is that presumed indices of the environment are themselves genetically compromised when it comes to evaluating environmental effects. The second is that biology in the form of DNA need not be destiny, given how environmental conditions can moderate genetic penetrance. But given this reality, doesn't that seem to imply that the documentation of Mendelian genetic effects reflects not so much the inevitable influence of DNA on human development, thinking and behavior, as so often presumed, but rather *a reflection of the environmental influence*? After all, if the heritability of a phenotype is not fixed in stone, such that genetic penetrance can be greater or lesser in one context than another, why shouldn't the conclusion be that what an index of heritability reflects is the following environmental effect: the degree to which *the developmental context in question affords the penetrance of genes*? Think about it.

Darwinian nature

Perhaps a good example of the need for and potential benefit of considering both Darwinian and Mendelian perspectives when it comes to understanding human development can be found in a long-ago study on the heritability of television watching (Plomin et al., 1990). To appreciate why this is the case it helps to recognize that like so much of developmental science today, the study of genetic influence is concerned with the how of development, whereas an evolutionary perspective is concerned with the why of development (Belsky & Pluess, 2013; Ellis et al., 2022).

What was not even considered in the television-watching work, just as remains true of so much heritability research to this day, is why genes for such a phenotype (and many others) would even have been selected into the human gene pool. Needless to say, such selection occurred long before television was even imaginable. Even if any answer to the question posed would be just speculative, some do come to mind and, it seems to me, merit consideration when documenting and discussing the heritability of behavioral and developmental phenotypes.

It is not hard to imagine that historically there could have been survival and reproductive benefits, especially via monitoring prey or invading enemies, for being able to sit still for lengthy periods of time without moving much–just like when watching TV–while just observing the world from some vantage point. Or consider the opposite tendencies that might help to explain the Mendelian genetics of ADHD. Might our ancestors have benefited from some individuals being genetically inclined to have trouble staying still and focused because such proclivities might have made them ideal for moving around and monitoring their tribe's or clan's borders? If nothing else, what these speculations illustrate is the potential utility of considering both how and why questions in developmental science. Why are our journals filled with estimates of heritability with virtually so little, if any, consideration of why there seem to be genes for particular phenotypes in the first place?

Many readers likely know that my own fascination with evolutionary-developmental (evo-devo) analysis was stimulated by a report by two anthropologists reinterpreting effects of father

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absence (Draper & Harpending, 1982). This introduced me to the claim that the passing on of genes to future generations is the fundamental purpose of life and that effects of nurture can reflect evolved and strategic responses to alternative early-life conditions, all in the service of this goal. In this section I first review the effect this insight had on my own thinking and, thereby, my own research and that of others, before moving on to an evolutionary challenge I confronted which led me to modify such evo-devo thinking.

The development of reproductive strategies

What the father-absence paper made me question was the implicit assumption so central to much developmental science, namely, that development is about well-being and even, apparently, the perfectibility of man. This seems so whether one considers Freud's claim that mental health involved the proclivity "to love and to work;" Maslow's assertion that fundamental needs change over time from initial biological and physiological ones required to sustain life (e.g., food, clothing, housing) to subsequent needs for belonging, for status and eventually for personal growth and fulfillment, that is, self-actualization; Erikson's eight stage model of development from basic trust in infancy to ego integrity in the aged years; and much theory and research on attachment stipulating that early security lays the foundation for later curiosity, autonomy, emotion regulation, resilience, capacity for intimacy, and sensitive parenting. In all cases, if and when such "optimal" development fails to progress, it is disturbance, dysfunction and disorder that is claimed to arise.

An evolutionary analysis challenges this view because what emerges when such idealized development fails to occur is not regarded as inherently problematic, even if at odds with prevailing Western, middle-class, humanitarian values. Because adversity has likely characterized human childhood since time immemorial, our species, like many others, is presumed to have been shaped by natural selection to adjust developmental responses to early-life conditions in ways that increased the probability of passing on genes to future generations. In other words, so-called problematic development is often an evolved response for making the best of a bad situation (Belsky et al., 1991; Del Giudice et al., 2015; Draper & Harpending, 1982), which is why some call attention to the previously unrecognized "hidden talents" of those growing up under conditions of adversity (Ellis et al., 2020; Frankenhuis and de Weerth, 2013; Frankenhuis & Nettle, 2019). Even if the ultimate fitness benefits of responding developmentally to adversity in particular ways are no longer realized by pursuing an alternative reproductive strategy or life history, the presumption is that the neurobiological "machinery" that once got that job done remains operative today, affecting how children still respond to the nurture they experience.

However attractive I initially found the reinterpretation of father-absence effects when cast in such evo-devo perspective, I remained to be convinced that it was more than just "old wine in a new bottle." And this was because such radical reframing of the nature of nurture–that it evolved to foster reproductive fitness and not just health, wealth and happiness–did not advance any novel, theory-distinguishing predictions. So why, then, embrace what seemed like a less parsimonious explanation–Occam's razor–of the way nurture shapes development than standard developmental ones? This concern eventually stimulated my "puberty hypothesis" which, when first supported, led me and my colleagues to advance an evolutionary theory of socialization (Belsky et al., 1991), often referred to now as psychosocial acceleration theory. I have always been uncomfortable with that label because the theory is not just about adversity accelerating development in the service of reproductive goals rather than well-being ones, but about more general contextual regulation of development. That is why what I referred to as quantity and quality (of children) reproductive strategies are today referred to as, respectively, fast and slow life histories.

The evolutionary theory of socialization built on existing evidence reasonably accounted for by standard development thinking, though offering an uncanny prediction that had never before been advanced and could not be explained by traditional ways of thinking should it prove true. In line with social learning theory, attachment theory and sociological life-course theorizing, the evolutionary theory predicted that supportive and adverse early-life conditions would, respectively, lead to mutually-beneficial or opportunistic-exploitative social orientations in childhood, later or earlier sexual debut, stable-enduring or serial-changing intimate relationships, supportive or unsupportive parenting, as well as fewer-better-cared-for or more-poorly-cared-for children.

Its theory-distinguishing prediction, however, was that that early-life adversity would also accelerate pubertal development, thereby increasing the chance that the developing child would be positioned to reproduce before becoming seriously compromised or, as subsequently made clear by Chisholm et al. (1993), dying. In contrast, if nurturance and support characterized early life, then rather than fostering a quickly developing reproductive strategy (i.e., fast life history), a slower one would emerge, enabling the developing child to embody the multiple resources to which he or she was likely exposed (e.g., psychological, educational, nutritional). The latter would enhance future mate value and ability to support children, thereby enhancing their eventual fitness, along with that of their parents.

The ultimate reason why the two reproductive strategies were hypothesized to yield differences in terms of number of offspring was the greater risk to the survival and capabilities of children growing up under conditions of adversity. Whereas a quality-of-offspring reproductive strategy or slow life history might require only two children, for example, to eventually yield four grandchildren, the quantity reproductive strategy or fast life history might require four children to yield the same reproductive success. This is the same logic that explains why, at the species level, mosquitoes bear numerous offspring – because most are likely to die before passing on their genes but lions bear just a few.

Over the past 30 years any number of studies have provided support for the puberty hypothesis central to psychosocial acceleration theory, even if the theory is about so much more than that. Let me provide some examples. Before doing so, I should make clear that too many still regard it as a theory exclusively about father absence and pubertal timing, when that is by no means the case, as should become apparent shortly.

It was in the previously mentioned Dunedin Study that I first tested and confirmed the puberty prediction, linking father absence as well as high levels of family conflict within the first seven years of life, the sensitive period stipulated in the theory, with earlier age of menarche (Moffitt et al., 1992). Subsequent work by Ellis and Essex (2007) found that family unsupportiveness during the preschool years reflecting authoritarian parenting and negative family relationships predicted advanced development of secondary sex characteristics, such as breasts, in fifth-grade females. Evidence also emerged that in a Danish study of some 16,000 children that father absence in pregnancy and during childhood was associated

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with earlier pubertal development in girls (Gaml-Sorensen et al., 2021).There is also evidence from the Great Smokey Mountains' Study in North Carolina that maltreated girls reach sexual maturity earlier than do their non-maltreated counterparts (Costello et al., 2007).

Given recent work underscoring the need to distinguish experiences of threat and deprivation, it is also notable that whereas the former predicts earlier pubertal development in girls, the latter predicts slower maturation (Sumner et al., 2019). The latter result seems likely to be due to insufficient energetic resources to sustain even an average rate of development (Ellis, 2004). This should make evident that certain kinds of adversities may be necessary but not sufficient to accelerate reproductive development.

I would be remiss if I did not point out that not each and every inquiry that has sought to link early-life experiences and exposures with pubertal timing has yielded results such as those just highlighted. Of significance, then, are the results of a meta-analysis of 43 studies comprised of 46 independent data sets. It found that the following adverse childhood experiences were all reliably associated with one or more indices of girls' accelerated pubertal development: sexual abuse, physical abuse, child neglect, low socioeconomic status, father absence and family dysfunction (Zhang et al., 2019)!

However promising such results would appear to be, findings from the cited observational studies raise several interesting questions, including ones about boys, about actual reproductive behavior, and about Mendelian-genetic confounding. With regard to males, it remains the case that most investigations have not included them, principally because reliably assessing male sexual maturity is more challenging than it is in the case of girls. Whereas females can recall, even after decades, when they had their first period (Ellis, 2004), this is much harder for males when asked about, for example, their first ejaculation. Nevertheless, there is some evidence that boys, too, have their sexual maturation accelerated by exposure to child adversity. One study of an Australian national birth cohort found greater socioeconomic disadvantage to be associated with earlier pubertal development in males as well as females (Sun et al., 2017). Another investigation, this one longitudinal in design, documented much the same in detecting an association linking childhood trauma before the age of eight with accelerated pubertal development over the following two years (Lei et al., 2018).

Because psychosocial acceleration theory is, fundamentally, about the developmental regulation of reproductive psychology and behavior, the question arises as to whether there is evidence of a developmental cascade from early-life adversity to accelerated pubertal development to actual sexual behavior. The only study to my knowledge that has been positioned to address this issue central to the theory was conducted as part of another longitudinal study, The NICHD Study of Early Child Care and Youth Development (Belsky et al., 2010) Results revealed that the more mothers of preschoolers engaged in harsh parenting, the earlier were girls' ages of menarche and, as a result, the more "sexual risk taking" they engaged in (i.e., oral sex, vaginal sex, STD diagnosis). In fact, an effort to determine whether this developmental cascade might have been initiated even earlier indicated that attachment insecurity at age 15 months predicted girls' pubertal development in a manner in line with psychosocial acceleration theory (Belsky et al., 2010).

However compelling even the findings just presented may appear, there remains the elephant in the room: Could this all be a Mendelian-genetic masquerade? Perhaps it is just that the same genes that affect children's developmental experiences and environmental exposures, including their parenting, also influence pubertal maturation? A number of efforts to at least partially discount this possibility have still found evidence consistent with theoretical expectations upon controlling for maternal age of menarche (Belsky et al., 2010) and GWAS-derived polygenic scores for menarcheal age (D. Belsky et al., 2010; Gaydosh et al., 2018; Schlomer & Marceau, 2020) and age of first birth (D. Belsky et al., 2010). Another strategy that yielded evidence in line with psychosocial acceleration theory used sisters who were full sibs, thus sharing 50% of their genes, finding that, due to a parental breakup, it was younger ones–who spent less time in a father-present family than their older sibs–who sexually matured at a younger age (Tither & Ellis, 2008).

But perhaps the most convincing evidence that links early-life conditions and pubertal development is to be found in two natural experiments. The first sought to test the puberty hypothesis by comparing the development of two groups of Finnish citizens who were children during World War II when the Nazis invaded their country (Pesonen et al., 2008). Whereas the "experimental" group were evacuated from Finland, sent by their parents to live with temporary foster families in Sweden and Denmark in hopes of keeping them safe, the comparison group continued to remain at home with their parents. In light of the stress and fear likely induced by separation from parents, evidence indicated, consistent with expectations, that the female evacuees experienced menarche at an earlier age and, perhaps even more important from a fitness standpoint, bore more children than did the those not relocated during war time.

The second natural experiment focused on the effects of childhood exposure to the devastating earthquake in Wenchuan, China in 2008 (Lian et al., 2018). Thus it did not involve any decisions parents had to make, perhaps compromising any causal conclusions that might be drawn from the Finish study. Whereas the "experimental" children in Wenchuan lived where the earth trembled, destroyed many buildings, and killed many people, their counterparts did not. Comparison of the two groups that were essentially randomly assigned by nature to be exposed to devastation or not revealed that earthquake exposure increased the risk of early puberty in both girls and boys. Especially interesting and consistent with psychosocial acceleration theory is that this effect of the earthquake on pubertal development was most pronounced when children were 7-years old or younger at the time of the disaster. Indeed, for the girls the risk of early puberty was four times as great as for agemates who did not have direct exposure to the quake.

In view of the fact that the earthquake study is one of the few positioned to test the claim that it is early-childhood and not later-childhood experiences and exposures that regulates reproductive-strategy development, let me call attention to another investigation positioned to do so, though it did not include measurements of puberty, only other features of reproductive strategy and life history (Simpson et al., 2012). In this work on an at-risk sample of children followed from birth, results revealed that it was exposure between the ages of 0–5, but not between ages 6–16, to more unpredictable, rapidly changing environments that forecast for both males and females more sexual partners, more aggressive and delinquent behavior, and more involvement in criminal activity. Many of these developmental outcomes are clearly in line with the opportunistic-exploitative social orientation predicted by the theory to be associated with early-life adversity.

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Differential susceptibility

Most research on factors and forces that shape human development is geared toward estimating central tendencies, quite often defined in terms of a group's mean score, but also in terms of the variance explained by Mendelian-genetic differences. In either case, such evidence does not mean that everyone in a particular subgroup scored the average or that all individuals carrying the same gene or set of genes experienced the same degree of genetic penetrance. This is why I think of experimentalists as eating the oyster and throwing away the pearl. While the average effect of some experimental manipulation–or of contrasting contextual conditions in an observational study–is heralded (i.e., the oyster), the variation *within* the index group is often ignored (i.e., the pearl). The field of medicine has come to appreciate that such within-group variation is of significance, which is why it has moved toward precision or personalized treatments. Thus, the question becomes "what accounts for the variation in response to some experience, exposure or treatment?"

Appreciation of the importance of this issue, coupled with reflection on an all-too-rarely stated explanation of why human development is presumed by many to be affected by early-life conditions–to prepare the child for her likely future environment–is what gave birth to the differential susceptibility hypothesis (Belsky, 1997a; 1997b; 2005; 2007). Because the future is inherently uncertain, when a mismatch occurs between the contexts of childhood and that of adulthood, something that has surely occurred throughout the course of human history, then being shaped by one's childhood experiences and exposures could prove counterproductive in terms of passing on genes to future generations. It would thus have been to the benefit of the child, as well as to its parents, siblings and kin more generally when it came to passing on genes for children in a family to vary in the developmental plasticity. In fact, the same logic that leads to diversifying financial investments–instead of putting all one's eggs in one basket–was simply being applied to the currency of genes.

This analysis led to the hypothesis that nature, via natural selection, would have hedged its bets when it came to children being susceptible to their childhood contexts, with some children in the family more and others less developmentally plastic. That way, metaphorically speaking, if "tomorrow" ended up different than "today," the highly susceptible child would be ill prepared for the future and at risk when it came to passing on genes to the next generation, but would have its fitness at least partially "insured" by siblings who were less likely to succumb to this contextual "trap." By the same token, if today and tomorrow proved similar, the susceptible child would serve to "insure" the reproductive success of its sibling who had been less shaped by childhood to fit the future environment. Note that this logic applies to parents, who also share 50% of their genes with their offspring, just as (full) siblings do with each other.

Over the past 15 years extensive evidence has been reported consistent with the differential susceptibility hypothesis (Belsky & Pluess, 2009; 2013; in press). Given the prior focus on Mendelian nature, I will restrict my focus to Mendelian-genetic "plasticity factors" and thus only relevant GXE research. Just to be clear, though, genes are not the only plasticity factors that have emerged in relevant research (Belsky & Pluess, 2009; 2013; in press). When it comes to research on the genetics of differential susceptibility, it is important to understand that differential susceptibility inquiry essentially flips the GXE approach in behavior-genetic research. It is geared toward evaluating whether environmental effects vary as a function of genetic make-up, not whether genetic effects vary across contexts, as in Turkenheimer's work. That GXE interaction does operate this other way has now been documented in any number of candidate-gene studies, as well as non-GWAS and even GWAS-derived polygenic investigations (Belsky, in press).

Especially important is that this is so when diverse environmental indices (e.g., parenting, neighborhood violence, SES, father absence) are used as predictors, with markers of reproductive strategy, the outcome to be explained, including opportunistic-exploitative behavior (Brody et al., 2011; Davies et al., 2019; Fine et al., 2016; Gibbons et al., 2012; Rogosch & Cicchetti, 2013; Schlomer et al., 2021; Simons et al., 2011) and pubertal timing (Hartman et al., 2015; Manuck et al., 2011; Schlomer & Cho, 2017; Schlomer & Marceau, 2020; Sun et al, 2020). Unfortunately, to my knowledge, there is no such differential-susceptibility-related work focused on age of sexual debut or first birth, unstable partner relationships or number of children born. There is, though, such differential-susceptibility-related GXE evidence in the case of two reproductive-strategy-related developmental outcomes, quality of marital relationships (Haase et al., 2013; Lei et al., 2016; Simons et al., 2013) and parenting (Baião et al., 2020; Fortuna et al., 2011; Masarik et al., 2014).

If I had only two such observational studies to highlight, I would herald investigations focused on children growing up in the same family. This is because my original differential susceptibility theorizing stipulated that siblings would vary in their developmental plasticity because, as already noted, each would "insure" the other, with parents benefiting, too, when it came to passing on genes to the next generation (Belsky, 2005). In one inquiry the effect of lower birthweight, considered a proxy for prenatal stress on adult IQ held strongly for siblings who scored higher on a three-gene index of cumulative plasticity than for those who scored lower on the index (Cook & Fletcher, 2015). In the other investigation the effect of sibling differences in the number of plasticity alleles they carried also accounted for differences in the extent to which family economic status while growing up, which was the same for each sibling, predicted their own economic status in adulthood (Rauscher, 2017).

Because observational studies, even those involving siblings, can raise questions about whether actual differential responses to truly causal effects have been documented, it is noteworthy that results of a series of intervention studies suggest that results of all the cited GXE work is unlikely to be an artifact of genetic confoundment. These RCTs evaluated gene-x-intervention effects on externalizing problems of providing high-quality foster care to children who spent their earliest years in a Romanian orphanage (Brett et al., 2015); the effects of parenting interventions on the same phenotype (Bakermans-Kranenburg et al., 2008; Chhangur et al., 2017; Shaw et al., 2019), as well as on ADHD (Van Den Hoofdakker et al., 2012), and attachment security (Morgan et al., 2017); and effects of an intervention program targeting both parents and their teens on the latter's substance use (Beach et al., 2010; Brody et al., 2014). In all cases, candidate genes and polygenic scores presumed to reflect heightened developmental plasticity based on prior observational evidence led to predictions which were confirmed as to which individuals would benefit most from the interventions. In some cases, in fact, it was not just those carrying one or more would-be plasticity alleles who benefited the most, but also that those genetically similar functioned most problematically when randomly assigned to the control group (Bakermans-Kranenburg et al., 2008; Beach et al., 2010; Brett et al.,

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2015; Drury et al., 2012; Morgan et al., 2017; Shaw et al., 2019). This, of course, is consistent with the signature, for-better-and-for-worse proposition central to the differential susceptibility hypothesis, that is, that more susceptible children are most negatively affected by growing up under conditions of adversity yet also most likely to benefit developmentally when childhood is supportive and enrichening.

Conclusion

In view of my goal to move developmental thinking and inquiry toward its own modern synthesis of Mendelian and Darwinian nature, I will conclude this essay by first highlighting what appears to be resistance to the evolutionary perspective and thus an impediment to the realization of the synthesis I have sought to promote. Thereafter, I call attention to potential implications of such a synthesis for the future study and understanding of developmental psychopathology, first with respect to Darwinian nature and then with respect to Mendelian nature.

While many developmentalists have embraced the fruits of evo-devo thinking, especially in the case of the puberty and differential susceptibility hypotheses, it surprises me how often this occurs without embracing the theoretical framework that gave birth to empirical discoveries. As I am want to say, it is like ignoring the flashlight that illuminated the darkness and only attending to the illuminated key that led to opening the locked door that resulted in a discovery. My suspicion is that this occurs because too many have limited understanding of modern, inclusive-fitness thinking when it comes to evolution by natural selection, perhaps regarding it as just warmed over biology-as-destiny thinking. But what is central to an evo-devo view of the nature of nurture is that development has evolved to be shaped by nurture, even if for some more than others.

So evo-devo analysis is not a repeat of simplistic and inaccurate Mendelian-genetic thinking. It underscores nurture in the service of nature, meaning the passing on of genes to future generations, thus heralding the developmental significance of early-life conditions. But to understand variation in response to nature's imperative–passing on genes–when it comes to how nurture operates, appreciating Mendelian genetics cannot be overlooked, as the cited differential susceptibility work makes clear. So here we see a case for thinking in terms of both the why, Darwinian nature, and how, Mendelian nature, of development. In both cases we are not documenting biology as destiny, but the complex and fascinating interplay of the two, something too long neglected or feared (due to potential eugenic implications).

It is not unreasonable to wonder whether embracement of the ideas advanced herein have implications for our field. Let me address this issue first with respect to Darwinian nature. To begin with, there is reason to question the view that differences from the (valued) norm in behavior and psychological functioning reflects disturbance, dysfunction, dysregulation or even disorder. In many cases, evolution has shaped children to develop in so-called "problematic" ways when early-life conditions have induced them. This is why I object to the terminology of "optimal" development that pervades our field when the focus is on phenotypes we value, too often implying that what we value is what nature intended. By metaphorical comparison, who would regard orchids as what nature intended and weeds as just the opposite? Note that abandoning developmental "optimal" thinking does not carry with it any inherent implication that nothing can or should be done when development takes the form of a "weed" rather than an "orchid". One thing reproductive-strategy thinking implies, quite consistent with so much standard developmental thinking, is that it may be the early years when we have the most chance of inducing developments that we prefer.

Another implication that might fundamentally alter how we at least think about developmental psychopathology concerns Mendelian nature and, specifically, the issue of GXE interaction. In the hands of too many students of psychopathology, genes associated with "disturbance, dysfunction and dysregulation" are often regarded as "vulnerability" genes. But if, as I have argued, heritability estimates are as much a reflection of environmental influences that afford the expression of genetic differences as of genetic influence, then perhaps discourse should highlight, perhaps even preferentially, "enabling" and "disabling" environments rather than genetic influence. And this is so whether the phenotype in question is widely valued (e.g., IQ) or not (e.g., depression).

The fundamental point to be made is whether genes related to prosocial or antisocial behavior, for example, become phenotypically expressed, such that their latent potential becomes manifest, depends on whether the environment in which the child develops "enables" or "disables" such expression (perhaps through epigenetic processes, as just one possibility). Accordingly, when genetic and phenotypic differences prove to be systematically related, as when heritability proves evident, discourse should make reference to "enabling" environments; and this is so whether a study has specifically addressed GXE interaction or simply documented a genetic main effect. By the same token, when individuals who might otherwise have proven very cooperative or aggressive fail to develop in such ways despite carrying genes for such phenotypes, we should speak of the environment "disabling" phenotypic manifestation that is, genetic penetrance.

Clearly, the story we tell about genes is quite different once we acknowledge and embrace the view that their phenotypic consequences are environmentally dependent. So why, then, should we emphasize vulnerability (or even plasticity) when characterizing genes instead of enabling and disabling environments? What would popular understanding of the complexity of development be like if developmental discourse underscored the role of the environment in affording the phenotypic manifestation of latent genetic effects. Think about it.

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