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## Life history theory and evolutionary psychology

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(Article begins on next page)

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## Chapter 2 Life History Theory and Evolutionary Psychology

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The evolution of life is the result of a process whereby variant forms compete to harvest energy from the environment and convert it into replicates of those forms. Individuals “capture” energy from the environment—for example through foraging, hunting, or cultivating—and “allocate” it to reproduction and survival-enhancing activities. Selection favors individuals who efficiently capture energy and effectively allocate it to enhance fitness within their ecological niche.

Energy does not come free. Were individuals able to expend unlimited energy at no cost, in principle they could grow and develop so rapidly they would begin reproducing immediately after birth, produce massive numbers of offspring, and preserve themselves such that they never age. In biological reality, however, individuals must live within finite energy “budgets”—themselves earned through energy and time expenditures—and can never spend more than they have available. Allocation of a finite budget entails trade-offs and hence forces decisions about the relative value of possible ways to spend. Acquiring one expensive item means giving up others; more consumption today may entail less tomorrow.

Selection favors organisms’ strategies for allocating energy budgets on the basis of one criterion: the strategy that leads to the allocation of energy that, on average, results in the greatest inclusive fitness (see West & Gardner, 2013) is the one that wins out over others. In this sense, selection is expected to result in fitness-maximizing or “optimal” strategies. Of course, they are optimal in a restricted sense, that is, under the constraints imposed by trade-offs between allocations of energy (see Parker & Maynard Smith, 1990).

Crucially, optimal allocations depend on the characteristics of an individual and its environment: newborns optimally allocate energy differently from adults; healthy individuals optimally allocate differently from those infected with disease; the best allocation strategy for individuals in stable circumstances differs from that of individuals whose future circumstances are unpredictable.

Life History Theory (LHT) provides a framework that addresses how, in the face of trade-offs, organisms should allocate time and energy to tasks and traits in a way that maximizes their fitness. Life history trade-offs have profound ramifications, affecting virtually every aspect of an organism's development and behavior. The concepts of LHT have steadily gained prominence within evolutionary psychology and are now core components of the discipline's toolkit, but with many potential avenues for further integration and application yet to be explored.

We begin with an overview of LHT. We then discuss the proximate mechanisms that enact allocation decisions, including hormonal systems and cognitive adaptations. Finally, we review current psychological applications of LHT and offer suggestions for advancing the integration of LHT into evolutionary psychology.

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# Life History Theory: An Overview

## Fundamental Trade-Offs in Life History Theory

Individuals can enhance fitness in two primary ways: They can invest either in traits that affect the age-schedule of survival, or in traits that affect the age-schedule of fertility (in this chapter, fertility refers to an organism's number of offspring rather than its ability to conceive). Ultimately, the influence of traits on inclusive fitness must be mediated through changes in survival or fertility or both (though they may do so by enhancing the survival and/or fertility of related individuals—e.g., offspring—as well as self). Because of allocation trade-offs, many if not most traits have opposing effects on survival and fertility, on the same fitness component at two different points in time, or on a fitness component of self (e.g., own fertility) and that of a related individual (e.g., offspring survival and/or fertility). For example, a trait that increases fertility by increasing mating frequency (e.g., a mating display) may simultaneously reduce survival by compromising immune function; energetic allocations to growth suppress fertility during youth, but may increase it later in life; allocations to offspring viability through parental investment may reduce one's own survival or future fertility.

Trade-offs between two traits do not necessarily lead them to be negatively correlated. Large individual differences in the availability of, ability to acquire, or efficiency in utilizing resources generate positive covariation among traits; e.g., individuals with larger budgets can invest more than others in both fertility *and* parental care. This positive covariation may overshadow negative covariation produced by trade-offs (see Reznick, Nunney, & Tessier, 2000).

Allocation problems can be conceptualized at multiple levels of detail (see Roff, 2002). We focus on three broad, fundamental trade-offs: *current vs. future reproduction*, *quality vs. quantity of offspring*, and *mating vs. parenting effort*.

## The Trade-Off Between Current and Future Reproduction

At any point in time, an organism can convert its available energy into a variety of activities. Some facilitate reproduction now (e.g., copulation, gestation). Others prolong life, thereby creating opportunities to reproduce later (e.g., additional energy harvesting, growth, predator avoidance, tissue repair, etc.). Allocation of energy to future opportunities draws it away from efforts to reproduce now, and vice versa. The first modern LHT framework for this trade-off was developed by Gadgil and Bossert (1970). Organisms capture energy (*resources*) from the environment. Their capture rate (or *income*) determines their energy budget. Through time, they can “spend” income on three different activities. Through *growth*, organisms can increase their energy capture rates in the future, thus increasing their future fertility. Through *maintenance*, organisms repair somatic tissue, allocate energy to immune function, engage in further energy production, and so on. Through *reproduction*, organisms replicate genes. How organisms solve this energetic trade-off shapes their life histories. Organisms typically have a juvenile phase during which fertility is zero, and then cease growth when allocation to reproduction increases fitness more than growth. Because maintenance and growth affect fitness through impacts on *future* reproduction, the tripartite trade-off collapses into a trade-off between current and future reproduction (Bell & Koufopanou, 1986; Hill, 1993; Lessells, 1991; Stearns, 1992). The loss of future survival, energy capture, and reproduction because of energy allocation to current reproduction is referred to as the *cost of reproduction* (Williams, 1966).

The current–future reproduction trade-off has been invoked to explain *senescence*, a pattern of gradual deterioration of somatic functionality and increased mortality occurring after reproductive maturity (Jones et al., 2014; Williams, 1957). According to *disposable soma theory*, senescence arises as a byproduct of optimal allocation design (Kirkwood, 1990). Perfect maintenance of somatic tissues would result in zero senescence, with no mortality due to internal deterioration. Because the organism is still subject to mortality due to external causes, however, it optimally diverts some resources away from maintenance and invests them in present reproduction. Accordingly, organisms invest less in maintenance than would be required to avoid senescence, thus allowing the soma to decay at a nonzero rate. Kaplan and Robson (2009) offer a model that explains differences in rates of senescence across the lifespan. Since maintenance costs increase as the *quantity* of tissue to maintain increases during growth, optimal allocations to maintenance progressively shrink across the life course. The

combination of early growth, decaying somatic quality, and reproductive trade-offs leads to a U-shaped mortality curve that decreases early in life but increases later on (Kaplan & Robson, 2009).

## The Trade-Off Between Quality and Quantity of Offspring

A second major life history trade-off, first discussed by Lack (1954, 1968), concerns a division within the resources allocated to current reproduction: allocation to increase offspring *quality* vs. allocation to increase offspring *quantity*. This trade-off arises because parents have limited resources to invest in reproduction and, hence, additional offspring must reduce average investment per offspring in terms of parental care, provision of resources, and so on. Models of the quantity–quality trade-off usually operationalize quality as offspring survival (e.g. Fischer, Taborsky, & Kokko, 2011; Harpending, Draper, & Pennington, 1990; Smith & Fretwell, 1974). More complex multigenerational models consider not only offspring survival but also the adult fertility of offspring, which can vary due to body size, health, skills, status, and so on, accrued as a result of parental investment (e.g., Kaplan, 1996).

The basic principle underlying the quality–quantity trade-off is that it is adaptive to increase investment in the quality of existing offspring until the fitness return on investment equals the return of a comparable allocation of resources to fertility (i.e., producing an additional offspring). This optimal level of investment is typically lower than the level that would ensure maximum offspring quality (Harpending et al., 1990; Pennington & Harpending, 1988). Specific solutions to the quantity–quality trade-off depend critically on the shape of the functions that translate parental investment into offspring quality, and, in particular, on whether those functions show diminishing returns (the benefit to offspring levels off as investment increases) or increasing returns (as investment increases, offspring benefit disproportionately more; see Kaplan, 1996).

## The Trade-Off Between Mating and Parenting Effort

Sexual reproduction adds another layer of complexity to life history allocations. To reproduce, individuals need to find potential mates, choose and be chosen by a specific mate, and secure copulation. All these activities take time and may involve substantial energy expenditures (e.g., costly displays, competition with rivals) as well as exposure to danger (e.g., increased predation risk). Individuals who already have offspring also can invest time and energy to increase their survival and quality. When mating effort and parental investment compete for time and resources, a trade-off arises so that the opportunity of gaining additional mating must be weighted against a reduction in the fitness of existing offspring (Trivers, 1972). For many sexual organisms, the mating–parenting trade-

off clearly overlaps with the quality–quantity trade-off, but only in part; offspring number can be regulated by many means other than mating frequency—for example egg production, spontaneous abortion, or even infanticide.

The mating-parenting trade-off is an important factor in the evolution of sex differences in patterns of mating competition and parental care (Kokko & Jennions, 2008). When mating and parenting conflict, the sex that experiences stronger sexual selection and higher mortality should invest more in mating competition, whereas the other sex should provide more parental care and become choosier. In addition, uncertainty of paternity is expected to select against male care (Kokko & Jennions, 2008). When the value of biparental care is substantial, females partly select males for their willingness to invest in parenting, leading to smaller sex differences in allocation toward mating and parenting and favoring the evolution of mutual mate choice (Edward & Chapman, 2011). Models suggest that strong female preferences for caring males may be able to overcome the effect of paternity uncertainty, leading to high levels of male care even in the face of a low probability of paternity (Alonzo, 2012).

Although trade-offs between mating and parenting are widespread, they are by no means inevitable; even the distinction between mating and parenting is not always a sharp one (Stiver & Alonzo, 2009). Most notably, when females base mating decisions on males' ability to care and invest in offspring, the same male behavior (e.g., protecting offspring) may simultaneously contribute to both mating and parenting effort. Conversely, when allocations to mating effort severely affect one's ability to invest in parenting (e.g., because of somatic investments in traits that aid competition), alternative reproductive strategies within a sex (usually males) may evolve, whereby some individuals invest heavily in parental effort whereas others specialize in mating strategies involving little if any parental investment (see Stiver & Alonzo, 2009; Taborsky & Brockmann, 2010).

## Embodied Capital

Growth and development can be viewed as investments in stocks of *embodied capital*: investments in self that can be translated into future reproduction. In a physical sense, embodied capital is organized somatic tissue (muscles, digestive organs, brains, and so on). In a functional sense, embodied capital includes strength, speed, immune function, skill, knowledge, and other abilities (Hill & Kaplan, 1999). Because allocations to maintenance counteract the depreciation of stocks of embodied capital with time, they, too, can be treated as investments in embodied capital (Kaplan & Robson, 2009). In this perspective, the current–future reproduction trade-off can be framed as a trade-off between investments in own embodied capital versus reproduction, while the quality–quantity trade-off is a trade-off between investments in the embodied capital of offspring versus their number (Kaplan, 1996).

When translated and extended into an embodied capital framework, LHT allows one to entertain possibilities not explicitly conceptualized by standard treatments. Standard models tend to treat investment in the future as physical growth. But growth is only one form of such investment, as illustrated by brain development. The brain has the capacity to transform present experiences into future performance. Brain expansion among higher primates represents an increased investment in this capacity (Fleagle, 2013; van Schaik, Isler, & Burkart, 2012). But this investment is realized not only in growth of neural tissue; substantial energy and time may be allocated to encountering experiences that, through changes in neural tissue, yield benefits realized over time—investments in the future.

How selection affects these investments depends on costs and benefits realized over an organism's lifetime. Growing and maintaining neural tissue entails substantial energetic costs (see Kuzawa et al., 2014) and, by curtailing “preprogrammed” behavioral routines, compromises performance early in life (consider for example the motoric incompetence of human infants). Hence, the *net* benefits of learning are only fully realized as the organism ages. In a niche where there is little to learn, benefits never offset early costs and smaller brains are favored. In a more challenging niche, small brains might be better early in life but much worse later, such that large brains are favored. Other systems may similarly become more functional through time—for example, the immune system, which requires exposure to antigens to become fully functional. The concept of embodied capital can address the evolution of any form of investment in a stock of capital that pays off over time.

## **Life History Strategies**

Taken together, the allocation decisions made in response to life history trade-offs constitute an organism's *life history strategy*. A common approach to life history evolution employs demographic (age-structured) models of population growth (Charlesworth, 1994). In this modeling framework, a life history strategy is ultimately defined by three basic or “direct fitness” traits (Roff, 2002): age at maturity, age-specific fertility, and age-specific survival (or, equivalently, age-specific mortality). These traits are sufficient to determine the fitness of a given strategy, operationalized as the population growth rate associated with the strategy; they also determine the organism's lifespan and lifetime fertility. Other traits that have been classically investigated in LHT include size at birth, rate of physical growth, size at maturity, and offspring size (Stearns, 1992), with body size often used as a proxy for phenotypic quality. Although the age-based approach is adequate to model the current–future reproduction trade-off (as well as many narrower trade-offs; see Roff, 2002), investigating the quality–quantity trade-off requires tracking an individual's state in addition to age (McNamara &

Houston, 1996). Individual quality can be recast as embodied capital, which extends the logic of LHT to traits such as health, skills, and status (e.g., Kaplan, 1996). In a broader perspective, life history strategies are expressed as synergistic combinations of co-adapted *morphological*, *physiological*, and *behavioral* traits (Braendle, Heyland, & Flatt, 2011). For example, in many organisms the transition to reproductive status involves a range of motivational and behavioral shifts, including the onset of sexual receptivity and competitiveness and the activation of behavioral systems that support parental care (e.g., nest building, offspring protection). Life history strategies that delay reproduction should be characterized by protracted behavioral immaturity and inhibition of reproduction-related behavioral systems. Moreover, delayed reproduction should usually be associated with risk aversion, so as to minimize the likelihood of dying before reaching maturity.

The bottom line is that life history strategies organize behavior in multiple domains—including risk-taking, self-regulation, aggression, exploration, mating, and caregiving (see Del Giudice, 2014a; Réale et al., 2010; Stamps, 2007; Wolf, van Doorn, Leimar, & Weissing, 2007). In species with complex social lives, life history strategies have deep implications for behaviors that depend on future rewards—such as long-term cooperation and reciprocity—as well as behaviors that affect investment in offspring quality, including pair-bonding and the multigenerational transmission of knowledge and resources. In addition, different life history strategies likely benefit from different arrays of cognitive traits involved in learning, memory, and decision-making (Réale et al., 2010; Sih & Del Giudice, 2012).

## Evolution of Life History Strategies at the Population Level

Variations in ecological factors (e.g., food supply, mortality hazards) imply different optimal allocation strategies, leading to across- and within-species variation in life histories. Mathematical models can be developed to predict the evolution of life history strategies. The standard approach is to model life history outcomes as a function of age-specific rates of *extrinsic mortality*—the risk of death due to difficult-to-avoid causes such as predation, accidents, epidemics, and so on (see Charlesworth, 1994; Roff, 2002). The broader concept of *extrinsic morbidity-mortality* (Ellis, Figueredo, Brumbach, & Schlomer, 2009) includes unavoidable causes of deterioration and disability (e.g., the long-term consequences of nonlethal injuries and diseases) that limit an organism's reproductive potential. Another important factor is the degree of *unpredictable variation* in environmental conditions (e.g., unpredictable mortality rates). Finally, the *availability of resources* sets the baseline for all sorts of allocation problems.

In general, high levels of extrinsic adult mortality select for early maturation and reproduction, early senescence (Kirkwood & Rose, 1991), and

concentration of reproductive effort in a shorter period of time; high mortality in juveniles also favors early maturation, but promotes life history strategies that spread reproductive effort over an extended window (Charlesworth, 1994; Roff, 2002). The effects of unpredictable temporal variation are more complex. Like high mortality, variation in adult mortality selects for concentrated reproductive effort and, typically, early reproduction (Murphy, 1968), although the latter effect depends on patterns of correlation between risks across time. By contrast, unpredictable variation in juvenile mortality favors delayed maturation and an extended reproductive schedule (Charlesworth, 1994). In general, the impact of unpredictable variation on the distribution of reproductive effort should be small compared to effects of average extrinsic mortality (Roff, 2002).

Another adaptive response to unpredictable variation in juvenile survival/fertility is *bet-hedging* (Roff, 2002; Ellis et al., 2009). Bet-hedging reduces the average individual fitness of offspring in the short term, but enhances the long-term reproductive success of the genetic lineage by decreasing fitness variance across generations (see Starrfelt & Kokko, 2012). *Diversified bet-hedging* does so by generating stochastic variation in life history traits across offspring, thereby increasing phenotypic diversity. *Conservative bet-hedging* produces a “generalist” phenotype that does relatively well in a broader range of environments and is thus less vulnerable to unpredictable fluctuations in fitness (Ellis et al., 2009; Starrfelt & Kokko, 2012). When temporal or spatial environmental variation can be anticipated by relying on predictive cues, selection often favors *plasticity* in life history strategies (e.g., Roff, 2002). As prediction is typically imperfect, plasticity and bet-hedging are not mutually exclusive; they may coexist in the same species or population (e.g., Donaldson-Matasci, Bergstrom, & Lachmann, 2013).

**[“bet-hedging” (hyphenated) is the standard usage in the literature]**

In models of the quality-quantity trade-off, high extrinsic mortality in both juveniles and adults favors lower levels of investment in somatic capital (Harpending et al., 1990; Kaplan, 1996). Moreover, optimal fertility derives from the available investment budget divided by the optimal investment per offspring (Kaplan, 1996; Smith & Fretwell, 1974). All else equal, then, higher resource availability increases optimal fertility, whereas lower mortality tends to decrease it.

In sexually reproducing species, males and females usually face different trade-offs as a result of sexual selection, which leads to the evolution of sexually differentiated life history strategies. For example, when male-male contests determine male access to mates, males tend to mature later than females to accumulate competitive ability (Roff, 2002). More generally, species-typical patterns of sexual selection and competition determine systematic sex differences in reproductive timing, allocation to mating and parenting, age-specific mortality,

and investment in different components of embodied capital (e.g., McDonald, 1993; Promislow, 1990).

## Development of Life History Strategies at the Individual Level

Individual differences in life history strategy are routinely observed within species and populations. Individual strategies reflect the combination of genotypic effects, plasticity in response to environmental inputs, and stochastic processes. Genotypic variance in life histories can be maintained by various processes including mutation-selection balance (Roff, 2002), frequency-dependent selection (e.g., the fitness of a parenting-oriented strategy may depend on the frequency of mating-oriented strategists in the population; see Sinervo, Clobert, Miles, McAdam, & Lancaster, 2008), and shifting selective optima due to environmental variation across space and time (e.g., Del Giudice, 2012).

Plasticity in life history traits in response to environmental states and individual conditions is widespread. Plastic organisms have *reaction norms*, which reflect contingent phenotypic expression. For reaction norms to be adaptive, the cues used to predict the future state of the environment must have sufficient reliability, and the benefits of matching the phenotype to the environment must exceed the costs of plasticity (e.g., maintaining the relevant physiological machinery, energetic costs). The evolution of reaction norms in life history traits and allocations can be modeled explicitly (e.g., Fischer et al., 2011). For example, Berrigan and Koella (1994) showed that, in a simple developmental model, the optimal strategy in response to high juvenile mortality is early maturation and, in response to energetic scarcity, delayed maturation. More generally, developmental responses to recurrent changes in environmental characteristics can be often expected to parallel evolutionary responses to the same characteristics. Thus, the logic of population-level models can usefully inform predictions about developmental plasticity in life history traits (see Ellis et al., 2009; West-Eberhard, 2003).

Individual reaction norms can be affected by genotypic factors. Two individuals may show a similar amount of plasticity, but different *average* levels of the trait. Conversely, one individual may be more plastic than the other, his or her phenotypes more responsive to environmental variation. The reaction norms of males and females typically differ, so that the two sexes respond differently to the same environmental cues. For example, when females invest heavily in offspring, they are more likely than males to delay reproduction (e.g., by suppressing fecundity) in response to cues of temporary energetic scarcity (Beehner & Lu, 2013; Wasser & Barash, 1983; on humans, see Ellison, 2001, 2003).

Chance affects life history development in various ways. The probabilistic nature of life history events inevitably produces large stochastic variations in

direct fitness traits, such as longevity and lifetime fertility (Steiner & Tuljapurkar, 2012). At the same time, bet-hedging strategies in response to unpredictable chance events may adaptively increase offspring diversity. Notably, sexual organisms can increase their offspring's diversity by simply having more of them, and by mating with multiple partners. Increased offspring quantity and promiscuous mating may constitute adaptive bet-hedging in response to unpredictable variation in juvenile survival (e.g., Fox & Rauter, 2003; see Ellis et al., 2009).

## The Fast-Slow Continuum

Life history traits do not evolve independently from one another; both within and across species, different traits covary in clusters. At the broadest level of analysis, the life history strategies of different species can be arranged on a continuum from “fast” (early maturation and reproduction, fast growth, small body size, high fertility, short lifespan, and low investment in offspring quality) to “slow” (late maturation and reproduction, slow growth, large body size, low fertility, long lifespan, and high investment in offspring; Promislow & Harvey, 1990; Sæther, 1987). Within-species variation often falls along the same continuum (see Réale et al., 2010).

The fast-slow continuum captures the pattern initially described by models of *r-K selection* (MacArthur & Wilson, 1967; Pianka, 1970). Those models assumed that life history evolution was driven by density-dependence, with “*K*-selection” (slow growth, late reproduction, low fertility) occurring in stable and densely populated ecologies and “*r*-selection” (fast growth, early reproduction, high fertility) resulting from fluctuating, sparsely populated ecologies. These claims have since been largely rejected or revised, as factors such as costly-to-avoid mortality risks and their unpredictability are seen as more important drivers of life history variation (see Ellis et al., 2009; Jeschke, Gabriel, & Kokko, 2008). The existence of a fast-slow continuum has nonetheless proven empirically robust.

When body size is controlled for, the fast-slow continuum has been claimed to either dissolve into two independent dimensions (Bielby et al., 2007) or be defined by markedly different life history traits (Jeschke & Kokko, 2009). However, reanalysis of the same data shows that, despite some meaningful differences between taxa—for example, high fertility is a “slow” trait in fish but not in birds or mammals—the fast-slow continuum is a stable dimension of life history variation, even controlling for differences in body size (Appendix in Del Giudice, 2014b). That said, the fast-slow does not fully account for life history variation. Comparative data invariably show the existence of other meaningful axes of variation, such as the “lifestyle” dimension identified by Sibly and Brown (2007), or the two dimensions of reproductive timing (current versus future) and

reproductive output (quality versus quantity) identified by Bielby et al. (2007; see also Del Giudice, 2014b).

In part, the fast-slow continuum emerges from fundamental constraints on the relationship between mortality and age at maturity (e.g., Roff, 2002; see also Brown et al., 2004, on constraints on metabolic rates). But life history traits may also coevolve because they adaptively respond to the same characteristics of the environment: for example, high levels of extrinsic morbidity mortality typically favor early maturation and reproduction, higher fertility, lower levels of investment in offspring quality, and, often, additional investment in mating effort.

## Limitations of Standard LHT

As noted earlier, the standard approach in LHT assumes an extrinsic component of mortality not subject to selection, which then explains variation in other life history traits. Ultimately, this approach is theoretically unsatisfying. Organisms, after all, exert control over virtually all causes of mortality (e.g., by altering patterns of travel to avoid predators, by investing in immune function). By treating a component of mortality as assumed rather than explained, this approach fails to offer a full understanding of how mortality rates evolve. A more complete approach assumes that ecological factors do not directly entail mortality rates, but rather affect the *functional relationships* between mortality and efforts allocated to reducing it (Figure 2.1). They do so, at least in part, by imposing particular “assault” types and rates on the organism. For example, warm, humid climates favor the evolution of disease organisms and, therefore, increase the assault rate and diversity of diseases affecting organisms, which in turn affect the relationship between efforts to combat disease and mortality reduction. Mortality reduction can then affect the pay-offs of other efforts; for example, dynamic optimization modeling (see Frankenhuis, Panchanathan, & Barrett, 2013) shows that growing larger brains should co-evolve with the allocation of effort to reduce mortality (Robson & Kaplan, 2003). Relatedly, standard models lump all causes of mortality into a single mortality rate. In fact, allocations to different components of somatic capital (e.g., immune function versus antipredator defenses) may track different sources of mortality in a finer-grained way (see Kaplan, 1996).



**Figure 2.1 Mortality as a function of investments.**

Adapted with permission from Kaplan and Gangestad (2005).

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## Mechanisms of Life History Allocation

Thus far, we have considered forces of selection that shape the evolution of life histories. We now turn to the proximate mechanisms that evolve to enact life history decisions.

### Endocrine Systems

Adaptive allocation typically requires coordinated tuning of multiple physiological and behavioral systems. Increased allocation to reproduction, for instance, should be coordinated with less allocation to growth. Increased effort to immune function in response to infection may best be synchronized with lower

overall expenditure. Such adaptive coordination usually requires systems of communication and control distributed across a variety of somatic systems. These roles are often filled by endocrine systems (Finch & Rose, 1995; Lancaster & Sinervo, 2011). Indeed, the primary function of endocrine systems, giving rise to them and shaping their specific nature, may well be the adaptive, coordinated allocation of energetic and other resources in the face of trade-offs.

Endocrine systems are internal communication devices. Hormones released at one site (e.g., the gonads, the adrenal cortex) are “picked up” by receptors at multiple other sites (e.g., brain structures), thereby affecting them in a modular fashion. Accordingly, hormonal signals can simultaneously regulate many different features and modulate allocation decisions at various timescales, from short-term adjustments to major transitions between life stages. Consider, for instance, reproductive hormones during human puberty. In females, mechanisms regulating energy balance lead to fat storage and regular menstrual cycling. As mediated by estrogen and other hormones, increased energy is allocated to reproductive traits and functions, including secondary sexual characteristics. Males begin producing androgens in substantial quantities, leading to greater musculature and investments in forms of mating effort, including social competition and physical performance. Simultaneously, other investments (e.g., in certain immune functions) are withdrawn. For both sexes, modulation of psychological processes (e.g., desires, motives, situation-specific responses) is integral to the matrix of coordinated responses (see Ellis, 2013; Ellison, 2001).

Reproductive hormones also regulate differential investments on shorter time scales. For example, testosterone levels decrease when men enter committed romantic relationships (e.g., marriage), arguably facilitating reallocation of reproductive effort from mating to parenting (e.g., Burnham et al., 2003; Gettler, McDade, Agustin, Feranil, & Kuzawa, 2013). As well, individual differences in the timing and amount of hormone production partly mediate the development of individual differences in life history strategy; for example, male testosterone levels show robust associations with status-oriented competitiveness and lifetime number of sexual partners (e.g., Eisenegger, Haushofer, & Fehr, 2011; Pollet, van der Meij, Cobey, & Buunk, 2011).

The same developmental mechanisms that mediate species-specific transitions between life history stages may mediate individual plasticity by acting as *developmental switches* (West-Eberhard, 2003). A developmental switch is a regulatory mechanism activated at a specific point in development. Based on input about the external environment and state of the organism, it shifts the individual along alternative pathways, ultimately resulting in the development of alternative phenotypes. Human puberty involves two major transition points, *adrenarche* (the onset of androgen production by the adrenal glands) and *gonadarche* (the onset of androgen/estrogen production by the ovaries and testes), both potentially key

switches in the development of life history strategies (Del Giudice, 2014c; Ellis, 2013).

Endocrine systems involved in life history allocations are remarkably conserved across species. Testosterone typically regulates trade-offs between mating, parenting, and survival in male vertebrates (Hau & Wingfield, 2011). In vertebrates and invertebrates alike, insulin-like growth factor 1 (IGF-1) is involved in the trade-off between survival and growth/reproduction (Gerish & Antebi, 2011; Swanson & Dantzer, 2014). The major life history regulators in vertebrates include the hypothalamic-pituitary-adrenal (HPA), hypothalamic-pituitary-gonadal (HPG), and hypothalamic-pituitary-thyroid (HPT) axes, the insulin/insulin-like growth factor 1 (IGF-1) signaling system, and pathways involving prolactin, oxytocin, vasopressin/vasotocin, and immune cytokines (Lancaster & Sinervo, 2011).

These systems are characterized by extensive interplay and cross-regulation. Within the broader network they define, some nodes may play key roles in decision-making processes, by integrating information from multiple sources and redistributing it to other systems. In vertebrates, the HPA axis seems to play such a central role in life history development, as it encodes and integrates crucial information about many characteristics of the social and nonsocial environment (e.g., danger, unpredictable/uncontrollable events, crowding; see Crespi, Williams, Jessop, & Delehanty, 2013; Lancaster & Sinervo, 2011). The role of the stress response system in the development of human life history strategies has been explored in the Adaptive Calibration Model of stress responsivity (Del Giudice, Ellis, & Shirtcliff, 2011; Ellis & Del Giudice, 2014).

## **Psychological Processes**

Endocrine systems may play very important roles in modulating coordinated allocation decisions. Because adaptive allocations are often contingent on environmental circumstances, psychological processes—the perception, interpretation, and evaluation of life circumstances—ultimately guide many allocation decisions, regardless of whether they are mediated by endocrine processes.

Consider, for example, a cortisol response to a current or impending threat. Circulating cortisol causes changes in energy mobilization and allocation, as part of a system shaped by selection posited by life history theory. Prior to the release of cortisol, however, a cascade of processes occurs. An event must first be perceived, appraised, and judged to be a threat. Various cortical regions of the brain are involved in this perception, depending on the nature of threat. The amygdala, which receives input from, as well as directs output to these regions, plays a special role in interpreting the event as a threat and initiating the HPA

response (see Gunnar & Quevedo, 2007). Psychological processes, then, initiate the re-allocation of energy that cortisol entails; corticotropin-releasing hormone (CRH) and cortisol may feed back on the brain regions involved in appraising and evaluating threats, so that different levels of the control cascade influence one another. In a word, this system is psychoneuroendocrinological.

The same reasoning applies to changes in the HPG system regulating the production and secretion of men's testosterone in the testes. The reduction in gonadal secretion of testosterone that men experience when they enter romantic relationships (e.g., Gettler et al., 2013) is a final outcome of a series of steps, initiated in the brain. Though the precise proximate mechanisms are not fully understood, romantic relationships probably elicit appraisals of long-term commitment and relative exclusivity (e.g., McIntyre et al., 2006). These appraisals, whether experienced consciously or not, ultimately leads to down-regulation of the HPG axis—a process possibly mediated by oxytocin production in the brain (Weisman, Zagoory-Sharon, & Feldman, 2014).

Psychological processes may regulate life history allocations in a number of ways. Consider the optimal age of first birth for females. Nettle (2011a) examined several psychological processes that may be involved in the decision (conscious or not) to initiate reproduction. First, experiences during early childhood, such as poor maternal care or household instability, may affect timing of menarche through developmental induction (discussed in detail later). Second, social learning processes may affect decisions. Copying of close social others, for instance, may be adaptive, if an aggregate of multiple individuals' sense of, say, mortality rates has greater validity than a single individual's. Third, contextual factors such as mortality cues may trigger adaptive, domain-specific responses that take the form of relatively simple (and often unconscious) heuristics. For example, research has found that local birth rates increase following death-causing events (e.g., hurricanes; Cohan & Cole, 2002); even thinking about death can increase desires to have children (e.g., Wisman & Goldenberg, 2005). Finally, women may engage in conscious planning, reasoning about their life situation, and considering the costs and benefits of different options. Culturally transmitted knowledge and values should be especially relevant at this level. Of course, the subjective perception of goals, costs, and benefits involved in conscious decision-making is itself influenced by nonconscious evaluation processes taking place in the brain/body.

A concept that may offer a useful way to conceptualize the psychological processes that mediate life history trade-offs is that of the *internal regulatory variable*. Tooby, Cosmides, and their colleagues introduced this term as a means of explaining how motivational and emotional processes are instantiated (e.g., Lieberman et al., 2007; Tooby, Cosmides, Sell, Lieberman, & Sznycer, 2008). As Tooby et al. conceptualize them, they are “evolved variables whose function is to store summary magnitudes (or parameters) that allow value computation to be

integrated into behavior regulation” (Tooby et al., 2008, p. 253). Put otherwise, selection would have forged cognitive systems that adaptively direct behavior contingent on circumstances that recurred in our ancestral history. An internal regulatory variable functions as an index of a circumstance upon which adaptive behavior is contingent.

A next step toward understanding how psychological processes affect life history allocations would involve positing the internal regulatory variables involved—how the mind computes specific summary stores of experiences that affect pertinent decisions. For instance, how are accumulated stores of environmental harshness of the kind informative of mortality rates registered and represented psychologically? What kinds of short-term indexes of mortality risk become represented, and through what processes do they affect decisions? How does information about the behavior of others become synthesized with these personal experiences? Research in this area has identified some promising psychological variables such the perceived controllability of the environment (Mittal & Griskevicius, 2014) and the subjective estimate of one’s life expectancy (Chisholm, 1999). To date, however, there are no explicit models of how these variables may be computed and how they are used to regulate behavioral and physiological processes.

Here we illustrated how psychological processes may regulate life history allocations with the example of age at first reproduction. But there are countless decisions demanding explanation at a proximate, psychological level—for example, allocations of energy to immune function; allocations of effort to increase offspring quality, as a function of returns on investment; investment in skill acquisition, dependent upon usage; dedication to developing and strengthening particular social relationships, in light of time horizons; allocation of efforts to aid kin, dependent on likely relative returns to such investment versus investment in efforts enhancing self; male efforts to protect paternity, at the risk of cuckoldry, as a function of mortality rates; and many more. Scientists have available life history theoretic models specifying how selection might operate on how optimal decision-making in these instances is affected by circumstances (e.g., for an analysis showing how males’ tolerance of investment in offspring not their own—cuckoldry—should be influenced by mortality rates, in ways not intuitively obvious but understandable through life history modeling, see Mauck, Marschall, & Parker, 1999). Yet in most cases, we know very little about the psychological processes involved in these decisions. More generally, very little is now known about the precise nature of the adaptations by which people solve the major trade-off problems that life history theory identifies. A primary task for the future of evolutionary psychology, in our view, should be to specify the nature of these adaptations.

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# Psychological Applications

We now review several areas of application of LHT in psychological research, organized around four overlapping themes: species-typical patterns of growth and development; individual differences in developmental trajectories; personality; and psychopathology.

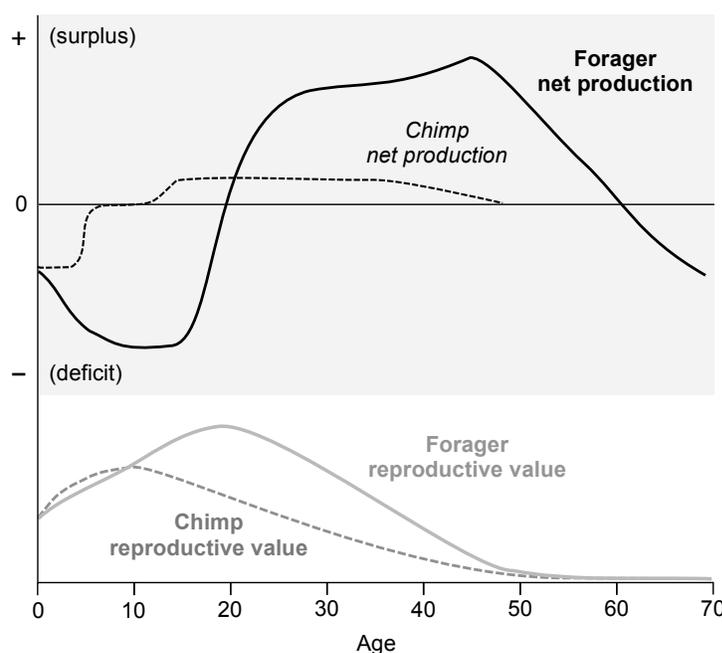
## Patterns of Growth and Development

### Human Life History and the Human Adaptive Complex

Humans have several distinctive life history features (Kaplan, Hill, Lancaster, & Hurtado, 2000)—a late onset of reproduction, an extended period of vulnerability and dependence during infancy and childhood, and a long lifespan with extended post-reproductive life (menopause). Relative to primate life histories, humans clearly fall at the slow end of the fast-slow continuum in most respects. At the same time, human populations that have not undergone the demographic transition show higher fertility and shorter interbirth intervals compared to close primate relatives.

This combination of traits can be understood in the context the *human adaptive complex*—a suite of coevolved traits that define humans' socioecological niche (Kaplan, Gurven, & Lancaster, 2007). Relative to chimpanzees, humans consume a diet consisting of nutrient-dense but difficult-to-extract foods such as meat, roots, and nuts (Kaplan et al., 2000). The techniques employed to acquire and process food (including hunting and fishing) are learning- and skill-intensive and often require extensive cooperation between related and unrelated individuals, with a special role played by pair-bonded couples (marriage). Attaining the skills to forage effectively and manage the complex social games that originate from cooperation and division of labor requires huge investments in embodied capital—including a large and flexible brain—and a long, slow phase of learning and dependency. As this way of thinking posits that social capabilities that permit one to choose and be part of cooperative ventures importantly affect foraging efficiency, it proposes that ecological and social intelligence coevolved and led to large investments in brains (Kaplan, Gurven, & Lancaster, 2007; Sterelny, 2007). It is compatible with data showing that both high-quality diet (emphasized by those who give priority to ecological intelligence) and social group size (emphasized by those who give priority to social intelligence, especially pertaining to close social bonds; e.g., Dunbar & Shultz, 2007) predict larger brains and slower development in primates (e.g., Walker, Burger, Wagner, & Von Rueden, 2006).

In humans, delayed maturation and intensive learning are made energetically sustainable by massive intergenerational transfers of resources from parents, grandparents, and others. Children do not pay their own way: they accumulate large calorie deficits that, in forager populations, are not repaid until about 20 years of age; after that, adults start producing large amounts of surplus calories, peaking around age 40 and continuing well into the seventh decade of life (Kaplan et al., 2000). By comparison, chimpanzees pay off their own calorie debt by age 5, generate relatively little surplus, and do so only while reproductively active (Figure 2.2).



**Figure 2.2 Net energetic production and reproductive value (expected future reproduction at a given age) in chimpanzees and human foragers.**

Adapted with permission from Kaplan and Gangestad (2005) and Gurven et al. (2012).

High-quality foraging, delayed development, and large energy debts entail considerable risks: returns from hunting and fishing can be highly variable, adverse conditions may reduce food availability, and one's parents may die before maturity. Complex cooperative strategies and resource transfers within and between generations absorb risk (Gurven, Stieglitz, Hooper, Gomes, & Kaplan, 2012). The costs of extended childcare are shared between mothers and others

such as grandparents and older siblings (*cooperative breeding*; see Hrdy, 2007); juveniles are routinely recruited to help with household activities and small-scale foraging, freeing parents to dedicate additional time and energy to high-quality foraging, breastfeeding, and so on (Kramer, 2011).

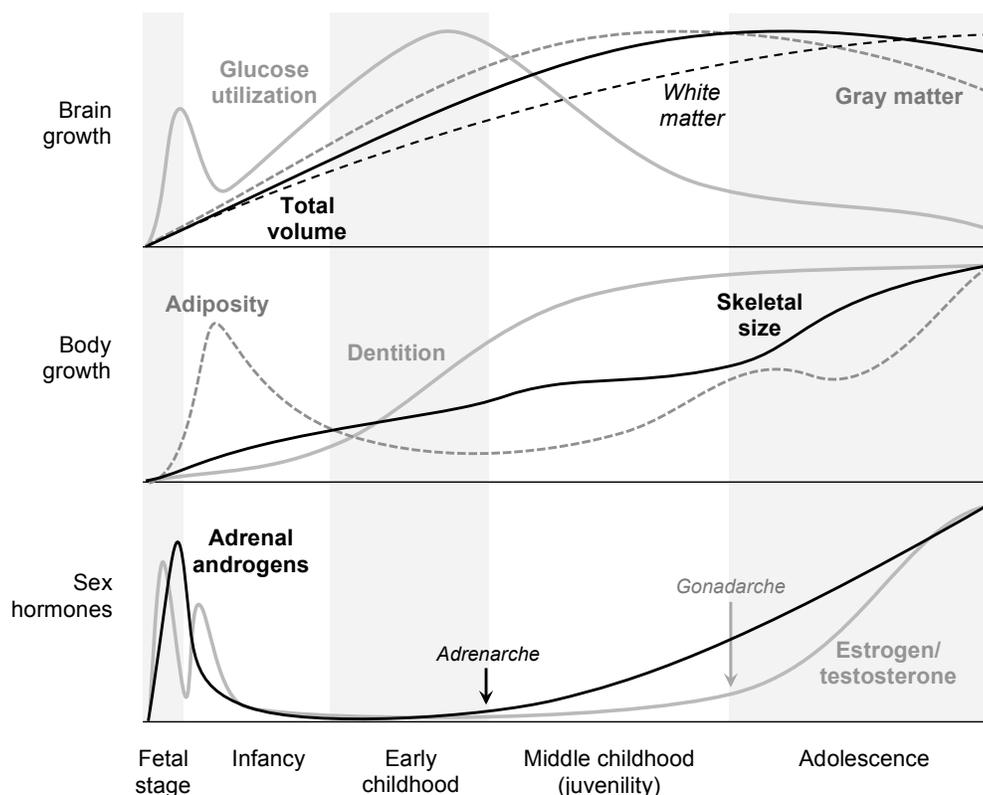
## Developmental Stages and Transitions

This analysis provides a background for conceptualizing human developmental stages and transitions in a LHT framework, one aspiring to offer an integrated model of physical and psychological development. For example, a central feature of early childhood (~2–6 years) is sustained, expensive brain growth; the proportion of glucose consumed by the brain peaks at age four, when it accounts for about 65% of the child’s resting metabolic rate (Kuzawa et al., 2014). These allocations deplete fat reserves accumulated during infancy, and entail a compensatory slowing of body growth (Kuzawa et al., 2014; Figure 2.3).

In turn, brain development permits the acquisition of language, the foundations of which are achieved by age 5. As language is arguably one of the most computationally complex processes in which humans engage, one may wonder why children acquire the ability to understand and produce a near-infinite number of utterances before they can even coordinate smooth running? An LHT framework offers a principled framework for answering such questions: Because language greatly increases the rate at which children learn about the world—such that benefits, post-acquisition, accrue rapidly—its development may be front-loaded, even at the expense of delaying the acquisition of other, computationally less demanding capabilities. Similar considerations apply to the development of basic mind-reading abilities (see Bjorklund, 2011).

Middle childhood (human juvenility; about 6 to 11 years) is characterized by intense learning. In traditional societies, children start practicing foraging techniques as well as social roles (Bogin, 1997). The transition to this phase is marked by adrenarche. Adrenal androgens shift energy allocation from the brain to the body, and trigger the accumulation of muscle and fat in preparation for sexual maturation (Campbell, 2011; see Figure 2.3). A cascade of cognitive and motivational changes accompany these changes: for example, marked increases in self-regulation, memory, and problem solving, the onset of sexual/romantic attraction, and the emergence and intensification of sex differences across domains (play, aggression, and so on; see Del Giudice, 2014c). Whereas language development in early childhood focuses on syntax and vocabulary, middle childhood witnesses a dramatic increase in pragmatic skills such as teasing, gossiping, joking, and verbal competition (Locke & Bogin, 2006). These remarkable physical, cognitive, and motivational changes can be understood in the light of shifting allocation priorities, both between different types of embodied capital and from exclusive investment in somatic effort to initial investment in

mating effort through social competition (Del Giudice, 2014c). Mating effort and sexual selection take center stage with the transition to adolescence, entraining yet another suite of coordinated physical and psychological changes (see Ellis, 2013; Hochberg & Belsky, 2013).



**Figure 2.3 Developmental trajectories of human growth and sex hormones production, from conception to adolescence.**  
[ch02f03]

Adapted with permission from Del Giudice (2014c).

## Individual Differences in Developmental Trajectories

Starting with seminal work by Belsky, Steinberg, and Draper (1991), LHT has been increasingly applied to explain individual differences in physical and psychological development. In Belsky et al.'s "psychosocial acceleration" theory,

harsh, insensitive parenting acts as a cue of ecological stress and promotes the development of fast life history strategies: earlier puberty, earlier sexual debut, higher investment in short-term mating effort, and an opportunistic-exploitative interpersonal orientation, typically expressed as aggression/noncompliance in males and anxiety/depression in females. They hypothesized that attachment security mediates the effects of parenting. Subsequently, Chisholm (1993, 1999) stressed the theoretical importance of local mortality rates (a proxy for extrinsic mortality) and argued that time preference—the preference for smaller immediate rewards versus larger, delayed rewards—importantly mediates life history development at the psychological level, (see also Kruger, Reischl, & Zimmerman, 2008).

Research has supported most of the theory's core predictions, while also guiding theoretical elaborations and modifications. In both sexes, early familial and ecological stress predicts earlier sexual debut and increased mating effort. At the same time, effects of early experience on pubertal timing appear largely specific to females (reviewed in Belsky, 2012; James & Ellis, 2013). Women's first potential reproduction is especially sensitive to sheer reproductive capability; moreover, women's reproductive window is shorter than that of men, and the requirements of pregnancy and lactation make women's fertility especially dependent on timing constraints. By contrast, male pubertal timing appears to be more strongly influenced by perceptions of mate quality (health, attractiveness, popularity) and availability of economic resources (James & Ellis, 2013; see also Copping, Campbell, & Muncer, 2014). The theory has been extended to incorporate systematic sex differences in insecure attachment styles (Del Giudice, 2009). Furthermore, research has aimed to unpack the construct of early stress by examining unique effects of environmental harshness and unpredictability (e.g., Belsky, Schlomer, & Ellis, 2012). Related studies have linked childhood illness, early sexual debut, and insecure attachment with preferences for exaggerated sex-typical features in opposite-sex faces and potential partners who display cues of short-term mating (e.g., Cornwell et al., 2006; de Barra et al., 2013; Kruger & Fisher, 2008).

Work inspired by LHT in this area has generally focused on developmental plasticity and focused on the family as a source of environmental cues. But other factors also play important roles. Genetic factors clearly affect developmental trajectories, including puberty timing and mating behavior (see Belsky, 2012). Some effects likely result from adaptively contingent development. For instance, as alluded to earlier, genetic factors affecting attractiveness and health may, in turn, affect life history outcomes. Gene-environment interactions are also possible; for example, attractiveness may be especially important in some environments (e.g., Gangestad, Haselton, & Buss, 2006), and certain genetic variants may increase life history plasticity by amplifying an individual's sensitivity to the

environment (see Belsky, Pluess, & Widaman, 2013; Ellis, Boyce, Belsky, Bakersmans-Kranenburg, & van IJzendoorn, 2011).

Family stress does not appear to fully mediate the effects of broader ecological factors such as mortality and violence rates. Other plausible mechanisms include social learning (e.g., copying one's mother's behavior) and direct observation of mortality cues (Copping, Campbell, & Muncer, 2013; Nettle, 2011a). Recently, Rickard, Frankenhuis, and Nettle (2014) advanced the intriguing hypothesis that early stress may speed up life history strategies not only because it predicts a dangerous future environment ("external prediction"), but also because it predicts increased morbidity-mortality due to stress itself and associated somatic damage ("internal prediction"). Internal prediction can be adaptive even when external prediction fails. The degree of stability required for successful external prediction remains a matter of debate (Nettle, Frankenhuis, & Rickard, 2013, 2014; Del Giudice, 2014d). In addition, these models have not been tested against predictions explicitly derived from embodied capital theory. For example, gains from investments in embodied capital, especially education, will correlate with early events and with community-level mortality rates. It still remains to be resolved whether the early events set the psychology or the costs and benefits realized over developmental time determine whether the gains from delaying pregnancy in terms of future life prospects are worth the costs.

## **Contingent Responses to Threat**

Recently, researchers have begun to investigate the effects of early experience on contingent responses to subtle threats of mortality and scarcity (e.g., Griskevicius, Delton, Robertson, & Tybur, 2011; Griskevicius et al., 2013; Mittal & Griskevicius, 2014; White, Li, Griskevicius, Neuberg, & Kenrick, 2013). Participants are asked about their childhood socioeconomic status (SES), then experimentally primed with stimuli suggesting threats of mortality or resource scarcity (i.e., news stories about rising homicide rates or looming economic recession). The hypothesis is that people raised in low-SES environments should have faster life history strategies and a tendency to appraise potential threats as unavoidable/uncontrollable (i.e., more "extrinsic"), whereas people with a high-SES upbringing should have slower life history strategies and a bias toward perceiving future threats as avoidable/controllable ("intrinsic").

People with low SES childhoods respond to mortality threats by expressing a desire for having children earlier, even at the cost of delaying one's education or career development, whereas those with high SES childhoods react with a preference shift in the opposite direction (Griskevicius et al., 2011). Mortality threats prompt participants with low-SES childhoods to choose riskier but more diversified options over safer and less diversified ones (e.g., different stock packages; White et al., 2013). Participants with low-SES childhoods respond to

scarcity threats with increased risk-taking and shorter time preferences (i.e., spending more now and saving less for the future), whereas participants with high-SES childhoods show increased risk avoidance and longer time preferences. Perceptions of personal control may mediate the psychological effects of the scarcity threat (Mittal & Griskevicius, 2014; Griskevicius et al., 2013). Intriguingly, behavioral differences between the two groups only emerge in the threat condition; absent threat, participants from different socioeconomic backgrounds make similar choices and express similar preferences.

Taken together, these studies open a window on the psychological mechanisms that mediate life history allocations through real-time behavioral adjustments to environmental change. They also offer an intriguing adaptationist alternative to the standard view that impulsivity and risk taking in low-SES environments are the outcomes of poor decision-making or deficits in coping strategies (see Frankenhuis & de Weerth, 2013). It remains unclear which aspects of a low-SES upbringing drive the development of threat-contingent strategies, because low income is associated with a wide range of life history-relevant experiences including—but not limited to—nutritional stress, harsh or neglectful parenting, household instability, exposure to violence, and exposure to infectious agents. Moreover, the association between SES and threat-contingent strategies may be partly mediated by genetic factors rather than induced by early experience.

## **Personality**

The idea that stable personality traits partly reflect individual differences in life history strategy has been gaining ground in biology and psychology. In their framework for understanding personality variation in nonhuman animals, Réale et al. (2010) proposed that fast strategies should typically be associated with increased boldness, activity, and aggression, lower sociability, and superficial (versus thorough) exploration. This list can be expanded to include impulsivity, risk taking, and neophilia (Del Giudice, 2014; Sih & Del Giudice, 2012; Wolf et al., 2007). These features may be expressed differently in different species.

In humans, the personality traits of agreeableness, conscientiousness, and honesty-humility consistently relate to reduced mortality, high investment in predictors of parental effort (e.g., relationship stability), reduced investment in mating effort (e.g., restricted sociosexuality and fewer sexual partners), and prosocial/cooperative behaviors. Conversely, impulsivity and some facets of extraversion and openness to experience (e.g., dominance, sensation seeking, imagination) predict fast life history traits such as increased mortality, relationship instability, unrestricted sociosexuality, larger numbers of sexual partners, and exploitative/antisocial behaviors (reviewed in Del Giudice, 2012, 2014a). How emotional stability (low neuroticism) contributes to life history strategies is less clear. There is initial evidence that anxiety and worry affect women's quality—

quantity trade-off through effects on parenting (Alvergne, Jokela, & Lummaa, 2010). A recent study in Tsimane forager-horticulturalists showed that individual variation in this population is best described by two personality dimensions (*prosociality* and *industriousness*) rather than a standard “Big Five” (Gurven, von Rueden, Hooper, Gomes, & Kaplan, 2013). Intriguingly, these dimensions largely reflect mixtures of conscientiousness, agreeableness, and aspects of extraversion, consistent with the idea that these traits reflect fundamental behavioral trade-offs.

Although the existence and meaning of a “general factor of personality” (GFP) are still debated in the literature, some scholars have argued that the GFP—essentially, a dimension of socially desirable personality emerging from the covariation between emotional stability, extraversion, conscientiousness, agreeableness, and openness—is associated with slow strategies (see Figueredo, Woodley, & Jacobs, Chapter 40, Volume 2). Also, profiles of personality and cognitive ability seem to become increasingly differentiated toward the slow end of the spectrum, perhaps reflecting benefits of behavioral specialization in slow strategists (Figueredo et al., Chapter 40, Volume 2).

All personality traits are at least moderately heritable (Ebstein, Israel, Chew, Zhong, & Knafo, 2010). Their associations with life history trade-offs leave open the question of what evolutionary processes have maintained genotypic variation. Personality traits may be subject to directional selection (maximal fitness associated with high or low levels of the trait) or stabilizing selection (maximal fitness associated with intermediate trait values). In either scenario, genetic variation is maintained through mutation-selection balance. Genetic variation may also be maintained by balancing selection, whereby selection pressures vary spatially, temporally, between the sexes, or depending on the frequency of a phenotype in the population (see Gangestad, 2011; Nettle, 2011b). In the Tsimane, personality traits predict fitness in ways that vary systematically across regions and between the sexes (Gurven, von Rueden, Stieglitz, Kaplan, & Rodriguez, 2014). Another potential source of balancing selection on personality is temporal fluctuation in local sex ratios; the relative proportion of males and females in the mating pool modulates the costs and benefits of life history allocations, such as that between mating and parenting effort (Del Giudice, 2012).

## **Psychopathology**

By organizing physiology and behavior across domains, life history strategies also contribute to increased or decreased risk for mental disorders. Some putative disorders may be best understood as adaptive behavioral strategies, albeit with socially or personally undesirable consequences. Several authors have argued that externalizing disorders such as psychopathy, antisocial personality disorder, and conduct disorder are (male-typical) behavioral manifestation of fast life history strategies (e.g., Barr & Quinsey, 2004; Belsky et al., 1991; Mealey, 1995).

Potentially, borderline personality disorder is a (female-typical) manifestation of fast life history strategy (Brüne, Ghiassi, and Ribbert, 2010; Brüne, 2014). For many other disorders, it is much less clear to what extent they represent adaptive strategies, maladaptive phenotypic extremes, or dysregulation of adaptive mechanisms. Nonetheless, individual differences in life history may play a role in their origin. For example, the spectrum of eating disorders appears to covary with increased sexual competition and fast life history indicators in women (Salmon, Figueredo, & Woodburn, 2009). Associations of attention-deficit and hyperactivity symptoms with fast life history indicators such as lower birth weight and unrestricted sociosexuality have been documented as well (Frederick, 2012).

Del Giudice (2014a, 2014b) advanced a comprehensive framework for psychopathology inspired by LHT. The framework identifies four pathways from life history strategy to psychopathology: First, adaptive life history-related traits may be regarded as symptoms; second, life history-related traits may be expressed at maladaptive levels (e.g., as a result of assortative mating between individuals high in the trait); third, adaptive strategies may yield individually maladaptive outcomes (e.g., defensive mechanisms may “misfire” with catastrophic consequences); finally, life history-related traits may increase vulnerability to dysfunction (e.g., upregulated defensive mechanisms may be more vulnerable to deleterious mutations or environmental insults).

Del Giudice (2014a) argued that many mental disorders can be classified as *fast spectrum* or *slow spectrum* conditions, depending on their correlates in the domains of motivation, self-regulation, personality, sexual maturation, and environmental predictors. Putative fast spectrum disorders include externalizing disorders, borderline personality disorder, schizophrenia spectrum and bipolar disorders (possibly a heterogeneous category), and specific subtypes of eating disorders, obsessive-compulsive disorder, and attention-deficit/hyperactivity disorder. Putative slow spectrum disorders include autism spectrum disorders (possibly heterogeneous), obsessive-compulsive personality disorder, and specific subtypes of eating disorders, obsessive-compulsive disorder, and attention-deficit/hyperactivity disorder (see Del Giudice, 2014a, 2014b). Depression appears to be a highly heterogeneous category, with some indications that fast life history strategies may be especially conducive to depressive disorders with high levels of somatic (stress-related) symptoms (Del Giudice, 2014a).

This proposal is theoretically ambitious, and much research is needed to flesh out the breadth of its applicability. Certain factors that increase vulnerability to disease (e.g., deleterious mutations) are likely to do so through pathways other than ones directly implicating life history strategies (e.g., compromised neural integrity, affecting schizophrenia and other neurodevelopmental disorders; see Yeo, Pommy, & Padilla, 2014). For this reason, the domain of adaptive *function* captured by life history strategies must be integrated with that of *functionality*, as instantiated in the efficiency and integrity of psychological and neurobiological

processes (see Del Giudice, 2014b). Classifying disorders based on motivation, self-regulation, and so on is complicated by the overdetermination of behavioral traits, which reflect life history strategies only in part (Gangestad, 2014). A strength of the framework, however, is its theory-grounded empirical generativity. Applications of LHT to psychopathology should continue to yield useful insights in the structure and meaning of mental disorders.

## **Present Limitations and Future Directions**

### **Theoretical Challenges**

LHT is a theoretical foundation of modern evolutionary biology, one that speaks very broadly to how selection operates on what organisms do and how they develop. The major concepts drawn upon to date within evolutionary psychology pertain to the fast-slow continuum of life histories, and specifically as they inform an understanding of developmental trajectories and individual differences.

Although this continuum is an important topic in biology, it is merely one aspect of the theory. Life history theory is far broader in scope and much more ambitious as an explanatory framework. It pertains to trade-offs between allocations of energy of many types, arguing that an understanding of how selection shapes organisms to execute them is a function of their fitness effects integrated across the life span. At a broad level, they may be few in type (e.g., current versus future reproduction, quality versus quantity of offspring). But at a more specific level, they are numerous; at any point in its existence, an organism could be allocating its energy to an extraordinary range of fitness-enhancing features and activities (e.g., bodily features with various impacts on survival and access to mates, brain structures, multiple elements of immune function, somatic repair, food search, mate search, mate retention, assisting kin—as merely a start). Within evolutionary psychology, the strong identification of life history theory with the fast-slow continuum limits appreciation of its richness and leads to an overly simplified understanding of its foundational nature.

Life history theory is expressed in mathematical models; work in evolutionary psychology could benefit from greater development of formal models of life history evolution and development in humans. For example, the approach to developmental trajectories inaugurated by Belsky et al. (1991) depends on the assumption that children can reliably forecast future conditions based on cues received during the first 5–7 years of life. However, only recently (Nettle et al., 2013) was this assumption formalized in a mathematical model. Subsequent debate (Del Giudice, 2014d; Nettle et al., 2014) has attempted to clarify the conditions under which the assumption may be plausible and, equally important, the kinds of empirical data that can test its validity. Future research should

combine evolutionary and cognitive modeling to better understand the psychological processes involved in life history allocations, as well as the origin and nature of relevant internal regulatory variables. Work in this area could benefit by interfacing with literatures on heuristics and decision-making, as well as with the expanding biological literature on the integration of adaptive functions and behavioral mechanisms (see McNamara & Houston, 2009).

## Empirical Challenges

Biologists interested in understanding the life history of a species often adopt a whole-organism approach that combines behavior, morphology, and physiology. Although behavioral components are often of key interest, they must be understood in combination with growth, metabolism, immune function, and so on. Psychologists have tended to focus on behavior at the exclusion of the other dimensions of life history allocation. Relatedly, research has often assessed life history strategies solely through questionnaire measures of behavioral and psychological traits, assuming that clusters of these variables map well onto allocations that define life histories. As individual behaviors are typically multiply determined, linkages with other dimensions of life histories may be modest (see also Copping et al., 2014). Research could benefit from a broader array of measures, including parameters of immune function, reproductive and metabolic hormones, energy utilization, growth trajectories, oxidative damage, and other indicators of somatic degradation. Metabolic regulators such as thyroid hormones and IGF-1 should be investigated alongside more commonplace reproductive and stress hormones.

Because of the focus on the fast-slow continuum and its emphasis on the transition from prereproductive growth to the reproductive phase of life, life history work in psychology has paid much attention to adolescence and early adulthood. Allocation decisions at other stages of the life course—the prenatal period, infancy and early childhood, the postreproductive phase, and the aging process more generally—have received much less attention (see Del Giudice & Belsky, 2011). Broadening the current perspective on life history trade-offs and decisions will be especially important in view of the disproportionate force of selection on early survival (Jones, 2009) and the severe metabolic trade-offs involved in brain growth through infancy and childhood (Kuzawa et al., 2014). Also, very little attention has been paid to rates of cognitive decline with age, and on changing endocrine profiles late in life in evolutionary life history models. Do we expect cognitive aging to proceed at the same rate as cardiovascular or immune system aging? Do changing endocrine profiles with age reflect dysregulation of those systems or are they adaptive responses to deteriorating phenotypic condition? Research designed to answer these questions is likely to be quite productive.

Increasingly, concepts and insights inspired by LHT permeate the field of evolutionary psychology, particularly with respect to individual differences and their developmental trajectories. We believe the discipline is ready to embrace the life history approach in its full richness, and look forward with excitement to the theoretical and empirical fruits of this integration.

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