Rethinking the fast-slow continuum of individual differences

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A B S T R A C T

The idea that individual differences in behavior and physiology can be partly understood by linking them to a fast-slow continuum of life history strategies has become popular in the evolutionary behavioral sciences. I refer to this approach as the "fast-slow paradigm" of individual differences. The paradigm has generated a substantial amount of research, but has also come increasingly under scrutiny for theoretical, empirical, and methodological reasons. I start by reviewing the basic empirical facts about the fast-slow continuum across species and the main theoretical accounts of its existence. I then discuss the move from the level of species and populations to that of individuals, and the theoretical and empirical complications that follow. I argue that the fast-slow continuum can be a productive heuristic for individual differences; however, the field needs to update its theoretical assumptions, rethink some methodological practices, and explore new approaches and ideas in light of the specific features of the human ecology.

1. Introduction

In this paper, I critically examine the idea that individual differences in behavior and physiology can be partly understood by linking them to a fast-slow continuum of life history strategies. In its original form, the fast-slow continuum denotes an empirical pattern of species differences in fitness-related traits such as fertility, mortality, and offspring size (Jeschke, Gabriel, & Kokko, 2008). Although there is still no complete, widely accepted theory of the fast-slow continuum, it is clear that some important functional principles are at play (Section 2). Similar principles may operate *within* species, and contribute to explaining differences in life history strategies between populations or even individuals. Following this line of reasoning, researchers in biology, anthropology, and evolutionary psychology have argued that within-species individual differences are partly organized along a fast-slow axis of variation. Crucially, this does not just apply to classic life history variables such as fertility and age at maturity, but also to the behavioral and physiological traits hypothesized to mediate the underlying trade-offs. In principle, the fast-slow continuum can help make adaptive sense of the *covariation* among behavioral and personality traits, their relations with physiological processes, and their developmental antecedents (e.g., early stress; see Belsky, Steinberg, & Draper, 1991; Del Giudice, Gangestad, & Kaplan, 2015; Ellis, Figueredo, Brumbach, & Schlomer, 2009; Figueredo et al., 2006; Réale et al., 2010; Wolf, van Doorn, Leimar, & Weissing, 2007). For convenience, I will refer to this set of general ideas as the *fast-slow paradigm* of individual differences.

Over the last decade, the fast-slow paradigm has become remarkably popular, and has spawned new research subfields and empirical literatures (Nettle & Frankenhuis, 2019). At the same time, this approach has inevitably come under closer scrutiny, and critics have started to point out problems and unresolved issues. To begin, research based on the fast-slow paradigm has become increasingly self-referential and disconnected from mathematical work on life history evolution (Nettle & Frankenhuis, 2019). Partly for this reason, tentative hypotheses have been treated as established theory, and researchers have come to rely on overly simplified predictions, without critically examining their assumptions (e.g., Baldini, 2015; Dammhahn, Dingemanse, Niemelä, & Réale, 2018; Mathot & Frankenhuis, 2018; Nettle, 2018; Stearns & Rodrigues, 2020). On the empirical side, the validity of measures of life history-related traits has been questioned, both in biology and psychology (Copping, Campbell, & Muncer, 2014a, 2014b; Copping, Campbell, Muncer, & Richardson, 2017; Figueredo et al., 2015; Niemelä & Dingemanse, 2018; Richardson et al., 2017, Richardson et al., 2017; Royauté, Berdal, Garrison, & Dochtermann, 2018). In humans, some widespread assumptions about the plasticity of life history strategies and the role of early experiences have been criticized, due to gaps in the underlying theory (e.g., Nettle, Frankenhuis, and Rickard, 2013; Del Giudice, 2014a) and contradictory findings from behavior genetics (e.g., Barbaro, Boutwell, Barnes, & Shackelford, 2017). The paper by Zietsch and Sidari (2020) is a useful compendium of critical arguments; I provide a concise point-by-point reply to their critiques in the supplementary material (S4).

In sum, it is high time for a reassessment, and this special issue is a great opportunity to move the conversation forward. My goal in this

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paper is to systematically examine the logic of the fast-slow paradigm. In Section 2, I review the basic empirical facts about the fast-slow continuum across species and the main theoretical accounts of its existence. In Section 3, I discuss the move from the level of species to that of individuals, and the theoretical and empirical complications that follow. To anticipate my conclusion, I argue that the fast-slow continuum can be a productive heuristic for individual differences, but needs to be developed further and embedded in a more sophisticated view of life history evolution. This means acknowledging the existing theoretical gaps and reconnecting with the biological literature, but also adapting the generic concept of fast versus slow strategies to the specific features of the human ecology. In total, I argue that the field needs to take criticism seriously, and use this opportunity to revise problematic assumptions, drop some bad habits, and start exploring new approaches and ideas.

2. The fast-slow continuum among species

2.1. Empirical patterns

The term "fast-slow continuum" was coined by Sæther (1987), but the empirical pattern it describes had been noted much earlier (e.g., Pianka, 1970; Tinkle, Wilbur, & Tilley, 1970), and initially explained with species differences in *r*- versus *K*-selection (favoring the evolution of faster vs. slower life histories, respectively; see Section 2.2). Species at the fast end of the continuum have high mortality and short lifespans; they mature and start reproduction early, produce small offspring at a fast rate, and show high fertility (at least in mammals and birds; see below for more discussion). Species at the slow end take long to mature and start reproduction, enjoy low mortality rates and long lifespans, and tend to produce few, large offspring at a slow rate. While faster species tend to be smaller and slower species tend to be larger, controlling for body size does not make the continuum disappear, and the overall pattern typically remains very similar (e.g., Del Giudice, 2014b; Stearns, 1983; see below). Fast-slow continua have been documented in mammals (including primates), birds, fish, reptiles, insects, and other animals (e.g., Bakewell, Davis, Freckleton, Isaac, & Mayhew, 2020; Healy et al., 2019; Jeschke & Kokko, 2009; Oli, 2004; Promislow & Harvey, 1990; Ross, 1988; Stearns, 1983); recent comparative studies have found a similar pattern in plants (Rüger et al., 2018; Salguero-Gómez, 2017; Salguero-Gómez et al., 2016).

2.1.1. Is life history variation one-dimensional?

There are two common misconceptions about the fast-slow continuum. The first is that, for the continuum to hold, there must be no other major axes of variation in life history traits; that is, life history differences between species must be well described by a one-dimensional model. In reality, it has been clear for decades that there are other important life history dimensions besides fast versus slow (e.g., Stearns, 1983). In mammals, the standard "map" of life history traits is defined by two main axes (Fig. 1): a fast-slow continuum that typically accounts for 70-80% of the variance in the traits (about 30-50% controlling for body size) and a secondary, largely independent axis that explains 10-15% of the variance (about 20-30% controlling for body size; see supplementary material S1). This secondary axis distinguishes between species with longer gestations that give birth to larger, precocial offspring (e.g., gazelles) and species with shorter gestations and smaller, altricial offspring that remain dependent for longer (e.g., kangaroos). (Note that the exact nature of the second axis depends on the variables included in the analysis; e.g., Dobson & Oli, 2007.) A widely cited study by Bielby et al. (2007) seemingly failed to recover the structure shown in Fig. 1, but the contradictory finding was due to a problem in the analysis—specifically, an inappropriate rotation of the axes in principal components analysis (PCA; see the supplementary material S1 for details). Upon reanalysis, the data showed the same pattern found by Stearns (Del Giudice, 2014b; Fig. S1.2).

2.1.2. The role of body size

The second misconception is that the nature of the fast-slow continuum within a given taxonomic group changes dramatically once body size is controlled for, to the point of becoming a conceptually distinct dimension of variation (or disappearing altogether). While the theoretical implications of partialing out body size are far from clear (Section 2.3), this has been interpreted as evidence that the fast-slow continuum is not a robust phenomenon (e.g., Crespi, 2014; Stearns & Rodrigues, 2020; Surbey, 2014; Zietsch & Sidari, 2020). The notion that body size has a major impact on the nature of the fast-slow continuum is largely based on the studies by Bielby et al. (2007) and Jeschke and Kokko (2009); it is a misconception because, in both cases, the conclusions of the study are not supported by the data. Again, the problems with the original analyses concern the orientation of axes in PCA; this issue is so pervasive that it deserves a dedicated treatment in the supplementary material (S1).

When the data are properly analyzed, the structure of life history traits turns out to be remarkably robust to the effect of body size (Figs. S1.2, S1.3, and S1.4). That said, some taxonomic differences are real and not merely artifactual. Most notably, slower species of fish, reptiles, and insects with larger bodies and longer lifespans also tend to show increased rather than reduced fertility (i.e., larger numbers of eggs; Bakewell et al., 2020; Jeschke & Kokko, 2009). A plausible reason is that, in these species, egg size is not a strong predictor of offspring quality and survival. As a result, parental investment mainly takes place through enhanced fertility; allocation to increase the number of eggs plays a role similar to allocation to increase offspring size in mammals and birds (see Bakewell et al., 2020; Jeschke & Kokko, 2009). A recent large-scale analysis of animal life histories by Healy, Ezard, Jones, Salguero-Gómez, and Buckley (2019) was largely consistent with the existence of a fast-slow continuum, with some exceptions and variations that probably reflect a mixture of true species differences and rotation artifacts (see supplementary material S1).

2.2. Models of r/K selection

The first theoretical explanation of the fast-slow continuum was proposed by Pianka (1970), based on MacArthur and Wilson's model of *r/K* selection (MacArthur and Wilson, 1967). As I discuss later, these initial contributions suffered from some important limitations. In the 1980s, the *r/K* framework—with its strong focus on density-dependent selection—fell out of favor, and was largely supplanted by density-independent "demographic" models based on age-dependent schedules of fertility and mortality (see Bassar et al., 2010; Jeschke et al., 2008; Reznick, Bryant, & Bashey, 2002; Roff, 2002). However, the notion that biologists have stopped using the concept of *r/K* selection (as suggested for example by Copping et al., 2014b; see also Zietsch & Sidari, 2020) is an exaggeration. In recent years, *r/K* models have been significantly updated (Engen, Lande, & Sæther, 2013; Engen & Sæther, 2016, 2017; Lande, Engen, & Sæther, 2009, 2017), and used to develop new ideas about the evolution of fast-slow variation within species (Wright et al., 2019; Section 3).

2.2.1. r-Selection

The letter *r* denotes the intrinsic rate of increase of a population, a central parameter in models of life history evolution (Roff, 2002). Selection maximizes *r* in stable environments when fitness does not depend on population density, or when density-dependence is present but only affects survival (as opposed to fertility) in an age-independent fashion (Dańko et al., 2018; Reznick et al., 2002). In stochastically variable environments, what is maximized is not *r* but a function of the expected *r* and its variance; as a result, more variable environments select for faster growth rates (Engen et al., 2013; Sæther & Engen, 2015). The conditions that lead to *r* maximization tend to coincide with high mortality rates, low population densities, and (often temporary) phases of unconstrained population growth. Theoretical models predict

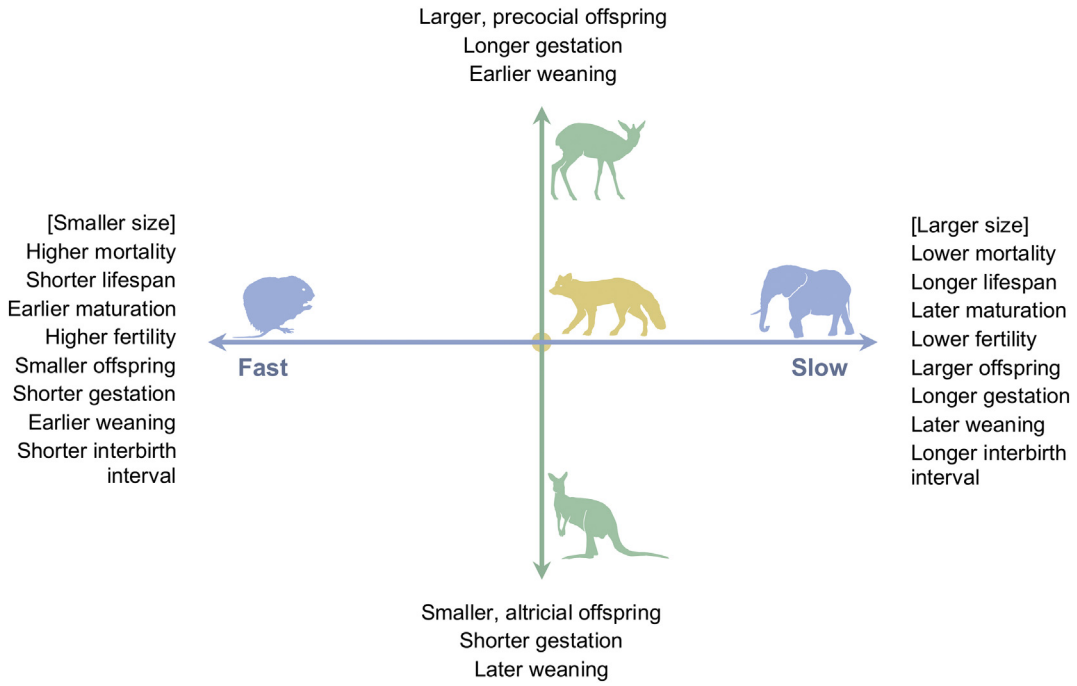

Fig. 1. A two-dimensional map of life history traits across mammalian species. Based on Stearns (1983); Promislow and Harvey (1990); Oli (2004); and the reanalysis of data from Bielby et al. (2007) and Jeschke and Kokko (2009) presented in the supplementary material (S1; Del Giudice, 2014b).

that *r*-selected species should mature and reproduce early, with high fertility and short lifespans (Engen & Sæther, 2016, 2017). However, selection to reduce the variance of *r* may also favor the evolution of plasticity and bet-hedging (Lande et al., 2017; Starrfelt & Kokko, 2013).

2.2.2. *K*-Selection

The letter *K* indicates the equilibrium size of a population, also known as the environment's "carrying capacity." The idea is that, as a population approaches its maximum size (and hence zero growth), density-dependent effects on fitness become stronger as individuals compete more intensely for resources and reproductive opportunities. As a result, *r* tends to zero, and the expected lifetime reproductive success R_0 becomes a more appropriate measure of fitness. Note that this general statement applies if population density affects fertility or age-dependent survival; if density only affects survival independent of age, selection maximizes *r* as explained earlier (see Dańko, Burger, Argasiński, & Kozłowski, 2018). *K*-selection scenarios tend to occur in stable ecologies, which allow populations to reach and maintain high levels of density. Modern *r/K* models describe a continuum of selection regimes in which environmental variability intensifies selection on *r*, whereas stability and density dependence favor the competitive traits that buffer the detrimental effects of population density (and increase *K* all else being equal; Engen et al., 2013; Engen & Sæther, 2016, 2017; Mylius & Diekmann, 1995; Sæther & Engen, 2015; see also Dańko et al., 2018, 2017). This does not mean that *K*-selected species will always have large populations; for example, if competition occurs via aggression and large bodies, the equilibrium population size may be small in absolute terms (see Wright et al., 2019).

A major problem with Pianka's original *r/K* framework was the assumption that *K*-selection would favor the opposite traits of *r*-selection—late maturation, delayed reproduction, low fertility, and a long lifespan. But things are not that simple: even under strong density dependence, selection may favor early reproduction and a shorter lifespan if unavoidable or *extrinsic* mortality[1] is high (Dańko et al., 2018, 2017). Other complications arise from the way in which density dependence and stochasticity jointly affect survival and reproduction (see Bassar et al., 2010; Reznick et al., 2002). In sum, density dependence does not favor a unique pattern of life history traits, and predictions may vary depending on the details of a species' ecology. This may explain the inconsistent findings that contributed to the downfall of *r/K* models in the 1980s (see Jeschke et al., 2008; Roff, 2002; Stearns, 1992).

2.2.3. *r/K* Models and the fast-slow continuum

Early *r/K* models postulated a direct trade-off between *r* and *K* without a compelling rationale (Jeschke et al., 2008; Reznick et al., 2002; Stearns, 1992). More recent *r/K* models are more explicit about the links between density dependence and specific life history traits; but they still suffer from a similar problem, as the functional basis of key trade-offs is left unspecified or described in ways that remain open to multiple interpretations. For example, Engen and Sæther (2016) assumed that phenotypes characterized by early reproduction and faster growth rates are more affected by population density; based on this assumption, they predicted that more variable environments will select for earlier maturation and reproduction. Engen and Sæther framed this trade-off as an alternative to standard models of allocation to current versus future reproduction. Wright, Bolstad, Araya-Ajoy, and Dingemanse (2019) based their work on the same model, but argued that the current-future reproduction trade-off is the functional *basis* for the trade-off between a faster growth rate (achieved through early reproduction) and the ability to withstand the detrimental effects of population density (achieved through delayed reproduction and

[1] The exact meaning of "extrinsic mortality" varies somewhat between disciplines. In evolutionary psychology and anthropology, "extrinsic" is used to

(*footnote continued*)
mean that mortality is unavoidable, i.e., insensitive to the allocation decisions of the organism (e.g., Del Giudice et al., 2015; Ellis et al., 2009; Quinlan, 2007). In theoretical biology, "extrinsic" often has the additional implication that mortality rates are independent of age (e.g., Caswell, 2007). The distinction is important because age-independent changes in mortality can only affect life history evolution if fertility and/or juvenile mortality are density-dependent. In and by itself, the level of extrinsic (age-independent) mortality experienced by an organism has no effect on the evolution of life history traits (André & Rousset, 2020; Dańko et al., 2017, 2018; Reznick et al., 2002).

investment in competitive traits). In conclusion, modern *r/K* models can explain some important aspects of the fast-slow continuum; however, they require additional functional assumptions and still do not provide a complete, first-principles account of the observed covariation patterns.

2.3. Other theoretical models

As density-independent models based on demographic schedules displaced the original *r/K* framework, the field moved away from all-encompassing theories and toward the exploration of narrower, well defined trade-offs (Jeschke et al., 2008; Roff, 2002). Accordingly, the demographic approach to life history evolution has not provided (or sought) a unified explanation of the fast-slow continuum. Still, key theoretical results point to extrinsic mortality as a driver of earlier maturation/reproduction and lower investment in offspring quality (summarized in Del Giudice et al., 2015). The role of stochastic (unpredictable) variability in mortality rates is more complex; models indicate that stochasticity in adult survival should typically select for early reproduction, whereas stochasticity in juvenile survival delays reproduction and favors the evolution of bet-hedging (Charlesworth, 1994; see Ellis et al., 2009).

From the standpoint of *r/K* models, extrinsic mortality (for example via predation or disease) reduces population density and hence tends to increase *r*-selection; even under density dependence, high mortality rates select for earlier reproduction and a shorter lifespan. Similarly, environmental variability can be expected to favor faster growth rates and earlier reproduction (Section 2.2). While life history models admit all sorts of complications and exceptions (Roff, 2002), these convergent predictions suggest the tentative generalization that extrinsic mortality and stochasticity play important roles in the fast-slow continuum (Del Giudice et al., 2015; see also André & Rousset, 2020).

From a different perspective, allometric models seek to derive large-scale life history patterns from basic energetic constraints related to growth and body size. For instance, the model advanced by Charnov (1991) is able to reproduce the general pattern of correlations observed among life history traits (controlling for body size), by assuming a stable population, density-dependent juvenile mortality, and certain allometric relations between adult size and age of maturity (see also Charnov & Berrigan, 1993; Charnov, Gislason, & Pope, 2013). Another well-known example is the *metabolic theory of ecology* by Brown et al. (2004); Brown & Sibly (2006); Sibly & Brown (2007). The core idea is that the metabolic rate of an organism scales as a constant power of its body mass, and in turn determines the pace of other biological schedules—from reproductive rate to age at maturity and longevity. The main stumbling block for the metabolic theory is the fact that the fast-slow continuum remains largely intact after body size is controlled for (Section 2.1). This suggests that broad patterns of covariation between life history traits may owe more to selection than to simple metabolic constraints (for recent data in this direction, see Boyce, Mouton, Lloyd, Wolf, & Martin, 2020; Malerba & Marshall, 2019). Moreover, body size is implicated in all sorts of trade-offs: a larger body can reduce predation risk, enhance mating success, buffer the effects of competition in high-density ecologies, and so forth (see Brown & Sibly, 2006). Controlling for body size in comparative analyses also removes these adaptive effects, raising the question of whether mass-corrected correlations make sense from a theoretical standpoint (see Jeschke & Kokko, 2009; Roff, 2002).

3. The fast-slow continuum among individuals

In this section I take a close look at the logic of the fast-slow paradigm. I do so in four connected steps. First, I discuss the move from *between*-species patterns of life history variation to *within*-species patterns that resemble the fast-slow continuum (Section 3.1). Next, I consider the move from demographic traits such as fertility, longevity, and age at first reproduction to the behavioral and physiological traits that are the main focus of the fast-slow paradigm (Section 3.2). Third, I review the genetic and developmental mechanisms that may produce individual differences in life histories, as well as adaptive covariation among traits (Section 3.3). Fourth and finally, I address some factors that may complicate or obscure trait correlations at the between-individual level (Section 3.4).

3.1. The functional structure of life history strategies and the ecological gambit

All the life history traits discussed so far show considerable variation *within* species—not just among populations but also among individual organisms. The hypothesis at the heart of the fast-slow paradigm is that the structure of individual differences in life history traits resembles in important ways the structure of variation across species. This hypothesis depends on what I will call the *ecological gambit*: the working assumption that relations observed at the group level will hold at the individual level, for similar functional reasons.[2] If the assumption is supported, it offers a useful heuristic for studying individual differences and facilitates empirical progress. The ecological gambit is analogous to other heuristics routinely adopted in evolutionary biology, most notably the *phenotypic gambit* (the working assumption that the genetic architecture does not constrain which phenotypes can evolve in the long run; Grafen, 1984) and the *behavioral gambit* (the working assumption that psychological mechanisms do not constrain the expression of adaptive behavior; Fawcett, Hamblin, & Giraldeau, 2013). While these often prove to be reasonable starting assumptions, they may or may not apply to any specific case and can lead to errors if applied unthinkingly (see Nettle, Gibson, et al., 2013). The evil twin of the ecological gambit is the *ecological fallacy*—the assumption that group-level relations automatically or necessarily hold at the between-individual level (Robinson, 1950; see also Pollet, Tybur, Frankenhuis, & Rickard, 2014). Without a detailed understanding of the mechanisms that generate covariation within and between populations, it is hard to anticipate whether (and to what extent) the gambit is likely to be productive. In general, the gambit becomes riskier if causal factors that affect multiple variables of interest are known to operate at one level of analysis, but not at the other (see Pollet et al., 2014). While there is no guarantee that the gambit will succeed in any particular case, there may be conceptual reasons to treat the assumption of cross-level similarity as more (or less) biologically plausible.

3.1.1. General arguments for cross-level consistency

There are both general and specific arguments that lend initial plausibility to the ecological gambit for life history strategies. On the general side, adaptive life history strategies require the integration of multiple traits, and often show coordinated plasticity to environmental conditions (Braendle, Heyland, & Flatt, 2011; Roff, 2002). For this reason, life history traits can be expected to be developmentally and genetically correlated, with extensive pleiotropic effects (more on this in Section 3.3); this makes it more likely that within-species patterns of covariation also exist among populations and closely related species (Peiman & Robinson, 2017; see Réale et al., 2010). Moreover, developmental plasticity and pleiotropy bias the distribution of individual phenotypes, channeling the effects of mutations and environmental changes along the existing reaction surface[3] (often with adaptive

[2] Note that, in this paper, I focus specifically on patterns of differences and covariation *between* individuals, and do not address the topic of variation *within* the same individual over time (e.g., Dingemanse & Dochtermann, 2013).

[3] A reaction surface is the multivariate generalization of a *reaction norm* (see Pigliucci, 2005). A reaction norm is the function that describes how different phenotypes develop in response to different values of a single environmental variable; a reaction surface generalizes this concept to multiple dimensions of

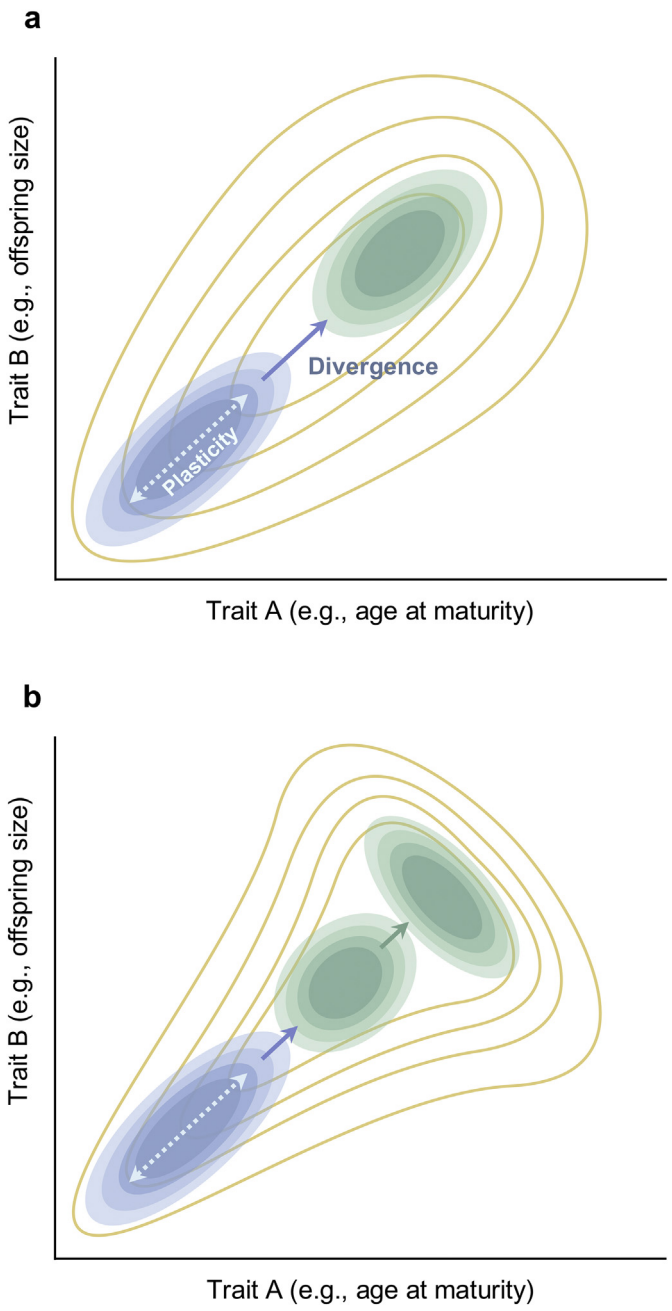

Fig. 2. Patterns of evolutionary divergence and the ecological gambit. In the ancestral population (lower left), developmental plasticity aligns with the genetic correlation between the two traits. Developmental and genetic correlations orient the initial response to selection along the axis of plasticity and facilitate adaptive evolution. In panel (a), the fitness landscape for the descendent population (upper right) maintains its features as the population diverges under selection. The trait correlation between populations mirrors the correlation within each population, and the ecological gambit is successful. In panel (b), the fitness landscape changes dramatically as the descendent population diverges; despite the initial bias produced by the genetic and developmental structure, selection eventually reverses the trait correlation. As a result, the ecological gambit succeeds in the ancestral population but fails in the descendent population.

consequences). The resulting developmental biases impose directionality on evolutionary trajectories, so that patterns of divergence between species and populations tend to align with correlations between individuals (Fig. 2a; Draghi & Whitlock, 2012; Ellis et al., 2009; Uller, Moczek, Watson, Brakefield, & Laland, 2018; West-Eberhard, 2003).

3.1.2. *The role of basic life history trade-offs*

To be sure, the arguments I just reviewed are merely suggestive: for example, even strong genetic and developmental correlations can break down relatively quickly under intense selection (e.g., Chippindale, Ngo, & Rose, 2003; Conner et al., 2011). The hypothesized similarity between within- and between-species patterns (e.g., Dammhahn et al., 2018) seems to require sufficient constancy in the underlying fitness landscape (Fig. 2). This is where the notion of basic life history trade-offs comes into play. Fundamental trade-offs such as those between current and future reproduction and between quality and quantity of offspring shape the allocations of individual organisms; but since population-level traits are averages of individual outcomes, the same trade-offs should be reflected at both the within- and the between-population levels (even if not identically). In other words, basic trade-offs contribute to defining a common fitness landscape for life history strategies across levels (see Wright et al., 2019). Another piece of the puzzle is that trade-offs are not functionally independent from one another. One of the key benefits of delaying reproduction is the ability to produce higher-quality offspring, which implies a functional link between the current-future reproduction trade-off and the quality-quantity trade-off (e.g., Wright et al., 2019). And for animals that increase offspring quality through parental care, the mating-parenting trade-off is going to overlap significantly with that between quality and quantity (see Del Giudice et al., 2015).

Even admitting exceptions and complications (Section 3.4), these functional links should tend to generate predictable relations among multiple life history traits, both within and across species. A much harder question is whether the same ecological factors that select for certain life history traits at the population/species level will also maintain genetic variation in the same traits among individuals, or prompt their development through mechanisms of adaptive plasticity. If one considers specific trade-offs, there are examples of developmental models that mimic the predictions of population-based demographic models. For instance, Berrigan and Koella (1994) found that the optimal plastic strategy involves early maturation in response to high juvenile mortality, and delayed maturation in response to energetic scarcity.

That said, there is an obvious theoretical gap regarding the fast-slow continuum as a whole. This is not surprising: as I noted in Section 2, even the fast-slow continuum across species is still waiting for a widely accepted formal explanation. The closest attempt so far is the recent verbal model by Wright et al. (2019). Drawing on recent versions of the *r/K* model (Engen et al., 2013; Engen & Sæther, 2016, 2017), these authors assumed that each species experiences a characteristic average level of density-dependent selection, leading to the emergence of a fast-slow continuum across species. On top of this average pattern, however, the intensity of density dependence within each species can be expected to fluctuate over time as populations grow and shrink. Wright and colleagues argued that fluctuating density-dependent selection explains the maintenance of individual variation on the fast-slow continuum (in combination with frequency-dependent selection within populations); crucially, the same process would also account for the existence of similar covariation patterns at the species and individual level of analysis.

None of the above implies that the between-individual fast-slow continuum of a given species should be *identical* to that of another, especially if distantly related. Even the comparative fast-slow continuum shows some meaningful differences among taxonomic groups, so a certain degree of variability is to be expected. For example, the strength of the functional link between offspring quality and future reproduction is likely to vary across species, and correlations between the relevant life history traits (e.g., age at maturity and offspring size)

(*footnote continued*)
the environment.

should change accordingly. In the recent biological literature on *pace-of-life syndromes* (POLS), some authors have defined the fast-slow continuum narrowly in terms of the current-future reproduction trade-off, partly to increase the precision of theoretical predictions (e.g., Araya-Ajoy et al., 2018; Dammhahn et al., 2018; Mathot & Frankenhuis, 2018). At the same time, the density-based model of POLS evolution by Wright et al. (2019) has brought the quality-quantity trade-off back into focus, thanks to its emphasis on density dependence. My point is that the trade-off between current and future reproduction should not be considered in isolation: trade-offs between offspring quality and quantity or between mating and parenting can be just as important, particularly for males—who can often make up for delayed reproduction by mating with more or higher-quality partners—and for long-lived species with flexible reproductive schedules and extended parenting, including humans (supplementary material S4).

To sum up: critics (e.g., Baldini, 2015; Schmitt, 2019; Stearns & Rodrigues, 2020; Zietsch & Sidari, 2020) are right to point out that patterns observed between species or populations cannot be automatically generalized to individuals within a species; the ecological gambit is only a working assumption, and the ecological fallacy is always a danger. In the case of life history strategies, there are arguments that lend face plausibility to the idea of a fast-slow continuum at the between-individual level, with the provision that the details of how trade-offs interact—and ife history traits correlate—are likely to show some variability across species. While this is encouraging for proponents of the fast-slow paradigm, these arguments have not been formalized in detail, and must be regarded as preliminary until then. Also, there is an important distinction between the weaker hypothesis that individual life history strategies will be partly described by a fast-slow continuum, and the stronger hypothesis that ecological factors—such as mortality, population density, and unpredictability—affect the development of individual strategies in the same way as they shape the evolution of strategies at the level of populations and species.

3.1.3. Baldini's critique

The role of ecological factors at different levels of organization is the topic of a widely read preprint by Baldini (2015), which in the words of Nettle (2018) has quickly become a "subcultural classic." The mathematical models in the paper are used to make two main points. The first is that various aspects of environmental harshness (e.g., mortality rates) can have different and even contrasting effects on the evolution of population-level life history traits (e.g., age at maturity), conditional on other factors such as the presence of density-dependent selection. While this is not a novel insight, it is true that density dependence moderates the impact of mortality on life history evolution (Section 2.2). This point has often been neglected in the human literature. However, there are exceptions: Ellis et al. (2009) discussed density dependence in considerable detail, and Sng and colleagues have started to explore the relations between population density and life history-related traits (Sng & Ackerman, 2020; Sng, Neuberg, Varnum, & Kenrick, 2017). A problem with Baldini's model—and a likely source of confusion about its implications—is that "extrinsic mortality" is defined so as to be preventable by investing resources in survival (with increasing marginal returns). This is not how extrinsic mortality is usually conceptualized (see Footnote 1); some counterintuitive results of the model follow directly from this inconsistency (André & Rousset, 2020).[4]

The second major point of Baldini's paper is that the optimal plastic response to a given ecological factor (e.g., mortality) at the individual level does not necessarily mirror the evolutionary response of the population to the same factor (e.g., Kawecki & Stearns, 1993). This is a valid concern that has not been adequately addressed in the human literature. However, Baldini's main result regarding the optimal response to variation in mortality depends on the same idiosyncratic definition of extrinsic mortality discussed above (André & Rousset, 2020). Moreover, the model assumes that ecological conditions vary across space but not over time, so that the population effectively contains multiple independent sub-populations at any given time. When the environment fluctuates over time, the entire population undergoes the same changes in conditions and the logic of Baldini's model does not apply. (In most realistic scenarios, spatial and temporal variation coexist; see Starrfelt & Kokko, 2013.) This is relevant because some models of individual variation in life history strategies (e.g., Del Giudice, 2012; Wright et al., 2019) are explicitly based on temporally variable selection (Section 3.3).

3.2. The role of behavior and physiology in life history strategies

In a narrow sense, life history strategies are patterns of allocation among fitness components; within the constraints of various trade-offs, these allocations determine the life history traits of individuals and species (summarized in Del Giudice et al., 2015). The basic life history traits are age at first reproduction, age-specific fertility, and age-specific mortality; these are also called *direct fitness traits* (Roff, 2002) because they are sufficient to calculate r and R_0, as well as other demographic traits such as the expected fertility rate and longevity. However, life history strategies do not exist in an abstract theoretical world: what actually mediates allocations and trade-offs are the organism's behaviors, physiological mechanisms, and physical characteristics. "Growth" is the outcome of a causal pathway that includes feeding and foraging behaviors, plus the metabolic and hormonal mechanisms that convert energy into tissues. "Survival" necessitates the activity of myriad processes, from immunity and other forms of body maintenance to the behavioral mechanisms that mediate fear, risk-avoidance, disgust, and so on. Besides its many physiological requirements, "reproduction" is brought about by a constellation of complex behaviors that may include courtship, parental behaviors, and pair-bonding. This broader, process-oriented view of life history strategies is illustrated in Fig. 3.

From this perspective, life history strategies are expressed as combinations of coadapted behavioral, physiological, and morphological traits (Braendle et al., 2011). Ultimately, these traits contribute to fitness through their effects on basic life history parameters—age at first reproduction, fertility, and mortality—within the constraints set by allocation trade-offs (Fig. 3). This is the foundation of the POLS concept and of the fast-slow paradigm more broadly: if life history strategies can be arranged on a fast-slow continuum within a species, this will partly explain the covariation among the behavioral and physiological traits that mediate the underlying trade-offs (e.g., Dammhahn et al., 2018; Figueredo et al., 2006; Réale et al., 2010). I suggest to refer to these traits as life history-*related* (or some equivalent label) to mark the distinction with demographic life history variables such as fertility and mortality. More specifically, life history-related traits should (a) be intra-individually stable enough to be treated as individual differences variables; (b) covary with basic life history traits and/or other outcomes of life history allocations (e.g., number of sexual partners, age of reproduction); and (c) plausibly contribute to mediating those allocations, or at least function as proxies of traits that do. As I discuss in Section 3.4, patterns of covariation may be complex and context-dependent, and the causal role played by any given trait may be quite indirect. The point is that putative life history-related traits must be validated against tangible outcomes to avoid circular reasoning (Copping et al., 2017; Zietsch & Sidari, 2020).

Fig. 3 helps to make an important but sometimes overlooked point: life history strategies are formally defined at the level of allocations and

[4] Another questionable aspect of the model is that the fertility rate is assumed to be directly proportional to the age at maturity, so that—all else being equal—later-maturing organisms produce more offspring per year, without any quality-quantity trade-off. This assumption runs counter the empirical pattern observed in mammals and birds, and may explain some counterintuitive predictions of the model in the density-independent scenario.

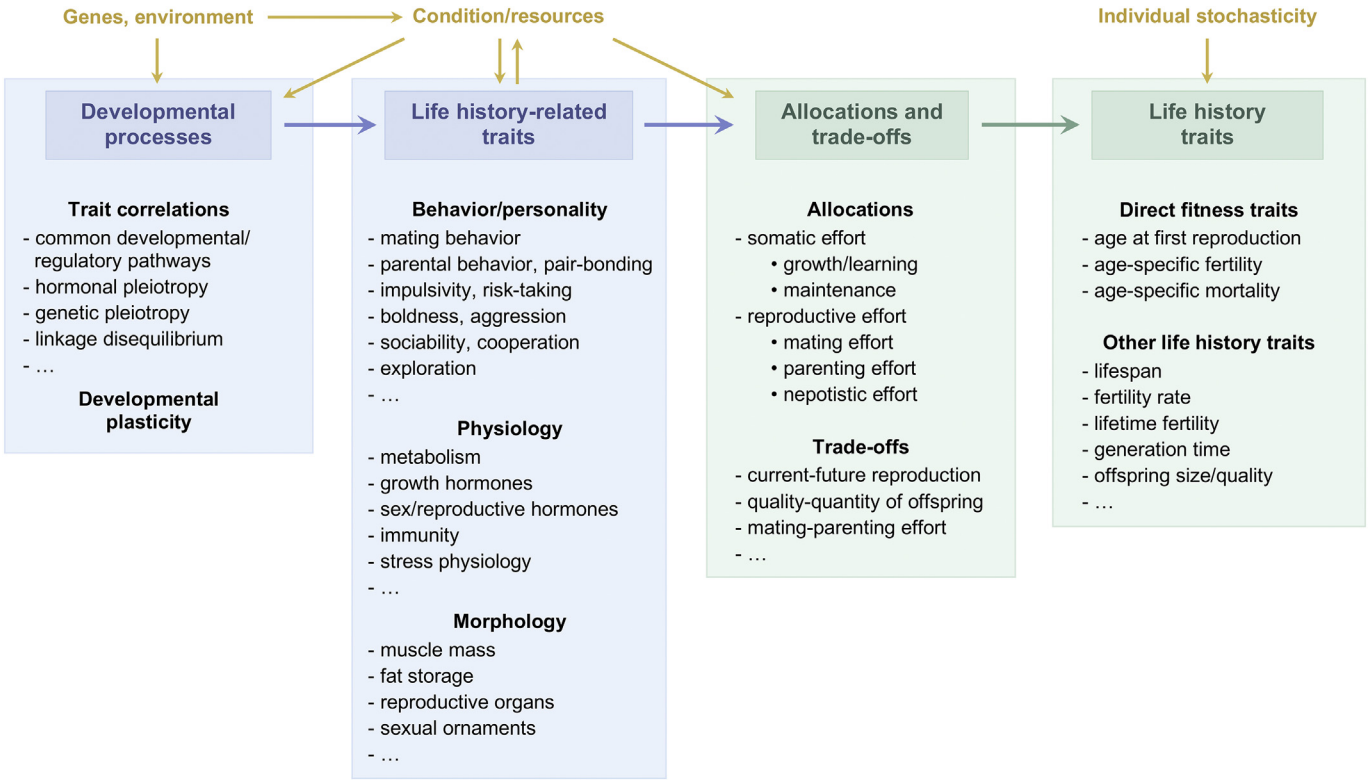

Fig. 3. A process view of life history strategies. Note how individual differences in condition and resources influence the development and expression of life history-related traits, are affected in return, and partly determine the efficiency and outcome of allocations.

trade-offs, *not* at that of specific behavioral and physiological traits. In many if not most instances, the same behaviors may be involved in different allocations, serve multiple functions, or even play functionally opposite roles in different contexts, individuals, or species (Section 3.4). For example, exploratory behaviors may reflect both risk-taking (e.g., risking predation and injuries while searching for food or mates) and risk-avoidance (e.g., searching the surroundings for cues of hidden dangers). Similarly, the "competitive" traits favored in high-density ecologies may take a number of rather different forms—from large body size and aggression to sociability and cooperation (Ellis et al., 2009; Wright et al., 2019).

Neglect of this crucial distinction has been a major problem in research on animal POLS, and has likely contributed to the many inconsistent findings in this literature (Royauté et al., 2018; Smith & Blumstein, 2008). When Réale et al. (2010) put forward a list of plausible life history-related traits (e.g., high activity and high sympathetic activity in "fast" individuals), they also warned that the list was tentative and unlikely to be widely applicable across species. Unfortunately, the list was quickly reified; investigators went on to test the suggested correlations in a variety of species, usually without testing key assumption about the functional role of the traits they measured. This is especially problematic given the dearth of formal models linking life history trade-offs to the evolution of specific behavioral/physiological traits (Dammhahn et al., 2018; Mathot & Frankenhuis, 2018). More recent work has started to address these questions more explicitly (e.g., Fenneman & Frankenhuis, 2020).

3.3. Mechanisms of trait variation and covariation

3.3.1. Genetic variation

To a first approximation, the genetic architecture of life history strategies is similar in humans and nonhuman animals. The heritability of life history traits such as longevity, age at first reproduction, and lifetime reproductive success tends to range between 10% and 40% (Briley, Tropf, & Mills, 2017; Kosova, Abney, & Ober, 2010; Madrigal, Relethford, & Crawford, 2003; Roff, 2002; Stearns, Byars, Govindaraju, & Ewbank, 2010; Tropf, Barban, Mills, Snieder, & Mandemakers, 2015), in line with the stochastic nature of these outcomes (Section 3.4). Likewise, the heritability of personality is 40-50% in adult humans and about 50% across species (Briley & Tucker-Drob, 2017; Dochtermann, Schwab, & Sih, 2015; Vukasović & Bratko, 2015). The distribution of genetic effects also follows a common template: with few exceptions (see Weitekamp and Keller, 2019), behavioral traits tend to be highly polygenic, with many loci of small effect and few (if any) loci with common alleles of large effect (Chabris et al., 2015; Sella & Barton, 2019; Weitekamp and Keller, 2019).

The two main processes that can maintain genetic variability in traits under selection are *mutation-selection balance* and *balancing selection* (see Gangestad, 2011; Keller, 2018; Sella & Barton, 2019). Deleterious mutations contribute substantially to individual condition (Section 3.4; Zietsch & Sidari, 2020); those with large detrimental effects are quickly purged by selection and hence remain rare, whereas slightly deleterious alleles can persist at high frequencies for a long time (Keller, 2018). In humans, rare and structural variants (e.g., copy number variations) may account for 50% or more of the genetic variance of traits such as height, intelligence, and neuroticism (Hill et al., 2018; Nolte et al., 2019; Wainschtein et al. 2019).

The role of mutations is not controversial, even if it has not always received the attention it deserves in the life history literature. The main point of contention is whether common genetic variation in life history-related traits is partly maintained by balancing selection—that is, selection that systematically changes direction across individuals, space, or time. Fluctuating selection and frequency-dependent selection are specific kinds of balancing selection. In animals with complex social systems, frequency-dependent selection can occur via social "niche-picking," whereby individuals seek out roles that match their phenotypes.

Critics argue that balancing selection should yield genetic

architectures in which high-frequency alleles account for most of the variance—a pattern inconsistent with the empirical data for most quantitative traits (Verweij et al., 2012; Zietsch & Sidari, 2020). Genomic studies have found relatively few loci matching the expected signatures of balanced polymorphisms (but see Bitarello et al., 2018); accordingly, many authors regard balancing selection as a marginal phenomenon, despite ample evidence of variable selection in nature (see Messer, Ellner, & Hairston Jr, 2016; Thompson, 2013). Skepticism is even stronger for hypotheses that involve temporal fluctuations, which—in contrast to spatial fluctuations—are often believed to be ineffective in maintaining genetic variation (Messer et al., 2016). This is important because fluctuations over time could plausibly generate individual differences along a fast-slow axis. For example, Wright et al. (2019) recently proposed fluctuating density-dependent selection as a general explanation for individual variation in POLS. But there are other features of the environment that may play a similar role. For example, I have argued that temporal fluctuations of the sex ratio determine shifts in the costs and benefits of life history allocations, particularly between mating and parenting (Del Giudice, 2012).

As it turns out, these widespread ideas about balancing selection and temporal variation have been substantially revised in the theoretical literature (see Del Giudice, 2012; Penke & Jokela, 2016). To begin, it has become clear that balancing selection takes an exceedingly long time to generate its classic signatures. In realistic scenarios involving highly polygenic traits, balancing selection is not expected to produce common alleles of large effect, and can be very hard to distinguish from recent positive selection or even neutrality (Connallon & Clark, 2013; Fijarczyk & Babik, 2015). Likewise, contemporary models show that temporal fluctuations can be quite effective in maintaining genetic variation, particularly in species with overlapping generations (including humans; Bertram & Masel, 2019; Ellner & Sasaki, 1996; Yamamichi and Hoso, 2017). A more detailed overview of these issues can be found in the supplementary material (S2).

3.3.2. *Developmental plasticity*

It should often be adaptive for organisms to adjust their life history allocations based on cues about the state of the environment and/or their own condition. However, sensitivity to the environment can also be detrimental—for example because it exposes individuals to dysregulation and mismatch—and adaptive plasticity is often assumed rather than convincingly demonstrated (e.g., Hendry, 2015). Whether plasticity is favored depends on a number of factors including the reliability of cues, the stability of the environment across an individual's lifetime, and the predictability of future states (Nettle & Bateson, 2015). The same factors determine the optimal balance in the integration of genetic and environmental information (McNamara, Dall, Hammerstein, & Leimar, 2016). Unfortunately, for many organisms—including humans—the relevant statistical features of the ecology are unknown or still poorly understood (Frankenhuis, Nettle, & Dall, 2019).

In the classic view of plasticity, the developing organism employs early life cues (e.g., nutrition, exposure to stressors) to forecast the future state of the environment, and match its phenotype to the predicted state ("external" predictive-adaptive response; see Bateson, Gluckman, & Hanson, 2014). An alternative possibility—especially when the environment is unpredictable or changes too quickly relative to the organism's lifespan—is to use early cues to forecast the future state of the soma instead of that of the environment ("internal" predictive-adaptive response; see Nettle & Bateson, 2015; Nettle, Gibson, et al., 2013). For example, early stress may work as a cue of a dangerous environment, but may also cause somatic wear and tear, which in turn may reduce the individual's expected survival (e.g., Chang et al., 2019; Dunn, Andrews, Nettle, & Bateson, 2019; Rickard, Frankenhuis, & Nettle, 2014). There is urgent need for realistic models; as I noted in Section 3.1, the assumption that plastic responses at the individual level should mimic evolutionary responses at the population level is not based on formal theory, and cannot be taken for granted.

Another urgent question in the human literature is how to reconcile theories of adaptive plasticity with the findings of behavior genetics. For most life history and life history-related traits, twin studies show a small to negligible role of shared environmental factors—that is, factors that act consistently on siblings from the same family, increasing their similarity (e.g., Bartels, Van den Berg, Sluyter, Boomsma, & de Geus, 2003; Briley et al., 2017; Briley & Tucker-Drob, 2017; Franz et al., 2010; Morris, Jones, Schoemaker, Ashworth, & Swerdlow, 2011; Ouellet-Morin et al., 2009; Polderman et al., 2015; Tropf et al., 2015; Tucker-Drob et al., 2017). In most cases, their estimated contribution in adulthood is less than 10% of the variance (for some notable exceptions see Kendler, Ohlsson, Lichtenstein, Sundquist, & Sundquist, 2019). On the face of it, this is a puzzling pattern. If developmental processes respond to life history-relevant factors such as mortality and resource availability, and if parental behaviors and family stress act as cues during childhood (Belsky et al., 1991; Chisholm, 1993, 1999; Ellis et al., 2009), what explains the surprisingly small impact of growing up in the same family?

One possibility is that developmental plasticity is mostly non-adaptive, or does not respond to the particular factors emphasized in the fast-slow paradigm (Zietsch & Sidari, 2020). Instead, the apparent effects of family variables may be mediated by shared genetic factors—a suspicion reinforced by the lack of genetic controls in most developmental studies (e.g., Barbaro et al., 2017). However, this is not the only possible explanation. For example, individuals may systematically differ in their level of plasticity, as postulated by theories of *differential susceptibility* (see Belsky & Pluess, 2013; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, M. H., 2011; Ellis & Del Giudice, 2019). Under some conditions, this type of G × E interaction may account for the results of twin studies, and even accommodate a major role of the shared environment in shaping early plasticity (Del Giudice, 2016). Natural experiments have shown that children respond to traumatic events (e.g., earthquakes) with accelerated maturation and increased reproductive effort (Lian et al., 2018; Pesonen et al., 2008); but the impact of weaker, common stressors that vary across families is still unclear. The tension between developmental models and behavior genetic findings is a major challenge that needs to be recognized and addressed.

For reasons of space, I will not discuss developmental factors further in this paper. For some recent (but not genetically informative) studies that relate childhood stress to life history-related traits in adolescence and adulthood see Chang and Lu (2018), Chang et al. (2019), Chang et al. (2019), Deater-Deckard, Li, Lee, King-Casas, and Kim-Spoon (2019), Holdsworth and Appleton (2020), Mell, Safra, Algan, Baumard, and Chevallier (2018), Sear, Sheppard, and Coall (2019), and Szepsenwol et al. (2017).

3.3.3. *Mechanisms of covariation*

From a genetic perspective, trait correlations can arise in a number of ways (Saltz, Hessel, & Kelly, 2017). Correlational selection for specific combinations of traits, assortative mating, and other forms of social selection may produce nonrandom associations between alleles (linkage disequilibrium). As noted by Zietsch and Sidari (2020), correlations due to linkage disequilibrium are usually volatile and temporary (Roff & Fairbairn, 2007; Saltz et al., 2017; see also Revell, 2007). However, it has been suggested that persistent correlational selection may favor the evolution of regulatory loci that produce the same association through pleiotropic effects (Peiman & Robinson, 2017; Roff & Fairbairn, 2007). Also, fluctuating selection that acts simultaneously on multiple traits (co-selection) can favor the evolution of genetic correlations via pleiotropy (Pavličev, Cheverud, & Wagner, 2011; see also Pavličev & Cheverud, 2015).

Especially when they get entrenched in developmental processes, pleiotropic effects can be stable and fairly robust. Deleterious mutations can also have broad pleiotropic effects, and tend to produce positive correlations among traits that reflect individual condition or quality

(Section 3.4). In principle, genetic correlations among traits could be suppressed or reversed at the phenotypic level by environmental factors acting in the opposite direction. In practice, phenotypic correlations generally have the same sign of the corresponding genetic correlations, and tend to be only somewhat smaller in magnitude (Dochtermann, 2011; Kruuk, Slate, & Wilson, 2008; Roff, 1996; Sodini, Kemper, Wray, & Trzaskowski, 2018). The implication is that genetic and environmental effects tend to act in the same direction; this suggests a confluence of adaptive plasticity and balancing selection on the same phenotypic traits, and/or a confluence of mutations and environmental disturbances on the same developmental mechanisms.

When pleiotropic correlations evolve as a result of trade-offs, they are usually produced by multiple loci of small effect rather than "master switch" genes (Saltz et al., 2017). Zietsch and Sidari (2020) cite the absence of large-effect pleiotropic alleles to dismiss the possibility of adaptive trait coordination via genetic mechanisms. It is entirely possible to envision polygenic regulatory mechanisms that receive inputs from multiple genetic and environmental sources, and adaptively coordinate the expression of suites of traits. In fact, this is precisely how endocrine systems work (Ketterson, Atwell, & McGlothlin, 2009; Vitousek & Schoenle, 2019). Hormones are agents of biological coordination: they regulate cell activity and gene expression across multiple tissues and achieve integration among physiological, morphological, and behavioral traits (Cox, McGlothlin, & Bonier, 2016). The ability of hormones to influence many target tissues at once has been called *hormonal pleiotropy*; in addition, hormones often act as physiological mediators of pleiotropy at the genetic level (Dantzer & Swanson, 2017; Ketterson and Nolan Jr, 1999; Ketterson et al., 2009).

The major endocrine systems that regulate life history allocations are remarkably conserved across species (Vitousek & Schoenle, 2019). For instance, testosterone and other androgens regulate trade-offs between mating, parenting, and survival in vertebrates (Hau & Wingfield, 2011; Ketterson et al., 2009). In both vertebrates and invertebrates, insulin-like growth factor 1 (IGF-1) regulates trade-offs between growth, fertility, and survival (Gerish & Antebi, 2011; Swanson & Dantzer, 2014; Vitousek & Schoenle, 2019). Other vital coordination roles among growth, metabolism, immunity, reproduction, and behavior are played by the HPA axis (Crespi, Williams, Jessop, & Delehanty, 2013; Ellis & Del Giudice, 2014; Hau, Casagrande, Ouyang, & Baugh, 2016), in concert with the hypothalamic-pituitary-gonadal (HPG) and hypothalamic-pituitary-thyroid (HPT) axes, the IGF-1 signaling system, and pathways involving prolactin, oxytocin, vasopressin/vasotocin, and immune cytokines (Del Giudice, 2018; Ellis, 2013; Lancaster & Sinervo, 2011; Vitousek & Schoenle, 2019). By virtue of their biological function, hormonal mechanisms are both plastic and highly pleiotropic (Vitousek & Schoenle, 2019). Much of the evolution of endocrine systems takes places through changes in the receptivity and gene expression of individual tissues, rather than at the level of the central signaling molecules (Dantzer & Swanson, 2017). Hence, their genetic basis is going to be quite complex, with many layers of upstream and downstream regulation and different roles for the same genes across organs and cell types.

Naturally, hormones are not the only coordination mechanisms that may generate adaptive trait covariation. Neurocognitive mechanisms collect, interpret, and integrate information from the environment; generate predictions about the future and evaluate their validity; and can regulate life history allocations in myriad ways—not just through behavior, but also via top-down regulation of endocrine systems (see Del Giudice et al., 2015). An example in humans is the interplay between social, cognitive, and physiological mechanisms in the process that leads to the start of reproduction (Nettle, 2011).

3.4. Patterns of covariation at the between-individual level

In Section 3.1 I reviewed reasons why between-species relations among life history traits can be tentatively used as a guide to relations within species (the ecological gambit). I now discuss some factors that, *even if the ecological gambit is valid at the functional level of trade-offs*, may attenuate or even reverse the observed correlations between traits. These factors are individual stochasticity in life history outcomes; individual differences in condition and resources; complex functional relations involving behavioral and physiological traits; and measurement error. Without careful consideration of these factors, it is easy to take a literal view of the ecological gambit, and assume that correlations at the between-individual level should be just as strong and clear-cut as those at the level of species. A related expectation is that, when individual life history and/or life history-related traits are subjected to factor analysis or PCA, the results should mirror those of comparative studies—with a strong, general axis of fast-slow variation accounting for a large proportion of trait variance. In my view, these misleading expectations have implicitly shaped the debate around the fast-slow paradigm in humans (e.g., Richardson, Sanning, et al., 2017; Zietsch & Sidari, 2020).

3.4.1. Individual stochasticity

Because life history events such as death and reproduction have a stochastic component, life history traits will show a considerable amount of variation even if all the individuals in a population are identical and make the same allocations (Caswell, 2009; Steiner & Tuljapurkar, 2012). This is the pervasive phenomenon of *individual* or *demographic stochasticity* (see Cam, Aubry, & Authier, 2016; Caswell, 2009; Sæther & Engen, 2015). As a result of individual stochasticity, between-individual correlations involving life history traits will be necessarily smaller than those between species or populations. The contribution of stochasticity can be estimated using demographic models, and is often the dominant component of variation. For instance, chance accounted for a median 65% of variance in longevity across invertebrate species (Hartemink & Caswell, 2018); in a seabird, the proportion was 66% of longevity and 61% of lifetime reproductive success (Snyder & Ellner, 2018).

Individual stochasticity does not affect correlations across species or populations, which are based on group-level averages. But at the between-individual level, it attenuates the correlations among some life history traits (e.g., age at first reproduction and fertility) and puts a low ceiling on the correlations between life history traits and the behavioral/physiological traits that mediate the underlying allocations—even in presence of strong causal effects (e.g., Araya-Ajoy et al., 2018). To illustrate, consider a hypothetical species in which 50% of the variance in longevity is due to chance, and a variable X that predicts individual survival. Even if X accounts for *all* the explainable variance in survival and is measured without error, its expected correlation with longevity is only 0.71. If variable X accounts for 25% of the explainable variance and is measured with 80% reliability, the correlation drops to only 0.32. Clearly, it is unrealistic to expect strong associations at the between-individual level when traits are substantially affected by stochasticity.

In humans, correlations between personality traits and longevity tend to be less than 0.10, and correlations between personality and measures of fertility rarely exceed 0.20 (e.g., Alvergne, Jokela, & Lummaa, 2010; Briley et al., 2017; Gurven, von Rueden, Stieglitz, Kaplan, & Rodriguez, 2014; Jokela, Alvergne, Pollet, & Lummaa, 2011; Međedović, Šoljaga, Stojković, & Gojević, 2018; Terracciano, Löckenhoff, Zonderman, Ferrucci, & Costa Jr., 2008). These effect sizes necessarily understate the true strength of the functional links between personality and life history. Similar considerations apply to traits such as puberty timing and age at menarche, which are the end results of stochastic physiological processes; but also to competitive outcomes such as mating success and dominance, which are partly determined by luck (see Frankenhuis and Del Giudice, 2012).[5]

[5] If accurate estimates of stochasticity are available, one may get a more

3.4.2. Individual differences in condition and resources

The next factor to consider at the within-species level is individual variation in condition and resources (Fig. 3). If some individuals enjoy higher genetic quality (fewer deleterious mutations) or a more favorable environment, they may be able to acquire and allocate more resources to multiple fitness components at once, or achieve fitness-related outcomes more efficiently (e.g., attractive individuals may obtain the same mating success with less effort). In other words, they are less constrained in their allocation decisions than worse-off individuals (Reznick, Nunney, & Tessier, 2000; van Noordwijk & de Jong, 1986). The interplay between resource acquisition and allocation can be quite complex (Roff & Fairbairn, 2007), and may involve feedback loops in which an organism's life history-related traits (e.g., risk-taking, aggression, foraging behaviors) partly determine its future condition and resources (see Fig. 3). Trade-offs may also be loosened when organisms have access to effectively unlimited resources without competition and danger, as is common in laboratory studies (see Reznick et al., 2000; Royauté et al., 2018).

All else being equal, a functional trade-off between competing components should give rise to a negative correlation at the genetic and/or phenotypic level. But when there are sizable differences in condition/resources, the predicted negative correlation can be attenuated, erased, or even reversed (see Reznick et al., 2000; Roff & Fairbairn, 2007; Stearns, 1989). If individual differences in condition/resources are caused primarily by environmental factors, trade-offs will be masked at the level of phenotypic correlations but manifest in genetic correlations. However, it is often the case that condition and resource acquisition have a genetic component, maintained at least in part through mutation-selection balance (Section 3.3). If so, trade-offs may also be masked at the level of genetic correlations (Houle, 1991; Wilson, 2014).

Simulation studies clearly illustrate how variation in condition/resources can obscure the role of strategic allocations (e.g., Araya-Ajoy et al., 2018). The take-home point is that phenotypic and genetic correlations between life history traits cannot always be taken at face value; methods that work at the level of species and populations (e.g., PCA of uncorrected life history traits; see Araya-Ajoy et al., 2018) may give misleading results when applied to individuals. This is highly relevant to interpreting matrices of genetic correlations among traits, such as the one shown by Zietsch and Sidari (2020).[6] A pattern of positive genetic correlations along an axis of individual "quality" does not falsify the hypothesis of a fast-slow continuum; in fact, the functional relations generated by life history trade-offs may become apparent only after the general quality factor has been statistically controlled for (see e.g., McLean, Archie, & Alberts, 2019; Wilson, 2014). More precisely, this is the expectation if the analysis is restricted to classic life history traits such as fertility and longevity. As I discuss next, behavioral and physiological traits often show complex functional relations that may not be adequately represented by linear correlations. Also, the role of individual condition/resources may be complicated by the presence of bidirectional relations with life history allocations (Fig. 3; Roff & Fairbairn, 2007). On the other hand, the behavioral and physiological traits that mediate life history allocations can be more revealing of the underlying trade-offs than the outcomes of those allocations. For example, the desire for short-term sexual encounters and that for stable, long-term relationships play a role in mediating the trade-off between mating and parenting at the behavioral level; these dispositions are negatively correlated (e.g., Holtzman & Strube, 2013; Jackson & Kirkpatrick, 2007), even if higher-quality (e.g., richer, healthier, more attractive) individuals have more access to short-term sexual partners *and* can provide better investment for their children.

3.4.3. Complex relations among traits

A common pitfall of the fast-slow paradigm is the (often implicit) assumption that relations between life history variables and their behavioral/physiological correlates should follow a simple linear pattern, so that fast versus slow strategies correspond to high versus low levels of the trait. For example, Réale et al. (2010) predicted—among other things—that "slow" individuals would tend to be nonaggressive, shy, and high in hypothalamic-pituitary-adrenal (HPA) reactivity, whereas "fast" individuals would be aggressive, bold, and low in HPA reactivity. Similar predictions in the domain of cognition were made by Sih and Del Giudice (2012). While this kind of pattern may apply to some traits with a relatively straightforward role in allocation trade-offs (e.g., impulsivity), as a general expectation it can be seriously misleading. Physiology and behavior often show complex and context-dependent relations, both with one another and with life history-relevant outcomes (e.g., Adkins-Regan, 2005; Salzman, McLaughlin, Westneat, & Crowley, 2018). Even if a trait is functionally related to the fast-slow axis of variation, the *form* of the relation can easily be nonlinear and/or interactive.

For an illustration, consider the *adaptive calibration model* of stress responsivity in humans (Del Giudice, Ellis, & Shirtcliff, 2011; Ellis & Del Giudice, 2014). A key prediction of the model is that the optimal level of HPA reactivity should vary nonlinearly as one moves from extremely safe and supportive to extremely dangerous environments, so that both "fast" and "slow" constellations of traits can be associated with high (or low) HPA reactivity. In addition, we predicted an interaction with sex, with males switching to low-reactivity patterns more readily than females when growing up in dangerous, high-risk environments. If the model is broadly correct (see Ellis & Del Giudice, 2019 for an empirical update), linear correlations between HPA reactivity and other life history-related traits will yield inconsistent findings, obscuring the role of the HPA axis in the regulation of life history strategies.

Nonlinear and interactive associations present obvious problems to analytic techniques based on linear associations, including standard varieties of PCA, factor analysis, and network analysis. Attempts to recover a fast-slow continuum by analyzing correlation matrices of behavioral and/or physiological traits are fraught with problems. This is unfortunate, but the relative simplicity of the fast-slow continuum does not automatically extend to the behavioral and physiological traits that mediate the underlying trade-offs. In some cases, the relations between traits and outcomes may be relatively straightforward and adequately described by simple linear models; but other times, the only solution is a detailed functional understanding of the trait and its costs, benefits, and constraints, which may vary across species and contexts (see Mathot & Frankenhuis, 2018).

3.4.4. A note on measurement

In the human literature based on the fast-slow paradigm, behavioral traits are typically assessed via self-report. In recent years, some widely used questionnaires have been criticized for lacking external validity,

(*footnote continued*)
realistic sense of the functional associations between traits by adjusting the observed correlation for the impact of chance, not unlike correcting for measurement error. The adjusted correlation would then refer to the relation between the "explainable portions" of the variables.

[6] The matrix in Figure 1 of Zietsch and Sidari (2020) includes a variety of physical symptoms/illnesses, some psychiatric disorders, and indicators of well-being, but no classic life history traits and none of the behavioral traits that are usually regarded as core life history markers in humans (e.g., impulsivity, risk-taking, sociosexuality). Thus, I am not suggesting that a different analysis of this particular matrix would recover a meaningful fast-slow continuum of variation. Some authors have proposed a general factor of mental and physical health ("covitality") as a component of slow life histories, with the rationale that slow strategists can invest more in somatic maintenance and are likely to receive better care from parents (Figueredo, Vasquez, Brumbach, & Schneider, 2007; Figueredo, Vásquez, Brumbach, & Schneider, 2004). However, this argument fails to consider the role of deleterious mutations. In later work, Sefcek and Figueredo (2010) acknowledged that mutation load must be included in the picture, and that the relation between health and life history strategies is likely more complex than they originally envisioned.

employing circular or otherwise inadequate validation procedures, and mixing conceptually distinct types of constructs within a single measure (for an overview of the debate see Black, Figueredo, & Jacobs, 2017; Copping et al., 2014b, 2017; Figueredo et al., 2015; Richardson, Sanning, et al., 2017; Zietsch & Sidari, 2020). To address the perceived limitations of self-reports, some researchers have started to measure putative life history-related traits such as impulsivity, risk-taking, and cooperation with standardized tasks instead of self-reports (for examples within the fast-slow paradigm, see Copping et al., 2014a; Wu et al., 2017). The available procedures include motor/attentional inhibition tasks, monetary choice tasks involving delays or variable outcomes, and economic games designed to elicit altruism, trust, and prosociality. These tasks lend themselves to computational analysis and promise to be more objective than traditional self-reports.

In practice, however, the validity of laboratory tasks turns out to be questionable. For example, executive tests of inhibition also measure general intelligence (Friedman et al., 2008), and delay discounting tasks tend to mistake risk aversion for impulsivity (Lopez-Guzman, Konova, Louie, & Glimcher, 2018). As a result, self-reported impulsivity and disinhibition predict real-world outcomes (e.g., substance use, sexual behavior) much better than standardized tasks (Creswell, Wright, Flory, Skrzynski, & Manuck, 2019; Leeman et al., 2018; Venables et al., 2018; Wilson & Daly, 2006). Likewise, economic games in the laboratory often fail to predict similar behaviors in the field (e.g., Galizzi and Navarro-Martínez, 2018; Voors, Turley, Kontoleon, Bulte, & List, 2012). Of course, games are very brief samples of behavior, and their individual reliability is low; for example, the correlations of economic games with other measures of cooperation and prosociality are usually smaller than 0.20, which helps explain the inconsistent results in the literature (e.g., Ferguson, Zhao, O'Carroll, & Smillie, 2019; McAuliffe, Forster, Pedersen, & McCullough, 2019; Zhao & Smillie, 2015; see also Dang, King, & Inzlicht, 2020). The standardized behavioral assays used to assess personality in nonhuman animals suffer from similar problems (e.g., Beckmann & Biro, 2013; Carter, Marshall, Heinsohn, & Cowlishaw, 2012; Niemelä & Dingemanse, 2018), but without the option of using self-reports. While laboratory tasks can be informative, they are far from "gold standards" and should be used and interpreted with caution.

3.4.5. Trade-offs and life history-related traits in humans

As I noted earlier, the general concept of the fast-slow continuum should be adapted to the particular ecology of each species to work as a useful heuristic. In the supplementary material (S3) I offer some reflections on certain notable features of the human ecology, and their implications for life history trade-offs and strategies. I also summarize my recent proposal for a descriptive model of life history-related traits in our species (Del Giudice, 2018).

4. Conclusion

Over the past thirty years, the fast-slow paradigm of individual differences has stimulated a remarkable amount of research. At the same time, empirical work in this area has too often drifted away from its theoretical premises, and many important gaps and questions have remained unaddressed. Now the paradigm is entering a new life stage—perhaps moving from a turbulent adolescence to the beginnings of maturity. In this paper I made a systematic attempt to lay out the logic of the paradigm, point out its current weaknesses, and identify opportunities for progress and improvement. I have argued that the fast-slow continuum can be a productive heuristic for individual differences, but there is clearly much work to do. I look forward to a new wave of research in this area, and to the insights and surprises it will certainly bring.

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