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Review

The juvenile transition: A developmental switch point in human life history

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ABSTRACT

This paper presents a new perspective on the transition from early to middle childhood (i.e., human juvenility), investigated in an integrative evolutionary framework. Juvenility is a crucial life history stage, when social learning and interaction with peers become central developmental functions; here it is argued that the "juvenile transition" is a developmental switch point in the human life history, when both sex-related and individual differences in reproductive strategies are expressed after the assessment period provided by early childhood. Adrenarche, the secretion of adrenal androgens starting at the beginning of middle childhood, is proposed as the endocrine mechanism mediating the juvenile transition. It is argued that, in connection with the stress system, adrenal androgens enable adaptive plasticity in the development of reproductive strategies through integration of environmental and genetic factors. Finally, evidence is reviewed of both sex-related and individual differences arising during the juvenile transition, in the domains of attachment and aggression. Juvenility plays a central role in the ontogeny of behavior and personality; this paper contributes to defining its place within an integrated model of human development.

Introduction

In this paper we present a new, integrative perspective on the ontogenetic transition from early to middle childhood, in the framework of evolutionary developmental psychology. We will argue that this transition (which we label the *juvenile transition*) represents a crucial turning point in human behavioral development, when the cues provided by the early environment combine with new genetic

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factors to affect the trajectory of the individual's reproductive strategy. Moreover, we will propose that this ontogenetic shift is regulated and coordinated by sex hormones through the onset of "adrenal puberty" or *adrenarche*, and explore the many interesting implications of this endocrine mechanism for the pattern of sex-related and individual differences arising in middle childhood.

In this perspective, the transition from early to middle childhood has broad-ranging implications for children's later development, and can be expected to affect many areas of behavior, from attachment to aggression and sexuality. Despite its importance, however, the middle childhood phase (which, as we will discuss later, is another label for human *juvenility*) has been traditionally neglected, not only in psychology but also in the fields of human biology and primatology, as researchers focused their attention on the study of early childhood and adolescence. This situation has been changing steadily in the past years, and now there is a rich base of empirical knowledge to draw upon. Still, the relevant data are not well organized, and lie scattered across various academic disciplines and subdisciplines; in this paper, we begin to integrate those data in a coherent biological framework, and argue that the juvenile transition deserves a privileged place in a complete evolutionary theory of human development. Our aim is twofold: on one side, we aim at describing the *normative* characteristics of the juvenile transition, as an universal stage of human development. On the other hand, we want to provide a framework for understanding how *individual differences* in behavior are expressed and modulated in the juvenile transition, thus contributing to plasticity in the developmental trajectory towards adulthood.

A sketch of our argument

In building our argument, we will take a number of side trips to disciplines and concepts that have traditionally received little attention in developmental psychology,[1] and may thus be unfamiliar to the reader. It is then useful to start by providing a sketch of the main points we will discuss. In synthesis, we will argue that

1. The human life history can be described as a sequence of stages, including infancy, childhood, juvenility, adolescence, and adulthood. Ontogenetic stages have at least two key functions for organisms: modularizing development and coordinating the timing of phenotypic changes related to life history patterns.
2. Human juvenility is a crucial life history stage, in which social learning and interaction with peers become central developmental functions. During juvenility, vital social abilities (such as parenting, competition, coalition building, and sometimes sexuality) are first practiced, and can significantly affect the individual's social standing and future opportunities. In contrast to early childhood, many behavioral traits in juvenility are strongly shaped by sexual selection, and are related to within-sex competition and other sex-specific activities.
3. The transition from childhood to juvenility requires a phenotypic switch, to coordinate the appropriate suite of traits and behaviors in an adaptive fashion. This switch is provided by the mechanism of adrenarche, the pre-pubertal secretion of adrenal androgens which usually starts between 6 and 8 years of age in boys and girls alike. Adrenal androgens can activate sexually differentiated neural and endocrine pathways, and thus powerfully influence behavior, even if they have minimal effects on physical development.
4. The onset of adrenarche induces a set of co-ordinated, adaptive changes in behavior. These changes are both sex-related (through activation of sexually differentiated pathways) and at the level of individual differences (through expression of inter-individual genotypic variation, and interaction with environmental factors such as early stress). In particular, we will discuss fascinating evidence

[1] We conceive of evolutionary developmental psychology in a broad sense, as the evolution-informed study of psychological and behavioral development. We thus do not emphasize finer distinctions between evolutionary psychology, human behavioral ecology, evolutionary anthropology and so on. Given the almost complete metatheoretical overlap among these approaches, we regard such distinctions as minor differences in perspective within the same basic framework.

of such changes in the areas of attachment and aggression. The timing of the transition itself appears to be adaptively plastic, as shown by recent research on the developmental effects of stress and parenting.

5. The juvenile transition can be conceptualized as a developmental switch point in human development, when adaptive individual differences are expressed and individuals adjust their reproductive strategies to their local environment and genetic dispositions. Adrenarche acts as a regulator of developmental plasticity, physiologically integrating ecological factors (such as stress and relationship with caregivers) with sex-related and genotypic differences, and setting the ontogeny of social behavior on alternative pathways. This function places juvenility in a crucial place within the bigger picture of human life history.

Ages or stages?

In developmental psychology, the concept of "stage" has been employed in many different ways, with occasional debate arising about the usefulness and adequacy of stage-based models (especially in the cognitive domain) for understanding development (Brainerd, 1978; Flavell, 1982). The concept of stage we employ is the biological one (commonly used in evolutionary anthropology and primatology), and is based on the major functional, morphological and physiological transitions in ontogeny (see below for the broader evolutionary meaning of stages). It should be stressed at the outset that, in current biological understanding, life stages: (1) may not occur at fixed ages; (2) may not even be bound to occur at all: an organism could have a set of alternative developmental pathways, some of which do not involve expression of a certain stage; and (3) can be sensitive to environmental input and show considerable plasticity, even when they are co-ordinated by physiological mechanisms (such as hormones). Thus, while basing our theory on a sequence of life stages, we do *not* imply developmental fixity, or any form of so-called "biological determinism".

In addition, we want to avoid tying the juvenile transition to a specific *age*: as we will explain in a later section, adrenarche is the hormonal switch initiating human juvenility, and its timing (just like that of puberty) is highly variable between individuals, usually in a range between 5 and 10. The modal transitional age in western countries is somewhere around 7 years, which corresponds to the conventional threshold usually employed to demarcate early from middle childhood; however, in addition to individual variation, it is reasonable to suppose (lacking specific research on this issue) that adrenarche timing should also show a degree of ethnic and regional variation similar to that observed in the timing of puberty (e.g., deMuinck Keizer-Schrama & Mul, 2001; Juul et al., 2006; Parent et al., 2003). For all these reasons, chronological age is an inadequate proxy as a marker of the juvenile transition. Consider as an example the notion of "5- to 7-years shift", referring to the phase of rapid cognitive (e.g., self-regulation, reflection, strategic planning) and social development observed in children of this age (Collins, 1984; Sameroff & Haith, 1996; Weisner, 1996; White, 1965). While this label roughly captures an interesting developmental phase, if we were to investigate changes during this age range the "shift" would appear fuzzy and highly variable (due to individual variation in developmental timing), and its descriptive usefulness could be reasonably questioned. An even more serious problem with age-based models is that they invite looking at individual variation as "noise"; in an evolutionary life history framework, on the contrary, part of the individual variation in transition timing can be seen as adaptive, thus helping to illuminate the functional significance of stages themselves.[2]

[2] This, of course, does not mean that *all* individual variation is adaptive; variation can also arise because of neutral (or harmful) mutations, by random environmental influences, or as a byproduct of other adaptive traits. Attributing adaptive significance to variation is no different from describing a trait as an adaptation, and requires the same kind of theoretical and empirical support (see Schmitt & Pilcher, 2004). Nevertheless, evolutionary reasoning often permits a deeper appreciation of the functional meaning of variation, even variation that is commonly conceived of as "pathological" (see for example Chisholm, 1999; Figueredo et al., 2006).

The interpretation of behavior genetic data

In this paper we will sometimes refer to data from behavior genetic studies. Some developmental theorists have challenged the usefulness of behavior genetics (e.g. Gottlieb, 1995) by arguing that, given the indirect and nonlinear mapping between genotype and phenotype, behavior genetic data say little or nothing on the complex causal mechanisms underlying development and can be interpreted in rigidly deterministic ways. Although such critiques have some merit, they do not imply that behavior genetics is invalid or systematically misleading; in addition, the idea that behavior genetics is fundamentally inconsistent with developmental theory is definitely overstated (see for example the responses to Gottlieb by Scarr (1995) and Turkheimer, Goldsmith and Gottesman (1995)). We believe that behavior genetic data, properly interpreted, can provide information relevant to evolutionary-developmental models (for a similar position see Belsky, 2005; Figueredo, Vásquez, Brumbach, & Schneider, 2004; Figueredo et al., 2006; Segal & Hill, 2005). However, some caveats and qualifications are in order. First of all, behavior genetic data are often little informative about the *processes* underlying the development of a given trait. Especially in the simpler designs, quantitative genetic models essentially attempt to partition the phenotypic variance of a trait in a population into a number of components, usually an additive genetic component (or narrow-sense *heritability*), a shared environment (or within-families) component, and a nonshared environment (or between-families) component. These labels, however, can mislead if interpreted nontechnically: for example, the nonshared component actually includes "objectively shared" aspects of the environment (e.g., parental divorce) that, for whatever reason, affect siblings in different ways. It also includes some kinds of gene–environment interactions and (importantly) all the measurement error. For an introduction to the concepts and vocabulary of behavior genetics see Carey (2003), Evans, Gillespie, and Martin (2002). A second caveat is that heritability, the proportion of individual variation attributable to additive genetic effects, is always a local measure that refers to a given population in a given environment. For example, restriction in the range of environments experienced by a population usually leads to higher heritability estimates. Thus, to properly interpret a heritability coefficient one should carefully consider the population and environment it refers to. In addition, the meaning of variance components may be distorted by the presence of unaccounted effects such as gene–environment correlations and interactions. Various kinds of gene–environment interplay can be modeled and tested (see Rutter, 2007; Rutter, Moffitt, & Caspi, 2006), but simple designs are usually unable to provide this information. Finally, genetic effects may often be detected more easily and reliably than environmental effects (Turkheimer, 2004; Turkheimer & Gottesman, 1996; Turkheimer & Waldron, 2000). Even when the environment has strong *causal* effects on the development of individual differences in a trait, these effects may turn out to be unsystematic, nonlinear, and subject to phenotype–environment matching across development; all these factors would render them very difficult to detect with standard statistical methods. Thus, even environmentally sensitive traits may show large heritabilities and little evidence of systematic environmental influence.

Despite their limitations and interpretive difficulties, behavior genetic data can nevertheless provide useful information to developmental researchers. Among the more informative data there are unusual patterns of heritability, changes in variance components across development, and changes in the correlations between genetic (or environmental) components at different ages. More recently, behavior geneticists have started to measure and model the effect of specific environmental effects, sometimes in conjunction with the direct assessment of individual genotypes (see Rutter, Pickles, Murray, & Eaves, 2001; Rutter et al., 2006).

Life histories and developmental stages

In this section, we will frame our theoretical perspective by briefly introducing the main concepts of life history theory and by discussing the evolutionary meaning of developmental stages.

Life history theory

Life history theory is a branch of modern evolutionary biology devoted to the formal analysis of the patterns of growth, development and reproduction of living organisms. Its fundamental question is,

how do organisms allocate their limited resources to vital functions (such as growth and reproduction) during their lifetime, so as to maximize their expected fitness? In fact, all organisms live in a world of limited resources. The energy that can be extracted from the environment in a given amount of time, for example, is limited. Time itself is also a limited good: the time spent by an organism looking for mates cannot be used to search for food, or to care for already-born offspring. Since all these activities contribute to an organism's evolutionary fitness, devoting time and energy to one will typically involve both benefits and costs; and natural selection will strongly favor organisms that are able to adopt an optimal scheduling of activities.

Life history theory (see Hill, 1993; Kaplan & Gangestad, 2005; McNamara & Houston, 1996; Roff, 2002) uses mathematical modelling to solve the complex optimization problem of how, and when, to allocate limited resources to gain the maximum reproductive success. Life history strategies (also called *reproductive strategies*[3]) are, in a nutshell, adaptive solutions to a number of simultaneous fitness trade-offs. The most basic trade-offs are between *somatic effort* (i.e., growth, body maintenance, and learning) and *reproductive effort*; and, within reproductive effort, between *mating* (i.e., finding and attracting mates, conceiving offspring) and *parenting* (i.e., investing resources in already conceived offspring). From another perspective, the crucial decisions involved in a life history (or reproductive) strategy can be summarized by the trade-offs between *current* and *future reproduction*, and between *quality* and *quantity of offspring*. Is the organism going to reproduce as soon as it can, or to wait longer, in order to accumulate resources that can then increase offspring "quality" and reproductive value? The more time spent waiting, the more resources (e.g., energy reserves, but also ability and social status) could become available, but the risk of dying before reproducing will increase as well. And is the organism going to put all of its reproductive effort into increasing the number of offspring, or will it channel resources and parenting effort into increasing the quality and long-term prospects of a few, selected descendants?

One of the most important findings of life history theory is that no strategy can be optimal in every situation; more specifically, the optimal (i.e., fitness-maximizing) strategy for a given organism depends on its ecology and on a series of factors such as resource availability, mortality risk and environmental uncertainty. Indeed, organisms usually embody mechanisms that allow them to fine-tune their life histories according to the environmental cues they encounter during development. In other words, life history strategies show *adaptive developmental plasticity*.

Adaptive plasticity in life history strategies

Due to the variety of ecological niches they live in (and the corresponding trade-offs), different species show impressive variation in their life history strategies, and organisms differ wildly in their growth rate, size, lifespan, fertility, and number of offspring. Life history traits (e.g., growth rate and age at reproduction) do not only differ between species, however: a remarkable degree of variation is usually observed within the same species as well. Why is it so? First of all, in sexually reproducing species the sexes face different trade-offs. For example, in many animals (including humans; see Ellison, 2001; Geary, 1998) males take longer to reach maturity, since they have to compete with other males for reproduction and need to accumulate the size, strength and ability needed to succeed. Males also devote more resources to mating, and less to parenting, than females; they generally pay lower costs for reproduction, and can potentially reproduce at a faster pace (Trivers, 1972). Thus, the optimal life history strategy in a given species is usually not the same for males and females.

Another reason for variability is that, even within the same species, individuals find themselves in different environmental conditions, which may result in different strategic trade-offs. Some individuals, for example, may face higher mortality risks, perhaps because of predation or diseases; others may live in a place or time in which food is scarce; and others still may live in groups where the ratio of females to males is especially low, thus engendering fiercer competition among males for access to females. Due to such environmental variability, reproductive traits and strategies tend not to be genetically fixed, but rather evolve to show adaptive developmental plasticity (see Ellis, Jackson, & Boyce, 2006, for an introduction; West-Eberhard, 2003, for a comprehensive account). Organisms assess their

[3] Note that reproductive strategies are different from "mating strategies": as usually conceptualized in evolutionary psychology, mating strategies only refer to long- vs. short-term mating preferences, thus representing a very narrow subset of the behaviors involved in a reproductive strategy.

local environments and adjust their strategic allocation choices, following evolved rules that maximize expected fitness in different ecological conditions.

What constitutes an "optimal" reproductive strategy is, to a degree, contingent on local conditions: for example, when mortality is high and unavoidable, the optimal strategy is to mature faster and engage in earlier reproduction, investing the available resources in a higher number of offspring (even at the expense of their quality). In safer environments, however, the optimal strategy may be that of maturing late, and investing more resources in a smaller number of offspring. It is important to note that different reproductive strategies will not be limited to differences in sexual maturation or fecundity, but will often involve a suite of reproduction-related *behavioral* traits such as risk-taking, dominance seeking, aggression, altruism/cooperation, and long-term attachment to mates. These traits are functional to implementing a given strategy (e.g., increased mating effort usually requires increased aggression to compete with rivals), and can be expected to covary in broad "clusters" along the main life history dimensions, such as current vs. future reproduction and parenting vs. mating (Belsky, Steinberg, & Draper, 1991; Chisholm, 1999; Figueredo et al., 2006; Kaplan & Gangestad, 2005). For example, recent theoretical modelling by Wolf, van Doorn, Leimar, and Weissing (2007) shows that individual differences in present- vs. future-oriented reproductive strategies can be expected to result in consistent personality differences in a suite of risk-related traits, such as boldness and aggression. A similar concept, although not explicitly grounded in life history theory, has been proposed by Korte, Koolhaas, Wingfield, and McEwen (2005): they described two general behavioral phenotypes found in many animal species, labeled "hawks" (risk-prone, aggressive, bold) and "doves" (fearful, nonaggressive, and shy), characterized by specific functional differences in the stress response and (presumably) by related genetic differences. There is also initial evidence that, in humans, a broad life history-related factor (labeled the *K*-Factor) could account for 70–90% of reliable variance in a cluster of traits including attachment security, mating style, impulsivity, risk-taking, and altruism (reviewed in Figueredo et al., 2006).

Adaptive plasticity refers to those organismic responses to the environment that enhance the organism's fitness (plasticity *per se* can also be fitness-neutral, or even maladaptive). This implies that the relevant variables in the environment must be detected with some reliability, and that the organism must be equipped to assess them and to respond appropriately. While adaptive plasticity is widespread, it may not always be the best option: for example, if the cost of maintaining the mechanisms that regulate plasticity is high, or if there are no reliable cues in the environment on which to base the organism's strategy, natural selection can favor fixed alternative phenotypes based on genetic polymorphism.[4] This may also be the case when there are multiple ecological niches in the environment, and individuals are free to select the niche that best fits their phenotype (Wilson, 1994). As a general rule, genetic polymorphism in a population is maintained when the resulting phenotypes have equal fitness, and thus represent different ways of solving the same ecological problem with equivalent success. Plastic (condition-dependent) phenotypes, on the other hand, can also be ways of "making the best of a bad job", and thus be maintained even if their outcomes in terms of fitness are unequal.

A crucial question is, then, to what degree should life history traits be developmentally contingent and plastic, rather than canalized and more strictly determined by genotype. The answer is definitely not a simple one; what is typically found in organisms is a mixture of the two, and theoretical models suggest that we should often expect a balance between genetic and environmental determination of phenotypic individual differences. At the population level, the opportunity for habitat choice plus temporal variation in environmental conditions can maintain a polymorphic population composed of both "specialists" (fixed phenotypes) and "generalists" (plastic phenotypes), as shown by Wilson and Yoshimura (1994). At the individual level, a recent model by Leimar, Hammerstein, and Van Dooren (2006) shows that, in a broad range of conditions, plasticity switches should evolve so as to integrate both genetic and environmental information in phenotype determination. Also working from an evolutionary perspective, Belsky (1997, 2000, 2005); see also Belsky, Bakermans-Kranenburg, & van Ijzendoorn, 2007) argued that it may be adaptive for individuals to differ in their degree of

[4] Genetic polymorphism is the presence, in a population, of two or more discrete phenotypes (underlied by genotypic differences) that are maintained by natural selection (i.e., do not result exclusively from new genetic mutations).

susceptibility to the rearing environment. Since the behavior of parents sometimes constitutes a poor (i.e., unreliable) guide to future environmental conditions, there is an amount of risk in being shaped by parental input; thus, it would be advantageous for both parents and offspring if the development of at least some children was relatively unaffected by parental behavior. This form of "bet-hedging" would lead to differential susceptibility: while some children should be sensitive and developmentally plastic in response to their environment, others should be less responsive and more similar to fixed strategists. As we will see in a later section, recent molecular genetic studies are providing initial support for this hypothesis.

Consistent with the expectation that individual differences in plastic traits should also show genotypic effects, the life history factor described by Figueredo and colleagues (2006) has substantial heritability (.65 in a middle-age twin sample; Figueredo et al., 2004), as do other life history-related traits such as age at menarche (eg. Campbell & Udry, 1995; Rowe, 2000; Treloar & Martin, 1990).[5] In the present paper, we argue that the juvenile transition in human children is a *developmental switch point* (West-Eberhard, 2003; see also Ellis et al., 2006; Leimar et al., 2006), where environmental cues are integrated with genotypic factors, resulting in individual differences in reproductive strategies and in the related behavioral traits.

The emergence of developmental stages

It is apparent from the above discussion that, both in the course of development and in adult life, organisms must set their priorities and schedule their growth and activities according to a locally optimal life history strategy. This requires achieving tight coordination between physiology and behavior, and the emergence of stages is an effective solution to this problem. For example, if an organism's optimal life history involves a period of growth and resource accumulation before reproduction, there have to be evolved mechanisms that (1) promote bodily growth; (2) keep the neural-behavioral machinery devoted to mating shut off, or at least reduce its activity; (3) intensify behaviors related to resource seeking and acquisition; and possibly, (4) track the level of available resources to adjust the duration of the current stage and/or influence future mating behavior. Although life history trade-offs need not lead to mutually exclusive choices (e.g., mating and parenting effort may coexist up to a point), the likely interference between different fitness-related activities encourages a degree of developmental specialization. Of course, not all development proceeds by stages; but it is reasonable to expect that the main trade-offs related to growth, resource acquisition (including "social" resources such as status and ability), mating, and parenting will be co-ordinated by means of developmental stages and transitions.

Evolutionary modularity and the role of hormones

To gain a deeper understanding of the biological meaning of life stages, it is useful to consider them from the perspective of *evolutionary modularity*. At different times, an organism expresses different traits (morphological as well as behavioral); the traits expressed in a given stage, which make up the individual's phenotype at that time, contribute to the organism's fitness together and can be the target of natural selection independently from those expressed in other stages. Thus, each life stage can respond to selection (and evolve) in relative autonomy from the others, permitting a variable

[5] As indicated above, high heritability (often coupled with small shared environmental effects: about zero for the K-Factor) does not rule out trait plasticity; it does suggest, however, that the effects of the environment are not systematic and/or linear, for example because of gene-environment interactions (Rutter et al., 2006) and phenotype-environment interplay during development (Turkheimer, 2004; Turkheimer & Gottesman, 1996; Turkheimer & Waldron, 2000). Extensive gene-environment interactions, leading to small shared-environmental effects in biometric models, are predicted by Belsky's differential susceptibility hypothesis (since genotypes differ in their sensitivity to the environment). In addition, as people grow up they can select their environments to match their phenotypes, especially in modern societies where specialized niches abound (e.g., a highly aggressive adolescent boy may join a gang and be imprisoned, with each of these events further increasing his aggressiveness), leading to a kind of genotype-environment correlation that would raise estimated heritability. It would be interesting to see whether the heritability of the K-Factor, which was high in the middle-age sample studied by Figueredo and colleagues, increased from childhood to adulthood as it would be expected in this case.

degree of "disconnection" between stage-specific phenotypes (West-Eberhard, 2003; Wilkins, 2002). At the molecular level, the selective expression of different traits is permitted by expression of different sets of genes: thus, transitions between life stages involve the turning on and off of co-expressed genetic networks. The specific suite of traits expressed in a given life history stage fulfills the definition of *evolutionary module*: a set of phenotypic features that are highly integrated by pleiotropic effects of the underlying genes,[6] and relatively isolated from other such sets by a paucity of pleiotropic effects (Wagner & Altenberg, 1996; Wagner, Mezey, & Calabretta, 2005). Of course, genetic disconnection between stages needs not be complete; in this perspective, modularity is a matter of degree rather than an all-or-none property of a phenotype. There are also sets of genes that are expressed over many (or all) an organism's life stages, and that promote continuity and integration across time (needless to say, stability in the environment can also promote developmental continuity).

Finally, we should note that, when a set of genes is expressed only (or predominantly) during a given stage, the effects of genetic variation on the corresponding trait will be "hidden" until that stage is reached. Imagine an allelic variant of a gene involved in a butterfly's wing color determination, which has the effect of making the wings blue instead of white (the example is fictitious). If this gene is only expressed in the adult stage, genotypic variability between individuals will be invisible until they become adults, and all larvae will look the same irrespectively of the allele they carry. Put in another way, individual differences in genotype will not translate into individual differences in phenotype until the corresponding genetic network is activated and expressed.

What, then, could be the mechanism that coordinates stage-specific gene expression and regulates the transition between different life stages? For most organisms this role is played by hormones (Adkins-Regan, 2005; Heyland, Hodin, & Reitzel, 2005). Hormones are very special molecules in this respect: they can reach virtually every cell in the body, thus carrying signals to different tissues (e.g brain, muscle, bone, fat reserves, immune system) at the same time. And steroid hormones, which bind to intracellular nuclear receptors (NRs) in the cytoplasm and then to DNA (where they regulate gene transcription), can literally "talk to the genome". This property of hormonal signalling systems allows them to act as crucial nodes in complex regulatory networks, co-ordinating the expression of traits in the whole organism (Dufty, Clobert, & Møller, 2002; Heyland et al., 2005). Since hormone secretion can be controlled top-down by brain centers, hormonal regulation of between-stage transitions allows for remarkable plasticity, making them sensitive to social and environmental cues. As a result, life history transitions in animals (including humans) are usually mediated and regulated by endocrine mechanisms (see Adkins-Regan, 2005, for a thorough discussion). More generally, it is becoming increasingly clear that the endocrine system is crucially involved in the regulation of developmental plasticity in most species, by integrating and "interpreting" environmental variation and adaptively shaping the development of the whole organism (Dufty et al., 2002; Kaplan & Gangestad, 2005; Nijhout, 2003; Ricklefs & Wikelski, 2002).

The evolution of childhood and juvenility

The slow primate

Among primates, *Homo sapiens* shows a rather peculiar combination of life history features. It has a long lifespan, a big brain, gives birth to big babies, and has a relatively high fertility (with inter-birth intervals of about 2.5–3.5 years). More than any other primate, humans develop slowly—with an extended period of juvenile dependence, late puberty, and years (even decades) of protracted and intensive parental effort (Bogin, 1999; Flinn & Ward, 2005; Mace, 2000). Biologists and anthropologists have long been fascinated by this pattern, and have tried to track the evolutionary forces that relate it to the ecological, social and cognitive characteristics of the human species. Some anthropologists (Kaplan, Hill, Lancaster, & Hurtado, 2000; Kaplan & Robson, 2002) propose that the entire pattern of

[6] A gene is pleiotropic when it is involved in the expression of more than one phenotypic trait, so that allelic variation can affect many traits simultaneously. With respect to stages, pleiotropy would refer to genes that affect behavior at different time points, thus creating genetic correlations across stages.

human life history traits can be explained as a co-evolved response to a dietary shift toward high-quality food, acquired through skill-intensive practices (e.g., big game hunting, food processing). In this model, extended juvenility evolved as a learning period in which to acquire sophisticated foraging skills; in return, the low productivity of juveniles would have been compensated for by quality food provision from males and older, post-reproductive relatives (thus explaining the long lifespan). The key to human adaptation would be the intergenerational resource flow from the old to the young, and skill learning (described as investment in "embodied capital") would be the primary function of our long juvenility.

Other theoretical models are instead based on mortality reduction, or "ecological risk aversion" (Janson & van Schaik, 1993): if juveniles could reduce their risk of starvation and predation by growing at a slow rate, a long juvenility would be favored by natural selection. Recently, Gurven and Walker (2006) proposed a model in which slow juvenile growth followed by an adolescent growth spurt maximizes fertility, by lowering nutritional demands and freeing resources that can be devoted to feeding younger siblings, thus allowing for bigger family sizes (see also Bogin, 1997, 1999, for a similar view). During this slow-growth period, the child's energetic resources can be spent to produce a bigger brain and to boost immune function. In this theoretical framework, juvenility did not evolve primarily for skill-learning, but it would nonetheless *permit* extensive learning: according to Pereira and Fairbanks (1993), primate juveniles are "specialized for the task of surviving the wait until reproduction and of using that time wisely". However, once extended skills/social learning became possible thanks to a long juvenility, a self-reinforcing cycle could have ensued, in which the advantages of learning generated an evolutionary pressure to increase juvenility even further and promote the growth of even bigger brains (Pagel & Harvey, 1993).

Finally, some authors stress the importance of social skills learning to the point of making it the primary evolutionary function of human juvenility (Alexander, 1989, 1990; Flinn & Ward, 2005; Joffe, 1997). In this perspective, fast and sustained brain growth coupled with a long juvenile phase is not due to the need of practicing foraging skills, but to that of learning how to manage the complexity of human social relationships. From the time humans became an ecologically dominant species, it is argued, they also became the main selective force driving their own evolution, in a within-species coevolutionary arms race based on coalition formation (Alexander, 1990). The delay in achieving adult size and aspect would also be advantageous for social learning, since it would reduce juvenile competition with adults and allow for relatively risk-free social experimentation.

As it is apparent from the brief synthesis above, there is still substantial uncertainty about the phylogeny of our life history pattern, especially regarding the exact sequence of evolutionary pressures leading to our present developmental trajectory. The picture is rendered more complex by the likelihood of strong coevolutionary dynamics, in which different factors (e.g., brain size, growth rate and food provisioning) mutually reinforce each other's evolution. In addition, different aspects of childhood and juvenility might be primarily due to different adaptive reasons; for example, brain growth and body growth may respond in part to different selective pressures (e.g., social learning vs. mortality reduction and increased fertility). Testing these competing hypotheses about the evolution of the human life history has become a major enterprise for evolutionary anthropologists, one that involves diverse lines of inquiry ranging from hunter-gatherer studies (e.g., Bock, 2002; Hawkes, O'Connell & Blurton-Jones, 1995; Kramer, 2005) to comparative analysis (e.g., Dunbar & Shultz, 2007; Holekamp, 2007) and mathematical modelling (e.g., Gurven & Walker, 2006; Kaplan & Robson, 2002).

In the following paragraphs, we examine two crucial stages of human development, childhood and juvenility (see Fig. 1), and following Bogin (1997, 1999; Bogin & Smith, 1996; Locke & Bogin, 2006) we examine their likely adaptive functions in more detail.

Childhood

Childhood can be defined as the period following infancy, in which the youngster is weaned from nursing but still depends critically on older people for feeding and protection (Bogin, 1997). In traditional societies, this goes from about 2–3 to 7 years of age. In other primates, when infants are weaned they are relatively independent and begin to feed for themselves; in humans, there is a striking 4-year period in which the child cannot forage and must be protected and fed by means of specially prepared

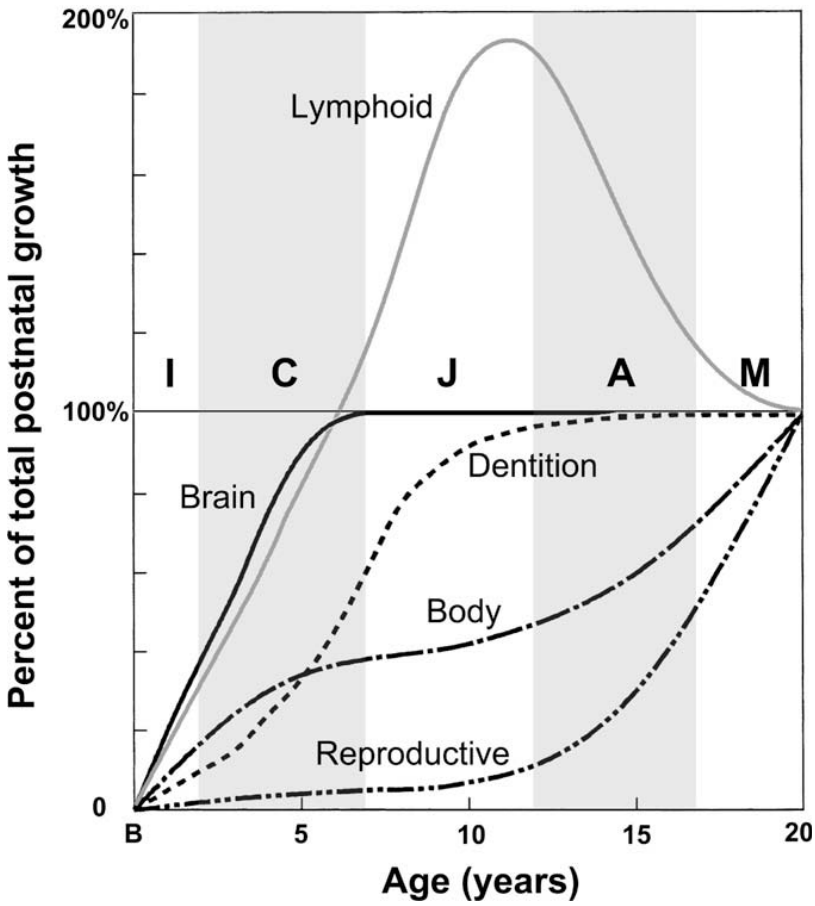

Fig. 1. Schematic growth curves of different parts and tissues of the human body: brain weight, lymphoid tissue weight, stature growth (body), weight of the gonads and primary reproductive organs, and dental maturation. I = infancy, C = childhood, J = juvenility, A = adolescence, M = mature adult. Adapted from Bogin (1997) and Tanner (1955).

food. Fossil evidence suggests that childhood evolved in *Homo habilis*, and that it preceded the evolution of adolescent growth spurt (first found in the late *Homo erectus*, about 1.5 millions of years ago; see Bogin & Smith, 1996).

After infancy, bodily growth decelerates; children grow slowly, at a nearly constant rate. At the same time, however, brain growth proceeds steadily, without signs of deceleration from birth. At age 5, the energetic requirements for brain growth and maintenance still amount to 40–50% of total metabolism, compared with almost 90% in newborns and 20–25% in adults. Children's teeth are still not suitable for chewing adult food, and special food must be prepared to feed the youngsters. Behaviorally, childhood is marked by relatively poor motor ability and coordination, and general cognitive immaturity (which could have an adaptive role in itself; see Bjorklund, 1997). Language, on the other hand, develops quickly: from age 3 to 7 fluency increases dramatically, children become apt at storytelling, and experiment with verbal creativity and language games. In addition, the first instances of verbal competition appear, mirroring the emergence of early dominance hierarchies among children around age 5 (discussed in Locke & Bogin, 2006).

The function of childhood

Bogin (1997) proposed a set of specific adaptive roles of childhood in human life history: (1) to free the mother from direct provisioning, thus enhancing fertility; (2) to stimulate caregiving and nurturing, by maintaining an infantile appearance; (3) to reduce nutritional needs, thus avoiding competition with adults for food resources and the risk of starvation; (4) to allow for "babysitting", since children can be cared for by older juveniles and other relatives; and finally, (5) to permit increased developmental plasticity: childhood allows for 4 more years in which the child can assess the ecological conditions (e.g., parental support, environmental risk, available social and material resources, and

so on) and match his/her phenotype to the local environment (see also Belsky et al., 1991). In the remainder of this paper, we will elaborate on this last point and argue that a crucial function of the juvenile transition is to act as a plasticity "switch point" following the assessment period provided by childhood. Of course, humans are long-lived animals with complex social structures, and they are expected to assess their local environment at multiple points during their lifetimes. In this framework, the juvenile transition is one of the crucial switch points (another being puberty), but individuals can probably readjust (within limits) their reproductive strategies following cues of strategy success/insuccess (e.g., actual mating opportunities, relationships quality, birth of offspring) and of environmental change.

Juvenility

Juvenility is defined as a pre-reproductive life stage in which the youngster is independent from parents for survival, but is still sexually immature. In humans, this phase lasts until the onset of puberty: from 7 to 10 years in girls, and from 7 to 12 years in boys (on average), with considerable variation worldwide. In traditional societies, children in this age range become relatively self-sufficient with respect to feeding and protection from predators and diseases (reviewed in Bogin, 1999; Kramer, 2005).

The juvenile growth pattern of humans is marked by a slight acceleration at the beginning of this stage (known as the "mid-growth spurt"), followed by a further deceleration that brings juvenility to the slowest growth rate from birth. Physically, little happens: the first pubic and axillary hair appears, and there is a beginning of sexual dimorphism in vocal characteristics (Ellison, 2001; Wuyts et al., 2003). All of these features (growth spurt, hair growth and voice change) follow from the secretion of androgens by the adrenal glands, known as *adrenarche*, which is the physiological mechanism underlying the juvenile transition. The eruption of the first permanent molars also occurs at about 6 years, allowing juveniles to process adult-type food. Notably, brain growth (in weight) is almost complete by age 7, so that the brain-related metabolic expenditure of juveniles is relatively low; in contrast, there is a peak in the activity of the immune system, consistent with the "risk aversion" hypothesis advanced by Janson and van Schaik (1993).

At the behavioral level, there is a significant increase in motor coordination and maturation, and important cognitive progress takes place in many areas, from attention control and planning to perceptual acuity (the "5- to 7-years shift"). Many of the cognitive changes in juvenility appear to be relatively independent from schooling (e.g., Morrison, Griffith, & Frazier, 1996). Remarkable progress also occurs in language development, not only in verbal fluency but also in pragmatic abilities such as gossip, argumentation and verbal duels; sex differences in language use become increasingly apparent, with males engaging in more competitive verbal exchanges and females "specializing" in gossip (a trend that will further increase in adolescence; see Locke & Bogin, 2006). Most importantly for our discussion, juvenility is characterized by a dramatic increase in (1) children's social activities with peers, and (2) sex differentiation in these activities. Between 6 and 11 years, for example, there is a peak in fighting and rough-and tumble play (especially boys), play parenting (usually girls), and sex segregation between groups of boys and girls. Boys also engage in more locomotor and exploratory play,[7] with a wider play range than girls (reviewed in Geary, 1998; Smith, 2005). In the same period, the aggression patterns of males and females start to differ significantly, with females engaging in more indirect and relational aggression than males (Pellegrini & Archer, 2005).

The function of juvenility and sexual selection

Whether or not learning is the primary adaptive function of juvenility, it is clear that a remarkable amount of social learning does take place in this stage, and the child's almost full-grown brain provides the equipment for engaging in the complexities of life in the peer group. In a comparative study, Joffe (1997) found positive correlations among social group size, volume of "social" brain areas, and

[7] Exploratory play is defined as play involving active exploration of new places and objects. Of course, play and exploration are distinct concepts and their developmental trajectories do not completely overlap.

duration of juvenility in primates (see also Dunbar, 1998). Social learning and experimentation, however, do not imply that juvenile peer relationships are "cost-free" or without consequences: in this phase of development, children engage in intense social competition for status and dominance within their group, and the outcomes of this competition can carry over well into adulthood. Longitudinal studies of dominance and peer acceptance, for example, suggest that ranks acquired in childhood may be relatively stable over many years (reviewed in Weisfeld, 1999). Of course, in humans (children included) social status is not determined solely by physical dominance, though the latter may have a bigger role than sometimes acknowledged by developmental researchers (e.g., Pellegrini & Bartini, 2001; Rodkin, Farmer, Ruth, & Acker, 2006; Weisfeld, 1999). Successful competition for status can involve a mixture of aggression and cooperation (Hawley, 1999, 2003; Prinstein & Cillessen, 2003), the ability to be chosen as a group member (e.g., Geary, Byrd-Craven, Hoard, Vigil, & Numtee, 2003), and displays of intelligence and linguistic abilities in addition to physical qualities (Locke & Bogin, 2006; Miller, 2000). With reference to the main life history trade-offs, the juvenile stage is largely devoted to somatic effort, although in a different way from childhood (i.e., acquisition of information and social/practical abilities); however, social competition can also be seen as a form of anticipatory mating effort, thus underlining the complex functional role played by juvenility.

The social activities in which juveniles engage are highly sexually differentiated, and prepare them to face the social problems and tasks they will later encounter as adult males and females. Much more than early childhood, juvenility shows signs of *sexually selected* behavioral traits, i.e., traits shaped by within-sex competition and between-sex mate choice (see Geary, 1998, 2002, for an introduction). The differences in play activities, language use, and aggression styles of boys and girls observed in juvenility are a likely result of sexual selection, and it has been hypothesized that sexual selection also drives the sex dimorphism in attachment patterns arising in middle childhood (Del Giudice, in press-a, in press-b; see below). The length of the juvenile stage itself differs between males and females, with boys entering adolescence an average of 2 years after girls. This is consistent with a sexually selected pattern of slower male maturation, with males investing more time in acquiring competitive abilities before they can successfully engage in adult mating behaviors.

While many behaviors in juvenility appear to be shaped by sexual selection, there is no sex dimorphism in height and weight before puberty (Geary, 1998), which may seem puzzling given the role of size in physical competition. This might reflect a trade-off between sexual selection and other adaptive functions of juvenility (e.g., risk aversion, avoiding starvation and/or competition with adults); however, lack of gross sex differences in body shape does not mean that sexual dimorphism in competition-related physical characters is missing altogether. Large sex differences in throwing distance and velocity are apparent from 4-7 years of age (Thomas & French, 1985); boys also have larger arm muscles and stronger hand grip than girls already at six (Henneberg, Brush, & Harrison, 2001; Ruff, 2003). Prepubertal boys have stronger bones than girls (Macdonald, Kontulainen, Petit, Janssens, and Mckay 2006), and sex differences in body composition (less fat and more muscle tissue in boys) increase from 5 to 10 years of age (Shaw, Crabtree, Kibirige, & Fordham, 2007; see also Nagy et al., 1997). Thus, it may be that some physical characteristics are sexually selected in juveniles even in absence of adult-like dimorphism in size.

Adrenarchef

Adrenarche is a maturational phase characterized by several hormonal and structural changes, usually occurring between age 6 and 8 (probably with some ethnic and regional variation; see the introduction). Whereas middle childhood has been traditionally conceived as a hormonally quiescent period, it is becoming apparent that the hormonal changes of adrenarche are crucial to understanding interpersonal and psychological development in this life stage (e. g. McClintock & Herdt, 1996). From an endocrine perspective, adrenarche can be defined as the "awakening of the adrenal glands" (Dorn & Rotenstein, 2004): the adrenal glands produce a large quantity of hormones during fetal development; then, after birth, their activity decreases rapidly and remains low for the first six years of life (Rainey, Carr, Sasano, Suzuki, & Mason, 2002). Around age 6, a gradual rise in adrenal androgen secretion begins, after a long period of inactivity. The developmental pattern of adrenarche is only found in a small

number of primates (Spear, 2000): adrenal androgen levels and their developmental course differ markedly among species, but only chimpanzees and gorillas (who also experience an unusually long juvenility) have been found to exhibit an adrenarche similar to that of humans (Cutler et al., 1978; Ibáñez, Dimartino-Nardi, Potau, & Saenger, 2000).

Structural and hormonal changes in adrenarche

Adrenarche involves both structural and hormonal changes. From the structural point of view, there is a progressive broadening of the size and mass of the adrenal cortex and the expansion of one of its three regions, the *zona reticularis* (Auchus & Rainey, 2004; Dhom, 1973). The zona reticularis synthesizes adrenal androgens, especially dehydroepiandrosterone (DHEA) and dehydroepiandrosterone sulfate (DHEAS; Endoh, Kristiansen, Carson, Buster, & Hornsby, 1996). Starting from about age 6, production of DHEA and DHEAS keeps increasing gradually for the first two decades of life, with higher levels in men than in women (Orentreich, Brind, Rizer, & Vogelman, 1984); it reaches peak levels in the third decade (Parker, 1991) and declines thereafter, in a process often called *adrenopause* (Gray, Feldman, McKinlay, & Longcope, 1991).

Physical consequences of adrenarche, determined by the weakly androgenic effects of DHEA and DHEAS, are the appearance of pubic and axillary hair, changes in body odor, and increased oilness of skin and hair. Adrenal androgens directly affect the central nervous system, with consequences on neural plasticity as well as on memory and emotional behavior, as shown by animal studies (Wolf & Kirschbaum, 1999). In particular, those studies have shown memory-enhancing and antiamnestic properties of DHEA and DHEAS in rodents (mice and rats) using different test paradigms (i.e., Maurice, Su, & Privat, 1998; Melchior & Ritzmann, 1996), and effects of DHEA on aggression in mice (Haug et al., 1989). DHEA and DHEAS modulate the activity of β-aminobutyric acid (GABA) receptors, which in turn regulate aggression (Majewlka, 1992; Simon & Lu, 2006); however, the specific action mechanism is still unclear.[8]

However, the biggest effects of adrenal androgens on behavior are probably not the direct ones: in fact, DHEA and DHEAS can be converted into the more potent androgen testosterone and/or estrogens in some tissues, including the CNS. This *intracrine* production of androgens and estrogens provides target tissues with a mechanism to adjust the formation and metabolism of sex steroids according to local requirements (Adkins-Regan, 2005; Labrie, 1991; Labrie, Luu-The, Labrie, & Simard, 2001). According to recent estimates, the intracrine production of sex hormones in peripheral tissues account for about 75% of total estrogen in adult women and 50% of total androgens in adult men (Labrie et al., 2005). In children, adrenal steroids can exert powerful behavioral effects in the domains regulated by testosterone and estrogen while having minimal effects on physical appearance, thus driving development along sex-specific developmental pathways even before full reproductive maturity.

The trigger of adrenarche

Several hormones have been proposed as possible triggers initiating adrenarche, including adrenocorticotropic hormone (ACTH), prolactin (PRL), and other peptides (Parker, 1991); however, as yet there is no convincing evidence that any of these is the actual causal factor of adrenarche. Recent data from a longitudinal study suggested that adrenarche may not be triggered by any particular hormone, rather resulting from a gradual maturational process: DHEAS concentration seems to increase exponentially starting from early childhood, in parallel with gradual alterations in the activity of key steroidogenic enzymes (Palmert et al., 2001). Other studies have shown that, as children grow up, the level of 3β-hydroxysteroid dehydrogenase (3β-HSD) in the adrenal reticularis decreases, which may contribute to the increase in DHEA and DHEAS production (Gell et al., 1998).

[8] Some data suggest that, by reducing the concentration of pregnenolone sulfate (an excitatory neurosteroid with GABA-antagonistic activity), DHEA increases GABA-ergic tone which in turn increases aggression control (i.e., Robel et al., 1995), thus highlighting the anxiolytic effects of DHEA and DHEAS caused by a GABA agonistic action. Other studies, however, have documented an antagonistic action on GABA receptors (i.e., Majewska, 1992). This suggests that DHEA might have additional, yet unknown, mechanisms of action; the precise effect of DHEA on neuro-transmitter receptors is still incompletely understood.

Adrenarche and gonadarche

The increase in adrenal androgens characterizing adrenarche occurs when the hypothalamic–pituitary–gonadal (HPG) axis is at its lowest level of activity, without the increase in gonadotropins typical of gonadarche (i.e., the activation of ovaries and testes during puberty); the two processes can thus be regardered as separate maturational events (Sklar, Kaplan, & Grumbach, 1980). The dissociability of adrenarche and gonadarche is apparent in children with atypical development, when one process can take place without the other. Adrenarche does not occur in many girls with Addison's disease (Kim & Brody, 2001), who undergo puberty but show minimal or no signs of adrenarche; conversely, girls affected by Turner's syndrome may manifest normal adrenarche, but they never undergo a complete gonadal puberty (Teller, Homoki, Wudy, & Schlickenrieder, 1986). Recently, however, a genetic study found evidence that the timing of adrenarche and that of gonadarche are largely regulated by the same set of genes, while environmental factors have unique effects on the timing of the two processes (Van den Berg et al., 2006). There is also some evidence that early adrenarche predicts early gonadarche, although the relationship may be stronger in girls (see Ellison, 2002).

Factors affecting the timing of adrenarche

The timing of adrenarche, like that of gonadarche, is rather variable. In investigating the factors potentially affecting adrenarche timing, most researchers have focused on fetal or childhood body mass and related endocrine signals (such as insulin). Adrenarche begins at about the same time as a rise in body mass index (BMI; Rolland-Cachera, 1993), with increases in insulin and insulin-like growth factor I (IGF-I) serum levels (Juul et al., 1997). Case-control studies have found that IGF-I and insulin levels are higher in both girls and boys with premature adrenarche (Denburg et al., 2002; Silfen et al., 2002). In a longitudinal study, Remer and Manz (1999) showed that changes in nutritional status, measured as Δ-BMI (variation in BMI), are strongly associated with increases in DHEAS secretion during adrenarche, independently from age or developmental stage; however, the direction of causality remains unknown. Recent data suggest that fetal growth may modulate adrenarche; starting from the studies of Barker and colleagues (1993), it has been shown that growth-retarded fetuses adapt to under-nutrition by altering endocrine and metabolic processes that remain altered also postnatally. More recently, low birth weight has been related to low fetal concentration of DHEAS in both males and females (Francois & de Zegher, 1997; Ong et al., 2004), supporting the idea of early endocrine programming. Other studies have found that a pattern of precocious puberty, pronounced adrenarche, ovarian hyperandrogenism and hyperinsulinemia was associated to reduced fetal growth, indicating that these occurrences may indeed have a prenatal starting point (Ibáñez, Potau, Francois & Zegher, 1998). The exact mechanisms controlling such relationships are still unknown; perhaps, low birth weight can serve as a marker for the subsequent abnormalities (Ibáñez et al., 2000). Interestingly, pronounced adrenarche has been linked to insulin resistance and reduced fetal growth in girls (Ibáñez, Potau, Marcos, Francois, & de Zegher, 1999; Ibáñez et al., 1998), and, more generally, extant evidence suggests that IFG-I and insulin resistance are related to the mechanism of adrenarche in girls, but not in boys (Guercio, Rivarola, Chaler, Maceiras, & Belgorosky, 2002; Guercio, Rivarola, Chaler, Maceiras, & Belgorosky, 2003; Potau, Ibáñez, Riqué, Sanchez-Ufarte, & de Zegher, 1999). The physiological basis for this sexual dimorphism is unclear; it has been suggested that gender specificity of endocrine levels in premature adrenarche could be a consequence of sexual dimorphism in prenatal growth, which is thought to result from androgen action (de Zegher et al., 1998). Sex differences in the relationship between DHEA levels and insulin sensitivity persist in adulthood, with men (but not women) showing a positive correlation between the two variables (Nestler, Beer, Jakubowicz, & Beer, 1994).

When adrenal androgens production starts earlier than usual (sometimes as early as 3 years), the condition is often called *premature adrenarche*. Premature adrenarche is usually diagnosed by precocious pubarche (i.e., growth of pubic hair before 8 years in girls and 9 years in boys; Silverman, Migeon, Rosenberg, & Wilkins, 1952). The incidence of premature adrenarche is much higher in girls than in boys (about 10:1; see Ibáñez et al., 2000), although there is still no explanation for this sex difference. While the causes of premature adrenarche remain uncertain, some studies have docu-

mented that children with low birthweight are more likely to manifest this pattern (Ghirri et al., 2001; Oppenheimer, Linder, & DiMartino-Nardi, 1995). Moreover, premature adrenarche is more frequent in children with cerebral dysfunction (Thamdrup, 1955) and obesity (Jabbar, Pugliese, Fort, Becker, & Lifshitz, 1991). In addition to nutritional factors and birthweight, adrenarche timing in both sexes can be anticipated by early stress and, in particular, by stressful family relationships and inadequate parenting (see below). This was shown in a recent longitudinal study by Ellis and Essex (2007). The same stress-related factors can lead to earlier puberty, but apparently only in girls (reviewed in Ellis, 2004; see also Belsky et al., 2007-a, 2007-b, 2007-c). This may contribute to explain why the timing of adrenarche and that of gonadarche correlate more strongly in girls than in boys (Ellison, 2002): while early stress anticipates both adrenarche and gonadarche in girls (thus increasing the correlation between the two), it only anticipates adrenarche in boys (thus reducing the correlation).

Psychobiology of the juvenile transition

With the juvenile transition, children enter a phase of increased social learning and competition; in contrast to early childhood, the juvenile life stage is shaped by sexual selection pressures, resulting in different social strategies for boys and girls. We propose that adrenarche acts as a regulatory switch, co-ordinating changes in a suite of behavioral traits related to the evolutionary functions of juvenility. In this section, we describe three ways in which adrenarche can affect children's behavior: (1) by inducing sex differences via activation of sex-hormones pathways; (2) by activating previously unexpressed genotypic variation in those pathways, which then contributes to within-sex individual differences; and (3) by interacting with other endocrine and neural systems to integrate environmental cues in the process, resulting in adaptive developmental plasticity.

Sex differences

The onset of adrenarche activates sex hormones-related pathways in the brain, opening the way to increased sexual differentiation at the cognitive and behavioral level before puberty. In the standard model of sexual differentiation, prenatal and perinatal hormone levels have an *organizational* role on the nervous system, that is, they act on the structure of the maturing brain by rendering it (permanently) sexually dimorphic and priming it for the action of future endocrine signals. On the contrary, the pubertal secretion of sex hormones following gonadarche would have an *activational* role, acting on the brain so that previous organizational differences are expressed and start affecting behavior (Goy & McEwen, 1980). More recent studies have shown that the preceding account is inaccurate in at least two respects: first, adrenarche can activate the relevant pathways before puberty; second, puberty (and the same is likely true of adrenarche) does not have a merely activational role, but can permanently alter brain function with organizational effects (see Arnold & Breedlove, 1985; Romeo, 2003; Wilson & Davies, 2007). Thus, the expected course of sex differences in behavior, resulting from both organizational and activational effects, is a stepped function with two large increases following adrenarche (juvenile transition) and gonadarche (puberty)[9] Of course, endocrine factors are compounded by social learning, which in turn is directed and shaped by hormone-induced sexual differentiation in a dynamic feedback loop.

Genotypic variation

When adrenal androgens begin to circulate in the blood flow, they activate a signal transduction pathway which has been virtually silent for years. This pathway involves a large set of molecules, with specific roles in the chain that leads from the synthesis of hormones to their effects on behavior. From

[9] A minor peak could be also predicted in the first 4-5 months of life, when male infants' testes produce high levels of testosterone, and adrenal glands produce adrenal androgens (see McIntyre, 2006). Of course, the limited behavioral repertoire of months-old infants only allows for limited manifestation of sex differences; however, high levels of circulating sex hormones at the beginning of life could explain the findings of marked sex differences in the perceptual preferences of neonates (e.g., Connellan, Baron-Cohen, Wheelwright, Batki, & Ahluwalia, 2000).

now on, genetic variation at any of these steps can influence the overall outcome by affecting hormone levels, availability, or physiological effect. In other words, an amount of genotypic variation becomes active in a quasi-modular fashion and starts contributing to the development of individual differences. Here is a nonexhaustive list of the main steps in the adrenal androgens pathway (Fig. 2), with a brief description of the effects that genetic variation can have at each one of them (see also Dufty et al., 2002; Knapp, 2004):

1. *Steroidogenic enzymes*. The synthesis of DHEA and DHEAS in the adrenal gland requires the action of a number of enzymes. These enzymes can be more or less active and can vary in concentration, thus influencing the overall level of androgens in the plasma. Behavioral effects related to steroidogenic enzymes have been found in many animal species: for example, differences in steroidogenic enzymes have been related to courtship and mating patterns in various teleost fishes, to sex change in bluehead wrasse, and to dominance in male rats (reviewed in Knapp, 2004). In humans, however, research has typically focused on pathological rather than normal variation (e.g., deficiencies in the steroidogenic enzyme 21-hydroxylase causing congenital adrenal hyperplasia).
2. *Binding globulins*. Globulins are transport proteins that bind to androgens in the plasma. The concentration and binding capacity of globulins can significantly affect the availability of hormones in target tissues, since only the "free" portion of circulating hormone can pass through the cell membrane and induce physiological effects. Despite their potential importance, there is little research on the behavioral effects of globulins; to date, a relation between globulin variants and male stress reactivity has been documented in tree lizards (Jennings, Moore, Knapp, Matthews, & Orchnik, 2000).
3. *Conversion enzymes*. DHEA and DHEAS are weakly androgenic, but in order to fully affect behavior they need to be converted into testosterone, its metabolite 5-α-dihydrotestosterone (5-α-DHT), and estrogen. This requires a set of other enzymes (see Labrie et al., 1998), whose efficacy and concentration can affect the potency of androgenic/estrogenic action in the brain. While the developmental course of conversion enzymes in humans has not been well studied, the conversion enzyme 17-β-HSD has been found already in the fetal brain (Milewich, Carr, & Frenkel, 1990).

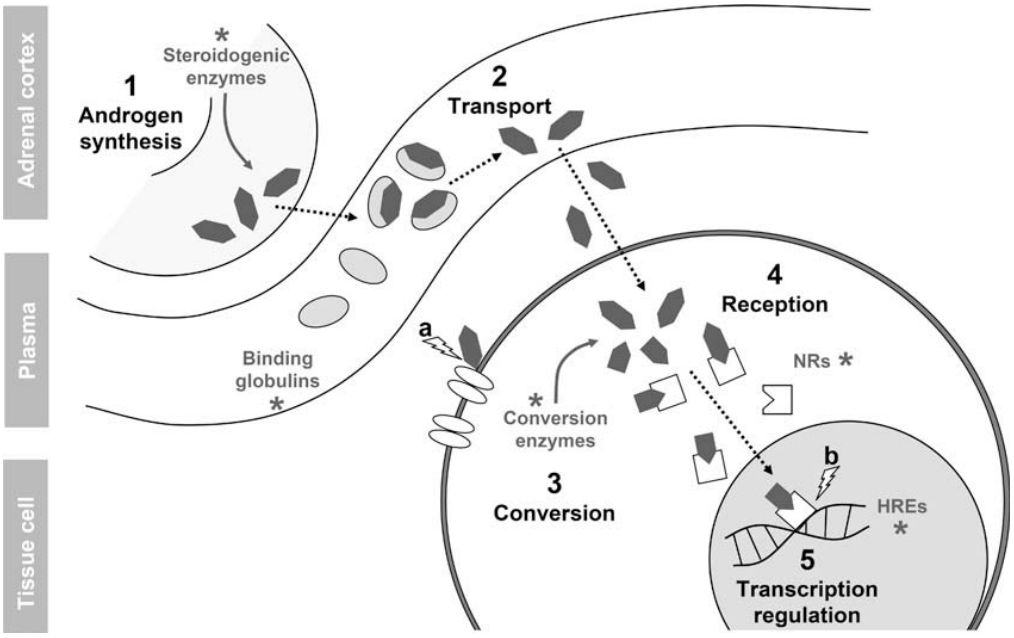

Fig. 2. The main steps in the adrenal androgen pathway. The figure shows two possible action mechanisms of adrenal androgens: (a) direct modulation of neuroreceptors (e.g., GABA receptors), and (b) conversion to testosterone and/or estrogens in the tissues equipped with the relevant enzymes, followed by regulatory action on DNA expression in the cell nucleus. Asterisks indicate possible sources of individual differences at the genotypic level.

4. *Receptors*. A major site of variability in hormone action is in the number and binding capacity of receptors themselves, with more active receptor variants (i.e., those which bind more easily to hormones) producing enhanced physiological effects. The X-linked androgen receptor gene (*AR*) has received the most attention in humans so far, and is a candidate for major behavioral effects in both males and females. Comings, Muhlemann, Johnson and Mac Murray (2002) found associations between carrying the short version of the allele and aggression, impulsivity, and number of sexual partners; in addition, in females the short version predicted parent's divorce, father absence and early menarche (these findings, however, were not replicated in a subsequent study by Jorm, Christensen, Rodgers, Jacomb, & Easteal, 2004). It has been suggested that variation in *AR* sequence might have an effect on intelligence as well (Manning, 2007). To date, there are no comparable data on estrogen receptors (ERs).
5. *Transcription promoters*. Ultimately, much of the effect of sex hormones comes from their ability to bind to promoter regions in the DNA (after forming hormone-receptor complexes with NRs) and thus influence transcription of other genes. Variation in the sequence of these DNA regions (called hormone response elements, HREs) can affect the rate of hormone-induced transcription (for example, by differential binding of the hormone-receptor complex). In humans and rats, the promoter of the vasopressin gene contains an HRE for the estrogen receptor (Adan & Burbach, 1992; Shapiro, Xu, & Dorsa, 2000), and vasopressin is critically involved in the regulation of sexual and aggressive behavior. Both the oxytocin gene and the oxytocin receptor gene appear to have HREs for the estrogen receptor in their promoter regions (see Gimpl & Fahrenholz, 2001), although the evidence is contested and it is possible that estrogen affects oxytocin synthesis in a more indirect way (Ivell & Walther, 1999). An interesting point about genetic variation in the "downstream" steps of the signalling pathway is that it can affect behavior in more specific and locally differentiated ways, e.g., exerting effects only on some specific behaviors rather than on generalized behavioral clusters (Badyaev, 2005).

Note that not all genetic variation in the endocrine pathway described above is expected to have a behavioral effect. In fact, most genotypic variation has no detectable effect at all on physiological processes, and only a certain number of allelic variants are actually able to influence behavior. Second, not all genotypic variation is adaptive, i.e., maintained by natural selection; genetic mutation can maintain variability in a population without it having any adaptive meaning (e.g., Roff, 2005). Accordingly, evolutionary psychology has traditionally downplayed the importance of heritable individual variation (e.g., Pinker, 1997; Tooby & Cosmides, 1992); however, there is accumulating evidence that heritable variation in personality and behavioral traits in humans and other animals can be the result of natural selection (see Dingemanse, Both, Drent, & Tinbergen, 2004; Dingemanse, Both, Drent, van Oers, & van Noordvijk, 2002; Groothuis & Carere, 2005; Nettle, 2006, 2007), leading to genetically influenced, adaptive individual differences.

Whereas genotypic differences can influence a quantitative trait in one direction or the other, e.g., different alleles can contribute to taller or shorter stature, sometimes the effect of genetic variation is to influence the degree of *plasticity* shown by the related traits. Recently, molecular genetic studies have uncovered a number of instances in which having a certain allelic variant makes one more susceptible to some environmental factors, so that the range of possible phenotypes (the genotype's *reaction norm*) is widened (Belsky, 2007-a, 2007-b, 2007-c; Ellis et al., 2006). For example, the 7-repeat variant of the *DRD4* dopamine receptor gene predicts higher levels of attachment disorganization in children exposed to maternal unresolved loss or trauma, but *lower* disorganization in children without such risk factors, compared with children carrying shorter allelic variants (van Ijzendoorn & Bakermans-Kranenburg, 2006). The shorter *DRD4* alleles are associated with a less plastic reaction norm, and make infants less susceptible to their rearing environment, consistent with the differential susceptibility hypothesis by Belsky (1997); see above).

Adaptive plasticity

As discussed above, adrenarche can affect behavior by activating sexually differentiated pathways and leading to the expression of individual genetic variation. These effects do not yet suffice to enable

adaptive plasticity; however, the sex-hormones pathway is deeply connected with other neural systems, which can track crucial cues in the environment and adaptively adjust the timing, intensity, and behavioral consequences of the juvenile transition.

The most important connection in this respect is that between two endocrine systems: the sex-hormones system (often described as limited to the hypothalamic–pituitary–gonadal axis, HPG, but actually including adrenal androgens as well) and the stress system (involving two main subsystems, the corticotropin releasing hormone—CRH system, which regulates the hypothalamic–pituitary–adrenal axis, HPA, and the locus coeruleus–norepinephrine system, LC–NE). There is extensive cross-talk between the two systems, since stress-related molecules such as cortisol and vasopressin (VP) affect the activity of the sex-hormones system (e.g., Tilbrook, Turner, & Clarke, 2002) and, conversely, sex hormones (including DHEA and DHEAS) powerfully regulate the activity of stress pathways. Androgens and estrogens affect the stress system at many hierarchical levels; animal studies show that sex hormones act (at least) on: (1) cortisol synthesis; (2) CRH and VP synthesis in the hypothalamus, mediated by effects on the amygdala (Viau, 2002; Viau, Bingham, Davis, Lee, & Wong, 2005); and (3) release and modulation of oxytocin (e.g., Jezova, Jurankova, Mosnarova, Kriska, & Skultetyova, 1996; McCarthy, 1995; McCarthy, McDonald, Brooks, & Goldman, 1996). Cross-talk between endocrine systems can even take place at the genomic level, since androgen and glucocorticoid receptors can interact in the regulation of gene transcription (Viau, 2002). Consistent with these findings, Taylor et al. (2000) gathered convincing evidence of a sex dimorphism in the stress response of mammals; the classical "fight-or-flight" response seems to be more typical of males, while females tend to increase affiliative and caregiving behaviors ("tend-and-befriend"). Furthermore, both stress hormones and sex hormones are involved in the regulation of dominance and aggression, with complex interplay between, for example, the circulating levels of testosterone and cortisol (see Booth, Granger, Mazur, & Kivlighan, 2006; Korte et al., 2005; Popma et al., 2007; Styne & Grumbach, 2002).

The connection between sex, stress and aggression in the endocrine system is especially important in the light of life history theory. Some of the crucial variables expected to affect reproductive strategies relate to *risk* and *unpredictability*, two aspects of the environment closely tracked by the stress system. In human children, the effect of environmental risk on stress levels (and on the regulation of the stress response) is strongly mediated by parental care and attachment security (see Cassidy & Shaver, 2008; Chisholm, 1999; Ellis, Essex, & Boyce, 2005; Flinn, 2006). Indeed, it has been proposed that security of attachment in the first 5–7 years encodes the perceived environmental risk and sets the child's reproductive strategy on alternative pathways, with secure attachment (indicating low risk) leading to reproductive strategies oriented to future reproduction, high parenting effort and offspring quality, and insecure attachment (indicating high risk) leading to maximization of current reproduction, mating effort, and offspring quantity (Belsky et al., 1991; Chisholm, 1993). This hypothesis has garnered remarkable empirical support so far (Belsky, 2007-a, 2007-b, 2007-c; Chisholm, 1999; Del Giudice, in press-b), and is consistent with the idea that early childhood affords an "assessment period" in which children gauge their local environment and adjust their future developmental trajectories accordingly (Bogin, 1997). Importantly, a recent longitudinal study showed that low quality of parenting in the first years anticipates the onset of adrenarche in both boys and girls, thus confirming the link between early attachment experiences and sexual development at the juncture of the juvenile transition (Ellis & Essex, 2007).

In the present paper, we take this concept a step further and argue that, with the transition to juvenility, the endocrine system integrates early environmental factors with genotypic variation (in sexually differentiated ways), thus switching the child's reproductive strategy on a given trajectory (with the possibility of later revision) and affecting a number of life history-related behavioral domains (Fig. 3). Reproductive strategies are expected to involve a co-ordinated suite of behavioral traits including sexual style, dominance-seeking/aggression, risk-taking, and altruism/cooperation; in the next section we will review evidence that the juvenile transition is associated with the emergence of both sex-related and individual differences in life history-related domains. In particular, we will focus on the well researched areas of attachment and aggression, while keeping in mind that other behavioral traits (such as risk-taking and altruism) are likely to show similar developmental trajectories.

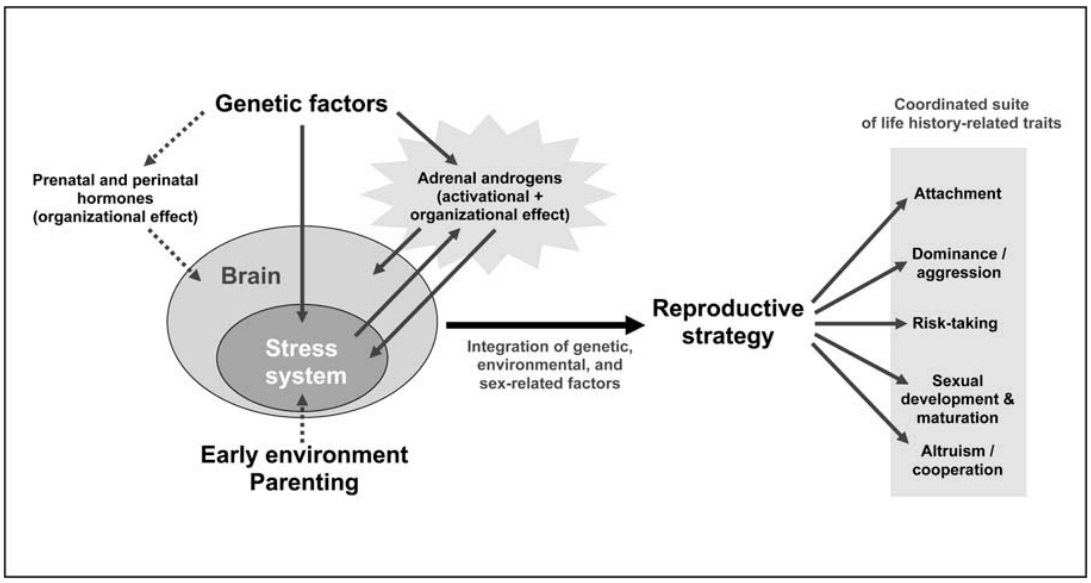

Fig. 3. The juvenile transition as a hormonally mediated developmental switch point. Dashed arrows represent effects in the past (prenatal development, infancy and early childhood); solid arrows represent effects taking place in the transition between early and middle childhood. Note the cross-talk between adrenal androgens and the stress system.

Changes in attachment and aggression during the juvenile transition

Changes in attachment

Attachment theory (Bowlby, 1969/1982, 1973, 1980; Cassidy & Shaver, 2008) is arguably one of the most wide-ranging accounts of human social development. It describes the formation of affectional relationships between infants and caregivers, the causes (and meaning) of individual differences in those relationships, their developmental course, and their consequences on other areas of behavior and personality. Whereas the original formulation by John Bowlby drew extensively on biological and ethological concepts, later theorists have tended to focus on the proximate level of explanation and have largely ignored the evolutionary underpinnings of attachment. In the 1990s, however, the life history models of Belsky et al. (1991) and Chisholm (1993) put attachment theory back in its place within the framework of evolutionary psychology (see Simpson & Belsky, 2008).

In these models, the adaptive function of attachment relationships is related not only to *survival* (i.e., keeping proximity between the child and the caregiver, ensuring protection and closeness), but to *reproduction* as well. In particular, attachment security acts as a "summary" of the safeness of the child's local ecology, thus critically contributing to shape the individual's future life history strategy; on the other hand, attachment patterns are themselves part of the cluster of traits that implement a given reproductive strategy, together with sexual styles, cooperation, aggression, and risk-taking (see above). Indeed, adult romantic relationships *are* (at least in part) attachment relationships, and their dynamics and neural substrates show considerable overlap (e.g., Carter, 1998; Feeney, 1999; Hazan & Zeifman, 1999; Insel & Young, 2001; Pedersen et al., 2005). This suggests that adult (romantic) attachment styles can be conceptualized as part of adaptive reproductive strategies. Consistent with this proposal (e.g., Kirkpatrick, 1998) is the fact that romantic attachment in adults shows marked sex differences (reviewed in Del Giudice, in press-a, in press-b): males tend to be more *avoidant* (detached, distancing, and with little desire for closeness) and females tend to be more *anxious* (intense desire for closeness and fear of rejection and abandonment). Higher avoidance in men has been interpreted as consistent with lower male investment in long-term couple relationships and parental effort (Kirkpatrick, 1998; Simpson & Belsky, 2008; the meaning of attachment anxiety in women has not been addressed directly by these theorists). A major problem remained, however: in infants and children, insecure attachment patterns (avoidant and ambivalent) showed virtually no evidence of sex differences. This was an obstacle for evolutionary-developmental theories, since it did not seem possible to link individual differences in attachment during childhood with adult reproductive

strategies, beyond a general relationship between attachment insecurity and current reproduction / mating effort (e.g., Chisholm, 1999).

Recently, a solution to this puzzle has been advanced. On the basis of previous literature and new data, Del Giudice (2008) found that large sex differences in children's attachment styles appear, cross-culturally, *starting from middle childhood*. In children samples aged 7 or more, a similar pattern to that observed in adults emerges, with most insecure boys displaying *avoidant* attachment and the majority of insecure girls classified as *ambivalent*. Del Giudice (in press-a) then proposed an updated life history model, describing the middle-childhood shift as part of a reorganization of the attachment system, driven by sexual selection in juvenility and coordinated by the endocrine switch of adrenarche. The model is rather complex, and is based on sexual selection theory and parental investment theory (Trivers, 1972) in the context of human reproductive ecology. Since this model has been extensively described elsewhere (Del Giudice, in press-a), here we will only summarize its most relevant points: (1) both avoidance and anxiety/ambivalence are seen as adaptive, sex-related facets of insecure reproductive strategies; while insecure males are expected to adopt mainly avoidant styles (related to low parental investment), the attachment styles of insecure females are expected to depend on the level of environmental risk, with moderate risk leading to anxious/ambivalent attachment (conceptualized as a strategy aimed at eliciting investment from kin and mates), and high risk leading to (low-investment) avoidance. (2) Male avoidance is not only adaptive in the context of adult couple relationships: during juvenility, insecure boys (who are adopting a mating-oriented life history strategy) can benefit from avoidant attachment and its behavioral correlates (e.g., aggression, inflated self-esteem, externalizing behaviors) as a high-risk dominance-seeking strategy in the peer group.[10] It could be that anxiety has some advantage for girls in the peer group as well, or that it contributes to between-sex attractiveness (for example by emphasizing immature and dependent behaviors); however, female peer dynamics are still poorly understood compared to those of male groups, and this makes the possible peer-related effects of anxiety less clear. (3) Secure attachment should relate to smaller sex differences in reproductive and mating strategies, since the interests of males and females tend to converge when adopting long-term, parenting-oriented life history strategies. (4) Finally, part of the within-sex individual differences in attachment styles could depend not only on early stress, but also on genotypic factors activated by adrenarche, as discussed in a previous section.

Changes in aggression

Aggressive behavior undergoes important changes during the transition from early to middle childhood, with the emergence and/or intensification of both between-sex and within-sex differences. Furthermore, and consistent with our theoretical expectations, these phenotypic changes seem to be partially driven by the activation, during the juvenile transition, of previously unexpressed genetic factors. Data from aggression research are well suited to investigate the effects of the juvenile transition, since: (1) individual differences in aggressive behavior are strongly influenced by genotypic factors (see Hyun Rhee & Waldman, 2002; Miles & Carey, 1997, for reviews); (2) they are mediated by neurotransmitters and hormones whose concentration and effectiveness can be modulated by adrenarche (see also Cashdan, 2003; Moore, Scarpa, & Raine, 2002; Simon & Coccaro, 1999); and finally, (3) aggression has an important function in juvenility, both because of its role in achieving social status in the peer group and because it can be functional to acquiring competitive abilities to be used in adult life (see Hawley, Little, & Rodkin, 2007 for an overview).

Whereas in past decades there have been few studies of aggressive behavior in juvenility (see Olweus, 1979, for a review), we can now rely upon a growing number of longitudinal and behavior genetic studies[11]

[10] Secure boys tend to be more cooperative, more friendly and less aggressive; this may count as an alternative route to social status (Hawley, 1999; Hawley, 2003; Pellegrini & Bartini, 2001), consistent with a parenting-oriented reproductive strategy and with delayed (and reduced) competition for mates. See Del Giudice (in press b) for more details.

[11] We will focus specifically on aggressive behaviors (i.e., behaviors that cause or threaten physical harm to others, Loeber & Hay, 1997), excluding deviant and antisocial non-aggressive behaviors (such as drug abuse and theft). However, we will also rely upon some studies of externalizing behaviors (which includes aggressive, disruptive, and delinquent behaviors; Achenbach, 1991), since most genetic research focusing on juvenility collapses aggression and delinquency.

stimulated by the finding that juvenile aggression is a good predictor of aggressive, antisocial and violent behaviors in adolescents and adults (e.g., Brame, Nagin, & Tremblay, 2001; Côté, Zoccolillo, Tremblay, Nagin, & Vitato, 2001; Lerner, Hertzog, Hooker, Hassibi, & Thomas, 1988; Loeber, 1991; Reiss & Roth, 1993).

Sex differences in aggression

Sex differences in aggression are nearly absent during infancy and toddlerhood, and begin to appear between the third and the sixth year of life (Coie & Dodge, 1997; Loeber & Hay, 1997). Boys manifest higher levels of physical aggression (e.g., instrumental grabbing of objects, hitting and physical fighting), girls more verbal and relational aggression (e.g., peer exclusion and gossip). Sex differences becomes even more pronounced during juvenility (Loeber & Stouthamer-Loeber, 1998; van Beijsterveldt, Bartels, Hudziak, & Boomsma, 2003), with boys scoring higher than girls in measures of both physical aggression and externalizing behaviors (e.g., Deater-Deckard & Plomin, 1999; Hudziak et al., 2003). Interestingly, sex differences are especially apparent in social contexts involving peer relations, such as school, as shown by the fact that they are clearly detected by teachers, but less so by parents (Kraatz Keiley, Bates, Dodge, & Pettit, 2000). In a sibling adoption study on children aged 7–12 it was also found that teacher ratings of children's aggression, but not parent ratings, were substantially heritable (Deater-Deckard & Plomin, 1999).

Not only sex differences in aggressive behavior become more pronounced in middle childhood, sex differences in the quantitative genetics of aggression appear as well. During middle childhood the relative contribution of genetic and environmental factors to individual differences varies as a function of both age and sex, with older children showing higher genetic variance than younger ones, and boys showing higher genetic variance than girls (Bartels et al., 2003, 2004; Miles & Carey, 1997; van Beijsterveldt et al., 2003). Whereas sex differences in the magnitude of genetic versus environmental variation in aggressive and externalizing behavior are already present in childhood, they become much stronger around age 7 (van Beijsterveldt et al., 2003).

Taken together, these data are consistent with the idea that aggression is part of a cluster of sexually selected behavioral traits expressed in the juvenile stage; accordingly, sex differences in aggression emerging in middle childhood tend to remain stable during the following years (Hymel, Rubin, Rowden, & LeMare, 1990; see Loeber & Hay, 1997, for a discussion).

Individual differences in aggression

Aggression as a trait is relatively stable from infancy to adulthood (see Olweus, 1979 and Lerner et al., 1988). In general, correlations between aggression ratings at different ages have been found to be moderately high (typically in the .50–.60 range; see Coie & Dodge, 1998 and Olweus, 1977, 1979); however, the strength of correlations is rather variable, depending on age at first measurement (with younger individuals showing less stability) and on the duration of the between-measurements interval (with shorter intervals yielding higher correlations). Behavior genetic studies have shown that, especially in males, the stability of aggressive and externalizing behaviors is mainly accounted for by additive genetic factors, with a smaller contribution of shared environmental factors, suggesting pleiotropic effects of the same genes at different ages (e.g., Bartels et al., 2004; Hudziak et al., 2003; Schmitz, Fulker, & Mrazek, 1995; van den Oord and Rowe,1997; van der Valk, van den Oord, Verhulst, & Boomsma, 2003).

On the basis of the early stability studies, in the past decades it was widely assumed that individual differences in aggression became established early in infancy, and remained stable through adulthood. The only exception to this pattern was represented by "late starters" (Patterson, Capaldi, & Bank, 1991) or "adolescent limited individuals" (Moffitt, 1993), a subgroup of children showing low aggression levels until early adolescence, then becoming significantly more aggressive. However, more recent findings have cast doubt on this view. First of all, little evidence has been found in favor of the late-onset hypothesis: only a small minority of individuals seem to undergo abrupt and unpredictable changes in aggression during adolescence (Brame et al., 2001; Lacourse et al., 2002). Rather, and most importantly in the context of the present paper, it has been found that major shifts in aggressive behavior take place *during middle childhood*. In this developmental phase, many individuals become significantly more or less aggressive than they were at previous ages, with the most abrupt changes in aggressive status taking place between age 7 and 9 (e.g., Aber, Brown, & Jones, 2003; Kovalesky-

Jones & Duncan, 1999). McFayden-Ketchum, Bates, Dodge & Pettit (1996) found that many children followed discontinuous trajectories in aggression from age 5 to 8; both increases and decreases in levels of aggressive behavior were observed. Nagin and Tremblay (1999, 2001), in a study of boys aged 6–15, found that fewer than 30% maintained a stable rank in physical aggression during the time interval considered; most boys showed decreasing trajectories, starting either from moderate or high aggression groups and ending up in the low aggression group. The same pattern was found in children aged 2–8 (Shaw, Gilliom, Ingoldsby, & Nagin, 2003; see Haselager, Cillessen, Van Lieshout, Riksen-Walraven, & Hartup, 2002 for similar results with peer rejected boys). In a study by Kingston and Prior (1995), about 25% of children showed an increase in aggression from age 2 to 8, with shifts often occurring around 5–6 years. Five different trajectories in the development of fighting behavior (two stable and three unstable) were identified by Haapasalo and Tremblay (1994) in children from age 6 to 12. Finally, Warman and Cohen (2000) found moderate instability in aggressive behaviors even in a sample of children aged 7–10 who were followed for a one-year period. In summary, most studies including the transition from early to middle childhood found a marked degree of instability; it is also noteworthy that, even in the few studies reporting stability, correlations were often lower compared with similar studies in different age groups (e.g., Moskowitz, Schwartzman, & Ledingham, 1985). It should be noted that the developmental instability found in the juvenile transition period is likely due to a complex interplay of genetic and environmental factors. During this stage, children undergo the hormonal and physiological changes described in previous sections, but also some important social changes, such as school entry, which may contribute to modify their level of aggression.

As can be expected from the psychobiology of the juvenile transition (see above), there is evidence that new genetic influences on aggression become active in the passage from early to middle childhood. van Beijsterveldt and colleagues (2003) performed a longitudinal twin study at ages 3, 7, 10, and 12. This design allows not only for variance partitioning at each age, but also for estimation of the stability of genetic and environmental influences across time. For example, at each time point the proportion of additive genetic variance can be further decomposed into genetic variance transmitted from the previous time point (i.e., the same genetic factors contribute to individual differences) and new, age-specific genetic variation. In this study it was found that, at age 7, nearly 50% of the additive genetic variance consisted of *new* genetic variance, and only a small part of the genetic effects was transmitted from age 3. On the contrary, only a small part of genetic variance after age 7 could be attributed to new genetic factors. Although such quantitative genetic models provide only an indirect estimate of stability in genetic effects, these results are clearly consistent with the hypothesis that substantial genetic variation is activated during the transition to juvenility.

Evidence from early-onset psychopathology

Additional evidence of behavioral changes in middle childhood comes from the field of psychiatric epidemiology. A longitudinal community study by Costello, Mustillo, Erkanli, Keeler, and Angold (2003), ranging from age 9 to 16, found that the overall prevalence of psychiatric diagnoses peaked at 9–10 years, then declined and started to rise again around 14 years; more specifically, the 9–10 years peak was due to high prevalence of anxiety disorders (including separation anxiety disorders) and ADHD; conduct disorders (involving aggression) were also fairly frequent. Another large study, drawing on the National Comorbidity Survey Replication sample (Kessler et al., 2005; see also Nock, Kazdin, Hiripi, & Kessler, 2007), showed that median age-of-onset of anxiety disorders and impulse-control disorders was 11 years on average, much lower than that of mood disorders (30 years on average). Notably, some anxiety- and aggression-related disorders had an age-of onset range that included, or was even limited to, middle childhood: Specific phobia, 5–12 years (interquartile range); social phobia, 8–15 years; separation anxiety disorder, 6–10 years; oppositional-defiant disorder, 8–14 years; conduct disorder, 10–15 years; ADHD, 7–8 years. In addition, the research literature on antisocial behavior shows that sex differences in prevalence of conduct problems begin to appear after age 5, with males showing higher conduct problems than females (reviewed in Silverthorn & Frick, 1999).

In synthesis, it has been reliably observed that some psychological disorders begin to appear (or even peak) in middle childhood; notably, these disorders all involve aggression, impulsivity, or anxiety, including attachment-related anxiety. There is initial evidence that adrenarche may play a specific

role in middle-childhood psychopathology: high levels of DHEAS were found in samples of children (mostly boys) diagnosed with conduct disorder and oppositional-defiant disorder (van Goozen, Matthys, Cohen-Kettenis, Thijssen, & van Engeland, 1998; van Goozen et al.,, 2000), and hyperactivity symptoms correlated with lower DHEA and DHEAS levels in a sample of children with ADHD diagnosis (Strous et al., 2001).

Conclusion

In this paper we proposed that the juvenile transition is a developmental switch point in the human life history, when sex-related and individual differences in reproductive strategies are expressed, and the relevant behavioral traits co-ordinated along adaptive trajectories. From the psychobiological point of view, we argued that adrenarche provides a hormonal switch between childhood and juvenility, and enables adaptive plasticity by integrating genetic and environmental influences in the shaping of individual reproductive strategies. We then reviewed evidence that, with the transition to juvenility, both sex-related and inter-individual changes take place in life history-related behavioral traits such as attachment and aggression.

While we think this is a promising first step, we are aware that our model is still far from complete, and that there are many crucial aspects that should be addressed by future research. Here we will conclude by highlighting those we think are the most interesting directions for further integration.

First of all, adopting a life history perspective leads one to consider traits in their functional relationships, rather than in isolation. Still, most of current developmental research only investigates one or two behavioral domains at a time. While this is certainly useful, it precludes us from seeing the forest from the trees—the integrated patterns of personality and behavioral traits engendered by life history trade-offs (Belsky et al., 1991; Figueredo et al., 2006). Future theoretical and empirical work will help elucidate the expected and observed patterns of integration among functionally related aspects of behavior such as stress and anxiety, dominance-seeking, aggression, attachment, parenting, and risk-taking, all of which are known to be influenced by sex and/or stress hormones (e.g., Cashdan, 2003; Wilson, Daly, & Pound, 2002). New evidence suggests that some previously unsuspected behavioral domains also fall under the influence of the stress and sexual endocrine systems: for example oxytocin, which has crucial roles in attachment, parenting and aggression and is strongly modulated by sex hormones, seems to be also involved in the regulation of interpersonal trust and cooperation (Kosfeld, Heinrichs, Zak, Fischbacher, & Fehr, 2005; Zak, Kurzban, & Matzner, 2005). This reinforces the idea of a broad cluster of covarying life history-related traits, under the influence of integrative endocrine systems (Fig. 3).

Second, whereas we argued that the juvenile transition represents a crucial moment in the shaping of individual reproductive strategies, developmental plasticity certainly does not stop there. Experience with peers during middle childhood and adolescence provides crucial feedback about one's social success and potential for competition, cooperation and so on; such feedback is likely to constitute important information to adjust (or switch) one's reproductive strategy. For example, Davis and Werre (2007) recently argued that exposure to agonistic stress in early adolescence can affect later mating behavior and fertility. A complete evolutionary theory of development will need to understand how the juvenile transition relates to adolescence (see Weisfeld, 1999) and, more generally, how reproductive strategies are modified (or persist) during all the major life stages (Del Giudice, in press-b).

Third, *stress* clearly emerges as a key factor in life history plasticity; the last few years have seen remarkable progress in theoretical and empirical research on stress from an evolutionary point of view. Individual differences in stress sensitivity arise during development, and result from integration of genetic and environmental factors (Boyce & Ellis, 2005; Ellis et al., 2005, 2006). In turn, differences in stress sensitivity interact bi-directionally with differences in the sex-hormones systems, resulting in complex (but co-ordinated) effects on mating, aggression, and risk-taking (Korte et al., 2005). Moreover, they do so in sexually differentiated ways (Taylor et al., 2000). One of the most exciting challenges in the future will be to integrate these findings and models in a broader life history framework (see also Cameron et al., 2005; Ellis et al., 2006), in order to gain the full potential of their explanatory and heuristic power.

Finally, our focus in the present paper has been on the endocrine and motivational mechanisms mediating life history strategies and transitions; but motivation is not separated from cognition, and further research is needed to understand how cognitive and motivational development unfold together in the life cycle. Promising steps in this direction have been taken by evolutionary-minded researchers (e. g. Ellis & Bjorklund, 2005; Locke & Bogin, 2006), but much remains to be discovered, especially about the link between neurobiological processes such as adrenarche and cognitive processes such as language, memory, planning, and so on. While evolutionary developmental psychology is still in its infancy, it has already made major contributions towards an integrative, consilient, and biologically rigorous theory of human development (see Ellis & Bjorklund, 2005, for an overview). We anticipate that, over the next years, it will provide the backbone for a long-awaited synthesis of evolution and development in the behavioral sciences, as is already happening in evolutionary biology; and, if successful, it will lay the foundation for the psychology of the future.

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