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Abstract

In this chapter we situate developmental psychopathology in an evolutionary perspective, and demonstrate how the discipline can benefit by embracing modern biological theory. We begin by presenting the integrative approach of evolutionary-developmental psychology and exploring the interplay between adaptation and maladaptation in the origin of disorders. We then review a host of recent theoretical developments that address the organization of individual differences, the nature of environmental risk, the role of early stress, the nature of gene-environment interactions, the classification of mental disorders, and many other critical issues. Together, these contributions paint the contours of an integrative theory of human development and provide a sophisticated evolutionary foundation for developmental psychopathology. We aim to show that, far from undermining the tenets of developmental psychopathology, the evolutionary framework we describe supports all its core principles, while also extending them, clarifying their underlying logic, and connecting them at a deeper level than previously possible.

Keywords

Developmental plasticity; differential susceptibility; evolutionary-developmental psychology; gene-environment interactions; life history theory; maladaptation; stress.

Right since its inception in the 1970s and 1980s, the discipline of developmental psychopathology has pursued an ambitious project of theoretical integration. The grand vision of developmental psychopathology is that of a truly multidisciplinary approach to the complex interplay of biological, psychological, and social-contextual aspects of both normal and abnormal development (Cicchetti, 1990, 2006; Hinshaw, 2013). As testified by this volume, the project has been remarkably successful, generating an impressive amount of empirical work while maintaining a shared language and a common theoretical background.

In this chapter we argue that—despite its achievements—developmental psychopathology has yet to realize its full potential, and that its integrative power is limited by the lack of an adequate metatheory. We contend that developmental psychopathology has much to gain by embracing modern evolutionary theory, the unifying metatheory of the life and behavioral sciences. We then review a host of recent theoretical developments in the field of evolutionary-developmental psychology (EDP) that address the organization of individual differences, the nature of environmental risk, the role of early stress, the nature of gene-environment interactions, and many other critical issues. Together, these contributions paint the contours of an integrative theory of human development and provide a sophisticated evolutionary foundation for developmental psychopathology. We aim to show that, far from undermining the tenets of developmental psychopathology, the EDP-based framework we describe supports all its core principles, while also extending them, clarifying their underlying logic, and connecting them at a deeper level than previously possible.

Toward an Evolutionary-Developmental Framework for Psychopathology. We begin the chapter by considering the role of evolutionary theory in developmental psychopathology. After reviewing the core points of the discipline and the historical reasons for its separation from mainstream evolutionary biology, we present the integrative approach of EDP and discuss its metatheoretical foundations. The basic concepts we introduce here provide a general introduction to evolutionary biology and the overarching background for the rest of the chapter. We also review the main tenets of developmental systems theory (DST; Griffiths & Gray, 2004; Oyama, Griffiths, & Gray, 2001), consider its potential role as an alternative metatheory, and conclude that EDP provides a suitable framework for developmental psychopathology.

Beyond Pathology: Adaptation, Maladaptation, and Disorders. The theme of this section is the interplay between adaptation and maladaptation in the origin of disorders. We build on the distinction between adaptive and desirable traits, and discuss how the concept of disorder can be specified in evolutionary terms. We then broaden our view to explore the many ways in which evolutionary and developmental processes—both adaptive and maladaptive—may result in undesirable outcomes at the individual level.

Beyond Mental Health: Conditional Adaptation and Life History Theory. In this section we introduce the concepts of developmental plasticity and conditional adaptation. We discuss how organisms make use of environmental cues to adaptively match their phenotypes to their developmental context, and the ways in which those processes can fail and result in maladaptive outcomes. We then present a non-technical overview of life history theory, the dominant biological theory of conditional adaptation and a general framework for understanding the organization of individual differences in physiology, growth, and behavior. Drawing on life history concepts, we take a closer look at the multidimensional nature of environmental risk, and

examine the logic by which physical and social environmental factors shape and direct individual development.

Beyond Allostatic Load: The Stress Response System as a Mechanism of Conditional Adaptation. Here we focus on the central role of stress in the development of individual differences and psychopathology. We argue that the standard framework employed in developmental psychopathology—the allostatic load model (McEwen & Stellar, 1993)—fails to capture the multiple roles of stress in development, and promotes a limited understanding of stress as a risk factor and a source of physiological and behavioral dysregulation. As an alternative, we propose the Adaptive Calibration Model (ACM; Del Giudice, Ellis, & Shirtcliff, 2011), a theory of individual differences in stress responsivity across the life span based on concepts from life history theory and the theory of conditional adaptation. The ACM offers a renewed understanding of the role of stress in development and illustrate the heuristic and integrative power of the evolutionary developmental approach.

Beyond Diathesis-Stress: Differential Susceptibility to Environmental Influences. People vary dramatically in the extent to which they respond to their developmental context. In recent years, it has become apparent that many of the genetic, temperamental, and neurobiological factors that make people more vulnerable to negative, stressful environments also make them more likely to benefit more from positive, supportive environments. Differential susceptibility to the environment is a source of systematic organism x environment interactions, with many implications for both normal and pathological development. In this section we explore the evolutionary logic of differential susceptibility and review the main theoretical models that have been proposed to explain it (Belsky, 1997, 2005; Boyce & Ellis, 2005; Del Giudice, under review).

Beyond the DSM: A Life History Framework for Mental Disorders. We conclude the chapter by showing how life history concepts provide the foundation for an integrative evolutionary approach to mental disorders (Del Giudice, in press). The framework we outline is based on the idea that individual differences in life history strategy set the stage for the development of psychopathology. The resulting taxonomy offers a promising alternative to both the atheoretical approach of the DSM and empirical classification systems based on the distinction between internalizing and externalizing disorders.

Each section in the chapter shows how an evolutionary developmental approach goes beyond current thinking and contributes to broaden our understanding of psychopathology. At the end of each section, we consider how the concepts and theories we discuss relate to the core points of developmental psychopathology. As our ultimate goal is to catalyze a paradigm shift in developmental psychopathology, we deliberately focus on general principles rather than specific disorders throughout the chapter.

Toward an Evolutionary-Developmental Framework for Psychopathology

The Missing Foundation of Developmental Psychopathology

Over the years, a consensus has formed around a set of core points—methodological commitments, goals, and theoretical principles—that define developmental psychopathology as a scientific field. Developmental psychopathology adopts a multidisciplinary perspective; pursues

integration across multiple levels of analysis; gives particular consideration to the social and cultural context, as well as to brain and neurobiological factors; and emphasizes person-centered designs in empirical research. Researchers in the field aim to describe, understand, and synthesize the interplay between normal and pathological development, between developmental continuity and discontinuity, and between risk and protective factors. Finally, developmental psychopathology adopts three key principles from systems theory and developmental biology: the twin principles of equifinality and multifinality, and a view of ontogenetic causality as probabilistic, nonlinear, and involving reciprocal interactions between the developing organism and the environment (see Cicchetti, 1990, 2006; Hinshaw, 2013).

These points are extremely valuable and we subscribe to all of them. At the same time, we recognize that something crucial is missing. Developmental processes are biological processes, and biology is ultimately about function. Yet while developmental psychopathology is highly attuned to the complexities of *how* humans develop, its core points are silent with respect to the *whys* of development. Why do developmental processes unfold in one way rather than another? Why, for example, have they evolved so as to be exquisitely sensitive to contextual factors? And why do different processes show different degrees of context sensitivity? More generally, what is development *for*? Nikolaas Tinbergen (1963) famously summarized the four types of explanation required for a complete understanding of a biological system. With an updated terminology, they can be described as: *mechanism* (what is the system like? How does it work?); *development* (how does it come to be over developmental time, and how does it change across the lifespan?); *phylogeny* (what is the evolutionary history of the system? How did it change across generations and species?); and *adaptation* (why is the system the way it is? What selective advantages does it confer, or used to confer, to the organism?).

Developmental and mechanistic explanations concern the way an organism works in the present, without reference to evolution and adaptation; collectively, they are called *proximate* explanations. In contrast, *ultimate* explanations (phylogenetic and adaptationist) consider the organism in relation to its past and to the evolutionary forces that shaped its body and behavior (Mayr, 1963). The four types of explanation are not mutually exclusive but complementary and synergistic: adaptive function crucially informs the study of mechanism and development, while development and mechanism constrain the range of plausible adaptive explanations (see Scott-Phillips, Dickins, & West, 2011; West-Eberhard, 2003). Restricting one's view to the proximate level of explanation can only result in a partial understanding of the investigated system, in direct contrast with the integrative stance of developmental psychopathology.

In spite of the growing influence of evolutionary theory on the study of human behavior and development (see Buss, 2005; Ellis & Bjorklund, 2005), developmental psychopathology has remained virtually insulated from mainstream evolutionary biology until very recently. Indeed, earlier authoritative introductions to developmental psychopathology (Cicchetti, 2006; Hinshaw, 2013) did not even mention evolutionary biology as one of its contributing disciplines. This state of affairs has historical reasons that should be considered, however briefly. The theoretical foundations of the discipline were largely laid down between the 1970s and the 1980s (Cicchetti, 1990). At the time, the inclusive fitness revolution (discussed in more detail below) was still underway in biology and had had only little impact on the human behavioral sciences. Debates within biology often pitted proximate (developmental) and ultimate (evolutionary) causation against one another rather than acknowledge their complementary nature (West-Eberhard, 2003, ch. 24), and early adaptationist thinking tended to ignore or downplay developmental dynamics.

Moreover, initial attempts to apply the logic of adaptation and inclusive fitness to human social behavior were surrounded by heated and often heavily politicized arguments (Segerstrale, 2000).

Coming of age in this intellectual context, developmental psychopathology embraced the holistic, organismic approach prevalent in embryology (Cicchetti, 1990), as did other sectors of developmental psychology and psychobiology. Organismic theories—epitomized by modern DST—stress the hierarchical, self-organizing, active nature of development and the dynamic, reciprocal relationship between the developing individual and its environment (Reese & Overton, 1970). Clearly, this viewpoint has much to offer, and captures some crucial features of developmental processes. However, acceptance of organismic theories has often gone hand in hand with wholesale rejection of the adaptationist approach that informs large part of mainstream evolutionary biology. Psychological theorizing based on adaptationist concepts has been rejected—incorrectly and prematurely, in our view—as implying genetic determinism, a simplistic and unidirectional conception of causality, and an inability to deal with ontogenetic change and transformation. This stance has since become entrenched in developmental psychology (see Lickliter & Honeycutt, 2003; Overton, 2006), and has contributed to insulate the discipline from contemporary evolutionary thinking.

In contrast with this view, and in line with more than two decades of biological and psychological research, we believe that the basic adaptationist approach can be extended and revised to accommodate a sophisticated view of developmental processes. Importantly, this can be done without renouncing its main tenets—such as inclusive fitness theory and the proximate/ultimate distinction (e.g., Bjorklund & Ellis, 2005; Bjorklund, Ellis, & Rosenberg, 2007; Frankenhuis, Panchanathan, & Barrett, 2013; Olson, 2012; Tooby, Cosmides, & Barrett, 2003; West-Eberhard, 2003). Reconciling the logic of natural selection with a truly developmental approach to human behavior is the central goal of EDP, a fast-growing field at the intersection of developmental psychology and evolutionary biology. We now turn to a more detailed analysis of EDP and its theoretical underpinnings.

Evolutionary-Developmental Psychology

EDP is the application of the principles of Darwinian evolution to explain contemporary human development (see Bjorklund & Ellis, 2005; Bjorklund & Pellegrini, 2002). The central assumption of EDP is that cognitive mechanisms, behavior patterns, and the developmental processes that produce them have been shaped by Darwinian selection processes across our phylogenetic history. The primary focus of the field is how evolved psychological mechanisms develop through bidirectional interactions between environmental and genetic factors. The approach of EDP is thus intrinsically interactionist, with a strong emphasis on contextual factors. Consistent with this outlook, EDP views the developing individual as a plastic organism that adapts to the local context while contributing to determine its own environment. Plasticity, however, is not understood as arbitrary malleability; rather, the plastic child responds to the environment following evolved rules that tend to guide development toward adaptive goals. In other words, developmental plasticity is to a large extent *adaptive*—and itself shaped by past selection history.

Universal, species-typical features of human development—such as play, extended immaturity, and language acquisition—are obviously a major research theme of EDP. At the same time, EDP aims to explain individual and gender *differences* in development and behavior—for example in play preferences, pubertal timing, and linguistic abilities. The emphasis

of EDP on adaptive variation has been a driving force in the recent surge of interest in the evolution of individual differences in behavior and personality (e.g., Buss & Hawley, 2011; Carere & Maestripieri, 2013). Given the pivotal importance of individual differences in psychopathology, the theories and models we review in this chapter focus on the developmental processes that make individuals different from one another, including more or less at risk for different mental disorders.

Metatheoretical Foundations of EDP

When scientists formulate theories and hypotheses and evaluate them against empirical data, they rely on basic, a priori assumptions that inform theory-building. Once they have been established (empirically or otherwise), these assumptions are usually not directly tested thereafter; instead, they are used as a starting point for further research. Philosopher of science Imre Lakatos (1970) referred to these basic a priori assumptions as the "hard core" of a research program. For example, Newton's laws of motion provide the metatheory for classical mechanics, and the principles of adaptation through natural selection provide a metatheory for evolutionary biology. Metatheories operate like maps of the scientific terrain explored by a discipline: they provide boundaries between plausible and unlikely explanations, guidance in formulating hypothesis and interpreting empirical data, and heuristic rules for discovery. Between metatheories and specific empirical hypotheses are middle-level theories, which have limited scope and are more directly exposed to empirical testing. A metatheory integrates the relevant middle-level theories into an organized, internally consistent whole. Within a given metatheoretical program, scientists build middle-level theories and even narrower conceptual models, which are then used to generate hypotheses and predictions that can be empirically tested (for extended discussion, see Ketelaar & Ellis, 2000).

The metatheoretical foundations of EDP comprise both general and special assumptions (see Bjorklund & Ellis, 2005; Bjorklund et al., 2007; Durrant & Ellis, 2003; Ketelaar & Ellis, 2000). The general assumptions of EDP concern evolution by natural selection and are shared with mainstream evolutionary biology. Special assumptions concern (a) the application of evolutionary principles to the psychological level of analysis, and (b) the conceptualization of developmental processes in terms of probabilistic epigenesis. Assumptions about psychological mechanisms are shared with the sister field of evolutionary psychology, while the assumption of probabilistic epigenesis originates in developmental systems theory (DST). However, it should be stressed that EDP embraces a "soft" version of DST that is compatible with an adaptationist stance, while rejecting the most radical claims of DST proponents (Bjorklund & Ellis, 2005; Bjorklund et al., 2007). Below we examine the distinction between "hard" and "soft" DST in more detail.

General Metatheoretical Assumptions

Natural selection. The Darwinian concept of natural selection is the cornerstone of evolutionary biology. For natural selection to occur in a population of organisms, three conditions must apply: (a) individuals differ from one another in their physical and/or behavioral traits (i.e., their *phenotype*); (b) at least some of these phenotypic traits affect an individual's ability to successfully reproduce in the next generation; and (c) phenotypic traits are heritable—that is, they are transmitted to descendants with some reliability. When these conditions are met, individuals that are better able to reproduce leave more descendants, which in turn carry the

physical and behavioral traits that favored reproduction in the previous generations. Over time, successful traits tend to become more common in the population—that is, they are "selected for" because of their positive effects on reproductive success or *fitness*. Traits that enhance fitness are called *adaptive*, while those that reduce fitness are called *maladaptive*; if a trait does not affect fitness, it is considered *neutral* with respect to natural selection. It is important to note that natural selection is an abstract process, and does not require specific inheritance mechanisms (such as DNA) in order to work. All is required is the combination of heritable variation and differential reproduction based on that variation.

The most basic measure of Darwinian fitness is an individual's reproductive success, that is, the number of that individual's offspring that survive to maturity. Of course, in order to reproduce an organism also needs to survive, but—despite popular renditions of natural selection as "survival of the fittest"—survival without reproduction is an evolutionary dead end. It doesn't matter how well an organism is able to survive; if it fails to leave descendants, the traits responsible for its enhanced survival abilities will not be represented in subsequent generations. Organisms thus need to trade off longer survival against increased reproduction, the latter being the ultimate currency of evolution.

In species that reproduce sexually—that is, by mating—physical and behavioral traits can be selected for because they increase the number and/or quality of an individual's mates. This can happen in two ways: by making individuals compete more effectively with rivals, and by making individuals more attractive to potential mates. When selection arises from competition over mates, it is termed *sexual selection*. While sexual selection is a special case of natural selection, it has its own peculiar dynamics and can have dramatic effects on the evolutionary trajectory of sexual species. As Darwin noted, sexual selection can drive the evolution of extravagant displays designed to attract mates (the peacock's tail is a prototypical example). Even more importantly, sexual selection may lead to remarkable divergence in form (e.g., size, color, natural weapons), behavior (e.g., aggression, sexual behavior, parental behavior), and development (e.g., growth rate, maturation timing, life span) between males and females of the same species.

Adaptation. By constantly weeding out unsuccessful variation, natural selection produces incremental modifications in existing phenotypes, leading to an accumulation of characteristics that are organized to enhance survival and reproductive success. These characteristics are termed *adaptations*. Adaptations are inherited and reliably developing characteristics that have been selected for because of their causal role in enhancing the fitness of individuals that possess them (Williams, 1966). Through this process, adaptations acquire biological functions and the appearance of purposeful design—they are *for* something. The immune system functions to protect organisms from pathogens, the heart functions as a blood pump, and the cryptic coloring of many insects has the function of preventing their detection by predators. The core idea of evolutionary psychology is that many psychological characteristics are adaptations—just as many physical characteristics are—and that the principles of evolutionary biology that are used to explain our bodies are equally applicable to our minds (for extended discussion see Durrant & Ellis, 2003).

While adaptations are the product of evolution, evolution does not always produce adaptations; likewise, not every characteristic of an organism is an adaptation. For example, traits may become fixated in a population by random drift, whereby neutral or even deleterious characteristics become more prevalent due to chance factors. A neutral or weakly maladaptive trait may also spread because it is developmentally or genetically linked to another, positively

selected trait (hitchhiking). In addition, many traits are not adaptations in themselves but rather by-products of other adaptations. The sound that hearts make when they beat, the white color of bones, and the human chin are all nonfunctional by-products of natural selection. Finally, random variation in traits can be maintained as residual noise, as long as it is selectively neutral. A variety of approaches can be employed—preferably in combination—to identify adaptations. They include making phylogenetic comparisons, directly measuring the fitness benefits of a trait, and building mathematical models of evolution. An especially useful method is looking for evidence of *special design*: economy, efficiency, complexity, precision, specialization, and reliability in service of a specific biological function (Williams, 1966).

The logic of adaptation has an important consequence: as evolution proceeds, individual organisms are selected to develop and behave in a way that maximizes their expected fitness. This optimization principle can be aptly described as the *individual-as-maximizing agent* analogy (Grafen, 1999), and is a critical component of the adaptationist approach in biology. Of course, the analogy does not imply that a given individual will *always* obtain high fitness; of equal importance, optimization does not by any means imply unconstrained "perfection"—fitness maximization always takes place within the constraints and trade-offs imposed by the physical and social environment, as well as those imposed by previous phenotypic evolution and entrenched developmental biases (more on this below). Finally, there is no assumption that individuals are intentionally or consciously maximizing expected fitness—they only tend to function *as if* they were attempting to do so.

Inclusive fitness. The basic account of adaptation given in the preceding paragraphs has an important limit: it utterly fails to explain altruistic traits—that is, physical and behavioral traits that reduce an individual's reproductive success while increasing that of another individual. However, altruism is widespread in nature, as strikingly illustrated by the sterile castes found in eusocial insects such as ants, bees, and wasps. In order to solve this puzzle, William Hamilton (1964) developed *inclusive fitness theory*, also known (somewhat improperly) as *kin selection theory*. Inclusive fitness theory started a revolution in evolutionary biology, and provided the first unified explanation of social behavior—from parental care and family dynamics to altruism and self-sacrifice in groups. Today, inclusive fitness theory is the bedrock of the study of social evolution, from bacteria to humans (see Grafen, 2009; West, Griffin & Gardner, 2007).

In a nutshell, the theory shows that what is maximized by natural selection is not individual fitness, but a different quantity termed *inclusive fitness*. Inclusive fitness is the sum of (a) an individual's contribution to its own reproductive success, and (b) the individual's contribution to the reproductive success of other individuals, weighted by a coefficient of relatedness. Relatedness is an index of genetic similarity, ranging from $r = 1$ between two genetic clones (for example identical twins) and $r = 0$ between two unrelated individuals. In simplified terms, the relatedness between parent and child ($r = 0.5$) is the same as that between two full siblings; while that between grandparent and grandchild ($r = 0.25$) is the same as that between two half-siblings; and so on[1]. Inclusive fitness theory is encapsulated by Hamilton's rule:

$$rb > c$$

The rule states that an actor's behavior (or any other phenotypic trait with social effects) will be selected for if the fitness benefit b enjoyed by the recipient, weighted by the relatedness r

[1] More precisely, relatedness is a regression coefficient that predicts the recipient's genotype from the actor's genotype. Relatedness can become negative if two individuals can be expected to be genetically *less* similar than two randomly selected members of the population (see Grafen, 2009; West et al., 2007).

between actor and recipient, is larger than the cost *c* incurred by the actor. Thus, costly altruistic behavior can evolve provided that the relatedness between actors and recipients is sufficiently high. The implications of inclusive fitness are not limited to altruism, and Hamilton's rule can be applied to a broad range of social dynamics, including competition and mutually beneficial cooperation (as distinct from purely altruistic behavior). Inclusive fitness theory leads to an updated version of the individual-as-maximizing agent analogy: when social interactions are involved, individuals will tend to behave as if they were maximizing their expected *inclusive* fitness (Grafen, 2006, 2009). Unlike individual fitness, maximizing one's inclusive fitness is equivalent to maximizing the replication of one's genes in future generations, since—by definition—related recipients (those for which $r > 0$) are carrying copies of the actor's own genes[2]. For this reason, inclusive fitness theory has sometimes been presented as a theory of the "selfish gene" (Dawkins, 1976)—a potentially misleading label, given its original focus on the evolution of altruism.

Inclusive fitness theory is also equivalent to *multilevel selection theory*, an approach to social evolution that focuses on group rather than individual dynamics (e.g., Sober & Wilson, 1998). While multilevel selection has often been viewed as alternative to inclusive fitness, it has since become clear that the two theories are mathematically interchangeable (see Marshall, 2011; West et al., 2007), and only differ in how they partition the costs and benefits of social traits. Whereas inclusive fitness partitions fitness effects between actors and recipients, multilevel selection partitions them between individuals and their broader social groups. Thus, in a multilevel framework, altruism toward group members (to the point of self-sacrifice or sterility) can be selected for if it is counterbalanced by an appropriate benefit to the group as a whole. The equivalent explanation in terms of inclusive fitness is that group formation mechanisms typically increase relatedness within groups relative to that between groups. Thus, helping group members leads to an indirect fitness benefit that can be so strong as to override large individual costs.

Special Metatheoretical Assumptions

Psychological mechanisms. Psychological adaptations, which govern mental and behavioral processes, are referred to by evolutionary psychologists as *psychological mechanisms*. Most research in evolutionary psychology focuses on identifying evolved psychological mechanisms because it is at this level where invariances occur. Indeed, evolutionary psychologists assert that there is a core set of universal psychological mechanisms that comprise our shared human nature (see Buss, 2005). The move to the level of psychological mechanisms is important to avoid a common fallacy—that of assuming that human behavior (a) has the conscious goal of maximizing inclusive fitness, and/or (b) actually maximizes inclusive fitness in current environments. At a very general level, natural selection does tend to produce organisms that behave as if they were trying to maximize their expected fitness (see above). However, actual behavior is ultimately mediated by a host of psychological mechanisms with local and sometimes conflicting goals (e.g., learning a language, finding and attracting mates, choosing food, avoiding diseases). There is no general "fitness maximization mechanism" anywhere in the brain. Each mechanism works and evolves within constraints (e.g., information availability, time constraints,

[2] The existence of epigenetic inheritance does not fundamentally change this picture. If epigenetic markings are reliably transmitted across generations, they are equivalent to genetic alleles from the standpoint of natural selection. If epigenetic markings are reversible and environmentally induced, they mediate short-term developmental plasticity and are irrelevant to inclusive fitness computations (see Shea, Pen & Uller, 2011).

coordination and conflict with other mechanisms, previous evolutionary history); as a result, the overall structure of the mind/brain is more akin to a gerrymandered contraption than an optimal, omniscient decision maker.

Even more importantly, the fact that a given adaptation was produced through differential reproduction does not imply that either (a) selection is *currently* favoring that adaptation or (b) variation in the expression of that adaptation will be associated with *current* reproductive success. For example, the dopamine-mediated reward mechanisms found in the mesolimbic system in the brain appear to have evolved to provide a pleasurable reward in the presence of adaptively relevant stimuli such as food or sex. In contemporary environments, however, these same mechanisms are subverted by the use of psychoactive drugs such as cocaine and amphetamines, which deliver huge dollops of pleasurable reward in the absence of the adaptively relevant stimuli, often to the user's detriment (Durrant & Ellis, 2003).

The concept of a psychological mechanism was updated by Bjorklund and colleagues (2007) to make it more consistent with EDP's metatheoretical assumption of probabilistic epigenesis (see below). These authors proposed a definition of *evolved probabilistic cognitive mechanisms*, that is,

> [psychological] mechanisms that are functionally organized to solve recurrent problems faced by ancestral populations, are highly probable when species-typical environments are encountered (i.e., when the developmentally relevant features of the environment are in the range typically encountered during a species' evolution), and are products of emerging developmental systems that have evolved over the course of the ontogenies of our ancestors (Bjorklund et al., 2007, p. 22).

This definition stresses the probabilistic nature of the ontogenetic processes responsible for building psychological mechanisms; it also makes it clear that, while evolved mechanisms prepare an organism for life in a species-typical environment, they are not "preformed" or specified in advance by a rigid genetic program.

Domain specificity. As is apparent from the preceding paragraphs, evolutionary psychology views psychological mechanisms as having some degree of functional specialization. More specifically, psychological mechanisms are composed of structures that (a) exist in the form they do because they recurrently solved specific problems of survival and reproduction over evolutionary history; (b) are designed to take only certain kinds of information from the world as input; (c) process that information according to a specific set of rules and procedures; (d) generate output in terms of information to other psychological mechanisms and physiological activity or manifest behavior that is directed at solving specific adaptive problems (see Buss, 2012). In short, psychological mechanisms are designed by selection to address specific *domains* of the physical and social world. Although evolutionary psychologists assert that the mind is not comprised primarily of content-free (domain-general) psychological mechanisms, it is likely that different mechanisms differ in their levels of specificity, and that there are some higher-level mechanisms that function to integrate information across more specific lower-level mechanisms. In addition, some general-purpose abilities (e.g., associative learning) may be co-opted in the context of different specialized functions. It is important to stress that functional specialization of a psychological mechanism does not imply clear-cut anatomical localization in the brain, nor complete functional independence from other mechanisms. Indeed, psychological mechanisms are expected to show a considerable degree of integration and reciprocal interaction.

The rationale behind the domain specificity argument is fairly straightforward: What counts as adaptive behavior differs markedly from domain to domain. The sort of adaptive problems posed by food choice, mate choice, and social exchange often require different kinds of solutions. A clear analogy can be drawn with the functional division of labor in human physiology. Different organs have evolved to serve different functions and possess properties that allow them to fulfill those functions efficiently, reliably, and economically: the heart pumps blood, the kidneys excrete urine, and so on. A super, all-purpose, domain-general internal organ faces the impossible task of serving multiple, incompatible functions. Analogously, a super, all-purpose, domain-general mind/brain mechanism faces the impossible task of efficiently and reliably solving the plethora of behavioral problems encountered by humans in ancestral environments. Thus, neither an all-purpose physiological organ nor an all-purpose psychological mechanism is likely to evolve.

Environment of Evolutionary Adaptedness (EEA). Biological adaptation is necessarily a historical concept, and all claims about adaptation are claims about the past. The environment in which a given trait evolved is termed its *environment of evolutionary adaptedness* (EEA). When we claim that the thick insulating coat of the polar bear is as an adaptation, we are claiming that possession of that trait advanced reproductive success in ancestral environments. However, traits that served adaptive functions and thus were selected for in past environments may not still be adaptive in present or future environments. In a globally warmed world, for example, the polar bear's pelt may become a handicap that reduces the fitness of its owner. While natural selection is expected—all else being equal—to weed out traits that have become detrimental to fitness, the process may often take a long time. This generates the potential for mismatch between an organism's adaptations and its present environment.

The possibility of mismatch raises a subtle but crucial point regarding the meaning of "adaptive." Broadly speaking, psychological and physiological processes can be described as adaptive if they result from the unimpaired functioning of adaptations. Thus, adaptive in the broad sense is a shorthand to describe the functioning of naturally selected processes and mechanisms, regardless of whether they are *currently* promoting reproductive success (i.e., adaptive in the narrow sense). For example, pursuit of mating relationships with fertile partners is guided by adaptive psychological processes, regardless of whether contraceptive technology prevents reproduction in present-day societies.

Within the same organism, different adaptations will often have different EEAs (for extended discussion see Durrant & Ellis, 2003). Consider the human adaptations of language and infant attachment. While the origin of language is firmly anchored in approximately the last 2 million years, infant attachment reflects a much lengthier evolutionary history and a shared heritage with other mammalian and primate species. While evolutionary timing helps define the EEA of a trait, the EEA itself is not a specific time or place; rather, EEAs capture the statistical regularities of the environment in which the trait evolved (Tooby & Cosmides, 1990). Environmental variation itself can be part of an EEA; for example, metabolic processes can evolve so as to maximize survival in an unpredictable environment, whereby food abundance is suddenly followed by starvation. In this case, metabolic adaptations evolve in an EEA characterized by a consistent pattern of unpredictable variation.

Over the last few millennia—the span of a few hundred generations—humans have experienced rapid and constantly accelerating rate of change in health, nutritional, social, and technological conditions. While genetic evolution has been accelerating as well (e.g., Hawks,

Wang, Cochran, Harpending & Moyzis, 2007), many of our evolved adaptations can be expected to be at least partly mismatched to modern lifestyles. At the same time, many adaptation-relevant aspects of our environment have probably remained the same: humans everywhere, for example, still find and attract mates, have sex, raise families, make friends, compete for status, gossip, and so forth (Crawford, 1998). Most important is that current and ancestral environments do not have to be identical in every respect to sustain the normal development and expression of evolved psychological mechanisms.

Probabilistic epigenesis. The concept of probabilistic epigenesis has a long history in embryology and is one of the central assumptions of DST (see Gottlieb, 2007). Probabilistic epigenesis holds that development involves continuous bidirectional influences between genetic activity, neural activity, behavior, and the physical and social environment (similar interactions take place in the development of non-neural mechanisms). In this view, neural structures begin to function when they are still developing, and their activity—both spontaneous and evoked by the environment—plays an important role in the ontogenetic process. This is contrasted with "predetermined" models in which genetic programs build neural structures, that only begin to function and interact with the environment when they are mature. The reciprocal, bidirectional interaction between multiple levels introduces a probabilistic element in the outcomes of developmental processes.

A key implication of probabilistic epigenesis is that genetic activity is influenced and regulated by neural, behavioral, and external events. Gene x environment (GxE) interactions in development, whereby the effects of an allelic variant are contingent on contextual variables, are prime examples of probabilistic epigenesis (Gottlieb, 2007). Probabilistic epigenesis provides reasons for expecting widespread plasticity in the outcomes of developmental processes; however, it is not sufficient to explain *adaptive* plasticity and phenotype-environment matching (Bjorklund et al., 2007). Understanding adaptive plasticity requires a synthesis between the proximate and ultimate level of analysis—where development meets adaptation.

Developmental Systems Theory: An Alternative Metatheory?

Researchers in developmental psychopathology often refer to developmental systems theory (DST) as a metatheoretical framework for the discipline. DST is a general approach to development and evolution rooted in the organismic concepts of embryology and developmental psychobiology. The major themes of DST are probabilistic epigenesis and developmental plasticity, with a strong emphasis on bidirectional interplay between genes and environment; an extended view of inheritance that goes beyond DNA to include epigenetic processes, cellular structures, scaffolded developmental environments (e.g., nests), and culturally transmitted information; and a view of the developing organism as actively involved in shaping its environment (see Griffiths & Gray, 2004; Oyama et al., 2001). Consistent with the metatheoretical framework we have presented, DST emphasizes the multiplicity of factors that jointly determine phenotypic outcomes and stresses the contextual, contingent nature of development.

Soft versus Hard DST

Much of the difficulty in discussing the role of DST stems from the fact that DST is not a single, unified theory; in fact, it is possible to recognize at least two versions of DST—a "soft"

version and a "hard" version—with vastly different implications for developmental science (Frankenhuis et al., 2013; Robert et al., 2001). Soft DST is essentially a theory of development; in this view, a developmental system comprises all the "resources" (e.g., genes, cellular structures, sensory experiences, physical parameters of the environment) that contribute to the ontogeny of the individual organism. However, the organism remains the main biological entity, and evolutionary processes acts on populations of organisms. In other words, soft DST reconceptualizes the causal structure of development—for example by placing genetic inheritance in a broader perspective and emphasizing bidirectional effects—but is otherwise consistent with inclusive fitness theory and the logic of individual adaptation (Pradeu, 2010). Indeed, many developmentally-oriented extensions of evolutionary biology already incorporate the main tenets of soft DST (e.g., West-Eberhard, 2003).

In contrast, hard DST is not so much a theory of development as a radical alternative to mainstream evolutionary theory. In hard DST, a developmental system comprises all the resources that produce the developmental outcomes that are *stably replicated* in that lineage. As a consequence, it is impossible to meaningfully distinguish between organism and environment, and what evolves are not populations of organisms but populations of replicating "organism-environment" systems. Such holistic reconceptualization of natural selection breaks down the individual-as-maximizing agent analogy, and makes adaptationist analysis all but impossible (Pradeu, 2010). This is because hard DST is inconsistent with inclusive fitness theory: selection is no longer assumed to act on individuals that can be more or less genetically related with one another, but on whole developmental systems (comprising every recurring influence on development, including social and bio-geographical factors) for which there is no meaningful definition of reproductive success or relatedness. In addition, hard DST does not only object to the concept of genetic programs, but—in a further break from mainstream biology—rejects the very idea that genes store *information* as unacceptable preformationism (Oyama et al., 2001).

In summary, DST comprises two related but partially distinct approaches. Soft DST is a developmentally oriented extension of mainstream evolutionary theory, and is fully consistent with the metatheoretical framework of EDP. In contrast, hard DST advances a radically novel theory of evolution, and constitutes an alternative metatheoretical framework with little overlap with that of EDP. Embracing soft DST does not commit one to also adopt the assumptions of hard DST. Unfortunately, the distinction between the soft and hard version of DST is often obscured in the literature, leaving many researchers confused as to the exact implications of the theory (see Frankenhuis et al., 2013; Pradeu, 2010).

Implications for Developmental Science

The distinction between soft and hard DST provides insight in the current theoretical status of developmental psychology and psychopathology. We surmise that, when developmental scientists embrace a DST perspective, they usually reason in terms of *soft* DST. However—and possibly without realizing it—they end up adopting the whole metatheoretical package of hard DST, with the added baggage of anti-adaptationism and a priori rejection of mainstream evolutionary thinking. As a result, developmental science is deprived of some of the most powerful tools in biology, such as inclusive fitness theory and the concept of adaptation. By contrast, we contend that the metatheoretical framework of EDP—a synthesis of adaptationism and soft DST—provides a suitable evolutionary foundation for developmental psychopathology.

In the remainder of the chapter we demonstrate the heuristic and integrative power of this approach.

Beyond Pathology: Adaptation, Maladaptation, and Disorders

In an evolutionary framework, the terms *adaptive* and *maladaptive* denote the effect of a trait or behavior on biological fitness. From the standpoint of the individual organism, adaptive traits are those that enhance inclusive fitness compared with potential alternatives. However, all adaptations have fitness costs as well as benefits; in order to be adaptive a trait does not have to be cost-free, but only to yield a positive overall contribution to the organism's fitness. This notion of adaptation and maladaptation contrasts sharply with how the same terms are usually employed in developmental psychology and psychopathology. In these disciplines, "adaptive" traits and behaviors are those that promote health, safety, subjective well-being, and mutually rewarding social relations. Socially undesirable, aversive, or health-damaging traits are viewed as "maladaptive". These definitions of adaptation and maladaptation are conceptually orthogonal and ought to be carefully differentiated. In this chapter we always refer to adaptation and maladaptation in the biological sense, and employ the terms *desirable* and *undesirable* to denote the implications of a trait for health, safety, well-being, and social values.

Unsettling as it may be, the logic of natural selection promotes reproductive success, not happiness or health (see Cosmides & Tooby 1999; Gluckman et al. 2011; Nesse, 2004a). Thus, biologically adaptive traits may or may not be socially desirable or conducive to health and well-being; conversely, a trait is not maladaptive just because it has negative effects on an individual's welfare. Traits that consistently reduce well-being and adversely impact an individual's health can be selected for as long as they result in enhanced reproduction—a highly counterintuitive notion in mainstream psychology. At the same time, adaptiveness and desirability—though conceptually distinct—are functionally connected to some degree. This is because positive emotions such as joy, excitement, and pride and are generally aroused by the fulfillment of fitness-enhancing goals, while threats to fitness are generally met with negative feelings such as sadness, anger, and shame (Nesse, 2004a).

The functional connection between threats to fitness and negative emotions lends intuitive plausibility to the implicit assumption—firmly entrenched in psychopathology and psychiatry—that aversive traits are by default pathological or "maladaptive" (see Nesse & Jackson, 2006). The evolutionary approach challenges this assumption, and unpacks the intuitive concept of disorder by separating adaptation from health and desirability. The result is a general framework for thinking about pathology that can be applied to both medical and psychopathological conditions.

What is a Disorder?

"Mental disorder" is a central concept of psychopathology, yet a satisfactory definition of disorder is notoriously difficult to achieve. In an influential paper, Jerome Wakefield (1992) built on previous biologically informed approaches to advance a definition of disorder as a *harmful dysfunction*. According to this definition, conditions are recognized as disorders when they (a) are caused by the failure of a biological mechanism to perform its evolved function, and (b) inflict

some harm or damage on the affected person, as judged by sociocultural standards. This is a hybrid definition that combines the objective dysfunction criterion with the subjective, culturally bound harm criterion. In Wakefield's account, people evaluate a condition as a disorder when the subjective perception of harm or undesirability is coupled with the idea that something in the body and/or mind is not working properly. In line with an evolutionary approach, what constitutes "proper" functioning of a biological mechanism can be correctly evaluated only by considering the evolved function(s) of that mechanism. In order to understand pathology, one needs to understand the function of the relevant biological mechanisms, as well as the structure of the environment in which they evolved (Nesse, 2001; Troisi & McGuire, 2002).

We believe that, correctly understood, the harmful dysfunction analysis is a useful heuristic for reasoning about pathology and disorders. Although Wakefield's proposal has been hotly debated, its core propositions have withstood criticism (see Wakefield, 1999, 2011). To avoid common misgivings, the following points should be kept in mind. First, evolved mechanisms are defined broadly; major organs like the heart are mechanisms, but so are specialized brain areas, microscopic cellular structures, and biochemical pathways. Accordingly, dysfunctions can occur in many ways and for a wide variety of causes (e.g., deleterious genetic mutations, pathogen infections, injuries and wounds, side effects of evolved defenses). Second, *dysfunctional* is not synonymous with *maladaptive*. Since dysfunctions interfere with evolved design, they can often be expected to reduce an individual's fitness; however, a reduction in fitness is not required to identify a dysfunction. It is quite possible for a dysfunction to be selectively neutral—for example because it occurs too late in life to impact an individual's reproductive success, or because changes in the environment reduce its damaging effects. This is why myopia—a failure of the crystalline lens to project a focused image on the retina—remains a dysfunction even if glasses and contact lenses eliminate its negative effects on survival. Third, the concept of dysfunction is a fuzzy one rather than all-or-none, and evolved mechanisms can show varying degrees of functionality (Wakefield, 1999). Thus, obvious instances of dysfunction are going to be surrounded by borderline cases for which there is no clear-cut demarcating criterion—as for example in the case of hypertension, extreme variation in height, and personality disorders.

A Taxonomy of Undesirable Conditions

Despite their theoretical significance, harmful dysfunctions are only a fraction of what people regard as diagnosable problems and/or seek treatment for (Cosmides & Tooby, 1999; Lilienfeld & Marino, 1999). Fever is an evolved defense against pathogens; with rare exceptions, it reflects a well-functioning system rather than a dysfunction—yet it is often treated with drugs. More generally, conditions that are not harmful dysfunctions in Wakefield's sense may nevertheless be labeled and treated as disorders, especially if their etiology and functional implications are incompletely understood. For example, it has been hypothesized that some forms of psychopathy are adaptive behavioral phenotypes that exist at a low frequency and thrive by exploiting others (e.g., Mealey, 1995). If this hypothesis were correct, a number of apparent dysfunctions (e.g., reduced empathy, lack of guilt, impulsivity) would be better understood as design features of the psychopathic strategy. Still, psychopathy is a source of trouble for society at large, and would be legitimately regarded as a condition in need of treatment even if it were established as a biologically adaptive variant rather than a "disorder" in the strict sense. This example also illustrates how conflicts of interest between social actors modulate the perception

and definition of a problematic condition; obsessional jealousy may appear desirable and useful to the affected individual, but harmful and undesirable to his/her partners (for a detailed analysis of this issue see Cosmides & Tooby, 1999).

We now take a wider perspective and consider the many ways in which evolutionary and developmental processes may result in undesirable conditions, including—but not limited to—harmful dysfunctions in the narrow sense (see Cosmides & Tooby, 1999; Gluckman et al., 2011). The taxonomy we present combines Wakefield's dysfunction criterion with the effects of a given condition on biological fitness (Figure 1). When considering the adaptiveness of a condition, we further distinguish between the fitness contribution of a *trait* or *mechanism*—averaged across all the individuals who express it—and the fitness of a *particular individual.* The distinction is useful because a mechanism may be fitness-enhancing *on average*, while imposing fitness costs on some individuals (e.g., Cosmides & Tooby, 1999). Distinguishing between individual and average fitness permits a fine-grained analysis of the interplay between adaptation and maladaptation in psychopathology (Frankenhuis & Del Giudice, 2012). Although we discuss them separately, the following categories are not mutually exclusive; a given condition or class of conditions may well reflect the interplay of multiple factors and require overlapping evolutionary explanations.

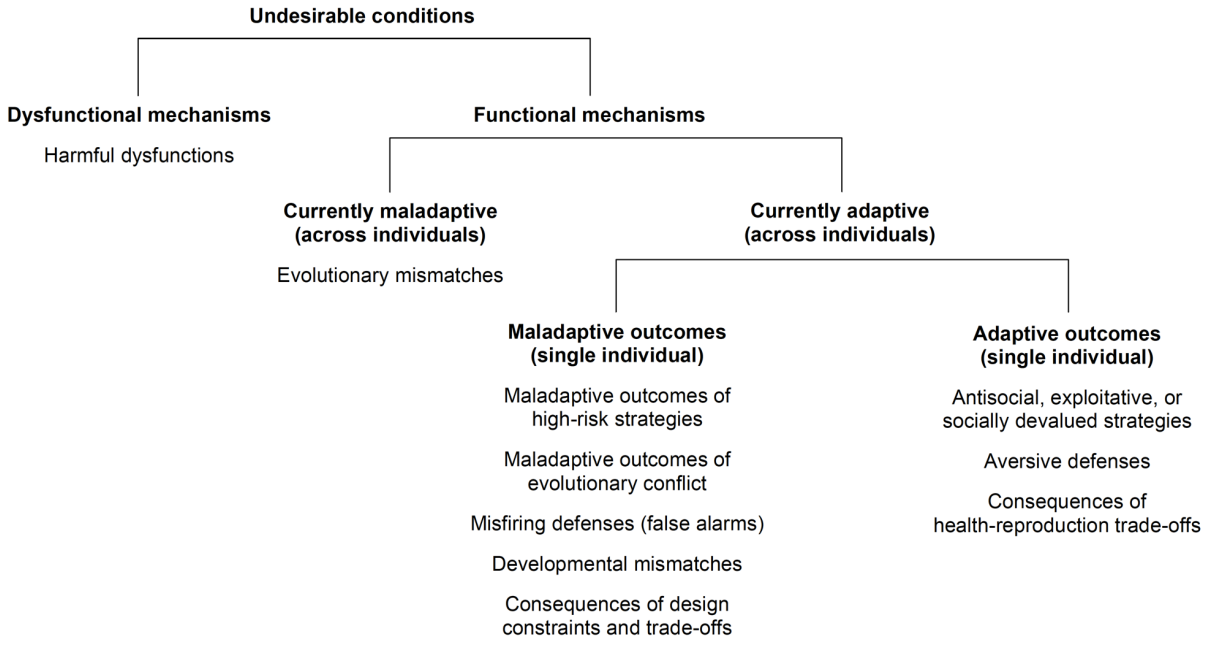

Figure 1. An evolutionary taxonomy of undesirable conditions.

Harmful Dysfunctions

All biological and artificial mechanisms—no matter how well designed—are vulnerable to malfunctions, failures, and breakdowns. Developmental pathways typically evolve canalization

properties (e.g., biochemical buffering mechanisms) that confer them robustness against accidents and perturbations. However, the accumulation of such events over time can affect development, resulting in random deviations from the target phenotype (*developmental instability*). More dramatically, an evolved mechanism may cease to perform its functions because of accidents or environmental insults beyond its regulatory capacity (e.g., brain injury, exposure to toxins), deleterious genetic/epigenetic changes (e.g., mutations and deletions), and attacks or manipulations by pathogens (see Crespi, 2000, 2010).

New deleterious mutations arise at every generation and they may be passed down to descendants, persisting for some time in a population until they are weeded out by natural selection. Harmful variants may be especially difficult to eliminate if they have recessive effects (i.e., they are only expressed when an individual inherits two copies of the same allele). The continuous process of creation and elimination of deleterious mutations is called *mutation-selection balance*; its dynamics determine the frequency and persistence of harmful variants in a population. Sometimes, a single mutation in a critical pathway is sufficient to cause a disorder; more often, disorders may result from the cumulative effect of many slightly deleterious mutations (*mutation load*), each with a small impact on phenotypic function. Mutation-selection balance has been proposed as a likely explanation for the persistence of common, heritable, and harmful mental disorders like autism, schizophrenia, bipolar disorder, and mental retardation (Keller & Miller, 2006). Since a large proportion of human genes are expressed in brain development, the likelihood that mutation load will have negative consequences on mental functioning is especially high. The role of mutation load in autism and schizophrenia is consistent with the high rate of new mutations in people with these disorders (e.g., Sanders et al., 2012).

Exposure to pathogens (harmful viruses, bacteria, and other parasites) is another common cause of biological dysfunction. Infectious diseases—especially when they occur in early development—have been associated with increased risk for a number of mental disorders including autism, schizophrenia, and depression (see Patterson, 2011; Benros, Mortensen & Eaton, 2012). The role of pathogens in the etiology of mental disorders does not contradict that of genetic mutations. Infections, like mutations, can perturb developmental processes at critical stages; accordingly, mutation load and pathogen load may ultimately converge on the same neurobiological pathways and exert a cumulative effect on the risk for psychopathology. In addition to their direct effects on individual organisms, pathogens may indirectly contribute to the risk of harmful dysfunctions through their effect on the evolution of defenses. Pathogens and hosts are constantly involved in coevolutionary "arms races", so that for every improvement in defensive mechanisms, new means of offense are going to be selected for on the other side (and vice versa). Coevolutionary arms races tend to produce increasingly complex offense/defense mechanisms (consider the intricacy of the immune system); in turn, increased complexity may render those mechanisms more vulnerable to failures and dysfunctions (Nesse, 2001).

Evolutionary Mismatches

Because of natural selection, evolving organisms tend to become progressively more successful at surviving and reproducing in their environment, broadly conceived to include not only physical factors but also social relations with conspecifics as well as interactions with other species (predators, prey, pathogens, and so forth). The environment, however, is not a static background: environments change all the time because of external events (e.g., geological change), social evolution within a species (e.g., increased population density), and coevolutionary

processes between species (e.g., new pathogens). When environmental changes are rapid and extensive, previously adaptive mechanisms may suddenly become maladaptive and generate all sorts of unintended and/or undesirable consequences. Thanks to cultural and technological evolution, humans have gained an unprecedented power to alter their social and physical environment, and in so doing have created enormous opportunity for evolutionary mismatch.

Evolutionary mismatch occurs when an organism encounters a *novel environmental context* (outside of the range that was recurrently encountered over its evolutionary history) that *disrupts normal development* and/or *impairs adult functioning*. Evolutionary mismatches are likely to be implicated—to various degrees—in the etiology of mental disorders. In modern societies, for example, the media expose girls and women to a relentless stream of images of unrealistically attractive "competitors"—an artificial, evolutionarily novel kind of social stimulus. It has been hypothesized that such exposure hyper-activates the evolved mechanisms that regulate female competition for attractiveness and status, contributing to the rising incidence of eating disorders (e.g., Abed, 1998). Other instances of potential mismatch are less obvious. For example, sanitation in developed countries determines a lack of exposure to common microorganisms ("old friends") during development. These novel hygienic conditions appear to interfere with the early ontogenetic processes that train the immune system and set its overall functioning parameters. The resulting states of chronic inflammation may increase the risk for a range of physical and mental disorders, especially depression (Raison, Lowry & Rook, 2010).

Although we have emphasized its negative consequences, evolutionary mismatch is an unavoidable and often vital aspect of evolution. By definition, all evolving organisms exhibit some degree of mismatch to their present environment—otherwise they would stop evolving altogether. The very process of adaptation generates subtle forms of mismatch that may contribute to the etiology of undesirable conditions. When a trait has been subjected to strong recent selection, the resulting adaptive changes may co-occur with maladaptive side effects on other traits that are genetically and/or developmentally linked to the selected trait. Similarly, recently evolved adaptations are likely to show increased scope for dysregulation because they have yet to be "fine-tuned" by natural selection (see Crespi, 2010).

Maladaptive Outcomes of Adaptive Mechanisms

So far, we have reviewed case in which undesirable conditions are caused by failures of evolved design. Harmful dysfunctions occur when a biological mechanism fails to perform its evolved functions; conversely, evolutionary mismatches occur when an intact mechanism becomes maladaptive because of novel environmental conditions. However, maladaptive outcomes at the individual level may systematically occur even when adaptive mechanisms perform their evolved functions in an environment that matches the EEA on the relevant dimensions. This is one of the central insights of evolutionary psychopathology: observing maladaptive outcomes at the individual level is not sufficient to infer maladaptation at the level of evolved mechanisms. We now review some important reasons why adaptive mechanisms may systematically yield maladaptive outcomes (for in-depth discussion see Cosmides & Tooby, 1999; Crespi, 2010; Frankenhuis & Del Giudice, 2012).

Maladaptive outcomes of high-risk strategies. An important source of maladaptation at the individual level is the evolution of risky adaptive strategies[3]. Risky behavior is part and parcel of daily life: many activities that contribute to survival and reproduction also increase the probability of harm, injury, loss, or death. Searching for food and competing for mates are both fraught with danger, but potential dangers are compensated by the potential fitness advantages of these activities. From an evolutionary perspective, we would expect natural selection to favor mechanisms that produce risk-taking when the fitness benefits outweigh the costs. Further insight in the dynamics of risky strategies can be gained by defining risk in its technical sense of *unpredictable variation in outcomes* (see Frankenhuis & Del Giudice, 2012; Ellis et al., 2012). Whereas some behavioral decisions offer a narrow range of possible outcomes (low-risk), others entail widely variable outcomes (high-risk), with the potential for large gains as well as large losses. Consider a predator that can choose between two types of prey: larger and hard-to-catch animals versus smaller and easily caught ones. Imagine also that the expected energetic returns associated with hunting each prey type are identical: one results in a high reward with a low probability, the other in a low reward with a high probability. In this scenario, hunting larger prey qualifies as more risky, because it entails more variable outcomes.

Broadly speaking, natural selection favors risk aversion when the relationship between behavioral outcomes and fitness is characterized by diminishing returns. For instance, a well-fed animal should look for low-risk food items (or not forage at all) when additional calories only slightly improve its condition. Conversely, when better outcomes yield increasing fitness returns, organisms may become risk-prone. For example, an animal on the brink of starvation may choose to forage in a nutrient-rich habitat, even if it is infested with predators, because it has so much to gain from additional calories (discussed in Frankenhuis & Del Giudice, 2012). An analogous logic applies to competition for reproduction. In some mating systems, reproductive benefits are highly skewed towards top-ranking individuals (i.e., "winner takes all" systems). In such conditions, males are intensely selected to compete for top rank, even if this implies a greater risk of injury; for instance, male elephant seals engage in ferocious fights that often cause harm and sometimes result in death. Still, males benefit—*on average*—from participating in fights because not participating implies being shut out from reproduction.

The logic of risky strategies can shed light on the interplay between adaptation and maladaptation in development and psychopathology. For example, externalizing behavior can be interpreted as a high-risk tactic of social competition (Del Giudice et al., 2011; Ellis et al., 2012; Martel, 2013). In some cases, aggressive children and adolescents become dominant, respected, and popular leaders in their peer groups; in other cases, they do not succeed and become unpopular or rejected, incurring physical and psychological harm. These outcomes can be individually maladaptive even if they result from an adaptive strategy designed to achieve dominance and social status. A similar logic may apply to schizotypal personality traits (e.g., the tendency to experience unusual perceptions, bizarre ideation, and reference thoughts). While schizotypal traits increase the risk of schizophrenia (a severe, harmful disorder), when schizotypal individuals do not develop a disorder, their enhanced creativity may facilitate high

[3] In an evolutionary framework, the term "strategy" denotes an organism's realized phenotype among a set of possible phenotypes. Adoption of a given strategy can depend on both environmental and genetic factors. It should be stressed that the term does not imply conscious planning, deliberation, or even awareness; an organism's "choice" between alternative strategies can be implemented by low-level physiological means, such as a hormonal switch or a change in genetic expression.

mating success (e.g., Nettle, 2001; reviewed in Del Giudice, Angeleri, Brizio & Elena, 2010); this would amount to a high-risk strategy with widely variable outcomes.

Maladaptive outcomes of evolutionary conflict. Conflicts of interest between individuals abound in nature—for example between mating rivals, or between dominants and subordinates in a hierarchy. While cooperation and even altruism can be favored by natural selection, it is often the case that a given individual can maximize its own fitness only at the expense of another individual's reproduction. An especially intriguing kind of evolutionary conflict is that between parents and their offspring (Trivers, 1974; see Schlomer, Del Giudice & Ellis, 2011). While a parent is equally related to all its offspring ($r = 0.5$), each offspring is more related to itself ($r = 1$) than to any of its present or future siblings ($r = 0.5$ in the case of full siblings). Offspring are thus selected to demand more than their "fair share" of their parent's investment in time, food, protection; conversely, parents are selected to resist such attempts, setting the stage for parent-offspring conflict over the distribution of parental investment. Of course, parents and offspring also have a lot of evolutionary interests in common, so conflict is tempered with a substantial amount of cooperation and altruism.

Although parent-offspring conflict is not maladaptive in itself—parents and offspring are both acting so as to maximize their own fitness—the dynamics of conflict often result in nontrivial costs for both parties. Furthermore, conflict may occasionally escalate to dangerous levels, yielding maladaptive outcomes for the parent, the offspring, or both. A dramatic example is provided by prenatal conflicts about fetal nutrition (Haig, 1993; reviewed in Schlomer et al., 2011). During pregnancy, the placenta—a fetal organ that only expresses the fetal DNA—releases massive amounts of hormones in the maternal bloodstream. These hormones affect maternal metabolism so as raise the nutrient content of maternal blood and increase the supply of blood to fetal circulation. The interplay between fetal hormones and maternal countermeasures may produce a range of undesirable side effects, including gestational hypertension and gestational diabetes. In rare cases, the physiological "tug-of-war" between mother and fetus may become dysregulated and result in life-threatening conditions such as pre-eclampsia (severe maternal hypertension). In a recent paper, one of us (Del Giudice, 2012) speculated that a similar conflict may arise about fetal exposure to maternal stress hormones, with mothers favoring higher levels of exposure than fetuses. Indeed, several puzzling features of stress regulation in pregnancy could be explained by the interplay between fetal attempts at manipulation and maternal countermeasures (for a detailed exposition see Del Giudice, 2012). Elevated prenatal stress has been associated with increased risk for a broad range of psychopathological outcomes in children—including anxiety, hyperactivity, autism, and schizophrenia (reviewed in Glover, 2011). To some extent, these undesirable outcomes may arise as maladaptive side effects of parent-offspring conflict in pregnancy.

Evolutionary conflict usually takes place between different individuals, but this is not always the case. Indeed, conflicts of interest can also arise between different genes within the same individual (*intragenomic conflict*; for a thorough review see Burt & Trivers, 2006). Intragenomic conflicts may involve sexual chromosomes, mitochondrial genes, or "selfish" strands of DNA that—for various reasons—follow inheritance rules that differ from those of the rest of the genome. Most relevant to the present discussion, the maternally and paternally inherited halves of an individual's genome may have divergent fitness interests when parental investment is involved. In species that are not perfectly monogamous—that is, most sexually reproducing species including humans—the occurrence of multiple paternity increases the chance that siblings in the same family have the same mother but different fathers. As a result, the genes

inherited from the father are—on average—less strongly related to those of one's siblings than the genes inherited from the mother. This generates complex dynamics in which maternal and paternal genes may favor opposite traits in the offspring (e.g., maternal genes may benefit from less demanding offspring, while paternal genes may benefit from *more* demanding offspring; see Schlomer et al., 2011). This latent conflict between the paternal and maternal genome is played out by *imprinted genes*, that is, genes that are differentially expressed depending on whether they were inherited from the mother or from the father (see Burt & Trivers, 2006; Schlomer et al., 2011; Wilkins & Haig, 2003).

Not surprisingly, imprinted genes have been found to be involved in prenatal conflicts about fetal nutrition (reviewed in Schlomer et al., 2011). In addition, many imprinted genes are expressed in the brain, and parent-of-origin effects have been detected in the key signaling pathways that mediate social behavior—including the dopaminergic, serotonergic, and oxytocinergic pathways (see e.g., Davies, Lynn, Relkovic & Wilkinson, 2008). It is quite possible that intragenomic conflict between imprinted genes may contribute to the development of psychopathology. For example, Crespi and Badcock (2008) hypothesized that autistic spectrum conditions are characterized by over-expression of paternal genes, whereas psychosis spectrum conditions are characterized by over-expression of maternal genes. This "diametrical" model of autism and psychosis was revised and extended by Del Giudice and colleagues (2010) to account for non-clinical variation in autistic-like and schizotypal personality traits.

Misfiring defenses. Adaptive defenses are mechanisms designed to protect individuals from physical and/or social harm. Most negative emotions—including fear, anxiety, disgust, and shame—can be conceptualized as defensive mechanisms, as they play crucial protective roles against physical danger, contamination by pathogens, social exclusion, and so forth (see Nesse, 2004a; Nesse & Jackson, 2006). The calibration of defenses involves a trade-off between the rate of false negatives (failing to activate a defense mechanism when a threat is present) and that of false positives (mistakenly activating the mechanism when no threat is present). Defensive mechanisms are usually designed by natural selection to accept a high rate of false positives so as to avoid catastrophic false negatives; this is known as the *smoke detector principle* (Nesse, 2005).

The smoke detector principle suggests that defensive mechanisms will often "misfire" or activate with excessive intensity, even when no actual threat is present. Adaptive defenses—like fever, cough, and anxiety—are usually aversive and often disabling; occasionally, inappropriate activation of a defensive mechanism may cause serious harm to the individual. For this reason, misfiring defenses are a likely source of undesirable conditions, ranging from benign "false alarms" to dangerous over-reactions. The crucial point is that inappropriate activation of a defensive mechanism does not necessarily imply that the mechanism is dysfunctional or dysregulated—even optimally functional defenses may be designed to misfire from time to time. The logic of the smoke detector principle can be employed to shed light on the etiology of emotional symptoms such as panic attacks, anxiety, and phobic symptoms (Nesse, 2005; Nesse & Jackson, 2006).

Developmental mismatches. Conditional adaptation is the process by which developing organisms make use of contextual cues to direct their developmental trajectory, so as to increase the likelihood that their future phenotype will match the state of the environment. Conditional adaptation is a manifestation of adaptive plasticity, and—when successful—it can dramatically increase the reproductive success of an organism across a broad range of environments. However, the predictive accuracy (i.e., validity) of contextual cues is usually far from perfect; even when

accuracy can be improved by sampling the environment more thoroughly, the potential benefit must be balanced against the required investment of time and effort. For these reasons, conditional adaptation is a fallible process, and a proportion of individuals end up developing a mismatched phenotype. Natural selection can favor conditional adaptation even if the fitness costs of mismatch are high, as long as the average benefits of plasticity are larger than the average costs across individuals. We will deal more extensively with the costs and benefits of developmental plasticity in a later section.

Constraints and trade-offs. The design of an organism is always shaped by countless physical constraints that limit the range of phenotypic change and burden evolved adaptations with undesirable side effects. For example, the erect posture of humans necessarily increases the impact of falling; a larger body size makes organisms more vulnerable to starvation, and so forth. Physical constraints are compounded by the legacy of previous evolutionary history: natural selection builds incrementally on previous designs, and its inability to "start from scratch" introduces further constraints on adaptive design (*path dependence*). For example, the fact that human babies are delivered through the pelvic canal poses severe constraints on head size at birth; conversely, selection for larger head size at birth is the biggest ultimate source of maladaptive obstetrical complications. Equally important is the ubiquity of design trade-offs: increasing the functionality of one system may interfere with the functionality of another; increasing the efficiency of a system early in life may lead to decreased efficiency when the organism gets older; enhanced defenses against a given disease may increase vulnerability to another; and so forth. Specifically, a "risk factor" for disorder A may often protect the individual from disorder B. For example, it has been suggested that the long allele of the serotonin transporter gene promoter (*5-HTTLPR*) may offer protection against depression but increase the risk for psychopathy (Glenn, 2011).

Undesirable Adaptations

The last category in this taxonomy is also the most intriguing from an evolutionary standpoint. As we just discussed, undesirable conditions often reflect the individually maladaptive outcomes of otherwise adaptive mechanisms. However, it may also be the case that *adaptive* outcomes are perceived as undesirable conditions, or even classified as bona fide disorders (Nesse, 2004a; Nesse & Jackson, 2006). Distinguishing undesirable adaptations from maladaptive outcomes can be theoretically and empirically challenging (see Nesse, 2011), but is an essential step to correctly understand the meaning and etiology of the relevant conditions.

Antisocial, exploitative, or socially devalued strategies. In complex social species like ours there are many potential routes to reproductive success, and not all of them involve cooperation and prosociality. Individuals who develop antisocial, exploitative behavioral strategies may often reap considerable rewards—especially in harsh and unpredictable social contexts (see below). Of course, the enhanced reproductive success of (some) antisocial individuals may come at a cost to their own emotional well-being as well as the welfare of their victims. We already mentioned the hypothesis that some types of psychopathy represent an adaptive strategy of this kind; the hypothesis is supported by the robust association between psychopathic traits and a pattern of precocious sexuality, promiscuity, and sexual coercion (see Barr & Quinsey, 2004; Del Giudice, in press; Glenn, Kurzban & Raine, 2011; Mealey, 1995). A similar case has been made for borderline personality disorder, a pervasive pattern of impulsivity and emotional, affective, and relational instability that is more common in females (Brüne,

Ghiassi & Ribbert, 2010). The heterogeneous category of personality disorders is likely to include other biologically adaptive behavioral variants that are treated as problematic, for example because they cause harm or distress to an individual's social partners.

Aversive defenses. When defenses activate inappropriately and/or respond with excessive intensity, the outcome may be correctly recognized as maladaptive. However, many protective mechanisms have strongly aversive effects (e.g., vomiting, panic); for this reason, they may give rise to undesirable conditions not only when they misfire but also when they respond appropriately in presence of actual threats. Sometimes, defensive processes can be altogether mistaken for disorders, especially if their logic is incompletely understood and if the correspondence between threat and response is imperfect (because of the smoke detector principle). Indeed, the "fallacy of mistaking defenses for diseases" is a pervasive feature of current psychopathological approaches (Nesse & Jackson, 2006). Many diagnosable instances of emotional disorders—involving low mood, anxiety, and so forth—may be better understood as unpleasant but adaptive responses to contextual factors.

As already noted, distinguishing adaptive defensive reactions from maladaptive outcomes and/or dysfunctional responses is not an easy task (Nesse, 2011). This is exemplified by the debate on evolutionary models of depression. Some authors have argued that major depression can be adaptive as a mechanism of motivational disengagement from unproductive goals, signaling of social submission, and solicitation of help from family and friends (e.g., Sloman & Price, 1987; Watson & Andrews, 2002). However, while low mood has a number of crucial adaptive functions, the available evidence is more consistent with the idea that major depression usually reflects a maladaptive dysfunction of the systems involved in mood regulation (e.g., Nesse, 2006; Nettle, 2004).

Health-reproduction trade-offs. Antisocial strategies and aversive defenses do not exhaust the potential range of undesirable adaptations. The more general point is that, since natural selection maximizes fitness rather than health, traits that increase reproductive success may often have substantial health costs. For example, many health problems associated with aging are the price we pay for more efficient functioning earlier in life (See Nesse, 2001). In developmental psychology, risk-taking and impulsivity in adolescence are often viewed as dysfunctional; however, they are better explained as behavioral adaptations to the stronger mating competition faced by human males (e.g., Ellis et al., 2012; Nesse, 2001).

Implications for the Core Points of Developmental Psychopathology

The mutual interplay between *normal and pathological development* is one of the core points of developmental psychopathology. An evolutionary perspective offers a deeper understanding of how "normality" and "pathology" can be defined in the first place, and provides researchers with a conceptual toolkit for analyzing the full spectrum of undesirable conditions—from harmful and/or maladaptive dysfunctions to adaptive but undesirable mechanisms that may be erroneously mistaken for disorders. In between lies a range of explanatory categories in which adaptation and maladaptation coexist to various degrees. All too often, models in developmental psychopathology converge on "dysregulation" as the default explanation of undesirable conditions (see the next section). As we have shown here, dysregulation is only one of many potential explanations of psychopathological outcomes; a biologically informed taxonomy like the one we presented (see also Cosmides & Tooby, 1999; Nesse, 2001, 2011) can be a useful guide to formulate alternative hypotheses and build more sophisticated explanatory models.

An especially intriguing case is that of high-risk strategies characterized by unpredictable outcomes. Strategic risk provides a powerful explanation of *multifinality*, since—by definition—individual variables associated with risky strategies can be expected to predict positive outcomes in some individuals *and* negative outcomes in others. Furthermore, the outcomes of high-risk strategies are often determined in large part by chance factors, highlighting the connection between multifinality and *probabilistic causality* in developmental trajectories. A similar picture emerges if one considers the calibration of adaptive defenses and the probabilistic trade-offs involved in the balance between misfiring and appropriate responding.

An evolutionary approach also provides a nuanced view of the interplay between *risk and protective factors*—another defining point of developmental psychopathology. In particular, the logic of constraints and trade-offs suggests that some putative "risk factors" for a given condition may actually protect individuals from other (and perhaps more severe) conditions. Similarly, the logic of adaptive defenses should alert researchers to the possibility that some putative "protective factors" involving defense down-regulation may actually interfere with an individual's ability to protect itself from rare but potentially severe threats. In sum, the approach we advocate goes beyond intuitive notions of risk and resilience and contributes to draw a more realistic picture of the complex, layered relations between health and pathology.

Beyond Mental Health: Conditional Adaptation and Life History Theory

A widespread set of assumptions in developmental psychology is that children raised in supportive and well-resourced environments (e.g., who live in communities with social networks and resources for young people; who have strong ties to schools and teachers; who benefit from nurturing and supportive parenting; who are exposed to prosocial peers) tend to develop normally and express optimal trajectories and outcomes. By contrast, developmental processes among children raised in high-stress environments (e.g., who experience poverty, discrimination, low neighborhood attachment, and community disorganization; who feel disconnected from teachers and schools; who experience high levels of family conflict and negative relationships with parents; who are exposed to delinquent peers) put them at risk for dysregulation, leading to impaired functioning and problem behaviors that are destructive to themselves and others. This set of assumptions is powerful and pervasive, if usually implicit, and underlies what we call the "mental health model" of developmental psychopathology.

In contrast with the mental health model, theory and research in evolutionary biology have come to acknowledge that, in most species, single "best" strategies for survival and reproduction are unlikely to evolve. Instead, the locally optimal strategy normally varies as a function of three overarching parameters. First, the costs and benefits of different strategies depend on the physical, economic, and social parameters of an organism's environment (e.g., food availability, mortality rates, quality of parental investment, social competition). This context-dependency means that a strategy that promotes success in some environmental contexts may lead to failure in others. Second, the success and failure of different strategies depends on an organism's condition or relative competitive abilities in the population (e.g., age, body size, health, history of wins and losses in agonistic encounters); that is, the cost-benefit trade-offs of different strategies varies depending on an organism's internal condition and competitive status.

Third, an organism's sex often has important implications for the range of available strategies and their relative costs and benefits.

In this section we discuss how developmental processes increase adaptation by matching an organism's phenotype to local environmental conditions and individual characteristics. We begin by reviewing the general concepts of plasticity and conditional adaptation. We then introduce evolutionary life history theory and show how it provides a general framework for adaptive plasticity, as well as an integrative understanding of the development of individual differences in physiology, growth, and behavior.

Developmental Plasticity and Conditional Adaptation

Because the viability of different survival and reproductive strategies is so context- and condition-dependent, natural selection tends to maintain *adaptive developmental plasticity*: biological systems that reliably guide the development of alternative phenotypes (including anatomy, physiology, and behavior) to match an organism's internal condition and external environments (see West-Eberhard, 2003). Developmental plasticity involves "durable biological change in the structure or function of a tissue, organ, or biological system" (Kuzawa & Quinn, 2009, p. 132). Importantly, adaptive developmental plasticity is a non-random process; it is the outcome of structured interplay between the organism and its environment , shaped by natural selection to increase the capacity and tendency of individuals to track both their internal condition and external environments and adjust the development of their phenotypes accordingly. Developmental plasticity is ubiquitous throughout the animal world (see extensive reviews in DeWitt & Scheiner, 2004; West-Eberhard, 2003).

Developmental plasticity is critically important for enabling organisms to adapt to stress, which has always been part of the human experience. Indeed, almost half of children in hunter-gatherer societies—the best model for human demographics before the agricultural revolution—die before reaching adulthood (e.g., Volk & Atkinson, 2013). Thus, from an evolutionary-developmental perspective, stressful rearing conditions, even if those conditions engender sustained stress responses that must be maintained over time, should not so much impair neurobiological systems as direct or regulate them toward patterns of functioning that are adaptive under stressful conditions (see Ellis et al., 2012; Frankenhuis & de Weerth, 2013).

Because developmental plasticity involves durable change, it is inherently forward-looking; that is, it involves predicting—and preparing—for future environments. Boyce and Ellis (2005) make this explicit in their definition of *conditional adaptation*: "evolved mechanisms that detect and respond to specific features of childhood environments, features that have proven reliable over evolutionary time in predicting the nature of the social and physical world into which children will mature, and entrain developmental pathways that reliably matched those features during a species' natural selective history" (p. 290). During fetal development and infancy, important features of the environment are communicated to the child via the placenta and lactation in nutrients, metabolites, hormones, growth factors, and immune factors that reflect the mother's current and past experiences (Kuzawa & Quinn, 2009). Beyond these molecular signals from the mother, relevant features of the environment are detected and encoded through the child's ongoing experiences.

Developmental plasticity necessitates developmental trade-offs. For example, tadpoles (*rana sylvatica*) alter their size and shape based on the presence of dragonfly larvae in their rearing environment (Van Buskirk & Relyea, 1998). These alterations involve development of

smaller and shorter bodies and deep tail fins. Although tadpoles that do not undergo these morphological changes are highly vulnerable to predation by dragonflies, those that do but end up inhabiting environments that are not shared with dragonflies have relatively poor developmental and survival outcomes. In short, the predator-induced phenotype is only conditionally adaptive. This process highlights that in many cases, natural selection favors a primary phenotype that yields high payoffs under favorable circumstances and a secondary phenotype that "makes the best of a bad situation" (West-Eberhard, 2003).

The Role of Genotypic Variation

As should be clear from the tadpole example, in addition to the apparent benefits of developmental plasticity, there can be substantial costs. On the one hand, there is the cost of producing and maintaining the appropriate regulatory and assessment mechanisms to support alternative patterns of development. On the other hand, environmental cues may have limited validity, and thus developmental plasticity in response to current conditions may fail to correctly predict future environmental conditions. Consequently, while adaptive developmental plasticity is widespread (see below), it is not always be the best or only option. As an alternative to adaptive developmental plasticity, or in conjunction with it, natural selection may also maintain genetic variation as a solution to the critical adaptive problem of matching phenotypes to heterogeneous environments.

There are a variety of circumstances in which genetic contributions to alternative phenotypes are likely to be favored by natural selection. When individuals inhabit multi-niche environments, and they are able to choose the niche that best fits their phenotype, it may partly or fully obviate the need for developmental plasticity. Instead, a diversity of genetically-regulated phenotypes that are specialized to the different social or physical niches can thrive in this context (see Wilson & Yoshimura, 1994). In addition, genetic variation can be maintained through *balancing selection,* whereby selection for alternative phenotypes systematically changes across time, space, population states, and so forth. A common type of balancing selection is *frequency-dependent selection*, which occurs when the fitness of different phenotypes changes as a function of their frequency in a population. The most viable form of frequency-dependent selection is negative, selecting against a given phenotype as it becomes more common. For example, aggressive individuals may be very successful when they are surrounded by tame individuals; however, as they multiply and begin to "invade" the population, their reproductive success may drop as they now compete mainly with other aggressive individuals. Balancing selection can also result from *heterozygote advantage* (when individuals who are heterozygous at a certain locus have higher fitness than either of the homozygous types) or from changes in selection pressures over time and space (*fluctuating selection*). Fluctuating selection pressures, by definition, weaken directional selection and therefore enable higher rates of genetically-regulated phenotypic variation (including neutral and deleterious forms of variation).

A crucial question is, to what degree should phenotypic variation be more developmentally contingent and plastic versus more strongly regulated by genotypic variation? The answer is not simple; indeed, what is typically found in organisms is a mixture of the two. Theoretical models suggest that one should often expect a balance between genetic and environmental determination of phenotypic individual differences. Depending on the structure of environmental variation, the costs and benefits of plasticity, and the life history of an organism, a given selection regime—for example one of temporally fluctuating selection—may maintain

different proportions of developmental plasticity and genotypic variation (reviewed in Del Giudice, under review).

The reproductive strategies of the male swordtail fish provide an example of this complexity, demonstrating the importance of adaptive genetic variation, adaptive developmental plasticity, and their interplay (reviewed in Ellis, Jackson, & Boyce, 2006). In the swordtail, three alleles at the *P* locus on the Y chromosome correspond to three modes in size distribution of mature males (small, intermediate, and large). Although all three genotypes perform the range of species-typical mating strategies, they do so at different size-related frequencies. Specifically, small, intermediate, and large males generally sneak, sneak and court, and court females, respectively. Size is the primary mediating mechanism in this species through which allelic variations influence mating strategies.

In determining alternative mating strategies, the key developmental event in male swordtail fish is gonadarche (maturation of the gonads). Specifically, the three alleles at the *P* locus differentially influence timing of gonadarche, which occurs earlier in genotypically small than in genotypically large males. In addition to these genetic influences, timing of gonadarche is also sensitive to a number of environmental factors, such as temperature and agonistic interactions with other males. These environmental influences can result in genotypically small males that are larger than genotypically intermediate males, and alternative mating strategies correlate more strongly with size than with genotype. In addition, mating strategies of male swordtail fish are competition-dependent in relation to interaction with other males. For example, males of intermediate size will sneak and chase females rather than court when in the presence of larger males.

In sum, both genomic and environmental factors influence timing of gonadarche, which in turn coordinates patterns of gene expression involved in the developmental cascade that induces sexual maturation and halts or dramatically reduces growth. Timing of gonadarche strongly influences size, and size is a major developmental factor in entrainment of alternative mating strategies. At the same time, mating strategies are conditionally adjusted in response to current physical and social dimensions of the environment. Thus, although there are strong genotypic influences on size and developmentally-linked mating strategies, the development of the alternative phenotypes in fact emerges through a complex series of gene-environment interplay. Importantly, these developmental interactions occur through integrated effects of gene products and environmental conditions on the developing phenotype.

Reaction Norms

A useful tool for thinking about developmental plasticity is the concept of a *reaction norm*. A reaction norm is a function describing how a single individual may express different phenotypes in response to a range of environmental conditions. While reaction norms are often treated as a property of genotypes (see Schlichting & Pigliucci, 1998), genotypic effects on development—including individual differences in plasticity—are always mediated by the pre-existing phenotype. Moreover, genetically different individuals may develop the same phenotype following different developmental trajectories. Thus, reaction norms may be legitimately employed to map phenotypic change on pre-existing phenotypic (rather than genotypic) differences (see Del Giudice, under review).

Figure 2 illustrates individual differences in developmental plasticity in the simple case of linear reaction norms. As can be seen in the figure, individuals may differ in the *elevation* and/or

in the *slope* of of their reaction norms. A steeper slope indicates higher susceptibility to environmental factors, as the same amount of variation in environmental conditions results in a larger change in the expressed phenotype. The *reaction range* of an individual is the difference between the minimum and maximum phenotypic score over a fixed range of the environmental variable, and denotes the individual's overall potential for plasticity. When the reaction norms of different individuals are not parallel (different slopes; Figures 2b and 2c), the result is a statistical genotype x environment (GxE) or phenotype x environment (PxE) interaction, whereby the effect of the environment is moderated by an individual's genotype/phenotype (and vice versa).

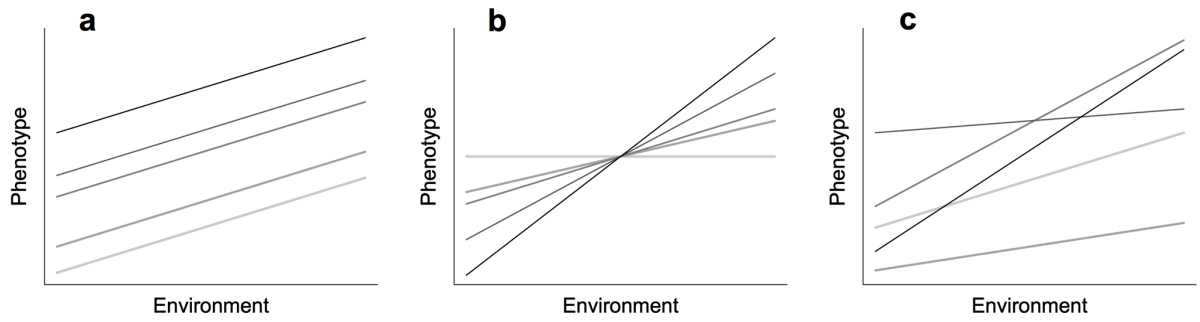

Figure 2. (a) Individual differences in phenotypic elevation but not in slope and reaction range; all genotypes have the same plasticity. (b) Individual differences in slope and reaction range (differential plasticity); all genotypes have the same elevation at the environmental mean. (c) Individual differences in elevation, slope, and reaction range. Reprinted from Del Giudice (under review).

Adaptive Plasticity in the Development of Life History Strategies

From Life History Trade-offs to Life History Strategies

A major framework in evolutionary biology for explaining patterns of developmental plasticity and individual differences is life history theory (see Kaplan & Gangestad, 2005; Stearns, 1992). All organisms live in a world of limited resources; for example, the energy that can be extracted from the environment in a given amount of time is intrinsically limited. Time itself is a limited good; the time spent by an organism looking for mates cannot be used to search for food or care for extant offspring. Due to these structural and resource limitations, organisms cannot maximize all components of fitness simultaneously and instead are selected to make trade-offs that prioritize resource expenditures, so that greater investment of time and/or resources in one domain occurs at the expense of investment in competing domains.

For example, resources spent on mounting a robust inflammatory response to fight infection cannot be spent on reproductive effort. Thus, the benefits of inflammatory response are traded off against the costs of lower ovarian function in women and reduced musculoskeletal function in men (Clancy et al. 2013; Muehlenbein & Bribiescas, 2005). Trade-offs between reproductive effort and health go in the opposite direction as well, as early reproductive

maturation is linked to more physical health problems in adulthood (e.g., Allsworth, Weitzen, & Boardman, 2005). Each trade-off constitutes a decision node in allocation of resources, and each decision node influences the next decision node (opening up some options, foreclosing others) in an unending chain over the life course (Ellis, Figueredo, Brumbach, & Schlomer, 2009). This chain of resource-allocation decisions—expressed in the development of a coherent, integrated suite of physiological and behavioral traits—constitutes the individual's life history strategy.

Life history strategies are adaptive solutions to fitness trade-offs within the constraints imposed by social conditions, physical laws, phylogenetic history, and developmental mechanisms. An organism's life history strategy coordinates morphology, physiology, and behavior in a way that maximizes expected fitness in a given environment (Braendle, Heyland, & Flatt, 2011; Réale et al., 2010). At the most basic level, the resources of an organism must be distributed between *somatic effort* and *reproductive effort*. Somatic effort can be further subdivided into growth, survival and body maintenance, and developmental activity (Geary, 2002). Developmental activity includes play, learning, exercise, and other activities that contribute to building and accumulating *embodied capital* – strength, coordination, skills, knowledge, and so forth (Kaplan & Gangestad, 2005; Kaplan, Hill, Lancaster, & Hurtado, 2000). Reproductive effort can be subdivided into *mating effort* (finding and attracting mates, conceiving offspring), *parenting effort* (investing resources in already conceived offspring), and *nepotistic effort* (investing in other relatives, for example siblings and grandoffspring).

The critical decisions involved in a life history strategy can be summarized by the fundamental trade-offs between current and future reproduction, between quality and quantity of offspring, and—in sexually reproducing species—between mating and parenting effort (see Ellis et al., 2009). By delaying reproduction, an organism can accumulate resources and/or embodied capital, thus increasing the quality and fitness of future offspring; however, the risk of dying before reproducing increases concomitantly. When reproduction occurs, the choice is between many offspring of lower quality and fewer offspring of higher quality. Although intensive parental investment is a powerful way to increase the embodied capital (and long-term prospects) of one's descendants, the fitness gains accrued through parenting must be weighed against the corresponding reduction in mating opportunities. Different life history strategies solve these problems in different ways by determining how organisms allocate effort among fitness-relevant traits. The same basic framework can be used to describe differences between species, as well as differences between individuals of the same species.

At the broadest level of analysis, life history traits covary along a dimension of *slow* versus *fast* life history strategies. Variation along the slow-fast continuum is observed both between related species and between individuals of the same species (see Ellis et al., 2009; Réale et al., 2010). Slow growth and late reproduction correlate with long lifespan, high parental investment, fewer offspring of higher quality, and low juvenile mortality. Conversely, fast growth and early reproduction correlate with high juvenile mortality, short lifespan, larger numbers of offspring and reduced parental investment in each (Figure 3). Fast life history strategies are comparatively high risk, focusing on mating opportunities (which typically involves more risky and aggressive behavior), reproducing at younger ages, and producing a greater number of offspring with more variable outcomes.

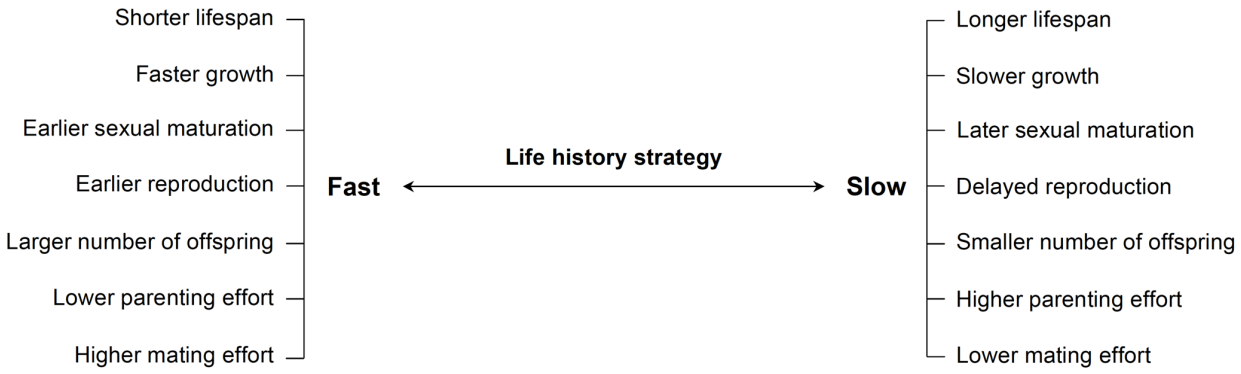

Figure 3. The fast-slow continuum of life history variation.

Sex Differences in Life History Trade-offs

The asymmetries introduced by sexual reproduction have important implications for the life histories of males and females. For example, in most species males tend to engage in higher mating effort and lower parental effort than females (Geary, 2002; Trivers, 1972). In addition, males usually undergo stronger sexual selection, that is, their reproductive success is more variable than that of females; they also tend to mature more slowly, in order to gain the competitive abilities and qualities needed for successful competition for mates. Sexual asymmetries in life history strategies can be attenuated in species with monogamous mating systems and when both parents contribute to offspring care. Compared with other mammals, humans show an unusually high degree of paternal investment; we are clearly adapted for the possibility of monogamous, long-term relationships. However, human paternal care is also highly variable and facultative (e.g., Geary, 2005; Quinlan, 2008), and strict monogamy is rarely if ever found. Overall, human mating is best characterized as strategically flexible (Gangestad & Simpson, 2000), with a widely documented tendency for men to engage in higher mating effort than women.

As a result, the trade-off between current and future reproduction is more pressing for women than for men: women's reproductive rate is limited by the long duration of gestation and the considerable energetic investment of pregnancy and lactation, and their window for successful reproduction necessarily ends with menopause. In contrast, men can potentially sire many offspring in a very short time, as well as for a more extended period of their lives. Men's crucial trade-off is the one between mating and parenting: the payoffs of high mating effort are potentially much larger for males, who can benefit directly from having access to a large number of partners; women can usually have only one child at a time, and thus benefit comparatively less from mating with multiple partners.

Environmental Determinants of Life History Strategy

Developmental calibration of slow versus fast life history strategies is a prototypical case of developmental plasticity. Key dimensions of the environment that regulate the development of life history strategies include energy availability, extrinsic morbidity-mortality, and predictability of environmental change (Ellis et al., 2009; Kuzawa & Bragg, 2012). Energetic resources—caloric intake, energy expenditures, and related health conditions—set the baseline for many developmental processes. Energy scarcity slows growth and delays sexual maturation and reproduction, resulting in a "slow" life history strategy. However, when bioenergetic resources are adequate to support growth and development, then proximal cues to extrinsic morbidity-mortality and unpredictability generally promote faster life history strategies.

Extrinsic morbidity-mortality refers to external sources of disability and death that are relatively insensitive to the adaptive decisions of the organism. Environmental cues indicating high levels of extrinsic morbidity-mortality cause individuals to develop faster life history strategies. Faster strategies in this context—a context that devalues future reproduction—function to reduce the risk of disability or death prior to reproduction. Moreover, high extrinsic morbidity-mortality means that investing in parental care has quickly diminishing returns, which favors reduced parental investment and offspring quantity over quality. Accordingly, exposure to environmental cues indicating extrinsic morbidity-mortality (i.e., observable cues that reliably covaried with morbidity-mortality risks during evolutionary history) can be expected to shift life history strategies toward current reproduction by anticipating maturation and onset of sexual activity. In humans, these cues may include exposure to violence, harsh childrearing practices, premature disability and death of other individuals in one's local ecology, and so forth.

In addition to extrinsic morbidity-mortality, environmental unpredictability—stochastic changes in ecological and familial conditions also regulates development of life history strategies (Ellis et al., 2009). In environments that fluctuate unpredictably (e.g., changing randomly between Conditions A and B, so exposure by parents or their young offspring to Condition A does not reliably forecast whether offspring will mature into Condition A or B), long-term investment in a development of a slow life history strategy does not optimize fitness; all of the energy invested in the future is wasted if the individual matures into an environment where life expectancy is short. Instead, individuals should detect and respond to signals of environmental unpredictability by adopting faster life history strategies. In humans, cues of unpredictability may include erratic neighborhood conditions, frequent residential changes, fluctuating economic conditions, changes in family composition, and so forth.

Belsky and colleagues (1991) were the first to hypothesize that harsh parenting, conflictual family relations, and insecure attachment would predict early sexual maturation, impulsivity, reduced cooperation, and exploitative interpersonal styles—the expected correlates of a fast life history strategy in humans. Empirical studies have confirmed these associations and detailed how harsh family relations, insecure attachment, and high levels of mortality in the immediate environment predict early puberty (in females), precocious sexuality, unstable couple relationships, and promiscuous mating styles (see special section of *Developmental Psychology*; Ellis & Bjorklund, 2012). Key psychological mediators of fast life history strategies include insecure attachment styles, impulsivity, present orientation (the inability to delay gratification and/or wait for larger rewards in the future), and a short subjective life expectancy. These variables are reliably associated with earlier onset of sexual activity, unrestricted sociosexuality (a desire for short-term, promiscuous sexual relationships), larger number of sexual partners,

earlier age at first birth in women, increased risk-taking, reduced cooperation, and antisocial behavior (reviewed in Belsky, 2012; Chisholm, 1999; Del Giudice, 2009; Del Giudice, in press; Figueredo & Jacobs, 2010; Figueredo et al., 2006). At the level of personality traits, slow life history strategies are robustly associated with agreeableness and conscientiousness (Del Giudice, in press). Taken together, these results strongly support the existence of a fast-slow dimension underlying a broad spectrum of individual differences in humans.

Because extrinsic morbidity-mortality and unpredictability are distinct, developmental exposures to each of these environmental factors should uniquely contribute to variation in life history strategy (Ellis et al., 2009). Longitudinal analyses of the National Longitudinal Study of Adolescent Health, the National Institute of Child Health and Human Development (NICHD) Study of Early Child Care and Youth Development, and the Minnesota Longitudinal Study of Risk and Adaptation (MLSRA) support this prediction (Belsky, Schlomer, & Ellis, 2012; Brumbach, Figueredo, & Ellis, 2009; Simpson, Griskevicius, Kuo, Sung, & Collins, 2012). For example, in the NICHD and MLSRA studies, exposures to environmental unpredictability in the first 5 years of life (e.g., parental changes, residential changes) uniquely predicted faster life history strategies in adolescence and emerging adulthood, independent of the effects of unpredictability in later childhood and indicators of extrinsic morbidity-mortality.

The Centrality of the Phenotype

All developmental processes are ultimately the product of structured organism-environment interplay. Development is always modulated by the organized phenotype, which is initially provided by the parents in the form of a zygote and then changes during ontogeny in response to both genetic and environmental influences.

Consider a central life history trait: timing of sexual maturation. As discussed above, sexual maturation is regulated by energetic conditions, so that—on average—individuals in well fed populations experience early puberty and poorly fed populations experience late puberty. The effects of energetic conditions, however, are modulated by the organized phenotype. For example, food-getting ability (a behavioral phenotype), metabolic efficiency (a physiological phenotype), and energy stores in the form of body fat (a morphological phenotype) all contribute to regulation of puberty; that is, these phenotypic traits modulate the effects of the critical environmental factor (energy availability) on maturation and functioning of the reproductive axis. The same logic applies to genetic effects: genes provide templates for the production of particular molecules that become incorporated into the phenotype, depending on the responsivity of the phenotype to those molecules and the presence of the necessary environmental building blocks (substances from outside the organism) to support gene expression (West-Eberhard, 2003). The effects of genes, environments, and phenotypes are hierarchically organized: The preexisting phenotype is the transducer of both genetic and environmental sources of information. Specifically, genetic and environmental effects depend on the phenotype being organized to accept them, and the modified phenotype retains these effects as development proceeds. In this sense, the phenotype embodies one's own particular history of genetic and environmental effects.

The organizational role of the phenotype is critical to understanding the development of life history strategies. As we will discuss in detail in the next section, Del Giudice and colleagues (2011) proposed that one of the key functions of the stress response system is to regulate an organism's life history strategy. According to the Adaptive Calibration Model (ACM; Del

Giudice et al., 2011; Ellis & Del Giudice, 2013), the stress response system coordinates the development of alternative life history strategies by affecting a broad suite of physiological and psychological traits, including growth and maturation, sexual and reproductive functioning, social learning, aggression, competition and risk-taking, pair-bonding, and related factors. This occurs in part through extensive physiological linkages between the stress response system and the reproductive axis (Ellis, 2004). The key idea is that activation of stress, metabolic, and immune system responses during childhood provides crucial *information* about threats and opportunities in the environment, their type, and their severity. Over time, this information becomes biologically embedded in the parameters—recurring set points and reactivity patterns—of these systems. These parameters provide the developing person with statistical "summaries" of key dimensions of the environment. An alternative pathway for the effects of stress may revolve around somatic damage: if early stress causes permanent damage to the organism and thus reliably reduces life expectancy, it may be adaptive for individuals exposed to stress early in life to engage in faster life history strategies even if the environment improves later on (Nettle, Frankenhuis, & Rickard, 2013). In total, the stress response system operates as a mechanism of conditional adaptation: it collects and biologically embeds information from the environment, and makes use of that information to match the developing phenotype to local environmental conditions. In this manner, the environment becomes instantiated in the phenotype.

At the same time, the phenotype modulates environmental effects at all points in development. As a result of differences in extant phenotypes, individuals differ in their reaction norms (Schlichting & Pigliucci, 1998). Because reaction norms differ in slope across individuals, some people are more likely than others to experience sustained developmental change in response to environmental exposures, including change in the physiological parameters that mediate development of alternative life history strategies. Moreover, as a result of differences in the organized phenotype, life history-relevant physiological parameters already differ across individuals at birth (cf. temperament). Stated differently, people differ in the elevation of their reaction norms. This means that developmental change in the physiological bases of life history strategies and their behavioral outcomes are likely to occur around different points on the life history spectrum (i.e., around the faster range of life history in some individuals and the slower range in others).

In total, the organized phenotype is present from conception and can be described in terms of the steepness and location of its reaction norms along various dimensions. These reaction norms, which have already undergone significant development by the time a child is born, are both regulated by and constrain the effects of environmental and genetic factors. The organized phenotype incorporates and biologically embeds environmental and genetic inputs throughout the life course. This ongoing process translates into individual differences in such critical traits as body size, energy reserves, metabolic efficiency, susceptibility to environmental influence, immune function, fecundity, mate value, fighting ability, and so forth. Differences between individuals in these phenotypic traits influence the cost-benefit trade-offs of different life history strategies and thus play a central role in regulating the development of these strategies.

Consider the trade-off between mating effort and parenting effort in men. Sexual selection models, such as Gangestad and Simpson's (2000) *strategic pluralism theory*, emphasize social and sexual competition as important factors shaping adaptive variation in reproductive strategies. According to this perspective, individuals who are competitively advantaged relative to peers (i.e., who possess social and physical attributes that make them successful in same-sex

competition and targets of choice by the other sex) have more mating opportunities. These enhanced opportunities tend to bias resource allocations toward more mating effort at the expense of parental effort. Because male reproductive success is ultimately constrained by the ability to access, attract, and retain mates, alternative male mating strategies should be especially attuned to the demands and desires of women and the ability of men to successfully engage in intrasexual competition. To a large extent, this variable success arises from phenotypic traits that facilitate gaining status and attracting mates (e.g., size, aggressiveness, physical attractiveness, social relations with others). This leads to the hypothesis that intrasexual competitive abilities in men will regulate life history strategies, especially the mating-parenting trade-off.

This hypothesis has been supported by a large empirical literature showing that men who achieve high social status or who possess honest indicators of genetic quality (e.g., physical attractiveness, bilateral symmetry of body parts) engage in more mating effort. For example, anthropological evidence indicates that social status is directly related to male reproductive success in horticultural, agricultural, and pastoral societies (see Pérusse, 1993 for an extensive review). Men with higher status in industrial societies, as measured by education, occupation, and income, also report a greater number of sex partners than men of lower social status (Pérusse, 1993). In addition, men who report that they are more attractive to the opposite sex also report having sex at an earlier age, a greater number of sex partners, and an unrestricted sociosexual orientation (reviewed in James & Ellis, 2013). Finally, men who are more symmetrical in bilateral traits have been found to have more lifetime sexual partners as well as more extrapair sexual encounters during ongoing relationships (controlling for physical attractiveness). In contrast, no consistent relations have been found between women's symmetry and number of lifetime sexual partners or extrapair sexual relationships (reviewed in Thornhill & Gangestad, 2004).

Another critical life history trade-off that is regulated by phenotypic condition is current versus future reproduction. Effort put into reproducing now will use energy or resources that cannot be used or saved for future reproduction. The costs of current reproduction may be paid in terms of reduced number, quality, or survival of future offspring, as well as reduced growth and survival of the parent. This decision whether to pay these costs critically depends of the physical condition of the individual. If either external conditions (e.g., infectious diseases, warfare) or internal state (e.g., poor health, oxidative stress) indicate a heightened probability of premature disability or death, then individuals should shift resource allocations toward current reproduction (presuming adequate bioenergetic resources to support a fast strategy).

Research examining relations between exposures to stress, biological aging or health, and reproductive strategies has provided preliminary support for this hypothesis. For example, Bleil and colleagues (2012, 2013) found that heightened psychosocial stress was associated not only with ovarian reserve depletion in older women, but also earlier puberty and higher antral follicle count in younger women, indicating a faster life history strategy. Likewise, women who were exposed *in utero* to the Dutch famine of 1944–1945 not only have increased risk of chronic degenerative disease, but also start reproducing at a younger age, have more offspring, more twins, and are less likely to remain childless (Painter et al., 2008), again indicating a faster life history strategy. An important qualification to these findings is that individuals with life-expectancy-reducing chronic disease diagnosed during childhood also shift toward current reproduction (e.g., early age at first reproduction), even though the incidence of serious chronic disease was uncorrelated with family and ecological stressors (Waynforth, 2012). These data indicate that internal factors, such as compromised phenotypic condition (i.e., damage), in and of

itself, can contribute to accelerate life history strategies. Once again, this underscores the centrality of the phenotype in organizing adaptive plasticity through the reciprocal interplay of environmental and genetic factors, either of which can have more decisive effects in different developmental contexts (see extended discussion in West-Eberhard, 2003).

Implications for the Core Points of Developmental Psychopathology

While the mental health model of developmental processes resonates with the intuitions of many researchers, its narrow view of adaptation and maladaptation is an obstacle toward the goal of synthesizing *normal and pathological development* in a single framework. The concepts of adaptive plasticity and conditional adaptation offer a better appreciation of the logic of individual differences and that of gene-environment interplay in development. The crucial idea is that adverse environmental conditions often direct developmental processes along alternative adaptive pathways, rather than simply impair or dysregulate them.

The framework of life history theory adds a layer of specificity to this general picture. Life history concepts can be employed to make remarkably accurate predictions about the structure of individual differences in physiology, growth, and behavior, and the environmental factors that shift development along alternative trajectories. In particular, life history theory delineates basic dimensions of environmental stress and support that underlie the multitude of *risk and protective factors* described in developmental psychopathology—resource availability, morbidity/mortality risk, and unpredictability. This is especially useful given the confusing abundance of environmental/contextual variables that might be measured and correlated with developmental outcomes. It is also important to stress how adaptive plasticity and life history theory offer a thoroughly *contextualist* view of development, though one that is compatible with a major role of genetic factors and genotype x environment interactions.

Finally, the centrality of the phenotype in enabling and structuring adaptive plasticity has a number of implications for the prospect of integrating developmental psychopathology and EDP. First of all, it does away with the notion that EDP is wedded to any sort of "genetic determinism" and shows how it is possible to integrate a sophisticated view of developmental mechanisms within an explicitly evolutionary framework. Second, it suggests a deep theoretical rationale for the prevalence of *probabilistic causality* in development. Third, it affords a principled way to investigate the connection between behavioral strategies and *brain and neurobiological factors*, thanks to the concept of biological embedding. In the next section we will further illustrate this point by reviewing the role of the stress response system in collecting environmental information and regulating physical and behavioral development.

Beyond Allostatic Load: The Stress Response System as a Mechanism of Conditional Adaptation

How does repeated or chronic childhood adversity shape biobehavioral development and, through it, mental and physical health? Consistent with the mental health model, there is a widely accepted answer to this question in the field of developmental psychopathology. Instantiated in models of "toxic stress" (Shonkoff et al., 2012) and "allostatic load" (Lupien et al., 2006; McEwen & Stellar, 1993), that answer posits a striking duality: biological responses to

stress are usually adaptive in the short term, but protracted activation of stress response systems is maladaptive and toxic in the long term. Toxic stress causes disruptions of brain structure and function, resulting in dysregulation of physiological mediators—autonomic, neuroendocrine, metabolic, and immune—"that are the precursors of later impairments in learning and behavior as well as the roots of chronic, stress-related physical and mental illness" (Shonkoff et al., 2012, p. e236). As eloquently stated by Juster and colleagues (2011), the wear and tear of toxic stress and altered stress hormone functioning "inexorably strains interconnected biomarkers that eventually collapse like domino pieces trailing toward stress-related endpoints" (p. 725).

These models of toxic stress and allostatic load, however, only tell half of story. The other half is the central role of the stress response system (SRS) in orchestrating physical and psychosocial development of both humans and nonhuman species (Ellis et al., 2006; Korte, Koolhaas, Wingfield, & McEwen, 2005), both in terms of species-typical development and individual differences. One of the most remarkable features of the SRS is the wide range of individual variation in its physiological parameters. Some individuals respond quickly and strongly even to minor events, whereas others show flat response profiles across most situations. Furthermore, the balance of activation among primary SRS subsystems—the sympathetic nervous system (SNS), the parasympathetic nervous system (PNS), and hypothalamus-pituitary-adrenal (HPA) axis—can vary considerably across individuals.

In developmental psychopathology, the standard framework for understanding the development and meaning of individual differences in stress responsivity is that of the *allostatic load model* (ALM; see Juster et al., 2011; Lupien et al., 2006; McEwen & Stellar, 1993). A guiding assumption of the ALM is that there is an optimal level of stress responsivity; accordingly, both "hyperarousal" and "hypoarousal"—recurring over or under activity of physiological mediators—are routinely described as dysfunctional deviations from the norm, usually caused by a combination of excessive stress exposure and genetic or epigenetic vulnerability. In this framework, environmental stress is treated as a risk factor for all kinds of symptoms and disorders (Compas & Andreotti, 2013). While some authors have argued that optimal adaptation is fostered by environments that contain *moderate* amounts of stressors (e.g., Rutter, 1993; Seery, 2011), the underlying assumption remains that a single "best" environment exists, and that deviations from that optimum cause dysregulation and pathology.

In this section we argue that acceptance of these assumptions, without placing them in a larger evolutionary-developmental framework, has impeded our understanding of the role of stress response systems in adaptively regulating development (for a detailed exposition see Ellis & Del Giudice, 2013). Specifically, models of allostatic load focus on the long-term *costs* of childhood stress and adversity—the "wear and tear" on multiple organ systems induced by chronic stress—but do not address the *benefits* of calibrating autonomic, neuroendocrine, metabolic, and immune systems to match current and future environments. We argue that this over-emphasis on costs misses something fundamental and thus weakens the conceptual power of the ALM perspective. The result has been an imbalanced approach to research that has yielded dramatically more empirical knowledge about dysfunction than adaptive function, making it difficult to gain a coherent "big picture" of the subject matter.

A promising alternative to the ALM is provided by the *adaptive calibration model* (ACM; Del Giudice et al., 2011), a theory of individual differences in stress responsivity that builds on the concepts of life history theory and developmental plasticity. The ACM supplements the ALM and revises some of its key assumptions, thus laying the foundation for a broad theory of

individual differences in stress responsivity. In this section we summarize the key tenets of the ACM, explicitly compare the ACM with the ALM, and discuss the implications of the two models for understanding adaptive and maladaptive developmental responses to stress (for more extended discussion, see Ellis & Del Giudice, 2013). Besides offering a broader perspective on the role of stress in development, the ACM exemplifies how the principles of EDP can be leveraged to achieve theoretical integration across multiple levels of analysis, from social behavior to neurobiology.

The Adaptive Calibration Model

The ACM is a theory of developmental programing focusing on calibration of the SRS and associated life history strategies to local environmental conditions. The ACM has its main theoretical foundations in life history theory and the theory of adaptive developmental plasticity (West-Eberhard 2003); it integrates and extends previous evolutionary models of stress (e.g., Boyce & Ellis 2005; Flinn, 2006; Korte et al., 2005; Porges, 2007) into a coherent theoretical framework. For a detailed presentation of the model, see Del Giudice and colleagues (2011).

The central tenet of the ACM is that the SRS operates as a mechanism of conditional adaptation, with a key role in regulating the development of individual life history strategies (Figure 4). In the ACM, the activation of autonomic, neuroendocrine, metabolic, and immune system responses during childhood provides crucial information about threats and opportunities in the environment, their type, and their severity. Over time, this information becomes embedded in the parameters—recurring set points and reactivity patterns—of these systems. These parameters provide the developing person with statistical "summaries" of key dimensions of the environment. For example, sustained activation of the HPA axis is generated by exposures to danger, unpredictable or uncontrollable contexts, and social evaluation, as well as energetic stress (see Dickerson & Kemeny, 2004; Gunnar et al., 2009); thus, the HPA axis tracks the key environmental variables involved in regulation of alternative life history strategies. Analogous arguments have been made regarding mesolimbic dopamine (Gatzke-Kopp, 2011). In turn, individual differences in SRS functioning regulate the coordinated development of a broad cluster of life history-relevant traits (Figure 4).

Although the ACM focuses on developmental plasticity, all developmental processes are the product of systematic organism-environment interplay. Because some individuals have steeper reaction norms than others, some individuals are more likely than others to experience sustained developmental change in response to environmental exposures. Further, as a result of differences in the organized phenotype, SRS parameter values already differ across individuals at birth. Consequently, individuals differ in the location of their reaction norms along SRS dimensions (Boyce & Ellis, 2005), with change more likely to occur for different individuals around higher versus lower ends of responsivity. Within these reaction norm constraints, the ACM articulates a theory of environmental regulation.

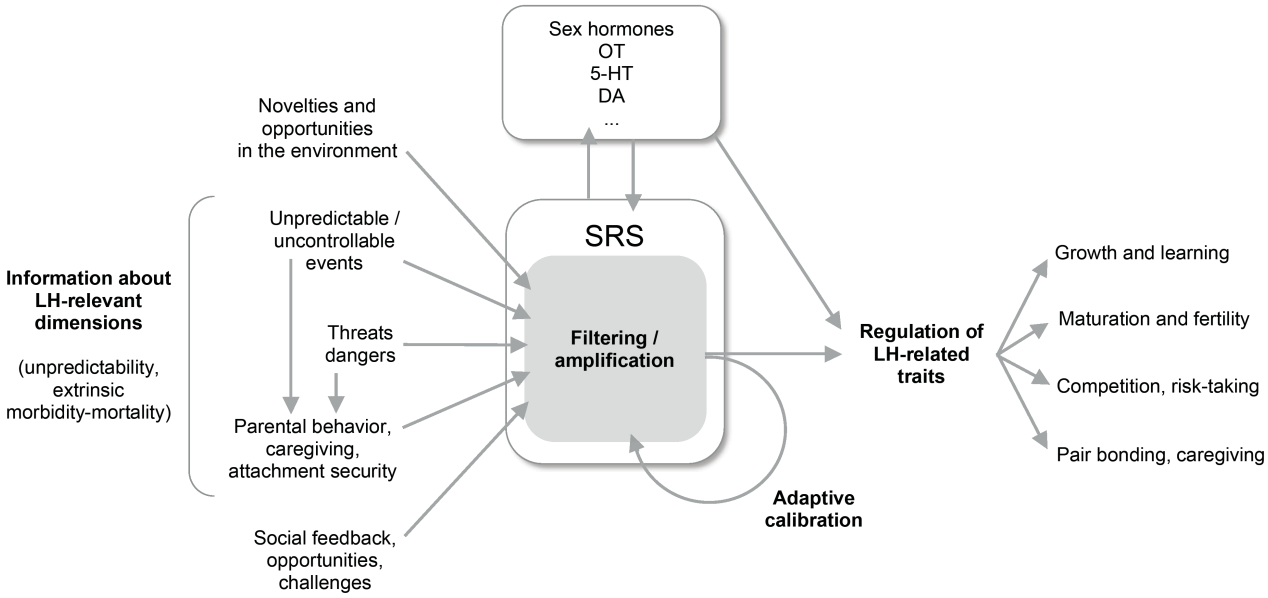

Figure 4. Core theoretical structure of the Adaptive Calibration Model. SRS: stress response system; LH: life history; OT: oxytocin; 5-HT: serotonin, DA: dopamine. Reprinted from Del Giudice et al. (2011).

In total, the SRS (a) collects and biologically embeds information from the environment and (b) makes use of that information to match the developing phenotype to local environmental conditions. A crucial aspect of this matching process is the (iterative) calibration of the SRS itself in the service of life history goals (curved arrow in Figure 4). SRS activity also feeds back on the system itself, resulting in responsivity patterns that are adaptively calibrated to current environmental conditions and the individual's overall strategy. This underscores the fact that responsivity patterns develop over time, and may change—within limits—if the local environment undergoes prolonged changes in safety and/or predictability (i.e., recalibration). Changes in responsivity are also expected to occur in tandem with key hormonal "switches" such as adrenarche, gonadarche, childbirth, and menopause. More details on pathways and transitions in development of responsivity patterns can be found in Del Giudice and colleagues (2011).

The Role of the SRS in Allostasis and in Transduction of Environmental Information

Environmental events signaling threats to survival or well-being produce a set of complex, highly orchestrated responses within the neural circuitry of the brain and peripheral neuroendocrine pathways regulating metabolic, immunologic, and other physiological functions. The SRS comprises primarily three anatomically distinct yet integrated and cross-regulated circuits: the PNS, SNS, and HPA axis. The general function of the PNS is to promote vegetative functions in the absence of stress (i.e., rest and restorative behavior) and reduce or downregulate cardiac activity. When a stressor is encountered, the PNS responds quickly by withdrawing this inhibitory influence (i.e., *vagal withdrawal*), allowing the SNS to operate unopposed and thus causing rapid increases in physiological arousal. The PNS promotes flexible responding to stress,

sustained attention, and coping with mild to moderate stressors (such as solving a difficult puzzle). More extreme defense reactions associated with "freeze/hide" behaviors also involve PNS activation, albeit via different efferent fibres (Porges, 2007).

If parasympathetic deactivation is not sufficient to cope with the present challenge, activation of the SNS occurs within seconds, providing a second layer of response in this hierarchy. Sympathetic activation mediates fight/flight responses following a fast, direct pathway via the noradrenergic innervation of visceral organs and a slower, hormonal pathway through innervation of the adrenal medulla (e.g., Gunnar & Vazquez, 2006). Following SNS activation, the adrenal medulla secretes epinephrine (E) and norepinephrine (NE) to increase heart rate, respiration, blood supply to skeletal muscles, and glucose release in the bloodstream.

The third component of the SRS is the HPA axis, which mounts more delayed, long-term responses to environmental challenge. The endpoint of the HPA response is cortisol release by the adrenal cortex, typically within 5 minutes after the triggering event, with a cortisol peak between 10-30 minutes (Gunnar & Vazquez, 2006). The main effects of cortisol are to (1) mobilize physiological and psychological resources (e.g., energy release, alertness and vigilance, memory sensitization), and (2) counter-regulate physiological effects of SNS activation, facilitating stress recovery.

The process by which the regulatory parameters of the SRS (as well as other neurobiological systems) are modified in the face of challenge is termed *allostasis* (i.e., "stability through change"). Allostasis refers to the moment to moment process of increasing or decreasing vital functions (i.e., adaptively adjusting physiological parameters within the organism's operating range) to new steady states in response to the demands of the environment and the organism's resources (McEwen & Stellar, 1993; see also Lupien et al., 2006). Allostasis functions to help the organism cope with challenging events or "stressors," enabling short-term adaptation to environmental perturbations. However, the term allostasis is not always used consistently; for example, some authors restrict the meaning of allostasis to long-term, potentially permanent changes in the system's parameters in contexts of protracted stress (what McEwen and Wingfield [2003] labeled *allostatic states* and is now more commonly referred to as *biological embedding*).

The SRS orchestrates whole-organism reactions to challenge through a suite of coordinated responses (i.e., allostatic adjustments). Depending on the intensity and duration of a stressor, SRS activation can reorient attentional focus, increase the organism's readiness for action (e.g., by increased heart/respiratory rate and changes in blood flow to various organs), shift the balance between different memory- and learning-related processes, release glucose into the bloodstream, suppress (or enhance) reproductive functioning, regulate immune function, and so on. The concept of allostasis represents a significant point of convergence between the ACM and the ALM. The ACM explicitly embraces the concept of allostasis and describes the coordination of allostatic responses as one of the main biological functions of the SRS.

The SRS responds not only to threats and challenges in the environment, but also novelties and positive social opportunities (e.g., unexpected or exciting rewards, opportunities for status enhancement, potential sexual partners). More generally, the SRS appears to mediate susceptibility to both cost-inflicting and benefit-conferring features of the environment, operating as an amplifier (when highly responsive) or filter (when unresponsive) of various types of contextual information (see extended discussion in Ellis, Del Giudice, & Shirtcliff, 2013). As we will discuss in the next section, this dual function of the SRS is captured by the concept of

biological sensitivity to context (Boyce & Ellis, 2005), which posits that a highly responsive SRS increases the organism's openness to environmental influence.

The Role of the SRS in Regulating Development of Life History Strategies

The ACM proposes that, across development, the environmental information collected by the SRS (in interaction with the child's genotype) canalizes physiological and behavioral phenotypes to match local ecological contexts (Figure 3). The SRS coordinates the development of alternative life history strategies by affecting a broad suite of life history-related physiological and psychological traits, including growth and maturation, sexual and reproductive functioning, social learning, aggression, competition and risk-taking, pair-bonding, and related factors. The assumption is that these traits and trade-offs are regulated in ways that once—even if possibly no longer—reliably enhanced fitness across different environmental contexts.

First of all, the SRS is crucially involved in the regulation of growth and metabolism, and chronic stress has been linked to individual differences in physical growth patterns. The SRS also modulates learning in a number of different ways: HPA and autonomic profiles have been associated with individual differences in cognitive functioning, memory, and self-regulation. Second, the SRS is functionally implicated in all the components of mating and parenting, beginning with sexual maturation. The autonomic systems, HPA, and gonadal axes are connected by extensive functional cross-talk, and HPA activity is linked to variation in pubertal maturation and fecundity. Variation in SRS functioning is also associated with romantic attachment styles. In turn, attachment styles predict relationship stability, commitment, and investment—all key determinants of parenting effort in humans. More directly, SRS functioning affects parenting behavior, including controlling and intrusive parenting practices, inconsistent discipline, and parental sensitivity to children's needs and demands. In men, cortisol and testosterone work together to direct somatic and behavioral effort toward mating or parenting. Finally, sexual competition is a crucial aspect of mating effort. Dominance-seeking, aggression, and risk-taking are all functionally connected to mating competition, and all are associated with SRS functioning in synergy and interaction with testosterone, serotonin, and dopamine. Furthermore, stress exposure regulates mating behavior by altering mate preferences and affecting the perceived attractiveness of potential sexual partners (reviewed in Del Giudice et al., 2011; Ellis & Del Giudice, 2013).

In summary, the SRS not only collects and encodes crucial life history-relevant information but is also involved in the regulation of all the major aspects of human life history strategies. Other systems that contribute to life history regulation include the hypothalamic-pituitary-gonadal axis, the serotonergic, dopaminergic, and oxytocinergic systems, and the immune system. Not coincidentally, all of these systems engage in extensive bidirectional cross-talk with the SRS (see e.g., Gatzke-Kopp, 2011; Miller, Chen, & Parker, 2011; van Goozen, Fairchild, Snoek, & Harold, 2007).

Patterns of Responsivity

Del Giudice and colleagues (2011) provide an extended theoretical and empirical treatment of the logic underlying the development of alternative responsivity patterns in the ACM (see Figure 5), including predicted relations between SRS physiology and serotonergic, dopaminergic, and oxytocinergic functioning. Here we briefly summarize this logic. In safe, low-

stress environments, a highly responsive SRS enhances social learning and engagement with the external world, allowing the child to benefit more fully from social resources and opportunities (Boyce & Ellis, 2005), thus favoring development of a *sensitive* phenotype (pattern I). The association between high parental sensitivity, positive family relations, and the development of a highly responsive SRS is supported by a number of studies (e.g., Ellis et al., 2005; Evans et al. 2013; Hackman et al., 2013). According to the ACM, sensitive patterns should be characterized by moderate HPA/SNS responsivity and high PNS responsivity. A sensitive phenotype makes children better at detecting positive opportunities and learning to capitalize on them. For example, high PNS responsivity has been linked to socio-emotional competence, engagement, and self-regulation (e.g., Stifter & Corey, 2001). Social learning and sensitivity to context are especially adaptive in the context of slow life history strategies, as a form of protracted somatic investment (Kaplan & Gangestad, 2005). In very safe and protected settings, sensitive individuals will rarely experience strong, sustained activation of the SNS and HPA systems; thus, the individual enjoys the benefits of responsivity without paying significant health costs (e.g., immune, energetic).

At moderate levels of environmental stress, the cost-benefit balance begins to shift as the potential advantages of high sensitivity decrease and the physiological and health costs of maintaining a hyper-responsive SRS increase. The optimal level of SRS responsivity is predicted to fall downward, favoring development of *buffered* phenotypes (pattern II) characterized by moderately low reactivity and a slow life history strategy. Buffered responsivity is expected to be the modal pattern in most populations (with most SRS parameters set around the mean), particularly in the low-risk, middle-class populations that provide a majority of research participants in psychology and neuroscience. The emergence of buffered responsivity patterns under conditions of moderate environmental stress is empirically consistent with the *stress inoculation hypothesis*, the idea that early exposure to repeated mild stressors down-regulates the SRS and leads to increased resistance to stress later on (e.g., Rutter, 1993). However, the ACM places stress inoculation in a broader theoretical perspective, in which moderate responsivity is only one out of many locally adaptive patterns of SRS functioning.

The benefits of increased responsivity rise again when the environment is perceived as dangerous and/or unpredictable. A responsive SRS enhances the individual's ability to react appropriately to dangers and threats while maintaining a high level of engagement with the social and physical environment. Moreover, engaging in fast life history strategies should lead the individual to allocate resources in a manner that discounts the long-term physiological costs of the stress response in favor of more immediate advantages. In this context, the benefits of successful defensive strategies outweigh the costs of frequent, sustained HPA and SNS activation, leading to *vigilant* phenotypes (pattern III). The predicted profile of vigilant individuals includes high HPA/SNS responsivity and low PNS responsivity (see Del Giudice et al. 2011). In turn, this physiological profile should be associated with fast life history-related traits such as fast maturation and high mating effort. Increased SRS responsivity in dangerous environments can be expected to go together with increased responsivity in other neurobiological systems; for example, hyper-dopaminergic function may contribute to the vigilant phenotype by boosting attention to threat-related cues and fast associative learning (Gatzke-Kopp, 2011).

Compared with their sensitive counterparts, vigilant individuals may show slower HPA recovery (i.e., they may take longer to return to baseline) and slower habituation (see Gunnar & Vazquez, 2006). For this reason, they are also likely to show stronger hypocortisolism following prolonged periods of stress. Although we argue that vigilant phenotypes represent biologically

adaptive responses to early stress, their "hair trigger" regulation may render them especially vulnerable to breakdown and/or persistent dysregulation following extreme stressors. On average, males and females are expected to differ in the predominant behavioral correlates of vigilant patterns—aggressive and agonistic behavior versus anxiety and withdrawal—because of the different costs and benefits of aggression, impulsivity, and risk-taking in the two sexes (Archer, 2009; see also Martel, 2013). Indeed, several studies suggest that acute HPA activation—typical of vigilant patterns—tends to promote risk-taking in males and risk aversion in females (see Mather & Lighthall, 2012; Starcke & Brand, 2012). Accordingly, high SRS responsivity can be associated with both internalizing and externalizing symptoms, especially in younger children (see Alink et al., 2008; Gunnar & Vazquez, 2006; van Goozen et al., 2007).

In very dangerous environments characterized by severe or traumatic stress, the balance shifts again toward low responsivity, especially for males who adopt a fast, mating-oriented life history strategy characterized by antagonistic competition and extreme risk-taking. Such a strategy requires outright insensitivity to threats, dangers, social feedback and the social context. For an extreme risk-taker, informational insulation from environmental signals of threat is an asset, not a weakness (see also Korte et al., 2005). In particular, adopting an exploitative/antisocial interpersonal style requires one to be shielded from social rejection, disapproval, and feelings of shame (all amplified by heightened HPA responsivity; reviewed in Del Giudice et al., 2011). An *unemotional* pattern of generalized low responsivity (pattern IV) can be evolutionarily adaptive at the high-risk end of the environmental spectrum – especially in males – despite its possible negative consequences for the social group and for the individual's subjective well-being. This type of chronic low responsivity should be carefully distinguished from temporary "exhaustion" periods, usually arising after prolonged SRS activation in highly responsive individuals exposed to enduring stressors. The association of risk-taking with low levels of SRS responsivity and basal activity is well documented, especially in males (e.g., Bubier & Drabick 2008; Halpern et al. 2002).

Unemotional profiles should be associated with high mating effort and early sexual maturation; this is consistent with the robust association between low SRS responsivity, externalizing behaviors (especially in male adolescents and adults), and callous-unemotional traits. As we discuss in a later section, this constellation of traits is associated with early maturation, precocious sexuality, and sexual promiscuity. Finally, the ACM hypothesizes two developmental pathways leading to unemotional responsivity patterns. In the first pathway, an initially responsive phenotype shifts toward unresponsivity following chronic severe stress. In particular, some children are expected to shift from pattern III to pattern IV during middle childhood or adolescence (Del Giudice et al. 2011). This prediction is consistent with the finding that associations between HPA activity and aggressive/externalizing behavior tend to be positive in preschoolers but negative in middle childhood and adulthood (Alink et al., 2008). In the second pathway, unresponsivity may develop even in low-stress environments because of strong genetic predispositions, and may be apparent already in early childhood.

The logic of sex differences in responsivity patterns is based on sexual selection theory informed by life history considerations. At the slow end of the life history continuum, both sexes engage in high parental investment, and male and female interests largely converge on long-term, committed pair bonds; sex differences in behavior are thus expected to be relatively small. As environmental danger and unpredictability increase, males benefit by shifting to low-investment, high-mating strategies; females, however, do not have the same flexibility since they benefit much less from mating with multiple partners and incur higher fixed costs through childbearing.

Thus, male and female strategies should increasingly diverge at moderate to high levels of environmental danger/unpredictability. In addition, sexual competition takes different forms in males and females, with males engaging in more physical aggression and substantially higher levels of risk-taking (e.g., Archer, 2009; Wilson, Daly, & Pound, 2002). As mating effort increases, sexual competition becomes stronger and sex differences in competitive strategies become more apparent. For these reasons, sex differences in responsivity patterns and in the associated behavioral phenotypes should be relatively small at low to moderate levels of environmental stress (patterns I and II) and increase in stressful environments (pattern III). Finally, we expect males to be over-represented as high-risk, low-investment strategists (pattern IV).

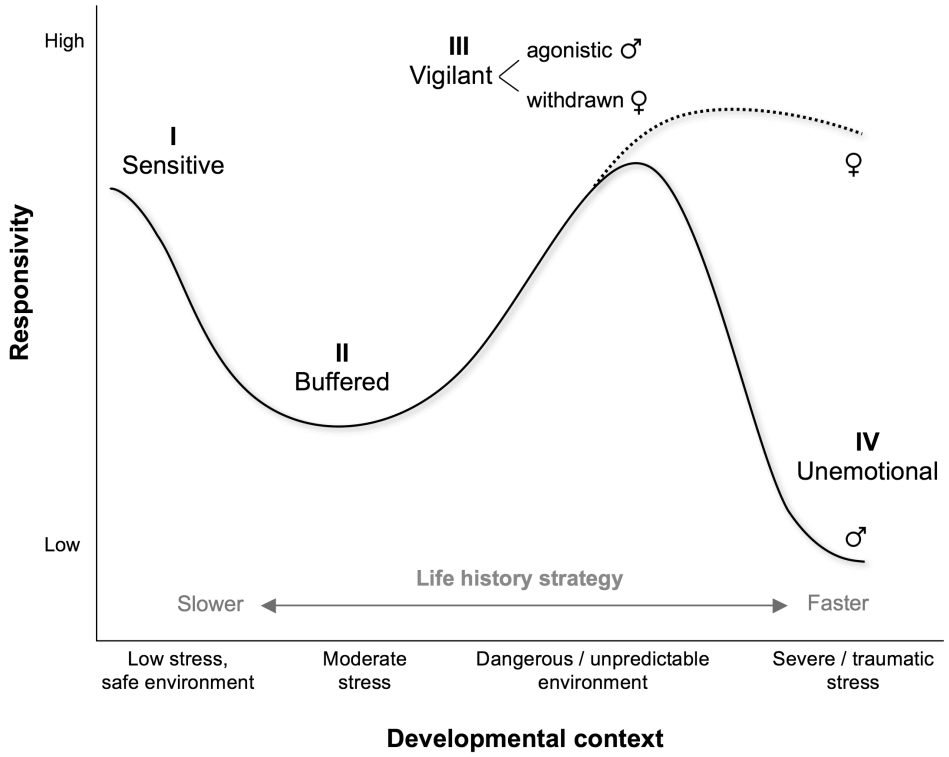

Figure 5. Predicted curvilinear relation between developmental context and optimal levels of stress responsivity. The figure does not imply that all components of the SRS will show identical responsivity profiles, nor that they will activate at the same time or over the same time course (see Del Giudice et al., 2011). Male/female symbols indicate sex-typical patterns of responsivity, but the model also predicts substantial within-sex variation. Adapted from Del Giudice et al. (2011).

The shifting equilibrium between costs and benefits of responsivity is predicted to result in a complex curvilinear relation between environmental stress and SRS responsivity (Figure 5). The ACM taxonomy of responsivity patterns is supported by preliminary empirical evidence on autonomic responsivity in middle childhood (Del Giudice, Hinnant, Ellis, & El-Sheikh, 2012).

While the SRS has a crucial role in directing the development of alternative life history strategies, it does so in interaction with other neurobiological systems. Accordingly, the four responsivity patterns described in the ACM are characterized not just by different profiles of SRS functioning, but also by functionally organized individual differences in behavior, in the physiology of the hypothalamic-pituitary-gonadal axis, in serotonergic and dopaminergic pathways, and other systems (Del Giudice et al. 2011). Each pattern reflects a unique combination of costs, benefits, and specific vulnerabilities to pathology. Finally, it should be noted that Figure 5 depicts an idealized, population-level reaction norm. In practice, different individuals may respond differently to the same level of environmental stress and show different degrees of plasticity—owing to different environmental sampling histories and the effects of genetic, epigenetic, and endophenotypic variation—as discussed in the section on differential susceptibility.

ALM and ACM: A Comparison

Allostatic load is a label for the long-term costs of allostasis; it is often described as the "wear and tear" that results from repeated allostatic adjustments (i.e., adaptation to stressors), exposing the organism to adverse health consequences. The ALM emphasizes that biological responses to threat, while essential for survival, have negative long-term effects that promote illness. The *benefits* of mounting biological responses to threat are traded off against *costs* to mental and physical health, and these costs (allostatic load) increase as the organism ages.

Basic tenets of the ACM an ALM are compared in detail in Ellis and Del Giudice (2013) and juxtaposed in Table 1. Both the ACM and ALM emphasize the adaptive nature of short-term physiological responses to stress (Table 1: *Activation of autonomic, neuroendocrine, metabolic, and immune systems*). Further, the ACM concurs with the ALM regarding the effects of childhood stress and adversity on regulation of allostatic mechanisms. Indeed, a substantial body of research has now documented biological embedding of early life stress in SRS parameter values (Table 1: *Changes in allostatic mechanisms*). In the ALM, however, this biological embedding is construed negatively, as the result of cumulative stress exposures that predispose the individual to morbidities and premature mortality. As shown in Table 1 (*Cognitive, behavioral, and emotional impairments in children*), some of these outcomes include lower performance on standard tests of intelligence and executive functions and increased mental health problems (reviewed in Ellis & Del Giudice, 2013). The ACM also acknowledges that chronic SRS activation carries substantial costs, in terms of biological fitness as well as health and well-being. While the ACM stresses conditional adaptation, it leaves open the possibility that, for a number of reasons, some developmental outcomes are biologically maladaptive. In terms of proximal responses to childhood adversity, The ACM and ALM mainly differ in their emphasis on the benefits (ACM) versus the costs (ALM) of allostatic adjustments (light shaded region of Table 1).

Table 1. Comparison of Adaptive Calibration Model (ACM) and Allostatic Load Model (ALM)

Responses to psychosocial stress/unpredictability	Examples of response	ACM	ALM
Activation of autonomic, neuroendocrine, metabolic, and immune systems	• Acute SNS and HPA responses mobilize energy reserves; protect against septic shock and nutrient deprivation; permit fight or flight responses that are normally protective against danger. • Inflammation accelerates the healing of wounds.	Central to theory	Central to theory
Changes in allostatic mechanisms	• Increased inflammatory tone • Elevated cortisol and catecholamines • Muted cardiovascular responses to stress	Central to theory	Central to theory
Cognitive, behavioral, and emotional impairments in children	• Reduced scores on standard tests of intelligence, language, memory, and other abilities • Early onset and increased prevalence of psychopathology	Not inconsistent with theory	Central to theory
Cognitive, behavioral, and emotional adaptations to stress in children	• Tailoring of emotion systems, arousal responses, and perceptual abilities to the detection and monitoring of danger • Development of insecure attachments, mistrustful internal working models, opportunistic interpersonal orientations, oppositional-aggressive behavior.	Central to theory	Not inconsistent with theory
Long-term deleterious outcomes	• Cognitive and physical impairments • Depression • Increased risk of cardiovascular disease and all-cause mortality	Not inconsistent with theory	Central to theory
Long-term adaptive changes in biobehavioral systems	• Adaptive calibration of autonomic, neuroendocrine, metabolic, and immunological systems • Regulation of alternative life history strategies to match ecological conditions	Central to theory	Beyond the scope of the theory

Note. Light shading indicates a difference in emphasis between the ACM and ALM. Dark shading indicates a qualitative divergence between the two theories. Reprinted from Ellis & Del Giudice (2013).

Cost-Benefit Trade-Offs in the Development of Alternative Phenotypes.

The ACM and ALM diverge considerably in how they deal with cost-benefit trade-offs, individual differences, and long-term developmental changes. From an evolutionary standpoint, the ALM makes no distinction between the two meanings of "adaptive" and "maladaptive," as conceptualized from a public health versus evolutionary perspective. Indeed, maladaptation is typically inferred whenever there are substantial costs to the organism. For example, if elevated

cortisol levels in adolescents are associated with an undesirable outcome, such as reduced working memory, then elevated cortisol is classified as a marker of allostatic load (see Juster et al., 2011). This reasoning ignores the crucial fact that *biological processes are maintained by natural selection when their fitness benefits outweigh the costs, not when they are cost-free*; indeed, even large costs can be offset by large enough expected benefits. Although there are practical reasons for identifying allostatic load biomarkers, this approach alone is incomplete because it only specifies dysfunction and not the adaptive functions of developmentally calibrated biological parameters.

Because of the failure to distinguish between (mal)adaptive and (un)desirable outcomes, most applications of the ALM do not adequately address the trade-offs involved in the development of physiological and behavioral phenotypes; as a consequence, the ALM literature often lacks a theory of adaptive individual variation in stress responsivity (see Korte et al., 2005, for a notable exception). Instead the ALM focuses on optimal SRS parameter values, as defined by covariation with desirable health outcomes; deviations from these optimal settings form the basis of "dysregulation." The applied goal of the ALM is to identify non-optimal autonomic, neuroendocrine, metabolic, and immune profiles that predict psychiatric and biomedical disorder (Table 1: *Long-term deleterious outcomes*).

In contrast, the ACM emphasizes adaptation in context and posits that optimal SRS parameter values vary as a function of environmental conditions. From this perspective, the notion of globally optimal baseline or responsivity levels for SRS parameters is highly problematic; indeed, the entire literature on biological sensitivity to context demonstrates that the value of hypo-responsivity versus hyper-responsivity is context-dependent (see extended discussion below). The ACM gives full consideration to the costs and benefits of SRS responsivity. For example, consider heightened stress responsivity in a dangerous, unpredictable environment. In the ACM, it is hypothesized that the costs of repeated SRS activation are offset by improved management of danger. Although the system is on a hair trigger, with a resulting increase in anxiety and/or aggression, few instances of actual danger will be missed. In addition, engaging in a fast, present-oriented life history strategy makes it optimal to discount the long-term health costs of chronic SRS activation if the immediate benefits are large enough (for in-depth discussion, see Del Giudice et al., 2011).

In the ALM framework, the same pattern of responsivity would be treated as dysfunctional because the stress response is deployed even in absence of true dangers (e.g., "excessive" response, "unnecessary" triggering; e.g., Lupien et al., 2006) and because of the associated undesirable states and health risks (e.g., interpersonal distress). However, this approach fails to consider that biological defenses are usually designed by natural selection to accept a high rate of false positives. In most instances, unnecessary responding is an adaptive feature of the system—though a costly one—rather than a sign of dysregulation or malfunction.

Long-Term Adaptations to Stress: The Developmental Regulation of Alternative Life History Strategies

According to the ACM, childhood adaptations to stress may eventuate in long-term adaptive changes in biobehavioral systems. Herein lies the key difference between the ACM and ALM (dark shaded region of Table 1). In the ALM, energy devoted to mounting autonomic, neuroendocrine, metabolic, and immune responses to threat is traded off against "wear and tear" on multiple organ systems. The ACM extends this logic by conceptualizing these trade-offs as

decision nodes in allocation of resources. It is through this chain of resource-allocation decisions—instantiated in the regulatory parameters of the SRS and related biological systems—that the developing organism adapts to local conditions. Thus, the ACM shifts the emphasis from dysregulation to conditional adaptation (Table 1: *Long-term adaptive changes in biobehavioral systems*).

From an evolutionary perspective, increased "wear and tear" is a cost of pursuing a fast life history strategy. The fast strategy is instantiated in a chain of resource allocation decisions over the life course that "make the best of a bad situation" by trading off survival for reproduction. Thus, many biologically embedded changes that the ALM conceptualizes as costs (e.g., heightened HPA reactivity) the ACM views as decision nodes in development of a faster strategy. Conversely, slower life history strategies involve greater allocation of resources toward enhancing growth, vitality, and long-term survival (e.g., DNA repair). Development of a fast life history strategy in dangerous and unpredictable contexts is not impairment or dysfunction; it is a coherent, organized response to stress that has been shaped by a natural selective history of recurring exposures to harsh and unpredictable environments.

Implications for the Core Points of Developmental Psychopathology

The integrative perspective on stress responsivity presented in this section touches on many core points of developmental psychopathology. First of all, the ACM illustrates that evolutionary principles are not just relevant to the behavioral level of analysis. On the contrary, they can be fruitfully applied to understand the role of *brain and neurobiological factors* in development, and employed to craft detailed models of neurobiological functioning. Like the ALM, the ACM helps synthesize *normal and pathological development* in a single framework; however, the ACM goes beyond the ALM with a broader view of costs and benefits and a detailed theory of adaptive matching between environment and phenotype. The ACM fully incorporates the *contextualism* of life history theory; in this perspective there is no single optimal phenotype or developmental trajectory—only locally adaptive, contextually sensitive strategies with their baggage of costs and trade-offs.

An important feature of the logic of responsivity patterns is that it invites—indeed, virtually requires—a *person-centered approach* to individual differences in stress physiology and neurobiology (e.g., Del Giudice et al., 2012). Even the schematic model depicted in Figure 5 shows that causal relations between environmental variables and developmental outcomes are predicted to be highly *nonlinear;* if the model is correct, attempts to model stress responsivity with standard linear models are doomed to yield inconsistent and misleading results. Responsivity patterns also provide a striking illustration of *equifinality* and *multifinality* in the interplay between environment, neurobiology, and behavior. In the ACM, dramatically different environments may entrain the development of similar responsivity profiles; for example, sensitive and vigilant patterns are both hypothesized to imply high HPA responsivity. In turn, similar responsivity profiles may predict remarkably different behavioral correlates—for example low impulsivity in sensitive patterns versus high impulsivity in vigilant patterns. While the resulting developmental trajectories may look empirically baffling, they become tractable when they are framed in the appropriate functional perspective.

Finally, the ACM potentially explains why, in the empirical literature, heightened and dampened stress responsivity seem to work as *risk factors* in some studies and as *protective factors* in others. Both sensitive and buffered phenotypes are associated with lower levels of

psychosocial stress and concomitant development of slower life history strategies, whereas both vigilant phenotypes and unemotional phenotypes are associated with higher levels of psychosocial stress and concomitant development of faster life history strategies (Figure 5). Depending on the specific outcomes under investigation, heightened reactivity may look like a protective factor in sensitive phenotypes and a risk factor in vigilant phenotypes, while dampened reactivity may look like a protective factor in buffered phenotypes and a risk factor in unemotional phenotypes. As we discuss in a later section, the picture is further complicated by the possibility that some disorders are actually more likely to occur at the *slow* end of the life history continuum. Taming this complex interplay of cause and effect will only be possible with the help of a detailed theory of biological and developmental trade-offs.

Beyond Diathesis-Stress: Differential Susceptibility to Environmental Influences

As discussed in the preceding sections, early experiences can have profound and lasting effects on psychological development. Nevertheless, people vary dramatically in the extent to which they respond to their social and physical environment. Similar developmental experiences may have profound effects on some individuals and slight or even negligible effects on others. The idea that some individuals are more susceptible than others to environmental adversity has a long history in psychology, and is captured by the complementary concepts of *vulnerability* and *resilience* (for a recent review see Compas & Andreotti, 2013). Individual differences in vulnerability may be determined by a combination of genetic factors and previous experiences, and explain why, for example, only some children develop such undesirable outcomes as aggression, insecure attachment, and cognitive difficulties in response to stressful or impoverished rearing experiences. The concept of vulnerability lies at the heart of the diathesis-stress model of psychopathology, which is arguably the dominant paradigm in the field.

Against this background, recent empirical and theoretical advances have brought about a conceptual revolution in the study of organism x environment interactions. As has become increasingly apparent, many of the same factors that determine increased vulnerability to stress and adversity also confer enhanced responsivity to the *positive*, supportive aspects of the environment (Belsky, Bakermans-Kranenburg, & Van IJzendoorn, 2007; Boyce & Ellis, 2005). No longer confined to vulnerability, susceptibility to environmental influence can be understood as a generalized trait that increases the range of potential developmental outcomes in a bivalent fashion. While some individuals are disproportionately likely to suffer damage if exposed to harshness and adversity, they are also disproportionately likely to benefit from nurturance and support. Here we introduce the concept of *differential susceptibility*, discuss its importance for developmental processes, and review the models that have been proposed to explain the evolution of systematic individual differences in susceptibility to the environment. For further discussion see Ellis, Boyce, Belsky, Bakermans-Kranenburg, and Van IJzendoorn (2011a) and Del Giudice (under review).

Differential Susceptibility: Orchids and Dandelions

In the differential susceptibility literature, highly susceptible children are referred to by the shorthand designation of *orchid children*, signifying their special sensitivity to both highly stressful and highly nurturing environments. For example, highly irritable infants tend to become less sociable than moderately irritable infants when they are insecurely attached, but *more* sociable if they experience secure attachment relationships (Stupica, Sherman, & Cassidy, 2011). Similarly, children with difficult temperament develop more behavior problems when they experience low-quality care, but *fewer* behavior problems when reared in a high-quality context (Pluess & Belsky, 2009). Children who are low in susceptibility to environmental influence, on the other hand, are designated as *dandelion children*, reflecting their relative ability to function adequately in species-typical circumstances of all varieties (Boyce & Ellis, 2005). Differential susceptibility can be distinguished from both vulnerability (specific sensitivity to negative environmental factors) and the more recent concept of *vantage sensitivity* (specific sensitivity to positive environmental factors; see Pluess & Belsky, 2012).

Converging evidence from different areas of research indicates that highly susceptible children share a cluster of interrelated traits including high physiological reactivity across multiple systems (including the SRS), negative emotionality, and "difficult" temperament (reviewed in Belsky et al., 2007; Boyce & Ellis, 2005; Ellis et al., 2011a). While the genetics of susceptibility is still incompletely understood, genes involved in serotonergic and dopaminergic pathways appear to play a central role (e.g., Bakermans-Kranenburg & Van IJzendoorn, 2011; Belsky & Beaver, 2011; Heiming & Sachser, 2011); as discussed above, serotonergic and dopaminergic pathways interact bidirectionally with the SRS. Together, these interconnected systems contribute to a general phenotypic dimension of *neurobiological susceptibility* to the environment (see Boyce & Ellis, 2005; Ellis et al., 2011a).

Figure 6 illustrates the concept of differential susceptibility by showing the reaction norms of high susceptibility and low susceptibility individuals in a population. Differential susceptibility requires that reaction norms cross somewhere in the middle range of environmental variation. Figure 6 may represent either a genotype x environment (GxE) or phenotype x environment (PxE) interaction, depending on whether individuals are distinguished by their genotype (e.g., variants of the serotonin transporter gene) or phenotype (e.g., high stress responsivity, negative emotionality). Note that reaction norms do not necessarily have to be linear as in Figure 6; indeed, mathematical models of differential susceptibility indicate that, in a broad range of conditions, GxE reaction norms can be expected to have a nonlinear shape (see Del Giudice, under review).

In summary, differential susceptibility is a special case of developmental plasticity; its defining characteristics are (a) a crossover interaction in which more susceptible individuals have a broader reaction range and steeper reaction norms (compare with Figure 2b), and (b) a clear positive-negative polarity of the relevant environmental dimension. These two factors together result in systematic variation in the strength of relations between environmental exposures and developmental outcomes that maps on to the orchid-dandelion distinction. Whether environmental conditions can be labeled as "positive" or "negative" in a biological sense depends on their likely effects on an individual's fitness. As we discussed in the section on life history theory, many of the negative environmental factors investigated by developmental psychologists are ultimately correlated with danger, unpredictability, and resource scarcity.

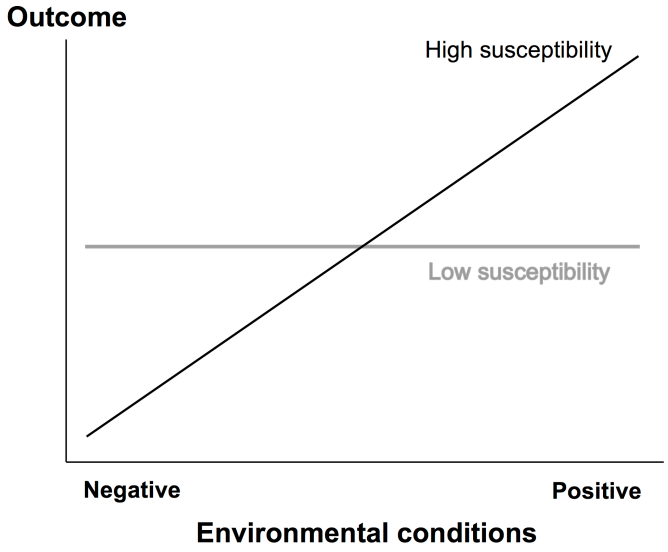

Figure 6. Reaction norms showing differential susceptibility. More susceptible individuals are disproportionally affected by both negative and positive environments.

Differential susceptibility has far-reaching implications for developmental psychology and psychopathology; it moderates the effects of environmental exposures on developmental and life outcomes. Ultimately, this means that the development of some individuals, more than others, will be influenced by their experiences and environments—even if these were exactly the same. Differential susceptibility to the environment, therefore, constitutes a central mechanism in the regulation of alternative patterns of human development. Over the last few years, the concept of differential susceptibility has generated considerable enthusiasm in developmental psychology, and has rapidly become the foundation of an expanding empirical literature. Evidence of differential susceptibility exists for a number of traits and outcomes including prosociality, sociability, externalizing and internalizing symptoms, depression, and timing of puberty (see Ellis & Boyce, 2011, and other papers in that Special Issue).

Evolutionary Models of Differential Susceptibility

Individual differences in neurobiological susceptibility to the environment raise many important questions. Why do they exist in the first place? Are they adaptive—and if so, what evolutionary processes are responsible for their maintenance in human populations? When do they emerge in development, and by what interplay of genetic and environmental factors? Evolutionary models of differential susceptibility (Belsky, 1997, 2005; Boyce & Ellis, 2005; Ellis et al., 2006) have explored different explanations for the maintenance of individual differences—balancing selection, selection for conditional adaptation, and selection for diversified bet-hedging. These explanations are not mutually exclusive, and indeed they can potentially be integrated within a single theoretical framework (see Del Giudice, under review). We now briefly review each in turn.

Maintenance of Differential Susceptibility through Balancing Selection

There is considerable evidence that individual differences in susceptibility can be attributed—at least in part—to individual differences in genotype. The best examples include allelic variation in the serotonin-transporter gene promoter (*5-HTTPLR*) and the dopamine D4 receptor gene (*DRD4*). Recent meta-analyses have demonstrated that these polymorphisms moderate environmental effects in a *for better and for worse* manner; that is, different variants of these genes are generally associated with differential susceptibility to both stressful and nurturing environmental conditions (Bakermans-Kranenburg & van IJzendoorn, 2011; van Ijzendoorn, Belsky, & Bakermans-Kranenburg, 2012). Other genetic variants involved in differential susceptibility have been identified, not only in dopaminergic and serotonergic pathways but also in the oxytocinergic system and the HPA axis (see Cicchetti & Rogosch, 2012; Ellis et al., 2011a).

As discussed in a previous section, adaptive genotypic differences in a population may be maintained by various forms of balancing selection, including negative frequency-dependent selection. Building on the work of Wilson and Yoshimura (1994), Ellis and colleagues (2006) proposed a negative frequency-dependent model of the maintenance of genetically-regulated variation in differential susceptibility to the environment. In this model, developmental specialists are defined by relatively flat reaction norms, such that phenotypic development is minimally sensitive to normal environmental variation. Different developmental specialists are instead characterized by different genetically-regulated behavioral types (e.g., shy vs. bold, slow vs. fast life history strategy). Although developmental specialists lack plasticity, their specialized personalities enable them to thrive in the niche that they are specialized to exploit. Developmental generalists, by contrast, are defined by relatively steep reaction norms, such that phenotypic development is highly context-dependent. Different developmental generalists thus experience different developmental outcomes based on their rearing experiences. All else being equal, the presence of multiple niches in a single environment will favor developmental specialists over developmental generalists when individuals can evaluate and select niches that increase their fitness. This is because specialists outperform generalists in their preferred niche. However, multi-niche environments are often characterized by negative density-dependence, meaning that as a given niche becomes more crowded (i.e., over-exploited relative to its size), the fitness benefits of specializing in that niche decrease. This is the cost of specialization.

Models of genotypic variation in susceptibility are still in the formative stages. The general hypothesis that frequency-dependent selection can maintain individual variation in susceptibility or responsiveness to the environment is well supported by mathematical models (Wilson & Yoshimura, 1994; see also Wolf, van Doorn, & Weissing, 2008) but has not yet generated empirical work in humans. Another plausible but less explored possibility is that other types of balancing selection—for example spatially and temporally fluctuating selection—may coexist with frequency-dependent selection and contribute to maintain genotypic variation in human populations (see Del Giudice, under review; Ellis et al., 2011a).

Development of Differential Susceptibility through Conditional Adaptation

Depending on the structure of the environment and of an organism's life cycle, conditional adaptation can be maintained alongside genetic variation by spatially and temporally fluctuating selection pressures (see Del Giudice, 2012, under review). The theory of Biological Sensitivity to Context (Boyce & Ellis, 2005) posits a *non-random distribution* of neurobiological susceptibility to the environment within populations that emerges through conditional adaptation to variable environmental conditions. From this perspective, differential susceptibility results, in part, from individuals tracking different environmental conditions and altering their development to match those conditions. The assumption is that this matching process promoted fitness across heterogeneous environmental contexts over human evolution.

Biological sensitivity to context theory identified physiological mechanisms of differential susceptibility—autonomic, adrenocortical, and immune reactivity to psychosocial stressors—and proposed that this psychobiologic reactivity moderated the effects of early environmental exposures on physical and mental health outcomes in the bivalent manner discussed above (reviewed in Belsky & Pluess, 2009; Ellis et al., 2011a). This dual function signified the need to conceptualize stress reactivity more broadly as biological sensitivity to context, which Boyce and Ellis (2005) defined as neurobiological susceptibility to both cost-inflicting and benefit-conferring features of the environment and operationalized as heightened reactivity in one or more of the stress response systems. In total, biological sensitivity to context theory proposed that individual differences in the magnitude of biological stress responses function to regulate openness or susceptibility to environmental influences, ranging from harmful to protective.

As discussed in the previous section, the ACM explicitly builds on the logic of biological sensitivity to context. In a nutshell, the theory of biological sensitivity to context proposes that heightened susceptibility is adaptive at both ends of the environmental continuum—in both highly stressful and highly protected environments. This results in a U-shaped curvilinear relation between early stress/adversity and susceptibility to later environmental effects (Boyce & Ellis, 2005). Consistent with the focus of biological sensitivity to context on adaptive plasticity, the ACM views early experience as a crucial determinant of individual differences in neurobiological susceptibility to the environment. Potential cues of danger and adversity versus safety and support include prenatal stress (e.g., fetal exposure to stress hormones), parenting quality, family stability and conflict, attachment security, neighborhood quality, and so forth.

These contextual factors are hypothesized to affect development mainly by up- or down-regulating the activity of infants' and children's stress response systems, as shown in Figure 5. Whereas the original conceptualization of biological sensitivity context focused primarily on the functions of high stress responsivity, the ACM expanded this conceptualization to encompass the functions of low stress responsivity—and thus added the unemotional pattern shown in Figure 5. This addition turned the original U-shaped curve into a more complex function (see discussion of the ACM responsivity patterns in the previous section). Like the theory of biological sensitivity to context, the ACM conceptualizes variation in neurobiological susceptibility to the environment as part of a conditional adaptation process that matches stress response profiles to local conditions. This conceptualization is supported by a large empirical literature demonstrating changes in SNS, PNS, and HPA parameter values over development in response to different developmental experiences (reviewed in Boyce & Ellis, 2005; Del Giudice et al., 2011),

including prenatal experiences such as infections, fetal undernutrition, and exposure to maternal stress hormones (Pluess & Belsky, 2011).

Differential Susceptibility as Adaptive Stochastic Variation

According to the theory of differential susceptibility advanced by Belsky (1997, 2005), the main adaptive function of differential susceptibility is spreading the risk of mismatch by making some individuals resistant to environmental influences, including—but not limited to—the influences of parental behavior on children's development. When early cues correctly predict future states of the environment, susceptible individuals benefit from enhanced phenotype-environment matching; but when prediction fails (for example because the environment undergoes sudden and unpredictable changes), they end up developing a mismatched phenotype and potentially suffering large fitness costs as a result. Individuals who follow a fixed developmental trajectory may avoid the fate of their more susceptible counterparts when ecological cues fail to predict later environmental states. Thus, for a parent, producing offspring with varying degrees of susceptibility works as an insurance against future unpredictability.

In evolutionary terms, this is an example of reproductive *bet-hedging*—a strategy that reduces average individual fitness in the short term, but enhances the long-term reproductive success of the genetic lineage by decreasing fitness variance across generations (Bull, 1987; Seger & Brockmann, 1987; Starrfelt & Kokko, 2012). More specifically, differential susceptibility is conceptualized as an instance of *diversified* bet-hedging, that is, a strategy that reduces fitness variance between generations by increasing phenotypic variability among offspring (Philippi & Seger, 1989). Such a strategy could be maintained by an evolutionary history of exposure to environments that fluctuated unpredictably over time. Bet-hedging strategies increase the probability of achieving some reproductive success in every generation while limiting success in good conditions and shielding against total failure in bad (for a detailed treatment see Starrfelt & Kokko, 2012).

Theory and data from evolutionary biology indicate that fluctuating selection pressures, if sufficiently strong, can support variable or random generation of offspring phenotypes ("adaptive coin-flipping") arising from a monomorphic genetic structure. This strategy can be implemented through a stochastic developmental switch (presumably instantiated by epigenetic mechanisms), which generates one of several alternative phenotypes according to a probabilistic rule. For example, in a range of animal species, when mothers cannot forecast the likely environment of their offspring, or environmental cues in the maternal generation suggest that the offspring environment is likely to vary unpredictably, mothers hedge their bets by increasing variation in offspring phenotypes (Crean & Marshall, 2009). Although temporally fluctuating selection pressures can select for stochastic phenotypic variation, diversified bet-hedging cannot be instantiated through *genetic* polymorphisms because the resulting phenotypes do not all have the same average fitness (Bull, 1987; Philippi & Seger, 1989). Thus, the bet-hedging hypothesis can explain adaptive stochastic variation in susceptibility but not genotypic variation, in contrast with early formulations of the theory (Belsky, 1997). More recent formulations of the theory (e.g., Belsky, 2005; Pluess & Belsky, 2011) also acknowledge the role of early environmental influences—especially prenatal exposure to stress—in shaping individual levels of susceptibility.

In total, the bet-hedging hypothesis of differential susceptibility is valuable in that it provides a plausible adaptive explanation for stochastic variation in susceptibility to environmental influence. More generally, the larger framework advanced by Belsky (2005)

emphasizes the potential adaptive significance of unsystematic within-family variability in susceptibility to the environment. However, adaptive stochastic variation coexists with many sources of neutral or maladaptive unsystematic variation, which result from the random effects of sexual recombination, non-adaptive phenotypic plasticity, random perturbations in developmental processes, harmful genetic and epigenetic mutations, and so forth. The empirical challenge for the theory advanced by Belsky (1997, 2005) is that it may be extremely difficult to distinguish between adaptive within-family variation that has been shaped by natural selection and within-family variation that is truly random and non-adaptive (see Ellis et al., 2011a).

Differential Susceptibility as a Model of Organism-Environment Interplay: The Case of Pubertal Development

The differential susceptibility framework returns us to the centrality of the phenotype and the complexity of organism-environment interplay. Susceptibility to the environment is instantiated in the biology of the nervous system; it is *neurobiological* susceptibility. Genetic susceptibility factors operate through neurobiological processes and behavioral indicators of susceptibility are grounded in neurobiology. Neurobiological susceptibility itself is not a static trait; it develops and changes in response to genetic and environmental influences, which become incorporated into the phenotype over time. Genetic factors, environmental factors, and the extant phenotype all interact in intricate ways that are not yet entirely understood, including both systematic effects and stochastic processes.

A good demonstration of the real-life complexity of differential susceptibility is the interplay of early life stress, stress responsivity, and polymorphic variation in the estrogen receptor α gene (*ESR1*) in the regulation of pubertal development. The timing of pubertal development is a central life history trait; as discussed in a previous section, Belsky and colleagues (1991) first predicted that early stress would entrain fast life history strategies, leading to accelerated puberty timing (the *psychosocial acceleration theory*; see Ellis, 2004). Specifically, the theory posits that ecological stressors in and around the family create conditions that undermine parental functioning and lower the quality of parental investment—such as by escalating marital conflict, increasing negativity and coercion in parent-child relationships, and reducing positivity and support in parent-child relationships. According to the theory, children respond to these familial and ecological conditions (particularly those experienced in the first 5-7 years of life) by developing in a manner that speeds up pubertal maturation, anticipates the onset of sexual activity, and promotes the development of a cluster of behavioral traits associated with fast life histories, including impulsivity and unstable pair bonds.

A study by Ellis and Essex (2007) investigated the psychosocial acceleration theory in the Wisconsin Study of Families and Work. Consistent with predictions, higher quality parent-child relationships in preschool—more parental warmth and family positivity, less parent-child stress and conflict—forecast slower pubertal maturation in daughters. Although this association proved robust, the unique effect of parent-child relationships on puberty was relatively small. However, theories of differential susceptibility suggest that the weak main effects of environmental variables on many developmental outcomes may often reflect the fact the children differ in whether, how, and how much they are affected by rearing experiences. As articulated by Belsky (2000), the weak main effects of parenting variables on pubertal timing may overestimate the impact of family environments in some children (dandelions, more fixed reproductive development) and underestimate it in others (orchids, more plastic reproductive development).

This hypothesis was supported in a later study of the same dataset (Ellis, Shirtcliff, Boyce, Deadorff, & Essex, 2011b). Specifically, higher SRS responsivity in 1st grade—a key facet of neurobiological susceptibility—moderated the effects of parental behavior on maturation: lower-quality parent-child relationships forecast faster initial tempo of puberty and earlier pubertal timing, but only among children showing heightened SNS/HPA reactivity. In other words, the data revealed a clear PxE interaction between parent-child relationships and SRS responsivity, consistent with the biological sensitivity to context account of differential susceptibility (Boyce & Ellis, 2005).

This, however, is only part of the story. Neurobiological susceptibility is not a fixed trait but develops over time through the interplay of various causal factors—including genetic variation and early environmental effects. In the same dataset, growing up in either highly supportive or highly stressful home environments predicted development of high SNS reactivity (consistent with the U-shaped curve predicted by biological sensitivity to context theory; see above), whereas growing up in a highly stressful home environment predicted heightened HPA activation (Ellis, Essex, & Boyce, 2005). Thus, under conditions of early environmental stress and uncertainty, indexed by coercive and unsupportive family relationships, individuals developed heightened neurobiological susceptibility to the environment (as indexed by SNS and HPA activation) and subsequently accelerated pubertal maturation in early adolescence, with family stress and neurobiological susceptibility acting synergistically in this process. Heightened neurobiological susceptibility thus enabled a stronger pubertal response to adversity. According to psychosocial acceleration theory, this response may represent a strategic—that is, functional—way of developing under stress.

The other side of the coin is that under conditions of early environmental protection and stability, indexed by positive and supportive family relationships, individuals also developed heightened neurobiological susceptibility, which in combination with high-quality parent-child relationships forecast slower initial pubertal tempo and later pubertal timing (Ellis et al., 2011b). In this case, heightened neurobiological susceptibility enhanced responsiveness to environmental resources and support. As suggested by life history models, the resulting pattern of late sexual maturation may also constitute adaptive variation. Specifically, Ellis (2004) hypothesized that children have been selected to capitalize on the benefits of high quality parental investment and reduce the costs of low quality parental investment by contingently altering the length of childhood. Given high family resources and support and biological sensitivity to these development-enhancing contexts, extending childhood by delaying onset of puberty or slowing pubertal tempo may function to improve socio-competitive competencies (i.e., embodied capital) that ultimately increase reproductive potential.

Timing of puberty does not only depend on the environment, and indeed, all measures of pubertal development show heritability values indicative of robust genotypic effects (see Ellis, 2004). As can be expected from the vantage point of differential susceptibility theory, recent evidence indicates that GxE interactions are also involved in the regulation of pubertal development (Manuck, Craig, Flory, Halder, & Ferrell, 2011). Consistent with past research (reviewed in Ellis, 2004), Manuck and colleagues (2011) found that women who reported being raised in families characterized by distant interpersonal relationships and high levels of conflict tended to reach menarche earlier than women raised in close families with little discord. However, this effect was moderated by variation in the gene coding for estrogen receptor α (*ESR1*). Among women who were homozygous for minor alleles of the two *ESR1* polymorphisms examined in the study, a childhood history of low-quality family relationships

was associated with earlier age of menarche compared with a childhood history of high-quality family relationships; no such effect was found among women with other *ESR1* genotypes. Intriguingly, estrogen receptor α is highly expressed in the hypothalamus, where it regulates the activity of the HPA axis (Bao, Meynen, & Swaab, 2008). Thus, among its other possible effects, variation in *ESR1* may affect pubertal development by contributing to the development of higher or lower levels of neurobiological susceptibility.

In summary, regulation of pubertal development depends on genetic factors and GxE interactions, such as between *ESR1* variation and family stress; on environmental factors, such as energetics and psychosocial stress; on interactions between these environmental factors and extant phenotypic characteristics that modulate neurobiological susceptibility to the environment, such as SRS responsivity; on the developmental calibration of these stress response systems, which filter and embed information about environmental stress and support, mediating the organism's openness to environmental inputs; and on genetic influences and GxE interactions in the regulation of neurobiological susceptibility to the environment (see extended discussion in Ellis, 2013). At the nexus of all of these processes is the organized phenotype, which exists from conception; modulates, integrates, and retains genetic and environmental effects; and is the basis of differential susceptibility. Yet even if all of these factors are taken into account, there is still be much unexplained variation due to stochastic developmental processes.

This is the tangled web of development. Theoretical models propose that differential susceptibility develops through a mixture of genetic regulation—maintained by balancing selection—and environmental regulation that enables conditional adaptation. Whereas balancing selection and conditional adaptation result in systematic variation in neurobiological susceptibility to the environment, unsystematic variation may also be maintained by natural selection as an insurance policy against unpredictably changing environments (diversified bet-hedging). Taken together, these evolutionary processes may result in large individual differences in whether, how, and how much people are affected by their experiences. Differential susceptibility, therefore, can be expected to play a key role in moderating the effects of environmental conditions on developmental outcomes, including the development of life history strategies.

Implications for the Core Points of Developmental Psychopathology

The theory of differential susceptibility brings about a fundamental change in the way one thinks about *risk* and *resilience* in development. First, the same genetic or phenotypic factor can behave as *both* a risk and a protective factor depending on the ecological context. Second, the very children whose heightened responsivity appears to make them vulnerable to developing psychopathology (orchid children) may also be most able to benefit from positive, supportive environments and interventions. The divergent outcomes associated with highly susceptible phenotypes also set the stage for pervasive, systematic manifestations of *multifinality* in development.

As we have seen, the development of susceptibility is itself a dynamic process, taking place through an intricate interplay of genetic, environmental, and stochastic effects. This perspective combines the *contextualism* of conditional adaptation with new and potentially fruitful explanations of *probabilistic causality*. Whereas standard approaches tend to see probabilistic causality as opposed to biological adaptation, the theory of bet-hedging shows how

natural selection can make adaptive use of developmental randomness as a response to unpredictable environmental change.

Still other implications for developmental psychopathology stem from the centrality of the phenotype in regulating differential susceptibility. First, a view of the phenotype as the result of sequential organism-environment interplay blurs the distinction between variable-centered and *person-centered approaches*. Second, the concept of neurobiological susceptibility is a powerful reminder that genetic, epigenetic, and environmental effects must ultimately be understood in terms of their influence on *brain and neurobiological* pathways; it also illustrates how multiple pathways and molecules—for example serotonin, dopamine, cortisol, and sex hormones—may exert synergistic effects by converging on general, adaptive dimensions of phenotypic variation.

Beyond the DSM: A Life History Framework for Mental Disorders

In the preceding sections we showed that EDP provides an integrative biological perspective on human development, and sheds light on the origin and function of individual differences in life history strategy, developmental plasticity, and physiological responsivity to stress. We now take this approach one step further and show how the principles of life history theory can be employed to outline a unifying framework for the analysis and classification of mental disorders (for a detailed exposition see Del Giudice, in press). For the sake of simplicity and consistency with standard usage, in this section we employ the term "disorder" as a synonym for "diagnosable condition"— regardless of whether the condition represents a harmful dysfunction in the narrow sense (Wakefield, 1992).

Limitations of Current Taxonomic Approaches

The dominant approach to the classification of mental disorders is that of the Diagnostic and Statistical Manual (DSM) of the American Psychiatric Association (2013). In the DSM, disorders are defined by lists of symptoms, and grouped together mainly on the basis of symptom similarity—in keeping with the atheoretical stance embodied by the Manual. Thus, DSM-5 categories include, for example, *anxiety disorders*; *disruptive, impulse-control, and related disorders*; *depressive disorders*; *obsessive-compulsive and related disorders*; and *feeding and eating disorders* (American Psychiatric Association, 2013).

While the DSM system has many undisputable qualities—diagnostic reliability above all—it also has a number of significant problems (see e.g., Beauchaine, Klein, Erickson, & Norris, 2013; Nesse & Jackson, 2006). The main limitation of the DSM is also its defining feature: the deliberate absence of a theoretical model of mental disorders. Since disorders and disorder categories are primarily defined by symptom similarity, many diagnostic classes are likely to include a heterogeneous mix of conditions with different etiological, developmental, and functional characteristics. More troubling, the DSM lacks a theory of normative mental functioning, and is therefore ill equipped to discriminate between adaptive defensive responses and disordered functioning (Nesse & Jackson, 2006; see above).

The main alternative to the DSM system comes from a family of empirical approaches based on patterns of genetic and phenotypic correlations between disorders (e.g., Kendler, Prescott, Myers, & Neale, 2003; Lahey, Van Hulle, Singh, Waldman, & Rathouz, 2011; Lahey et

al., 2008; Watson, 2005). Empirical taxonomic studies suggest the existence of broad, hierarchically organized clusters of disorders that overlap only in part with DSM categories. The fundamental distinction in empirical taxonomies is that between *internalizing* and *externalizing* disorders. Externalizing disorders are characterized by impulsivity, disinhibition, and high levels of aggressive, antisocial, and/or disruptive behavior. Internalizing disorders are marked by high levels of anxiety and negative emotionality. The internalizing spectrum comprises a cluster of *distress disorders* (depression, generalized anxiety disorder [GAD], post-traumatic stress disorder [PTSD]) and a cluster of *fear disorders* (panic disorder, agoraphobia, social phobia, and specific phobias; Clark & Watson, 2006). Obsessive-compulsive spectrum disorders are typically treated as a separate internalizing cluster; other disorders that are usually included in the internalizing spectrum are bipolar disorders and borderline personality disorder (BPD), although their exact placement is more problematic (see Beauchaine & Hinshaw, 2013; Lahey et al., 2008; Watson, 2005). A recent factor-analytic study by Caspi and colleagues (2013) supplemented the internalizing and externalizing categories with a *thought disorder* factor comprising schizophrenia, mania (bipolar spectrum), and obsessive-compulsive disorder (OCD). Moreover, the authors identified a general, higher-order factor of psychopathological risk they labeled the *p factor* (see Caspi et al., 2013).

The internalizing-externalizing distinction has received considerable empirical support, and has become a standard tool in developmental psychopathology (Beauchaine & Hinshaw, 2013). In the latest edition of the DSM, the authors explicitly recognized the usefulness of the broad distinction between internalizing and externalizing disorders (American Psychiatric Association, 2013, p. 13). However, this approach also has a number of important limitations. First of all, disorders are grouped based on their emotional and affective characteristics; however, emotions can serve multiple motivational goals, and associations between emotions and motivational processes are often remarkably non-specific (see Nesse, 2004a). For example, anger can be triggered by aggressive competition, by threats to one's dominance or status, by suffering or witnessing acts of injustice, by separation from an attachment figure, and so forth. Anxiety, shame, and sadness are prominently associated with psychopathology, but their motivational specificity is also extremely low. Thus, classifications of disorders based on emotion and affect are unlikely to reliably capture the underlying motivational structure.

Moving to the empirical level, there is mounting evidence that the internalizing-externalizing dichotomy is riddled with ambiguities and inconsistencies. To begin with, depression and GAD—often regarded as prototypical internalizing disorders—are in fact "bridge" diagnoses that overlap with both internalizing and externalizing disorders at the phenotypic, genetic, and developmental level (e.g., Lahey et al., 2008, 2011). Also, some disorders that are usually considered part of the internalizing spectrum—such as obsessive-compulsive disorder (OCD) and BPD—show atypically large correlations with externalizing disorders (Crowell, Kaufman, & Lenzenweger, 2013; Lahey et al., 2008). Finally, the internalizing-externalizing taxonomy excludes many important pathological conditions—notably schizophrenia, autism, and most personality disorders—because they are not primarily characterized by mood/emotional alterations and do not fit the conceptual distinction between "internalization" and "externalization" (the recent analysis by Caspi and colleagues [2013] is a partial exception).

A Life History Framework for Psychopathology

In the preceding sections we discussed how life history strategies play a central role in the organization of physiology and behavior. They define an organism's priorities and determine the allocation of effort and resources toward competing biological goals. Differences in life history strategy are the joint product of genetic and environmental influences on development, and are reflected in organized patterns of individual differences in motivation, affect, self-regulation, and personality. By organizing individual differences on such a broad scale, life history strategies set the stage for the development of psychopathology. More precisely, individual differences in life history strategy can be expected to determine individual differences in risk profiles for a broad range of mental disorders. As one moves along the fast-slow continuum of life history variation, some disorders and symptoms should become more frequent, while others should become less likely to occur.

The predictable association between life history strategy and risk for psychopathology offers a high-level functional criterion for the classification of mental conditions. This leads to the novel distinction between *fast spectrum* and *slow spectrum* disorders—that is, disorders that cluster at the fast or slow end of the life history continuum (Del Giudice, in press). Until recently, life history approaches to psychopathology have focused almost exclusively on the fast end of the fast-slow continuum. As widely recognized in the literature, fast life history strategies can predispose individuals to a variety of disorders, either as maladaptive outcomes of life history-related traits or potentially adaptive but undesirable behavioral strategies (e.g., Belsky et al., 1991; Brüne et al., 2010; Figueredo & Jacobs, 2010; Jonason, Li, Webster, & Schmitt, 2009; Salmon, Figueredo, & Woodburn, 2009). The framework advanced by Del Giudice (in press) extends this approach by addressing the role of *slow* strategies in setting the stage for the development of mental disorders.

It is important to stress that the functional connection between life history strategy and psychopathology is usually an *indirect* one. Causal pathways to psychopathology involve a multiplicity of traits and mechanisms—including temperament and personality, self-regulatory processes, and so forth. The general idea is that an individual's configuration of life history-related traits may increase the likelihood of developing a certain disorder or cluster of disorders—often in interaction with other causal factors including developmental insults, deleterious genetic and/or epigenetic mutations, infections, nutritional deficits, and psychosocial stressors. The power of life history theory lies in the ability to integrate these diverse etiological processes within a common frame of reference. The result is a large-scale map of the psychopathological landscape organized along the fast-slow axis of life history variation. Such a map is an invaluable guide in understanding comorbidity patterns, since functionally related disorders—for example different disorders in the slow spectrum—can be expected to co-occur more frequently in the same individual. At the same time, the fast-slow distinction can be used to tease apart functionally distinct conditions that coexist within the same descriptive category because of their phenotypic similarity.

In total, a functional analysis based on life history principles helps to "carve nature at its joints" by revealing commonalities between separate categories and suggesting important distinctions between phenotypically similar disorders (Keller & Nesse, 2006). Of course, mental disorders are complex biosocial phenomena, and as such they can be analyzed at many different levels. A life history analysis is only the first step toward a comprehensive functional account of psychopathology: the broad perspective afforded by the fast-slow distinction should be

complemented by narrower accounts focusing on specific motivational/behavioral systems, cognitive mechanisms, genetic pathways, and so forth.

Four Pathways from Life History Strategy to Psychopathology

The general statement that life history strategies set the stage for the development of psychopathology can be supplemented by a finer-grained analysis of the causal pathways that lead to the onset of mental disorders. First of all, some *adaptive life history-related traits may be regarded as symptoms*. This is most likely to happen with fast life history strategies characterized by impulsive, exploitative, or aggressive tendencies. The resulting phenotype may be classified as a disorder, even if it does not reflect maladaptive or dysfunctional processes. Even if they are biologically adaptive, or used to be adaptive in ancestral environments, such strategies may often involve substantial costs in terms of health and emotional well-being. Another important category of adaptive traits that may be diagnosed as symptoms of a disorder is that of aversive defenses. When defenses activate inappropriately and/or respond with excessive intensity, the outcome may be correctly recognized as maladaptive. However, many protective mechanisms have strongly aversive effects, and can be occasionally harmful to the individual. For this reason, they may give rise to undesirable conditions not only when they misfire but also when they respond appropriately in presence of actual threats.

The correlates of life history strategies often include up- or down-regulation of psychological and physiological defensive mechanisms. Up-regulated defenses have a lower threshold for activation and/or respond with higher intensity when they activate. Defense up-regulation can be associated with both fast and slow strategies, although the specific type of mechanism involved is likely to differ between the two. In the context of fast life histories, up-regulated defenses help protect the individual from immediate danger in risky, unpredictable environments. In the context of slow strategies, up-regulated defenses may help the individual prevent dangerous events and avoid potentially risky situations, even if the current environment is reasonably safe. Moreover, protecting oneself from even minor damages and losses contributes to the long-term maintenance of the soma (i.e., somatic effort)—a key priority for slow life history individuals. In contrast, down-regulation of defenses is most likely in the context of fast life history strategies, especially those involving a high degree of risk-taking. As discussed in a previous section, the underlying logic is that, in order to fulfill their purpose, such strategies require insensitivity to threats, dangers, and so forth.

The second pathway from life history strategy to psychopathology derives from the fact that *life history-related traits may be expressed at maladaptive levels*. Even phenotypic traits that are biologically adaptive within a certain range may become maladaptive if they exceed that range. Sometimes, the expected fitness associated with a trait may slowly increase up to an optimal level, then decrease abruptly following a “cliff-edged” function. In such cases, selection for optimal trait levels may result in a high frequency of maladaptive phenotypes that overshoot the fitness optimum (Nesse, 2004b). A trait can reach maladaptive expression levels owing to a combination of genetic, epigenetic, and environmental factors that contribute to push the phenotype in the same direction. In the simplest case, extreme levels of a trait may appear in the offspring of two individuals who are both high on that trait, yet still within the adaptive range. Thus, assortative mating—the tendency for sexual partners to be more similar than average on a certain trait—can increase the risk for psychopathology due to extreme trait values. Parent-offspring conflict and intragenomic conflict are other likely causes of maladaptive trait

expression. When conflict is present, phenotypic development can be conceptualized as the result of opposing forces, much like a game of tug-of-war. If for any reason this dynamic equilibrium is broken (for example, a mutation in the offspring may make it unable to counteract parental manipulation), the resulting imbalance may easily determine dysregulated or pathological outcomes.

In principle, the pathway leading from maladaptive trait expression levels to psychopathology may involve traits associated with both fast and slow life histories. However, there is some evidence that assortative mating on life history-related traits in humans tends to become stronger toward the slow end of the continuum (Figueredo & Wolf, 2009). If so, disorders that involve maladaptive expression levels of adaptive traits should occur more frequently in association with slow strategies, as similarity between parents increases the likelihood that offspring will inherit extreme genotypic combinations.

Third, as noted in a previous section, even *adaptive strategies may yield individually maladaptive outcomes*. Risky strategies are a prime candidate as a systematic source of individually maladaptive outcomes. Life history-related traits can steer individuals on high-risk pathways, thus increasing the likelihood of maladaptive and/or undesirable outcomes in case of strategy failure—even when the strategy is adaptive on average. This is more likely to happen in the context of fast life history strategies, which tend to promote risk-taking and favor the pursuit of large, immediate returns regardless of the potential costs. While some individuals engaging in high-risk strategies may end up developing mental disorders, other individuals expressing the same traits may enjoy desirable and/or biologically adaptive outcomes, often depending on chance and unpredictable contextual factors. Another important category of adaptive traits that systematically produce maladaptive outcomes is that of defensive mechanisms. Following the logic of the smoke detector principle, defensive mechanisms are usually designed to "misfire" occasionally, even in absence of threats. Individual differences in life history strategy are reflected in the calibration of behavioral and/or physiological defenses (see earlier discussion of the ACM), and indirectly affect the risk of inappropriate defense activation.

Fourth and last, *adaptive life history-related traits may increase vulnerability to dysfunction*. While life history traits are designed to promote adaptation, they can nevertheless increase vulnerability to some types of dysfunction as a side effect. For example, some configurations of personality traits within the adaptive range (for example schizotypy or autistic-like personality) may become especially conducive to psychopathology when they are coupled with high mutation load or brain-damaging infections (see Del Giudice, 2010). Also, fast life history-related traits such as risk proneness and future discounting may indirectly increase an individual's exposure to environmental factors such as pathogens. Finally, up-regulated defensive systems are not only more prone to misfiring—they also more vulnerable to actual instances of malfunction and dysregulation (Nesse, 2001). These four pathways from life history strategy to psychopathology are logically distinct but not mutually exclusive, and may coexist in the etiology of any given disorder.

Correlates of Fast and Slow Spectrum Psychopathology

The conceptual distinction between fast and slow spectrum pathology provides a powerful heuristic criterion for the functional classification of mental disorders. Whatever the specific causal pathway (or combination of pathways) that determine the onset of a given disorder, fast spectrum conditions will be associated with traits such as low agreeableness and

conscientiousness, impulsivity, disinhibition, and early sexual maturation (especially in females). Conversely, slow spectrum conditions will exhibit a "signature" of slow life history-related traits in the areas of motivation, self-regulation, personality, and sexual maturation. Correlations between life history-related traits and specific disorders may or may not imply a *causal* role of those traits in the etiology of the disorders. However, regardless of their role in the etiology of a given disorder, life history correlates can be employed as convergent *markers* of the underlying life history strategy. In principle, this approach can be extended to include genetic, epigenetic, and neurobiological markers (e.g., Del Giudice et al., 2011; Figueredo et al., 2004, 2006). A non-exhaustive list of markers of fast and slow spectrum psychopathology is presented in Table 2.

Table 2. Correlates of fast and slow spectrum psychopathology

	Fast spectrum psychopathology	**Slow spectrum psychopathology**
Motivation	Social antagonism	Social compliance, conformity
	Unstable attachments	Stable attachments
	Precocious sexuality	Delayed sexuality
	Sexual promiscuity, high sex drive	Sexual restraint, low sex drive
	Sensation/novelty seeking	Preference for routines
	Risk-taking	Risk aversion, harm prevention
Self-regulation	Disinhibition, impulsivity	Inhibition, restraint
	Discounting of future rewards	Discounting of immediate rewards
Personality traits	Low conscientiousness	High conscientiousness
	Low agreeableness	High agreeableness
Sexual Maturation	Early, fast maturation	Late, slow maturation
Environment	Harsh, unpredictable	Safe, predictable
	High exposure to stressors	Low exposure to stressors

Note. Reprinted from Del Giudice (in press)

A life history perspective yields novel predictions about the environmental correlates of mental disorders. Ecological harshness and unpredictability tend to entrain development of fast life history strategies, while slow strategies are favored in safe and predictable contexts. As a result, many classic risk factors for psychopathology—such as stressful life events, low socioeconomic status, negative family relationships, trauma, and abuse—are predicted to increase the occurrence of fast spectrum disorders, but not that of slow spectrum disorders. On the contrary, slow spectrum disorders should be associated—at least on average—with safe,

predictable environments, higher socioeconomic status, and *reduced* exposure to ecological and family stressors.

Sex Differences

If life history strategies set the stage for psychopathology, sexual asymmetries in life history trade-offs should produce consistent patterns of sex differences in the epidemiology of mental disorders. The first key asymmetry concerns the mating versus parenting trade-off. On average, human males invest more in mating effort and less in parenting effort than females. The intensity of mating effort increases sexual selection for competitive traits such as risk-taking, dominance-seeking, and physical aggression (see Archer, 2009; Wilson et al., 2002). In total, higher mating effort in males should predispose them to fast spectrum disorders characterized by high levels of risk-taking, such as those in the externalizing cluster (Martel, 2013). In contrast, females have generally less to gain and more to lose from high-risk strategies than males and can be expected to invest more effort in somatic maintenance and protection. As a consequence, they should be more prone to develop disorders that involve the up-regulation of protective defenses, and/or to exhibit more psychological and physiological symptoms reflecting defense up-regulation. This prediction applies to disorders across the fast-slow continuum, since up-regulated defenses can be functionally associated with both fast and slow life history strategies. The higher incidence of anxiety disorders in females (see Martel, 2013) is consistent with this prediction.

Another important asymmetry in life history strategy concerns the trade-off between current and future reproduction. As already discussed in the section on life history theory, this trade-off plays a more critical role in the organization of female life history strategies, since decisions concerning reproductive timing are more critical for females than for males. As a consequence, the timing of sexual maturation in females should be more sensitive to cues of danger and unpredictability (James et al., 2011). Indeed, the available data suggest that ecological stress in the first years of life anticipates gonadal puberty in girls, but not in boys (reviewed in Belsky, 2012). In addition, indices of sexual maturation in females can be expected to form a tighter cluster with other life history-related traits including motivation, personality, self-regulation, and so forth. It follows that maturation timing and rate should be stronger predictors of psychopathology in females than in males. This prediction is well supported by empirical research; the bulk of evidence indicates that individual differences in sexual maturation are more robustly associated with psychopathology in girls than in boys (see Ge & Natsuaki, 2010; Mendle, Turkheimer, & Emery, 2007).

Predictions about sex differences based on life history theory (Del Giudice, in press) can be integrated with those from a recent evolutionary model advanced by Martel (2013). Martel employed sexual selection theory to explain the male-biased prevalence of childhood-onset externalizing disorders and the symmetrical, female-based prevalence of adolescent-onset internalizing disorders. Because of differential sexual selection for social dominance versus interpersonal competence in males and females, she also predicted that males should be more sensitive to early environmental stressors related to the broader ecological conditions (including those occurring prenatally), whereas females should be more sensitive to interpersonal stressors occurring around puberty. These predictions are supported by considerable empirical evidence, and can be extended to the neurobiological level to yield insight in the role of prenatal

testosterone, dopamine, and serotonin in the etiology of common mental disorders in the two sexes (reviewed in Martel, 2013).

Toward a Life History Taxonomy of Mental Disorders

The general framework outlined in this section can be applied to individual disorders and categories of disorders, yielding an initial life history taxonomy based on the fast-slow continuum. In what follows, we briefly discuss how six common categories of mental disorders relate to the fast-slow distinction. For an extended analysis and review of the relevant empirical literature, see Del Giudice (in press).

Externalizing disorders

The externalizing spectrum comprises various disorders marked by aggressive, antisocial, and/or disruptive behavior, including oppositional defiant disorder (ODD), conduct disorder (CD), and antisocial personality disorder (APD). Externalizing disorders are also associated with high risk for substance abuse. Disorders in the externalizing spectrum are strongly male-biased and show high phenotypic and genetic correlations with one another, indicating the existence of a coherent, heritable dimension of externalizing behavior. At the same time, the development of externalizing behaviors is strongly conditioned by environmental factors, such as harsh-rejecting parenting, aggregation of high-risk youth in after-school programs, and exposure to neighborhood violence and criminality (reviewed in Beauchaine, Hinshaw, & Pang, 2010).

In a life history perspective, externalizing spectrum disorders are prototypical instances of fast spectrum psychopathology. Externalizing symptoms are associated with impulsivity and undercontrol, early puberty timing and fast sexual maturation in both sexes, earlier onset of sexual activity, and larger numbers of partners in adolescence and young adulthood. Low socioeconomic status, harsh or unpredictable parental discipline, parental conflict, family disruption, and child abuse—all cues of danger and unpredictability—are consistent predictors of externalizing behavior, consistent with predictions derived from life history theory.

Evolutionary models of externalizing spectrum disorders tend to stress the potential biological adaptiveness of aggressive, exploitative, and risky behavior—especially when coupled with promiscuous short-term sexuality (e.g., Barr & Quinsey, 2004; Figueredo & Jacobs, 2010; Jonason et al., 2009; Mealey, 1995). Accordingly, many evolutionary scholars see externalizing disorders as adaptive but undesirable constellations of traits. In some instances, externalizing disorders may represent maladaptive extremes of potentially adaptive traits (see MacDonald, 2012). It should be stressed that externalizing disorders can be adaptive even if their social outcomes are negative *on average*. This can happen if successful outcomes yield disproportionate fitness returns, even in a minority of cases. Finally, high-risk behavioral strategies are likely to involve down-regulation of defensive mechanisms; indeed, externalizing disorders in adolescents and adults are often associated with reduced anxiety, fearlessness, and dampened responsivity of the SRS.

Schizophrenia spectrum disorders

Schizophrenia is a family of mental disorders characterized by delusions, hallucinations, and cognitive disorganization. Given the severe reduction in reproductive success associated with

a schizophrenia diagnosis, most evolutionary scholars regard this disorder as a maladaptive outcome of dysregulated socio-cognitive processes (e.g., Burns, 2004; Keller & Miller, 2006). While schizophrenia spectrum disorders (SSDs) are highly heritable, schizophrenia risk is also increased by adverse environmental factors such as nutritional deficiencies, infections, and birth complications. This suggests that accumulated deleterious mutations and environmental insults may converge on common neurobiological pathways, increasing the risk of cognitive breakdown.

Even if SSDs are biologically maladaptive conditions, there may be evolutionary advantages associated with *schizotypal traits*—a constellation of personality traits associated with increased risk of psychosis. Most individuals who have psychotic experiences at some point in their life recover completely, and never transition to a diagnosable SSD. Various authors have proposed that schizotypal traits may be maintained by sexual selection processes based on mate choice. According to the sexual selection model of schizotypy (Nettle, 2001, 2006; Shaner, Miller, & Mintz, 2004), schizotypy-increasing alleles affect brain processes so as to increase traits such as verbal and artistic creativity, thus conferring mating advantages on those individuals who do not develop a psychiatric condition. However, the outcomes of schizotypy may be either beneficial (mating success) or harmful (schizophrenia), depending in part on the individual's genetic quality (i.e., lack of deleterious mutations) and developmental condition (e.g., good nutrition and low exposure to pathogens).

Consistent with the sexual selection model, positive schizotypal traits—unusual cognitive and perceptual experiences, tendency to magical ideation, reference and paranoid thoughts—are associated with verbal and artistic creativity, larger numbers of sexual partners, unrestricted sociosexuality, and reduced investment in long-term couple relationships. Large-scale studies of patients and their relatives show a robust familial association between schizophrenia and creativity. Schizotypal traits peak in adolescence/young adulthood and show a marked decline with age, mirroring typical changes in mating effort. In addition, positive schizotypy is associated with lower agreeableness and higher levels of aggression, suggesting a degree of overlap between the schizophrenia spectrum and the externalizing spectrum.

In light of this convergent evidence, SSDs can be provisionally classified as belonging to the fast spectrum of psychopathology, although there are reasons to expect a degree of functional heterogeneity (see Del Giudice, in press). According to sexual selection models, schizotypy can be understood as a high-risk strategy oriented toward short-term mating, whose negative outcomes become manifest as schizophrenia and other SSDs. Alternatively, the milder disorders of the schizophrenia spectrum (e.g., schizotypal personality disorder, brief psychotic disorder) may result from maladaptive levels of expression of potentially adaptive traits associated with fast life history strategies.

Autism spectrum disorders

The autism spectrum comprises disorders of variable severity characterized by impairments in social interaction, communication problems, and restricted and repetitive behaviors/interests. Severe autism is almost certainly maladaptive, and some theorists have focused specifically on the negative aspects of autism spectrum disorders (ASDs). For example, Shaner and colleagues (Shaner, Miller, & Mintz, 2008) hypothesized that autism—like schizophrenia—may represent the negative extreme of a fitness indicator, a hypothesis consistent with the large number of deleterious mutations found in ASD patients.

This negative emphasis should be balanced by accumulating evidence that autistic-like traits in the normative range—also known as the "broader autistic phenotype"—have a number of desirable and potentially adaptive correlates. Specifically, autistic-like traits predict higher systemizing abilities and attention to detail, better visuospatial skills, and enhanced low-level sensory processing in the visual and auditory domains. The autistic facets of repetitive behaviors, restricted interests, and detail-oriented cognitive style are associated with the development of outstanding talents in children. More generally, autistic-like traits are higher in people with technical-scientific interests and careers. Accordingly, several theorists have argued that ASDs can be seen as extreme and usually maladaptive manifestations of otherwise adaptive traits (e.g., Baron-Cohen, 2003; Crespi & Badcock, 2008).

In this perspective, Del Giudice and colleagues (2010) hypothesized that sexual selection may contribute to maintain autistic-like traits in the population despite the fitness costs of severe ASDs. Specifically, they argued that autistic-like traits in their non-pathological form contribute to a male-typical strategy geared toward high parental investment, low mating effort, and long-term allocation of resources—in other words, a male-typical manifestation of slow life history strategy. This hypothesis offers a parsimonious explanation of the male-biased distribution of both autistic-like traits and ASDs. In support of this hypothesis, autistic-like traits predict lower interest in short-term mating, increased investment of time and resources in one's partner, and stronger commitment to long-term romantic relations. People high in autistic-like traits report shorter duration of friendships but longer duration of romantic relationships, and their partners are on average just as satisfied as those of people low in autistic-like traits.

In a life history perspective, ASDs are thus likely candidates for inclusion in the slow spectrum of psychopathology. Further evidence comes from the finding that sexual maturation is delayed in women high in autistic-like traits as well as in women with ASD. Autistic-like traits may function adaptively as part of a slow life history strategy—especially in males—and only become maladaptive when they cross a certain threshold. Given the remarkable heterogeneity of ASDs, this functional explanation is likely to apply only to a subset of people diagnosed with autistic disorders. Different ASD subtypes may well require different explanations. The existence of functionally distinct subtypes of ASDs may explain the inconsistent correlation of autism risk with socioeconomic status in epidemiological studies.

Obsessive-compulsive spectrum disorders

Disorders in the obsessive-compulsive spectrum are primarily characterized by patterns of compulsive, repetitive thoughts and/or behaviors, usually associated with worry and anxiety. In addition to obsessive-compulsive disorder (OCD), the OC spectrum includes body dysmorphic disorder, hoarding disorder, grooming disorders (skin picking and hair pulling), and obsessive-compulsive personality disorder (OCPD)—a pervasive profile of orderliness, rigid perfectionism, and need to control one's self and environment.

In the evolutionary literature, OCD is usually treated as a maladaptive exaggeration of an adaptive trait or the result of a dysfunction in precautionary cognitive systems. However, the milder forms of the disorder are not necessarily maladaptive in the biological sense. Current models converge on the idea that the main functional substrate of OCD is an adaptive mechanism—the *hazard-precaution system* or *security motivation system*—specialized for dealing with potential low frequency threats such as food poisoning (e.g., Boyer & Lienard, 2006; Szechtman & Woody, 2004; Woody & Szechtman, 2011). The peculiar logic of potential

threats explains many features of compulsions (see Woody & Szechtman, 2011); obsessions can be explained as the involuntary generation of potential risk scenarios, a mechanism designed to increase future harm avoidance. Consistent with a threat prevention account and with the prediction that females should be more likely to develop symptoms reflecting up-regulated defenses, adult OCD patients are overwhelmingly women.

A life history analysis indicates that the OC spectrum is best understood as a functionally heterogeneous category comprising two clusters of disorders—a slow spectrum one and a fast spectrum one. Slow spectrum OCD is marked by *reactive* obsessions (Lee & Kwon, 2003); reactive obsessions concern "realistic" fears of contamination, mistakes, accidents, and/or disarray. They are triggered by cues of potential threats and are typically followed by preventive behaviors such as ordering or cleaning; anxiety is directed at the possible consequences of one's actions rather than at the obsession itself. Reactive obsessions are associated with high conscientiousness, perfectionism, heightened responsibility and personal standards, normal levels of motor and cognitive inhibition, and a prevalence of contamination/cleaning symptoms. Reactive OCD fits straightforwardly in the slow spectrum of psychopathology, as a combination of exaggerated trait expression, up-regulation of adaptive defenses, and dysfunctional protective responses. Obsessive-compulsive personality disorder (OCPD) also fits this classification, given its many overcontrol features and strong association with conscientiousness.

Fast spectrum OCD is characterized by *autogenous* obsessions—obsessions with sexual, aggressive, and/or blasphemous content. Autogenous obsessions tend to be bizarre, ego-dystonic, and threatening. They often have no apparent trigger, or are triggered by remote/bizarre thought associations (Lee & Kwon, 2003). Autogenous obsessions are associated with positive schizotypy, indices of psychotic thought disorganization, low conscientiousness, and reduced inhibitory control. The heterogeneous nature of the OC spectrum explains why OCD shows high comorbidity with both ASDs and SSDs. The two OC clusters can be expected to show markedly different epidemiological profiles; for example, traumatic events and low SES should be more strongly associated with fast spectrum OCD, whereas slow spectrum OCD should often arise in safe and predictable environments. Consistent with placement in the slow spectrum cluster, OCPD is uniformly associated with high education levels, and OCDP patients have the highest socioeconomic status of all personality disorders.

Eating disorders

Eating disorders (EDs) are defined by heightened concern with body shape/weight and associated behaviors such as dieting, binge eating, purging, and exercising. Eating disorders occur almost exclusively in females, and their age of onset peaks in adolescence. Most evolutionary models of eating disorders focus on the connection between dieting behavior and female reproduction. Two main alternative hypotheses have been proposed so far. First, dieting may work as a means to suppress fertility and delay or forego reproduction when the social environment is not optimal—for example when social support by relatives and partners is low, or when social competition is too harsh (e.g., Mealey, 2000; Surbey, 1987). Second, dieting may work primarily as a female strategy in mating and status competition (e.g., Abed, 1998; Ferguson, Winegard, & Winegard, 2011; Salmon et al., 2009). Thinness is a reliable signal of youth, and dieting can increase one's attractiveness because of men's strong preference for younger partners; in addition, dieting can enhance status in female groups (thus indirectly influencing mating success), especially when cultural emphasis on thinness is strong. This

hypothesis is supported by the robust pattern of associations among perceived sexual competition, dieting behavior, and eating symptoms. Under both hypotheses, the psychological processes that underlie dieting behavior are fundamentally adaptive, and lead to maladaptive outcomes (such as severe EDs) only when they become dysregulated or get trapped in vicious cycles.

The mating competition hypothesis of eating disorders can be easily reframed in a life history perspective. Both fast and slow strategists can face intense competition for mates; the main difference is that fast strategists compete primarily to become desirable sexual partners, whereas slow strategists compete primarily to be chosen as long-term partners in committed relationships. Thus, eating disorders can arise at both ends of the fast-slow continuum.

A life history analysis confirms that EDs are indeed a functionally heterogeneous category. Slow spectrum EDs are associated with *high functioning/perfectionist* personality profiles, low comorbidity rates (mostly with OCD and OCPD), and the most favorable clinical outcomes (see e.g., Thompson-Brenner et al., 2008; Westen & Harnden-Fischer, 2001). This cluster of eating disorders is associated with high self-esteem, relatively intact family and couple relationships, and a history of *fewer* stressful life events. Another profile that can be included in the slow spectrum is that of *overcontrolled* patients. This profile is associated with high rates of depression, low self-esteem and passivity, restricted emotionality, and comorbidity with OCPD. Overcontrolled ED patients might be engaging in reproductive suppression—an intrinsically future-oriented strategy—following loss of status and/or social support, as suggested by their depressed mood, low self-esteem, and acute sense of social exclusion.

In contrast, fast spectrum EDs are associated with *dysregulated* personality profiles (see Thompson-Brenner et al., 2008). Dysregulated ED patients show high levels of impulsivity and antisocial/externalizing behavior, high comorbidity (especially with borderline personality disorder), and many stressful life events including high rates of sexual abuse. While patients in the high functioning/perfectionist and overcontrolled groups can be diagnosed with either anorexia nervosa (AN) or bulimia nervosa (BN), the dysregulated subtype is strongly associated with BN (Westen & Harnden-Fischer, 2001). As a result, patients with BN—considered as a whole—show higher average levels of impulsivity, earlier sexual maturation, and earlier sexual debut than AN patients.

Depression

Depression is characterized by protracted episodes of distress and low, dejected mood. The clinical presentation of depression is quite heterogeneous; attempts to subtype depressive disorders based on empirical patterns of symptom co-occurrence consistently identify (a) a subtype characterized exclusively by depressed mood and feelings of worthlessness; (b) one or more subtypes characterized by somatic symptoms in absence of depressed mood; and (c) one or more subtypes in which depressed mood and somatic symptoms coexist. Somatic symptoms of depression include sleep disturbances (insomnia or hypersomnia), appetite disturbances (increased or decreased appetite), psychomotor disturbances (agitation or retardation), fatigue, and pain. All these symptoms are functionally related to the SRS, and in particular the HPA axis.

Most evolutionary theories of depression focus on low mood and its motivational and behavioral correlates. In the prevailing view, depressed mood is an adaptive defensive mechanism, whereas clinical depression is usually maladaptive and reflects a dysfunction of the same mechanism (e.g., Allen & Badcock, 2003; Nesse, 2006; Nettle, 2004, 2012). Some theorists

have argued that clinical depression may be an adaptation itself (e.g., Price, Sloman, Gardner, Gilbert, & Rohde, 1994; Watson & Andrews, 2002). Although this hypothesis appears reasonable in the specific case of postpartum depression (Hagen, 1999), there are many reasons to doubt its general applicability. The function of low mood as a protective mechanism is twofold. First, low mood helps people disengage from the pursuit of central life goals that have become unproductive. Second and more specifically, it promotes a risk-averse approach in unfavorable social circumstances—especially following losses in social support (typically in females), close relationships, and social status or dominance (typically in males). While affective reactivity determines one's susceptibility to episodes of low mood, stress reactivity is the crucial factor in the development of somatic symptoms. Thus, a complete evolutionary account of depression cannot be separated from evolutionary models of SRS functioning.

By synthesizing the ACM with evolutionary models of depressed mood, it is possible to predict a complex relation between depression and life history strategy. Both fast and slow strategists can fail to obtain or maintain crucial social resources—status, dominance, and support—resulting in episodes of depressed mood and risk for clinical depression. At the slow end of the continuum, males and females are both expected to develop relatively high levels of stress responsivity (Del Giudice et al., 2011), even if the actual intensity of stress responses is buffered by the availability of social support and lack of chronic stressors. As a result, symptom profiles at the slow end of the spectrum should not differ greatly between the sexes. Intriguingly, some subtypes of depression—in particular those characterized by pure depressed mood or pure somatic symptoms—are associated with very low rates of trauma, neglect, and abuse.

Moving toward the fast end of the continuum, both sexes face increasing threats to their ability to gain and maintain social resources. The availability of social support and stable, intimate relationships declines rapidly as environments become dangerous and unpredictable, exposing females to increased risk for depressed mood. At the same time, sex differences in stress responsivity can be expected to become proportionally larger, as more males develop unemotional responsivity patterns. Vigilant SRS profiles can be adaptive in dangerous and unpredictable contexts, especially in females; however, they also increase the risk of SRS dysregulation and dysfunction. In total, fast life history strategies should lead to increased risk for depression in both sexes, with females showing the highest rates of depressed mood and somatic symptoms. Consistent with these predictions, early and/or fast sexual maturation is a risk factor for depression in both sexes, with stronger effects in females. In addition, depression subtypes involving a combination of low mood and somatic symptoms are overwhelmingly more common in females, and are also associated with the highest rates of early trauma, neglect and abuse. In conclusion, depression may occur at both ends of the fast-slow continuum, suggesting the existence of functionally distinct clusters of depressive disorders. Unfortunately, the current literature defines depression subtypes exclusively in terms of symptom co-occurrence; further research in a life history framework should attempt to identify functional subtypes of depression based on motivation, personality, self-regulation, and comorbidity with other fast and slow spectrum disorders.

Summary and integration

A life history analysis of mental disorders reveals a coherent picture of associations between individual differences in life history strategy and specific patterns of risk for psychopathology. The constellation of fast spectrum conditions includes externalizing disorders,

schizophrenia spectrum disorders, OCD with autogenous obsessions, the dysregulated subtype of eating disorders (typically expressed as BN), and depressive disorders characterized by a combination of mood and somatic symptoms. These disorders tend to co-occur, both within families and within individuals; many of them share elements of impulsivity, disinhibition, and/or bizarre ideation. Slow spectrum psychopathology includes OCPD, OCD with reactive obsessions, autism spectrum disorders, the perfectionist and overcontrolled subtypes of eating disorders, and a cluster of depressive disorders of lesser severity. These comorbid disorders tend to share elements of inhibition, overcontrol, and cognitive rigidity. They are also characterized by lack of association with standard risk factors for psychopathology such as stressful life events, low SES, and early abuse; in some cases, they are actually associated with more favorable ecological and socio-economic conditions. The same approach can be easily extended to other disorders. For example, borderline personality disorder (BPD) bears the hallmarks of fast life history strategies—impulsivity, unstable attachments, risk-taking, promiscuous sexuality, antisocial and paranoid personality features, and high comorbidity with externalizing disorders (Brüne et al., 2010; see also Crowell et al., 2013). Similarly, disorders in the bipolar spectrum show substantial genotypic and phenotypic overlap with schizotypy and schizophrenia, including a familial association with enhanced creativity (see Del Giudice, in press). A provisional classification of slow and fast spectrum disorders is shown in Figure 7.

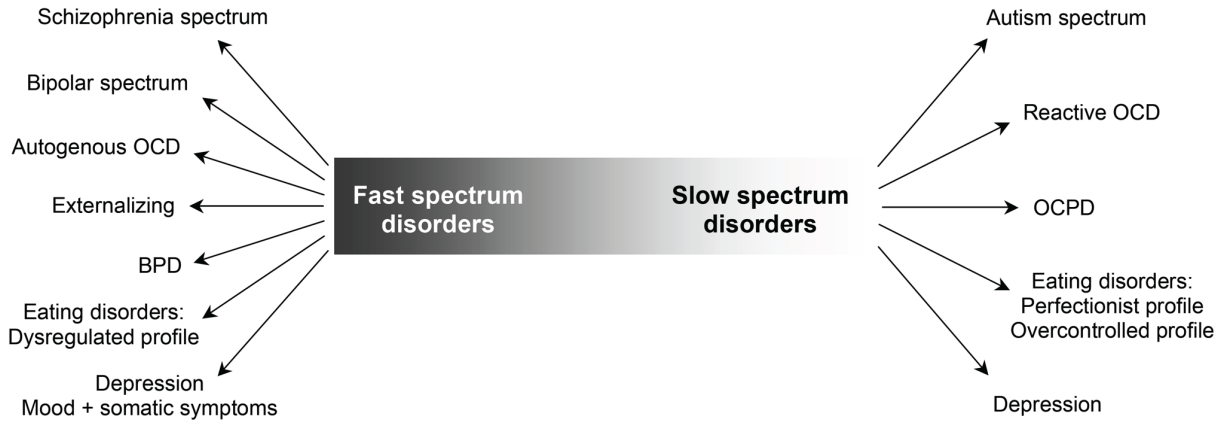

Figure 7. Provisional life history taxonomy of common mental disorders. BPD = borderline personality disorder, OCD = obsessive-compulsive disorder, OCPD = obsessive-compulsive personality disorder. Reprinted from Del Giudice (in press).

This classification is still tentative and incomplete, and many gaps and questions remain—for example about the possible functional heterogeneity of autism and schizophrenia, the role of reproductive suppression in disordered eating, or the identification of fast and slow spectrum subtypes of depression (see Del Giudice, in press). However, even this initial analysis illustrate how a life history framework can bring an integrative perspective to psychopathology, highlight connections between previously separate models, and suggest novel empirical questions. Even more importantly, this approach has the potential to overcome the limitations of

current taxonomic systems and offer a more solid foundation for the classification of mental disorders. In particular, the fast-slow distinction is both more *inclusive* and more *accurate* than the internalizing-externalizing distinction. It is more inclusive because it integrates mood and anxiety disorders with personality disorders, schizophrenia spectrum disorders, and autism spectrum disorders—all within the same conceptual framework. It is more accurate because it resolves many inconsistencies inherent in the basic internalizing-externalizing distinction. For example, the ambiguous placement of OCD in the internalizing spectrum is explained by the heterogeneity of OCD; specifically, the autogenous subtype of OCD is a fast spectrum disorder with strong functional connections with externalizing symptoms. More generally, the internalizing-externalizing distinction may be problematic because it is in large part illusory. The obvious genotypic and phenotypic coherence of the externalizing spectrum may have led researchers to assume that internalizing disorders must form a symmetrical category with similar properties of coherence. A life history perspective suggests that this assumption is probably mistaken, and that the "internalizing spectrum" may turn out to be a largely artificial collection of disorders with divergent functional properties.

Implications for the Core Points of Developmental Psychopathology

The life history framework discussed in this section has important implications for developmental psychopathology. Most crucially, it shows how *equifinality* and *multifinality* in the development of mental disorders can be explained in a functional perspective. On the one hand, the same kind of symptom—for example eating symptoms and obsessions—can arise in relation to different life history strategies. As a result, phenotypically similar disorders can be associated with opposite profiles of personality, sexual maturation, ecological factors, and so forth. On the other hand, the same basic dimensions of life history strategy can play a role in the etiology of functionally related but superficially different disorders. While these manifestations of equifinality and multifinality may be problematic in the context of the standard externalizing-internalizing distinction, they can be easily understood in terms of the fast-slow distinction advanced by Del Giudice (in press).

A closely related point is that, in this perspective, not all disorders are expected to arise in association with "typical" *risk factors* such as early stress, negative family relationships, and low SES. This helps make sense of the puzzling fact that some disorders seem to develop more frequently in safe, predictable ecologies and families with high socioeconomic status. The model presented here may also explain why insecure attachment—a robust psychological correlate of fast life history strategy—is consistently associated with externalizing symptoms but only weakly predictive of internalizing symptoms (Groh, Roisman, van IJzendoorn, Bakermans-Kranenburg, & Fearon, 2012). Finally, the coexistence of fast and slow spectrum subtypes within the same diagnostic category may go a long way toward explaining inconsistent or contradictory patterns of epidemiological findings.

Conclusion

We started this chapter with a promise—to show how an evolutionary approach can help developmental psychopathology realize its full potential, and to demonstrate how EDP provides

an integrative, powerful metatheory for the field. We hope we fulfilled our promise and succeeded in arousing the reader's interest in the EDP approach. Throughout the chapter, we sought to illustrate how an evolutionary-developmental perspective supports and extends the core points of developmental psychopathology. Consider for example the concept of *multifinality*. In the standard view, multifinality is an ubiquitous, general property of complex developmental systems. However, the mechanisms that generate of multifinality are usually left unspecified; as a result, the concept is often used to redescribe empirical findings rather than explain them. The EDP approach demystifies multifinality by grounding the concept in adaptive function, and provides the tools for predicting *when* multifinality should apply (or not) to a given developmental process, stage, or outcome. Thus, multifinality can be understood as a necessary consequence of differential susceptibility; the logic of the ACM predicts when similar physiological profiles may predict widely divergent behavioral outcomes; and life history theory (together with sexual selection theory) explains why similar developmental experiences may set the stage for phenotypically different but functionally related disorders. Even more importantly, these apparently disparate aspects of development can be understood in relation to one another and unified within an integrative theoretical framework.

Even if we covered a lot of ground, we barely scratched the surface of our topic. The evolutionary approach to development and psychopathology is a growing multidisciplinary enterprise, and new theories, models, and findings are published at an ever increasing pace. We therefore conclude by suggesting a reading list for further explorations of the field. Ellis and Bjorklund (2005) and Burgess and MacDonald (2005) offer a more complete overview of EDP, including discussion of cognitive processes such as memory and language. Special sections of *Development and Psychopathology* on differential susceptibility (Ellis & Boyce, 2011) and *Developmental Psychology* on conditional adaptation (Ellis & Bjorklund, 2012) provide useful collections of relevant work. An introduction to theories and models in evolutionary psychopathology can be found in Brüne (2008) and McGuire and Troisi (1998). Finally, Ellison and Gray (2009) show how evolutionary thinking can be applied to neurobiological and endocrinological processes. Explaining the development of psychopathology is a formidable task, calling for convergence and integration across myriad disciplines and levels of analysis. We believe that evolutionary theory offers invaluable tools for this task, and hope that developmental psychopathology will join forces with EDP toward a common understanding of human development in all its living complexity.

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