

AperTO - Archivio Istituzionale Open Access dell'Università di Torino

Middle childhood: An evolutionary-developmental synthesis

This is the author's manuscript

Original Citation:

Availability:

This version is available <http://hdl.handle.net/2318/1853338> since 2022-04-12T05:48:54Z

Publisher:

Springer

Published version:

DOI:10.1007/978-3-319-47143-3_5

Terms of use:

Open Access

Anyone can freely access the full text of works made available as "Open Access". Works made available under a Creative Commons license can be used according to the terms and conditions of said license. Use of all other works requires consent of the right holder (author or publisher) if not exempted from copyright protection by the applicable law.

(Article begins on next page)

Del Giudice, M. (2018). Middle childhood: An evolutionary-developmental synthesis. In N. Halfon, C. B. Forrest, R. M. Lerner, & E. Faustman (Eds.), *The handbook of life course health development* (pp. 95-107). New York: Springer.

Middle Childhood: An Evolutionary-Developmental Synthesis

Marco Del Giudice

University of New Mexico

Marco Del Giudice, Department of Psychology, University of New Mexico.
Address correspondence to Marco Del Giudice, Department of Psychology, University of New Mexico. Logan Hall, 2001 Redondo Dr. NE, Albuquerque, NM 87131, USA; email: marcodg@unm.edu

Middle childhood—conventionally going from about 6 to 11 years of age—is a crucial yet underappreciated phase of human development. On the surface, middle childhood may appear like a slow-motion interlude between the spectacular transformations of infancy and early childhood and those of adolescence. In reality, this life stage is anything but static: the transition from early to middle childhood heralds a global shift in cognition, motivation, and social behavior, with profound and wide-ranging implications for the development of personality, sex differences, and even psychopathology (Table 1).

In the last two decades, converging theories and findings from anthropology, primatology, evolutionary psychology, endocrinology, and behavior genetics have revolutionized our understanding of middle childhood. In this chapter, I show how these diverse contributions can be synthesized into an integrated evolutionary-developmental model of middle childhood. I begin by reviewing the main evolved functions of middle childhood and the cognitive, behavioral, and hormonal processes that characterize this life stage. Then, I introduce the idea that the transition to middle childhood works as a switch point in the development of life history strategies (Del Giudice, Angeleri, & Manera, 2009, 2012; Del Giudice & Belsky, 2011), and discuss three insights in the nature of middle childhood that arise from an integrated approach. This chapter was originally published as a short paper in the journal *Child Development Perspectives* (Del Giudice, 2014a). It is reprinted here with updated references and a new section on the model's implications for health development in a life course perspective (LCHD).

WHAT IS MIDDLE CHILDHOOD?

Middle childhood is one of the main stages of human development, marked by the eruption of the first permanent molars around age 6 and the onset of androgen secretion by the adrenal glands at about 6-8 years (Bogin, 1997). In middle childhood, body growth slows considerably, usually following a small mid-growth spurt. At the same time, muscularity increases and the body starts accumulating fat (the *adiposity rebound*; Hochberg, 2008), while sex differences in body composition become more pronounced (Del Giudice et al., 2009; Wells, 2007). Figure 1 places middle childhood in the broader context of human growth from conception to adolescence.

In biological terms, middle childhood corresponds to human *juvenility*—a stage in which the individual is still sexually immature, but no longer dependent on parents for survival. In social mammals and primates, juvenility is a phase of intense learning—often accomplished through play—in which youngsters practice adult behavioral patterns and acquire essential social and foraging skills. Indeed, the duration of juvenility in primates correlates strongly with the size and complexity of social groups, as well as with cortical brain volume (Joffe, 1997). Social learning in juvenility can be understood as investment in *embodied capital*—skills and knowledge that cost time and effort to acquire, but increase an individual's performance and reproductive success (Kaplan, Hill, Lancaster, & Hurtado, 2000).

Table 1. Development in middle childhood. See the main text for supporting references. BMI = body mass index; M = male; F = female; ADHD = attention deficit hyperactivity disorder.

Body growth	<ul style="list-style-type: none"> Eruption of permanent molars Mid-growth spurt, followed by decelerating skeletal growth Increased muscle mass Increased adiposity and BMI (adiposity rebound) Initial development of axillary hair and body odor Increased sex differences in adiposity (F>M), bone strength, and muscularity (M>F) Emergence of sex differences in vocal characteristics
Brain growth	<ul style="list-style-type: none"> Approaching peak of overall brain volume Peak of gray matter volume Continuing increase in white matter volume/integrity
Motor and perceptual skills	<ul style="list-style-type: none"> Increased gross motor skills (e.g., walking) Increased fine motor skills Local-global shift in visual processing preferences
Cognitive skills	<ul style="list-style-type: none"> Increased reasoning and problem-solving skills (e.g., concrete operations) Increased self-regulation and executive functions (inhibition, attention, planning, etc.) Increased mentalizing skills (multiple perspectives, conflicting goals) Increased navigational skills (working memory, ability to understand maps)
Motivation and social behavior	<ul style="list-style-type: none"> Acquisition of cultural norms (e.g., prosociality) Complex moral reasoning (conflicting points of view) Increased pragmatic abilities (gossiping, storytelling, verbal competition, etc.) Consolidation of status/dominance hierarchies Changes in aggression levels (individual trajectories) Development of disgust Changes in food preferences (e.g., spicy foods) Onset of sexual/romantic attraction Increased frequency of sexual play Increased sense of gender identity Peak of sex segregation Peak of sex differences in social play (including play fighting vs. play parenting) Increased sex differences in physical aggression (M>F) Emergence of sex differences in attachment styles
Psychopathology	<ul style="list-style-type: none"> Early peak of psychopathology onset (externalizing, anxiety, phobias, ADHD) Peak onset of fetishistic attractions Emergence of sex differences in conduct disorders (M>F)
Social context	<ul style="list-style-type: none"> Active involvement in caretaking, foraging, domestic tasks, helping Expectations of responsible behavior Attribution of individuality and personhood (“getting noticed”)
Behavior genetics	<ul style="list-style-type: none"> Increased heritability of general intelligence and language skills New genetic influences on general intelligence, language, aggression, prosociality

Human children are no exception to this pattern. Social learning is universally recognized as a key evolved function of middle childhood and is enabled by a global reorganization of cognitive functioning known as the *five-to-seven shift* (Weisner, 1996). By age 6, the brain has almost reached its maximum size and receives a decreasing share of the body's glucose after the consumption peak of early childhood (Figure 1; Giedd & Rapoport, 2010; Kuzawa et al., 2014). However, brain development proceeds at a sustained pace, with intensive synaptogenesis in cortical areas (gray matter) and rapid maturation of axonal connections (white matter; Lebel, Walker, Leemans, Phillips, & Beaulieu, 2008). The transition to middle childhood is marked by a simultaneous increase in perceptual abilities (including a transition from local to global visual processing), motor control (including the emergence of adult-like walking), and complex reasoning skills (Bjorklund, 2011; Poirel et al., 2011; Weisner, 1996). The most dramatic changes probably occur in the domain of self-regulation and executive functions: children become much more capable of inhibiting unwanted behavior, maintaining sustained attention, making and following plans, and so forth (Best, Miller, & Jones, 2009; Weisner, 1996). Parallel improvements take place in mentalizing (the ability to understand and represent mental states) and moral reasoning, as children become able to consider multiple perspectives and conflicting goals (Jambon & Smetana, 2014; Lagattuta, Sayfan, & Blattman, 2009).

In traditional societies, older relatives—especially parents and grandparents—are the main sources of knowledge for juveniles, supplemented by peers and—where available—professional teachers. Storytelling (both fictional and based in real events) is a powerful technology for transmitting knowledge about foraging and social skills, avoidance of dangers, topography, wayfinding, and social roles and norms (Scalise Sugiyama, 2011). Storytelling mimics the format of episodic memories, providing the child with a rich source of indirect experience (Scalise Sugiyama, 2011). Intriguingly, episodic memory shows dramatic and sustained improvements across middle childhood (Ghetti & Bunge, 2012).

However, children at this age are not just learning and playing. Cross-culturally, middle childhood is the time when children are expected to start helping with domestic tasks—such as caring for younger siblings, collecting food and water, tending animals, and helping adults prepare food (Bogin, 1997; Lancy & Grove, 2011; Scalise Sugiyama, 2011; Weisner, 1996). In favorable ecologies, juveniles can contribute substantially to family subsistence (Kramer, 2011). Thanks to marked increases in spatial cognition (reflected in the emerging ability to understand maps) and navigational skills, children become able to memorize complex routes and find their way without adult supervision (Bjorklund, 2011; Piccardi, Leonzi, D'Amico, Marano, & Guariglia, 2014). The important role of juveniles in collecting and preparing food may explain why the emotion of disgust does not fully develop until middle childhood (Rozin, 1990a).

The transition to middle childhood is typically associated with a strong separation in gender roles, even in societies where tasks are not rigidly assigned by sex. Spontaneous sex segregation of boys and girls peaks during these years, as does the frequency of sexually differentiated play (Del Giudice et al., 2009). On a broader social level, cross-cultural evidence shows that juveniles start “getting noticed” by adults—that is, they begin to be viewed fully as people with their own individuality, personality, and social responsibility (Lancy & Grove, 2011).

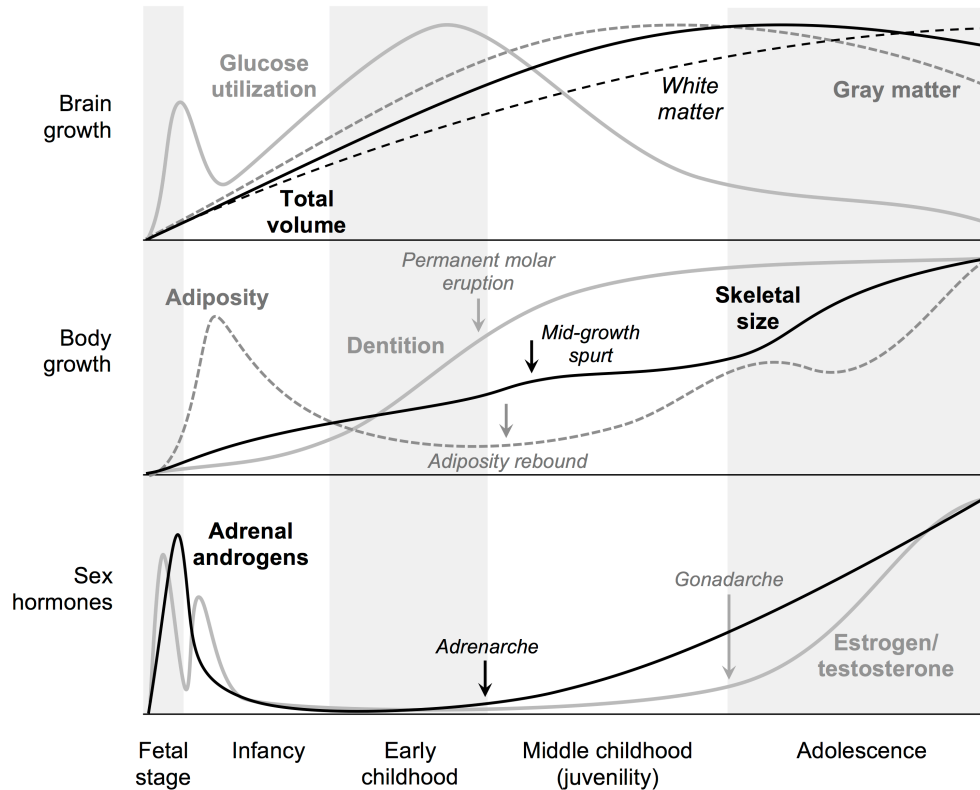


Figure 1. Developmental trajectories of human growth and sex hormones production, from conception to adolescence. Arrows show the landmark events that characterize middle childhood. Sources: Auchus & Rainey (2004); Bogin (1997); Giedd & Rapoport (2010); Hochberg (2008); Kuzawa et al. (in press); Ober et al. (2008); Wells (2007).

In summary, the life stage of juvenility/middle childhood has two major interlocking functions: *social learning* and *social integration* in a system of roles, norms, activities, and shared knowledge. While children are still receiving sustained investment from parents and other relatives—in the form of food, protection, knowledge, and so forth—they also start to actively contribute to their family economy. By providing resources and sharing the burden of child care, juveniles can boost their parents’ reproductive potential. The dual nature of juveniles as both *receivers* and *providers* explains many psychological features of middle childhood and has likely played a major role in the evolution of human life history (Kramer, 2011).

Adrenarche

The transition to middle childhood is coordinated by a remarkable endocrinological event: the awakening of the adrenal glands, or *adrenarche* (Auchus & Rainey, 2004; Hochberg, 2008). Starting at about 6-8 years—with much individual variation and only minor differences between males and females—adrenal glands begin to secrete increasing amounts of androgens (see Figure 1), mainly dehydroepiandrosterone (DHEA) and its sulfate (DHEAS). Adrenal androgens have only minor effects on physical development, but they have powerful effects on

brain functioning. DHEA and DHEAS promote neurogenesis and modulate GABA and glutamate receptors; moreover, DHEA can act directly on androgen and estrogen receptors. Even more important, adrenal androgens can be converted to estrogen or testosterone in the brain (Campbell, 2006; Del Giudice et al., 2009). As sex hormones, adrenal androgens play a twofold role: They activate sexually differentiated brain pathways that had been previously organized by the hormonal surges of prenatal development and infancy (Figure 1) and they further organize brain development along sexually differentiated trajectories (Del Giudice et al., 2009).

Adrenal androgens likely provide a major impulse for many of the psychological changes of middle childhood (Campbell, 2006, 2011; Del Giudice et al., 2009), including the emergence and intensification of sex differences across domains (see Table 1). Since the age of adrenarche correlates strongly with that of *gonadarche* (the awakening of the testes/ovaries that marks the beginning of puberty; Hochberg, 2008), human development shows a peculiar pattern in which sexually differentiated brain pathways are activated several years *before* the development of secondary sexual characteristics. This developmental pattern (shared by chimpanzees and, to a lesser extent, gorillas; Bernstein, Sterner, & Wildman, 2012) results in a temporary decoupling between physical and behavioral development, consistent with the idea of middle childhood as a sexually differentiated phase of social learning and experimentation (Geary, 2010). Moreover, adrenal androgens promote extended brain plasticity through synaptogenesis, and may play an important role in shifting the allocation of the body's energetic resources away from brain development and toward the accumulation of muscle and fat in preparation for puberty (Campbell, 2006, 2011; see also Kuzawa et al., 2014).

THE TRANSITION TO MIDDLE CHILDHOOD AS A DEVELOPMENTAL SWITCH POINT

The evolutionary model of middle childhood sketched in the previous section can be enriched and extended by considering the role of adrenarche as a *developmental switch* (Del Giudice et al., 2009). The concept of a developmental switch was introduced by West-Eberhard (2003); a switch is a regulatory mechanism that activates at a specific point in development, collects input from the external environment or the state of the organism, and shifts the individual along alternative pathways—ultimately resulting in the development of alternative phenotypes (morphological, physiological, or behavioral traits of an organism). For example, a switch may regulate the development of aggressive behavior so that safe conditions entrain the development of low levels of aggression, whereas threatening environments trigger high levels of aggression. Developmental switches enable *adaptive plasticity*—the ability of an organism to adjust its phenotype to match the local environment in a way that promotes biological fitness (West-Eberhard, 2003). In other words, plastic organisms track the state of the environment—usually through indirect cues—and use this information to develop alternative phenotypes that tend to promote survival or reproduction under different conditions.

Developmental switches work in a *modular* fashion (see Figure 2). Activation of a switch leads to the coordinated expression of different genes—both those involved in the regulatory mechanism itself and those involved in the production of the new phenotype. Moreover, alternative phenotypes (A and B in Figure 2) involve the expression of modular packages of genes specific to each phenotype. Another key aspect of developmental switches is that they

integrate variation in the environment with individual differences in the genes that regulate the switch. For example, different individuals may have genetically different thresholds for switching between aggressive and nonaggressive phenotypes. Finally, the embodied effects of past experiences and conditions (e.g., an individual's previous exposure to stress or nutritional conditions early in life) may also modulate how the switch functions, allowing the organism to integrate information over time and across different life stages (Del Giudice, 2014b; Ellis, 2013). In many instances, the effects of past experience on developmental switches may be mediated by epigenetic mechanisms (see Meaney, 2010).

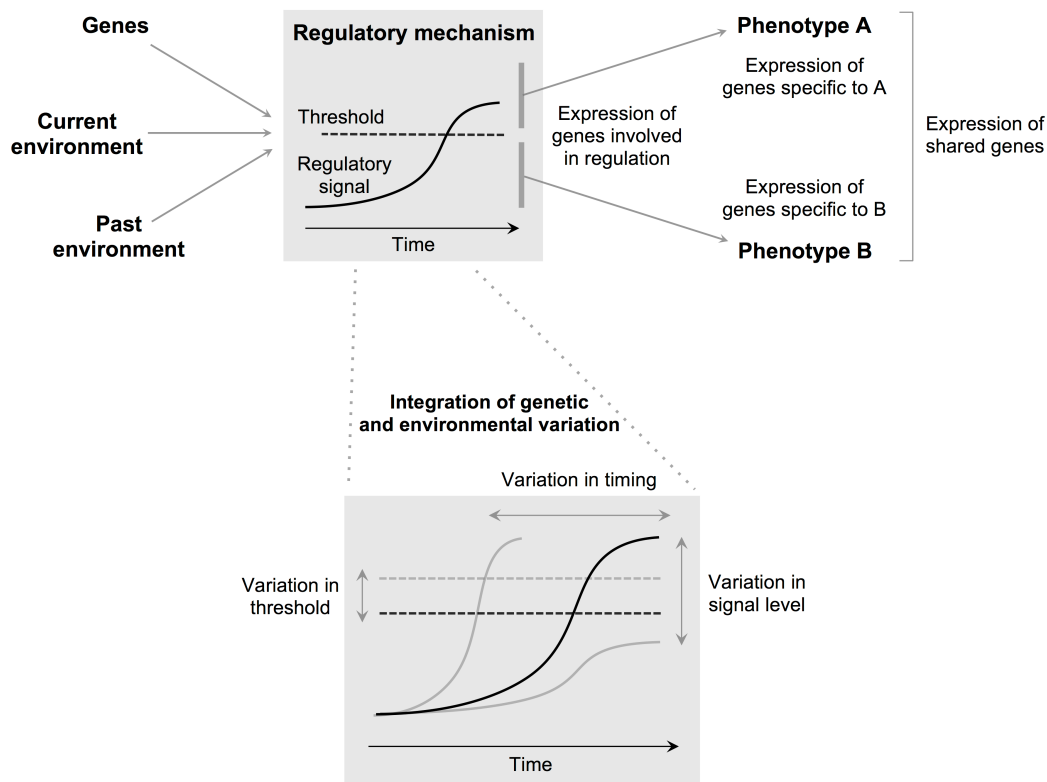


Figure 2. The concept of a developmental switch. A regulatory mechanism (which may operate through hormonal signals) integrates current and past information from the environment with the individual's genotype. As a result, the individual's developmental trajectory is shifted along alternative pathways—here, A and B—depending on whether a threshold is reached within the mechanism. The location of the threshold, the intensity of the signal, and the timing of the switch point all depend on the joint action of the current state of the environment, the embodied effect of past environmental conditions, and individual variation in the genes involved in the regulatory mechanism. Each alternative pathway involves the modular expression of a set of specific genes, in addition to the shared genes expressed in the new developmental stage. A developmental switch may integrate many sources of input from the environment or produce graded phenotypes rather than discrete alternatives such as A and B.

The concept of a developmental switch point resembles that of a *sensitive period*, in that the organism is maximally responsive to some environmental input. The crucial difference is

that, because genetic and environmental inputs converge in the regulatory mechanism, a developmental switch amplifies both environmental *and* genetic effects on the phenotype (West-Eberhard, 2003). Indeed, the activation of a developmental switch exposes many new potential sources of genetic variation, including the genes involved in the regulatory mechanism and in the expression of the new phenotypes (Figure 2).

A Switch Point in Life History Development

The role of adrenarche as a developmental switch is not limited to a single trait; in fact, the transition to middle childhood (or *juvenile transition*; Del Giudice et al., 2009) encompasses all the major domains of behavior—from learning and self-regulation to attachment and sexuality (see Table 1). My colleagues and I (Del Giudice & Belsky, 2011; Del Giudice et al., 2009, 2012) have argued that the transition to middle childhood is a switch point in the development of *life history strategies*, which are coordinate suites of morphological, physiological, and behavioral traits that determine how organisms allocate their resources to key biological activities such as growth, reproduction, mating, and parenting (for a non-technical overview of life history theory, see Del Giudice, Gangestad, & Kaplan, 2015). At the level of behavior, individual differences in life history strategy are reflected in patterns of self-regulation, aggression, cooperation and prosociality, attachment, sexuality, and so forth (Del Giudice & Belsky, 2011; Del Giudice, Ellis, & Shirtcliff, 2011; Del Giudice et al., 2009; Ellis, Figueredo, Brumbach, & Schlomer, 2009). Although life history strategies are partly heritable, they also show a degree of plasticity in response to the quality of the environment, including the level of danger and unpredictability (embodied in the experience of early stress) and the availability of adequate nutritional resources. In a nutshell, dangerous and unpredictable environments tend to favor *fast* strategies characterized by early reproduction, sexual promiscuity, unstable relationships, impulsivity, risk taking, aggression, and exploitative tendencies, whereas safe and predictable environments tend to entrain *slow* strategies characterized by late reproduction, stable relationships, high self-control, aversion to risk, and prosociality. Slow strategies are also favored by nutritional scarcity when danger is low (see Del Giudice et al., 2016; Ellis et al., 2009).

Our argument is that adrenarche coordinates the expression of individual differences in life history strategy by integrating individual genetic variation with information about the child's social and physical environment collected throughout infancy and early childhood (Belsky, Steinberg, & Draper, 1991). The stress response system plays a major role in gathering and storing information about environmental safety, predictability, and availability of resources; adrenarche contributes by translating that information into adaptive, sexually differentiated patterns of behavior (Del Giudice et al., 2011; Ellis & Del Giudice, 2014). Consistent with this view, both early relational stress and early nutrition have been found to modulate the timing of adrenarche (Ellis & Essex, 2007; Hochberg, 2008). It is no coincidence that the first sexual and romantic attractions typically develop in middle childhood, in tandem with the intensification of sexual play (Bancroft, 2003; Herdt & McClintock, 2000). By interacting with peers and adults, juveniles receive feedback about the effectiveness of their nascent behavioral strategies. The information collected during middle childhood feeds into the next developmental switch point, that of gonadarche (Ellis, 2013); the transition to adolescence offers an opportunity for youth to adjust or revise their initial strategy before attaining sexual and reproductive maturity (Del Giudice & Belsky, 2011).

The role of adrenarche as a switch point in life history development adds another level of complexity to the biological profile of juvenility. Figure 3 outlines an integrated evolutionary-developmental model that brings together the various strands of theory and evidence reviewed in this chapter.

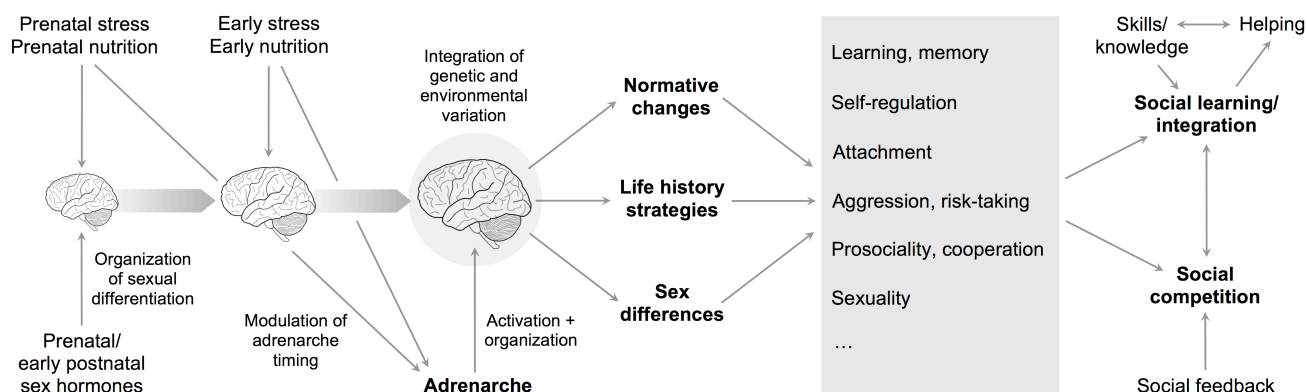


Figure 3. An integrated evolutionary-developmental model of middle childhood. Adrenarche is shown as a switch in the development of life history strategies, as well as a key mechanism underlying the normative changes of middle childhood and the emergence and intensification of sex differences. At a broader level, development in middle childhood serves two complementary functions, social *integration* and social *competition*.

THREE INSIGHTS IN THE NATURE OF MIDDLE CHILDHOOD

Insight #1: Social Integration and Social Competition are Complementary Functions of Middle Childhood

Evolutionary accounts of middle childhood typically focus on learning, helping, and other forms of social integration. A life history approach emphasizes the need to consider social competition as a crucial, complementary function of human juvenility. In the peer group, children compete for vital social resources—status, reputation, allies, and friends. While learning and play are relatively risk-free, they are not without consequences. The social position achieved in middle childhood is a springboard for adolescence and adulthood; popularity and centrality within the peer network put a child at a considerable advantage, with potentially long-term effects on mating and reproductive success (Del Giudice et al., 2009).

Physical and relational aggression are obvious tactics for gaining influence, but social competition also occurs through prosocial behaviors such as forming alliances, doing favors, and displaying valuable skills. Indeed, managing the balance between prosocial and coercive tactics is an important part of developing social skills (Hawley, 2014). More broadly, competition shapes many aspects of cognitive and behavioral development in middle childhood; for example, increased pragmatic abilities allow children to gossip, joke, tease, and engage in verbal duels—all forms of social competition mediated by language (Locke & Bogin, 2006). Intensifying social

competition also contributes to explain the early peak of psychopathology onset observed in middle childhood, characterized by increasing rates of externalizing disorders (e.g., conduct disorder), anxiety disorders (including social phobia), and attention deficit hyperactivity disorder (ADHD; Del Giudice et al., 2009).

Insight #2: Sexual Selection Contributes to the Emergence and Intensification of Sex Differences in Middle Childhood

By determining children's initial place in social networks and hierarchies, competition in middle childhood indirectly affects their ability to attract sexual and romantic partners later. In other words, middle childhood is a likely target for *sexual selection*—that is, natural selection arising from the processes of choosing mates (*mate choice*) and competing for mates (*mating competition*). My colleagues and I (Del Giudice et al., 2009) argued that sexual selection is one reason why sex differences emerge and intensify in middle childhood. In particular, sex differences in physical aggression increase markedly, in tandem with sex differences in muscularity and play fighting. At the same time, attachment styles begin to diverge between males and females, with insecurely attached boys becoming more avoidant and insecure girls becoming more preoccupied/ambivalent (Del Giudice, 2009; Del Giudice & Belsky, 2010). Different attachment styles are conducive to different social strategies, and may be adaptive in regulating children's nascent relationships with peers. There is initial evidence that attachment styles in middle childhood reflect the effects of prenatal sex hormones, which according to our model are activated by adrenal androgens (Del Giudice & Angeleri, 2016). Sexual selection also has indirect implications for the development of psychopathology; for example, marked sex differences in the prevalence of conduct disorders become apparent at the beginning of middle childhood, likely reflecting the stronger role of aggression in boys' social competition (see Del Giudice et al., 2009; Martel, 2013).

Insight #3: In Middle Childhood, Heightened Sensitivity to the Environment Goes Hand in Hand with the Expression of New Genetic Factors

When an organism goes through a developmental switch point, inputs from the environment combine with the individual's genotype to determine the resulting phenotype. For example, when adrenal androgens begin to increase during the transition to middle childhood, they activate many hormone-sensitive brain pathways that have been dormant since infancy. In doing so, they release previously hidden genetic variation (Del Giudice et al., 2009). Thus, middle childhood should be characterized by a mixture of heightened sensitivity to the environment (possibly mediated by newly activated epigenetic mechanisms; Meaney, 2010) and expression of new genetic factors.

Evidence of increased sensitivity to the environment in middle childhood is not hard to find. Two intriguing and little-known examples concern the development of food preferences and erotic fetishes. In cultures where chili pepper is an essential part of the diet, children tend to dislike spicy food until middle childhood then increase rapidly their preference for the flavor of chili as a result of social learning (Rozin, 1990b). Fetishistic attractions also tend to form in middle childhood, with the onset of pleasurable sensations toward the object of the fetish (e.g., rubber, shoes) that later become fully eroticized (Lawrence, 2009). The onset of fetishistic

attractions is part of a generalized awakening of sexuality in middle childhood (Table 1) and illustrates the potential for rapid plasticity with long-lasting outcomes. Enhanced sensitivity to the environment extends beyond individual learning to acquiring social norms: for example, cross-cultural differences in prosocial behavior are absent in young children but emerge clearly during middle childhood (House et al., 2013).

On the genetic side of the equation, general intelligence and language skills increase markedly in heritability from early to middle childhood. In both cases, new genetic factors come into play around age 7 (Davis, Haworth, & Plomin, 2009; Hayiou-Thomas, Dale, & Plomin, 2012). Studies of prosociality and aggression find the same pattern, with new genetic influences on behavior emerging during the transition to middle childhood (Knafo & Plomin, 2006; van Beijsterveldt, Bartels, Hudziak, & Boomsma, 2003). These genetic findings dovetail with converging evidence that individual changes in levels of aggression are especially frequent during the transition to juvenility (Del Giudice et al., 2009).

IMPLICATIONS FOR HEALTH DEVELOPMENT

The main focus of this chapter has been on psychological development, but the implications of the evolutionary-developmental extend to both mental and physical health. The transition to middle childhood seems to be a switch point for a number of growth and metabolic processes that have long-term impact on health, including the risk for obesity and type 2 diabetes (Hochberg, 2008, 2010). These processes become apparent in middle childhood (e.g., anticipated onset of the adiposity rebound, rapid weight gain, onset of insulin resistance), but respond to the accumulated effects of early nutrition and other sources of stress, starting from prenatal life (e.g., intrauterine growth restriction). From the standpoint of the model presented here, one can predict that metabolic changes in middle childhood will reflect both the “programming” effects of the early environment (Gluckman, Hanson, & Spencer, 2005) and the activation of new genetic factors. Consistent with this view, a recent study has documented significant genetic correlations between puberty timing, insulin levels, type 2 diabetes, and cardiovascular disease (Day et al., 2015). Moreover, some of those factors are likely to be expressed in sexually differentiated ways, and the different patterns of health risk associated with early adrenarche and puberty in boys and girls may be usefully interpreted in light of different constraints on life history trade-offs in the two sexes (see Hochberg, 2010). These predictions are consistent with the non-linear and multilevel nature of developmental processes—one of the guiding principles of LCHD emphasized in this volume.

Another intriguing implication of this perspective is that the juvenile transition may be a promising—and still virtually unexplored—developmental window for intervention. While intervening to change early life conditions may be desirable in view of their long-term effects, this approach is not always possible or realistic. In addition, prenatal factors such as fetal nutrition and gestational stress may be especially difficult to target, as they do not simply mirror the mother’s conditions but reflect a complex (and partially conflictual) interplay between fetal and maternal factors (e.g., Del Giudice, 2012; Gangestad, Caldwell Hooper, & Eaton, 2012; Haig, 1993). However, the logic of developmental switches (Figure 2) suggests that the activation of the mechanisms that initiate the switch (e.g., adrenarche) may correspond to a transient phase of instability and openness in the system. If so, it should be possible to exploit

that phase to maximize the efficacy of focused interventions (including pharmacological ones). Of course, this would require a better understanding of how different hormonal and neurobiological systems interact during the transition to middle childhood; the existing evidence points to a central role of the hypothalamic-pituitary-adrenal axis (HPA), the hypothalamic-pituitary-adrenal-thyroid axis (HPT), and the insulin/insulin-like growth factor 1 (IGF-1) signaling systems as mediators of life history allocations, not just in humans but in other vertebrates as well (see Del Giudice et al., 2015; Ellis & Del Giudice, 2014). Those systems might be used as direct targets for intervention, but also as “endophenotypes” or early indicators of the efficacy of interventions. Importantly, neurobiological systems such as the HPA axis regulate both metabolism and behavior (as components of coordinated life history allocations), so that many of the same processes may be relevant to both physical and mental health. The idea that intervening *during* a biological transition may afford more leverage to alter developmental trajectories resonates with two key principles of LCHD, that is, the nonlinearity of developmental processes and their time-sensitivity.

Probably the most important take-home point of an evolutionary-developmental approach is that researchers should be more cautious in assuming that undesirable developmental outcomes reflect dysregulation of a biological system, and more open to the possibility that those outcomes may be part of adaptive (or formerly adaptive) strategies for survival and reproduction. As I have argued in detail elsewhere (Del Giudice, 2014b, 2014c), a life history framework is especially useful in teasing out the logic of potentially adaptive combinations of traits, highlighting critical factors in the environment, and bridging behavioral development with physical growth trajectories. As an example, consider the association between intrauterine growth restriction and early maturation in children (Hochberg, 2008, 2010). This can be interpreted as a manifestation of physiological dysregulation due to prenatal adversity, or as an adaptive programming effect on children’s metabolic processes and life history trajectories. This alternative interpretation is supported by the association between low birth weight, anticipated puberty, and early childbearing in women (e.g., Nettle et al., 2013). A third possibility is that low birth weight partly reflects reduced energetic and metabolic investment by the mother during pregnancy, which may be adaptive as a component of a fast life history strategy. If so, the association between reduced fetal growth and earlier sexual maturation may not be fully causal, but rather result—at least in part—from shared genetic (or epigenetic) factors that influence life history strategy in both the mother and the offspring. Clearly, the implications for intervention are going to be quite different depending on which of these scenarios apply.

Another recent example is the finding that early adrenarche is associated with reduced white matter volume in the frontal lobe of children (Klauser et al., 2015). Again, the standard interpretation is that early DHEA exposure has a disruptive effect on neurodevelopmental processes; however, it is also possible that different trajectories of brain development—and even associated “symptoms” such as anxiety and aggressive behaviors—may instead reflect alternative strategies on a fast-slow continuum of life history variation. Ellis and colleagues (2012) present an extended analysis of adolescent risk-taking from this perspective, and discuss several implications for the design of interventions. The LCHD principle that evolution both enables and constrains plasticity is especially relevant in this regard; evolutionary scenarios are not just interesting explanatory “stories,” but can illuminate limits as well as opportunities for intervention. For example, when considering mother-fetus interactions, the existence of partial

conflicts of interest on nutrition, cortisol production, and so on suggests that interventions designed to favor the fetus may sometimes have detrimental side effects on the mother, and vice versa (see Del Giudice, 2014b; Haig, 1993)

In considering potential adaptive explanations for apparently pathological outcomes, it is important to remember that biologically adaptive traits may carry substantial costs. Fitness is ultimately about reproductive success; natural selection does not necessarily promote psychological well-being or physical health, and may even sacrifice survival in exchange for enhanced reproduction. Moreover, even adaptive developmental processes may result in genuinely maladaptive outcomes for some individuals (Frankenhuis & Del Giudice, 2012). It follows that the existence of even substantial psychological, social, or health costs does not automatically qualify a trait or behavior as biologically maladaptive (see Del Giudice, 2014b; Ellis et al., 2012; Ellis & Del Giudice, 2014).

CONCLUSIONS

We cannot make sense of human development without understanding middle childhood and its many apparent paradoxes. An evolutionary-developmental approach illuminates the complexity of this life stage and shows how different levels of analysis—from genes to society—can be tied together in a coherent synthesis. This emerging view of middle childhood can help developmental scientists appreciate its centrality in the human life history, and stimulate ideas for research and intervention. The study of middle childhood may finally be ready to come of age, opening up promising avenues for a better understanding of health development across the life course.

REFERENCES

- Auchus, R. J., & Rainey, W. E. (2004). Adrenarche—physiology, biochemistry and human disease. *Clinical Endocrinology*, *60*, 288–296.
- Bancroft, J. (Ed.) (2003). *Sexual development in childhood*. Bloomington, IN: Indiana University Press.
- Belsky, J., Steinberg, L., & Draper, P. (1991). Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. *Child Development*, *62*, 647–670.
- Bernstein, R. M., Sterner, K. N., & Wildman, D. E. (2012). Adrenal androgen production in catarrhine primates and the evolution of adrenarche. *American Journal of Physical Anthropology*, *147*, 389–400.
- Best, J. R., Miller, P. H., & Jones, L. L. (2009). Executive functions after age 5: Changes and correlates. *Developmental Review*, *29*, 180–200.
- Bjorklund, D. F. (2011). *Children's thinking: Cognitive development and individual differences* (5th ed.). Belmont, CA: Wadsworth.
- Bogin, B. (1997). Evolutionary hypotheses for human childhood. *Yearbook of Physical Anthropology*, *40*, 63–89.
- Campbell, B. C. (2006). Adrenarche and the evolution of human life history. *American Journal of Human Biology*, *18*, 569–589.

- Campbell, B. C. (2011). Adrenarche and middle childhood. *Human Nature*, *22*, 327-349.
- Davis, O. S. P., Haworth, C. M. A., & Plomin, R. (2009). Dramatic increase in heritability of cognitive development from early to middle childhood: An 8-year longitudinal study of 8,700 pairs of twins. *Psychological Science*, *20*, 1301-1308.
- Day, F. R., Bulik-Sullivan, B., Hinds, D. A., Finucane, H. K., Murabito, J. M., Tung, J. Y., et al. (2015). Genetic determinants of puberty timing in men and women: Shared genetic aetiology between sexes and with health-related outcomes. *Nature Communications*, *6*, 8842.
- Del Giudice, M. (2009). Sex, attachment, and the development of reproductive strategies. *Behavioral and Brain Sciences*, *32*, 1-21.
- Del Giudice, M. (2012). Fetal programming by maternal stress: Insights from a conflict perspective. *Psychoneuroendocrinology*, *37*, 1614-1629.
- Del Giudice, M. (2014a). Middle childhood: An evolutionary-developmental synthesis. *Child Development Perspectives*, *8*, 193-200.
- Del Giudice, M. (2014b). Early stress and human behavioral development: Emerging evolutionary perspectives. *Journal of Developmental Origins of Health and Disease*, *5*, 270-280.
- Del Giudice, M. (2014c). An evolutionary life history framework for psychopathology. *Psychological Inquiry*, *25*, 261-300.
- Del Giudice, M., & Angeleri, R. (2016). Digit ratio (2D:4D) and attachment styles in middle childhood: Indirect evidence for an organizational effect of sex hormones. *Adaptive Human Behavior and Physiology*.
- Del Giudice, M., Angeleri, R., & Manera, V. (2009). The juvenile transition: A developmental switch point in human life history. *Developmental Review*, *29*, 1-31.
- Del Giudice, M., Angeleri, R., & Manera, V. (2012). Juvenility and the juvenile transition. In R. J. R. Levesque (Ed.), *Encyclopedia of adolescence* (pp. 1534-1537). New York: Springer.
- Del Giudice, M., & Belsky, J. (2010). Sex differences in attachment emerge in middle childhood: An evolutionary hypothesis. *Child Development Perspectives*, *4*, 97-105.
- Del Giudice, M., & Belsky, J. (2011). The development of life history strategies: Toward a multi-stage theory. In D. M. Buss & P. H. Hawley (Eds.), *The evolution of personality and individual differences* (pp. 154-176). New York: Oxford University Press.
- Del Giudice, M., Ellis, B. J., & Shirlcliff, E. A. (2011). The Adaptive Calibration Model of stress responsivity. *Neuroscience & Biobehavioral Reviews*, *35*, 1562-1592.
- Del Giudice, M., Gangestad, S. W., & Kaplan, H. S. (2015). Life history theory and evolutionary psychology. In D. M. Buss (Ed.), *The handbook of evolutionary psychology – Vol 1: Foundations* (2nd ed.) (pp. 88-114). New York: Wiley.
- Ellis, B. J. (2013). The hypothalamic-pituitary-gonadal axis: A switch-controlled, condition-sensitive system in the regulation of life history strategies. *Hormones and Behavior*, *64*, 215-225.
- Ellis, B. J., & Del Giudice, M. (2014). Beyond allostatic load: Rethinking the role of stress in regulating human development. *Development and Psychopathology*, *26*, 1-20.

- Ellis, B. J., Del Giudice, M., Dishion, T. J., Figueredo, A. J., Gray, P., Griskevicius, V., et al. (2012). The evolutionary basis of risky adolescent behavior: Implications for science, policy, and practice. *Developmental Psychology, 48*, 598-623.
- Ellis, B. J., & Essex, M. J. (2007). Family environments, adrenarche and sexual maturation: A longitudinal test of a life history model. *Child Development, 78*, 1799-1817.
- Ellis, B. J., Figueredo, A. J., Brumbach, B. H., & Schlomer, G. L. (2009). The impact of harsh versus unpredictable environments on the evolution and development of life history strategies. *Human Nature, 20*, 204-268.
- Frankenhuis, W. E., & Del Giudice, M. (2012). When do adaptive developmental mechanisms yield maladaptive outcomes? *Developmental Psychology, 48*, 628-642.
- Gangestad, S. W., Caldwell Hooper, A. E., & Eaton, M. A. (2012). On the function of placental corticotropin-releasing hormone: A role in maternal-fetal conflicts over blood glucose concentrations. *Biological Reviews, 87*, 856-873.
- Geary, D. C. (2010). *Male, female: the evolution of human sex differences*. Washington, DC: American Psychological Association.
- Ghetti, S., & Bunge, S. A. (2012). Neural changes underlying the development of episodic memory during middle childhood. *Developmental Cognitive Neuroscience, 2*, 381-395.
- Giedd, J. N., & Rapoport, J. L. (2010). Structural MRI of pediatric brain development: What have we learned and where are we going? *Neuron, 67*, 728-734.
- Gluckman, P. D., Hanson, M. A., & Spencer, H. G. (2005). Predictive adaptive responses and human evolution. *Trends in Ecology & Evolution, 20*, 527-533.
- Haig, D. (1993). Genetic conflicts in human pregnancy. *Quarterly Review of Biology, 68*, 495-532.
- Hawley, P. H. (2014). Ontogeny and social dominance: A developmental view of human power patterns. *Evolutionary Psychology, 12*, 318-342.
- Hayiou-Thomas, M. E., Dale, P. S., & Plomin, R. (2012). The etiology of variation in language skills changes with development: A longitudinal twin study of language from 2 to 12 years. *Developmental Science, 15*, 233-249.
- Herd, G., & McClintock, M. (2000). The magical age of 10. *Archives of Sexual Behavior, 29*, 587-606.
- Hochberg, Z. (2008). Juvenility in the context of life history theory. *Archives of Disease in Childhood, 93*, 534-539.
- Hochberg, Z. (2010). Evo-devo of child growth III: Premature juvenility as an evolutionary trade-off. *Hormone Research in Paediatrics, 73*, 430-437.
- House, B. R., Silk, J. B., Henrich, J., Barrett, H. C., Scelza, B. A., Boyette, A. H., et al. (2013). Ontogeny of prosocial behavior across diverse societies. *Proceedings of the National Academy of Sciences USA, 110*, 14586-14591.
- Jambon, M., & Smetana, J. G. (2014). Moral Complexity in middle childhood: Children's evaluations of necessary harm. *Developmental Psychology, 50*, 22-33.
- Joffe, T. H. (1997). Social pressures have selected for an extended juvenile period in primates. *Journal of Human Evolution, 32*, 593-605.

- Kaplan, H. S., & Gangestad, S. W. (2005). Life history theory and evolutionary psychology. In D. M. Buss (Ed.), *Handbook of evolutionary psychology* (pp. 68–95). Hoboken, NJ: Wiley.
- Klauser, P., Whittle, S., Simmons, J. G., Byrne, M. L., Mundy, L. K., Patton, G. C., et al. (2015). Reduced frontal white matter volume in children with early onset of adrenarche. *Psychoneuroendocrinology*, *52*, 111-118.
- Knafo, A., & Plomin, R. (2006). Prosocial behavior from early to middle childhood: Genetic and environmental influences on stability and change. *Developmental Psychology*, *42*, 771-786.
- Kramer, K. L. (2011). The evolution of human parental care and recruitment of juvenile help. *Trends in Ecology and Evolution*, *26*, 533-540.
- Kuzawa, C. W., Chugani, H. T., Grossman, L. I., Lipovich, L., Muzik, O., Hof, P. R., et al. (2014). Metabolic costs and evolutionary implications of human brain development. *Proceedings of the national Academy of Sciences USA*, *111*, 13010-13015.
- Lagattuta, K. H., Sayfan, L., & Blattman, A. J. (2009). Forgetting common ground: Six- to seven-year-olds have an overinterpretive theory of mind. *Developmental Psychology*, *46*, 1417–1432.
- Lancy, D. F., & Grove, M. A. (2011). Getting noticed: Middle childhood in cross-cultural perspective. *Human Nature*, *22*, 281-302.
- Lawrence, A. A. (2009). Erotic target location errors: An underappreciated paraphilic dimension. *Journal of Sex Research*, *46*, 194-215.
- Lebel, C., Walker, L., Leemans, A., Phillips, L., & Beaulieu, C. (2008). Microstructural maturation of the human brain from childhood to adulthood. *NeuroImage*, *40*, 1044-1055.
- Locke, J. L., & Bogin, B. (2006). Language and life history: A new perspective on the development and evolution of human language. *Behavioral and Brain Sciences*, *29*, 259–280.
- Martel, M. M. (2013). Sexual selection and sex differences in the prevalence of childhood externalizing and adolescent internalizing disorders. *Psychological Bulletin*, *139*, 1221–1259.
- Meaney, M. J. (2010). Epigenetics and the biological definition of gene x environment interactions. *Child Development*, *81*, 41-79.
- Nettle, D., Dickins, T. E., Coall, D. A., & de Mornay Davies, P. (2013). Patterns of physical and psychological development in future teenage mothers. *Evolution, Medicine, and Public Health*, *2013*, 187-196.
- Ober, C., Loisel, D. A., & Gilad, Y. (2008). Sex-specific genetic architecture of human disease. *Nature Reviews Genetics*, *9*, 911-922.
- Piccardi, L., Leonzi, M., D'Amico, S., Marano, A., & Guariglia, C. (2014). Development of navigational working memory: Evidence from 6- to 10-year-old children. *British Journal of Developmental Psychology*, *32*, 205–217.
- Rozin, P. (1990a). Development in the food domain. *Developmental Psychology*, *26*, 555-562.

- Rozin, P. (1990b). Getting to like the burn of chili pepper: Biological, psychological, and cultural perspectives. In B. G. Green, J. R. Mason, and M. R. Kare (Eds.), *Chemical senses –Volume 2: Irritation*. New York: Dekker.
- van Beijsterveldt, T. C. E. M., Bartels, M., Hudziak, J. J., & Boomsma, D. I. (2003). Causes of stability of aggression from early childhood to adolescence: A longitudinal genetic analysis in Dutch twins. *Behavior Genetics*, *33*, 591–605.
- Weisner, T. S. (1996). The 5–7 transition as an ecocultural project. In A. Sameroff & M. Haith (Eds.), *The five to seven year shift: The age of reason and responsibility* (pp. 295–326). Chicago, IL: University of Chicago Press.
- Wells, J. C. K. (2007). Sexual dimorphism of body composition. *Best Practice & Research Clinical Endocrinology & Metabolism*, *21*, 415-430.
- West-Eberhard, M. J. (2003). *Developmental plasticity and evolution*. New York: Oxford University Press.