Rethinking the Fast-Slow Continuum of Individual Differences

Supplementary Material

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S1. The problem of axis rotation and its implications for the fast-slow continuum

In this supplement I address the pervasive but often overlooked problem of axis rotation in principal component analysis (PCA). As I note in the main text (Section 2.1), a number of seemingly inconsistent findings in the comparative life history literature are explained by irrelevant differences in the orientation of the axes used to summarize the data. Here I focus on PCA because it is the technique of choice in comparative studies; however, similar issues may arise in standard applications of exploratory factor analysis (EFA).

In comparative research, PCA is commonly used to identify the major axes of life history variation across species. For simplicity, in the examples that follow I assume that the data reflect two biologically significant axes of variation (the "true" axes), one of which represents a fast-slow continuum (Figure S1.1). This is a common pattern in the life histories of animals (e.g., Healy et al., 2019) and plants (e.g., Salguero-Gómez, 2017). In the unrotated PCA solution, the first component (i.e., the first axis of the transformed space) follows the direction of maximum variance in the data; the second component follows the direction of largest variance uncorrelated with the first; and so on. However, the space defined by these components is one possibility out of infinitely many, since any rotation of the axes around the origin yields a mathematically equivalent description of the data. The only thing that makes the unrotated PCA solution unique is that the axes are oriented so as to maximize the proportion of variance explained. There is no reason why the unrotated axes should represent the most biologically meaningful dimensions of variation, unless the latter also happen to explain the largest amount of variance in the data.

It follows that, if the main axis of variation in the data accounts for a large proportion of the total variance, it will be adequately captured by the first unrotated component (PC1; Figure S1.1a). The first component tends to be general, with sizable loadings (positive or negative) from most or even all the original variables. This is what happens with life history variables when body size is *not* controlled for: the first unrotated component accounts for most of the variance (about 70-80% in mammals), and in most cases it clearly represents a fast-slow axis of variation (for examples see Figures S1.2a, S1.3a, and S1.3c).

In their study of life histories in mammals, Bielby et al. (2007) did not report the unrotated solution, but used a standard rotation method (varimax) to automatically rotate the components. Rotated components no longer follow the direction of maximum variance but instead optimize some other criterion, usually with the goal of making the solution more readily interpretable. The problem with varimax and other standard rotations is that they seek a so-called "simple structure," whereby each component has a few large loadings for some variables and small (preferably zero) loadings for the remaining ones (see Browne, 2001; Sass & Schmitt, 2010). As a result, common rotation algorithms such as varimax and oblimin are *designed* to break up general components and factors; as a rule, they are unable to recover a general fast-slow continuum even if it exists. The main exceptions are quartimax and quartimin rotations, which typically produce a first general factor but are considerably less popular (see Browne, 2001; Darton, 1980; Sass & Schmitt, 2010).

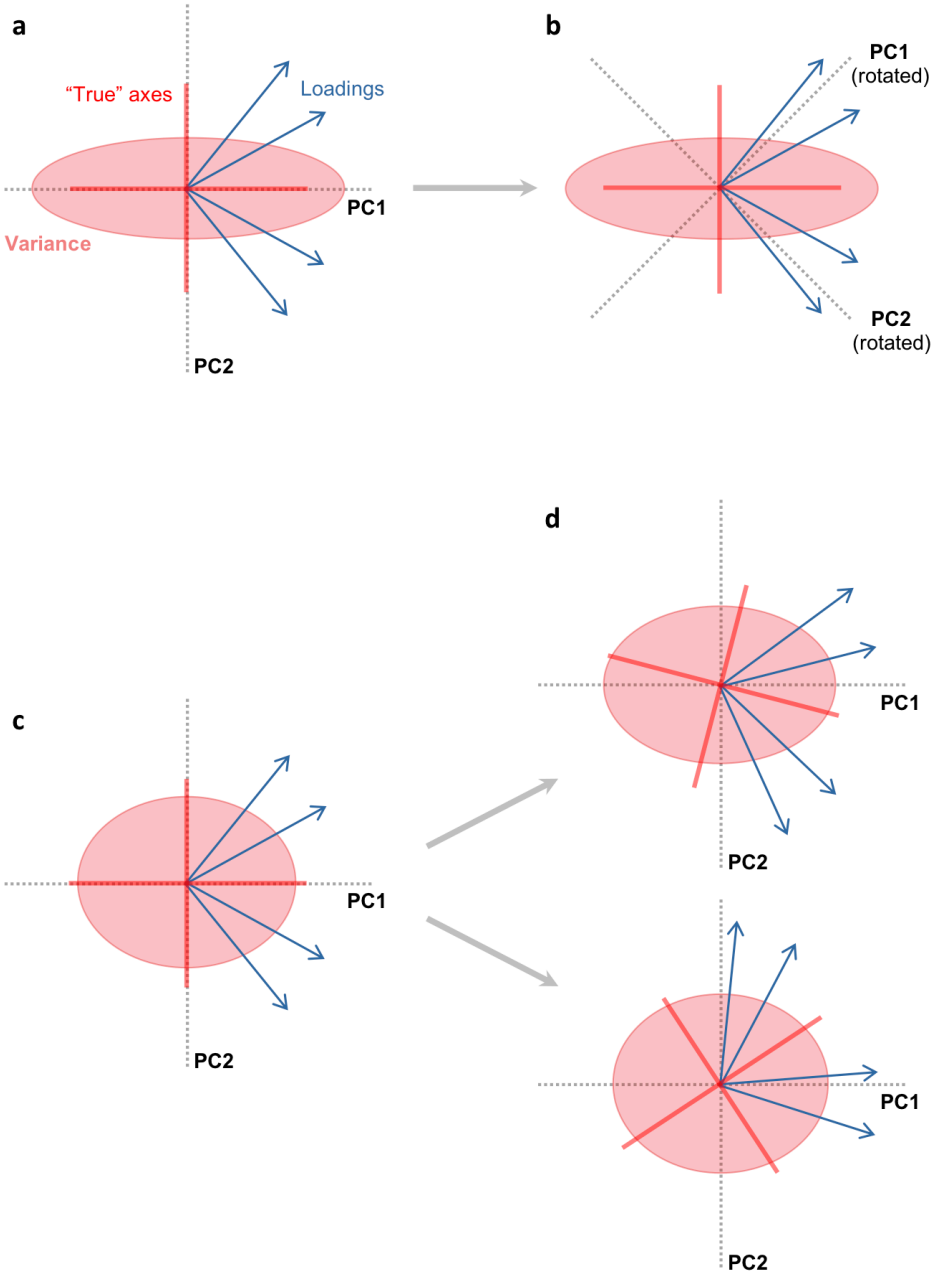

Figure S1.1. Schematic illustration of common rotation artifacts in PCA. In panel (a), the main axis of variation dominates the solution and aligns with the first unrotated component (PC1). Rotating the axes toward a simple structure (b) obscures the presence of a general dimension in the data; note that the rotated components are no longer aligned with the "true" axes. In panel (c), the two major axes explain similar proportions of variance in the data. The orientation of the axes becomes more sensitive to the composition of the sample, the specific variables included in the analysis, and so on (d). The two solutions shown in (d) are substantively equivalent, and differ only because of (irrelevant) changes in the orientation of the axes. Also note that the true axes of variation are not closely aligned with PC1 and PC2.

The effect of rotating the axes toward a simple structure is illustrated in Figure S1.1b. As has been noted before (e.g., Lykken, 1971), making the *a priori* assumption that the data will conform to a simple structure is not very reasonable when one is dealing with complex biological systems. And while this criterion may aid interpretation in other contexts, it is clearly inappropriate when the data are hypothesized to reflect a general dimension of variation. In

Bielby et al. (2007), the varimax rotation returned two axes corresponding to offspring size versus number and timing of reproduction, but—predictably—no unitary fast-slow continuum. However, the unrotated solution based on the same data (shown in Figure S1.2c) is extremely similar to the one found by Stearns (1983; Figure S1.2a); in both cases, the first component describes a classic fast-slow continuum (for more details see Del Giudice, 2014). The coefficient of congruence of PC1 loadings for the variables included in both datasets is > .99 in the uncorrected solution and .86 in the mass-corrected solution (see Abdi, 2007).

Of course, the main axis of variation does not always dominate the solution as in Figure S1.1a. An alternative scenario (illustrated in Figure S1.1c) is one in which the two major axes explain a similar amount of variance. When this is the case, the orientation of the components becomes particularly sensitive to the composition of the sample, the variables included in the analysis, and the effects of measurement error. All these factors contribute to determining the direction of maximum variance, and hence the direction of the axes identified by PCA. As a consequence, solutions tend to become less stable, and the "true" axes of variation may end up in directions that do not align closely with any of the components (Figure S1.1d). This is especially likely to happen when life history data are corrected for body size: after correction, the first component becomes smaller while the second becomes proportionally larger, so that PC1 and PC2 often account for similar amounts of (residual) variance. To illustrate, the first and second component in mammals explain 70-80% and 10-15% of the variance in uncorrected data; but after correction for body size, they account for about 30-50% and 20-30% of the variance, respectively (see Del Giudice, 2014; Healy et al.,2019; Jeschke & Kokko, 2009; Stearns, 1983). Thus, PCA solutions for mass-corrected data should often prove less stable than those for uncorrected data. It is also quite possible that the fast-slow continuum (if present) will not be neatly captured by either PC1 or PC2. Note that these problems may also occur with uncorrected data; but they become more likely when controlling for body size reduces the relative strength of the first component.

In the study by Jeschke and Kokko (2009), the uncorrected solution for mammals showed a clear fast-slow continuum on the first component (Figure S1.3a; the authors did not report loadings for PC2). In contrast, the first component of the mass-corrected solution looked rather different from the classic fast-slow continuum; this was interpreted as evidence that the uncorrected and mass-corrected continua are largely distinct constructs. However, Figure S1.3b tells a different story: the fast-slow continuum does not disappear in the mass-corrected solution—it simply aligns with PC2, rather than with PC1 as in the uncorrected solution (the coefficient of congruence between the uncorrected PC1 and the mass-corrected PC2 is .85). This "switch" between PC1 and PC2 is not surprising, since the two components account for very similar proportions of variance (37% and 29%).

Another interesting example of rotation artifact is shown in Figures S1.3e and S1.3f. Jeschke and Kokko (2009) compared the first component of the uncorrected and mass-corrected solutions in birds, found them to be markedly different, and interpreted this as further evidence that the nature of the fast-slow continuum changes after correction for body size. In fact, the two solutions are extremely similar, and only seem different because they are rotated in different directions (also, the sign of PC2 loadings is reversed in the mass-corrected solution—another irrelevant difference that contributes to masking the overwhelming similarity). For clarity, Figure S1.4 shows the same loading plots with two rotated axes superimposed on the original solutions. The axes were rotated so as to align with two key markers of the fast-slow continuum in birds and mammals, namely age at first reproduction and average fertility (labeled "fecundity" in the graph; more on this below). It is easy to see that—despite the superficial dissimilarity of the

original solutions—the two rotated axes are almost identical and describe the same fast-slow continuum.

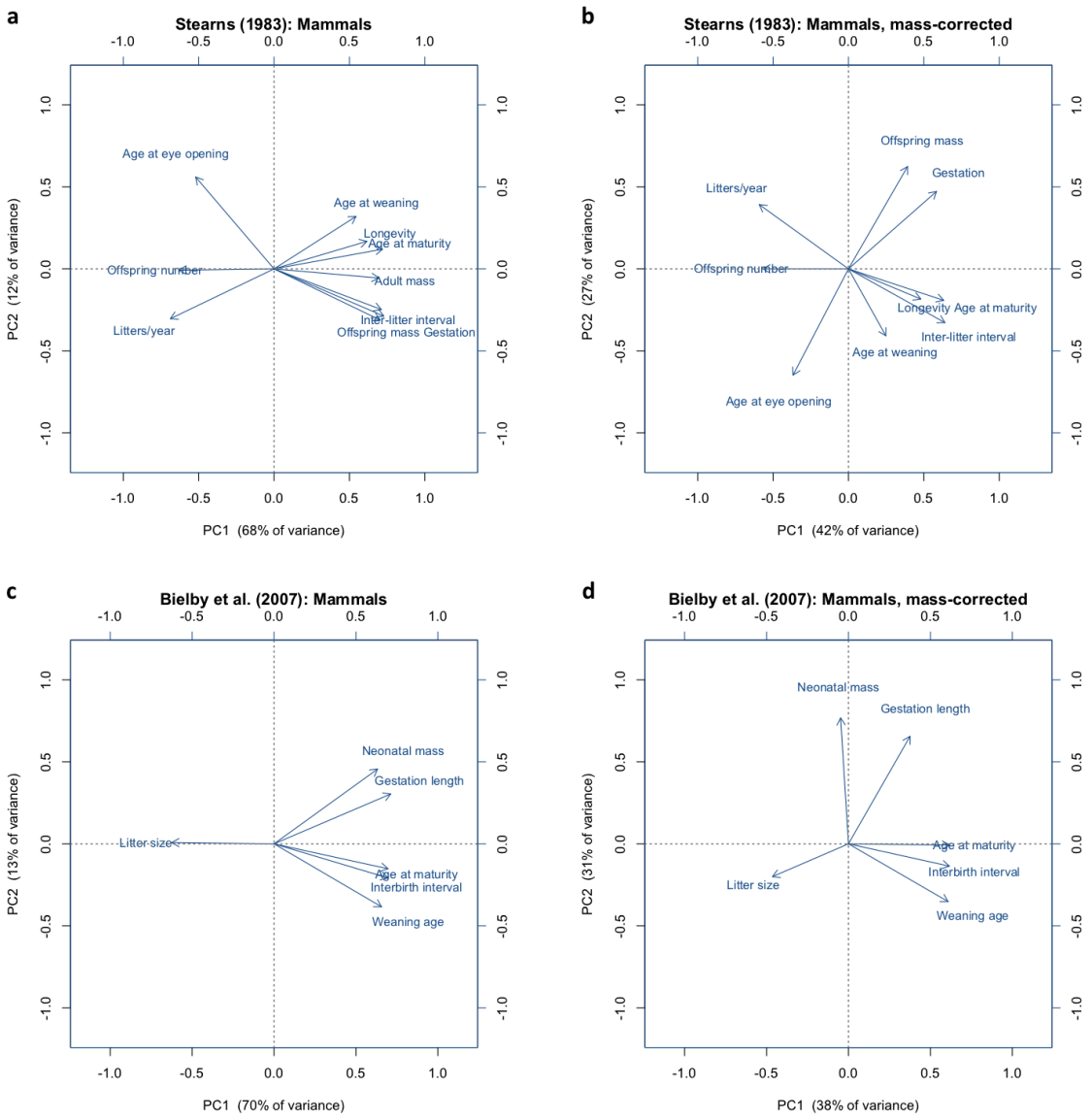

Figure S1.2. Unrotated loading plots based on Stearns (1983) and Bielby et al. (2007; the data were reanalyzed in Del Giudice, 2014). Note that the sign of PC2 loadings in panel (a) is reversed with respect to the other panels.

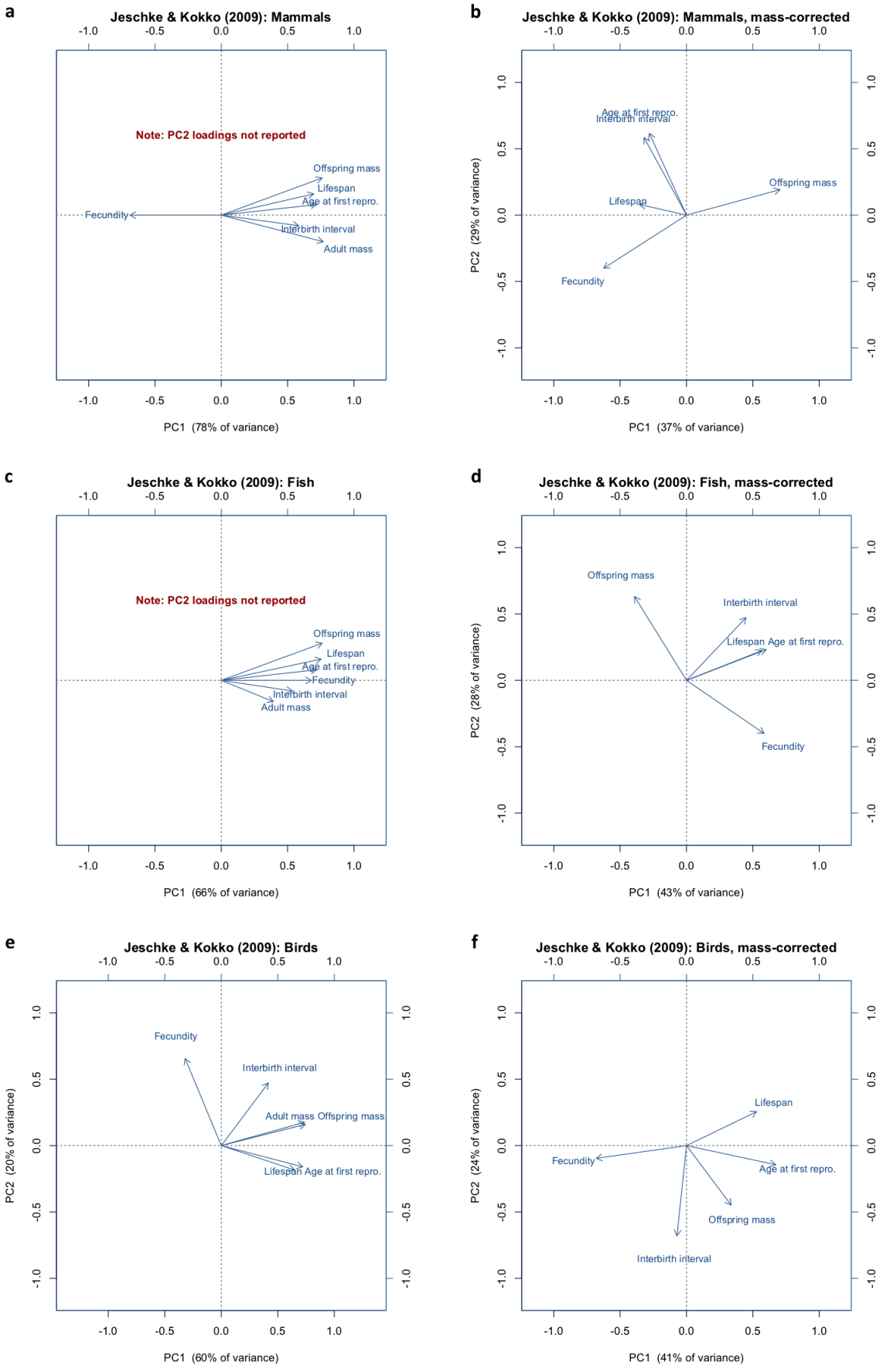

Figure S1.3. Unrotated loading plots based on Jeschke & Kokko (2009).

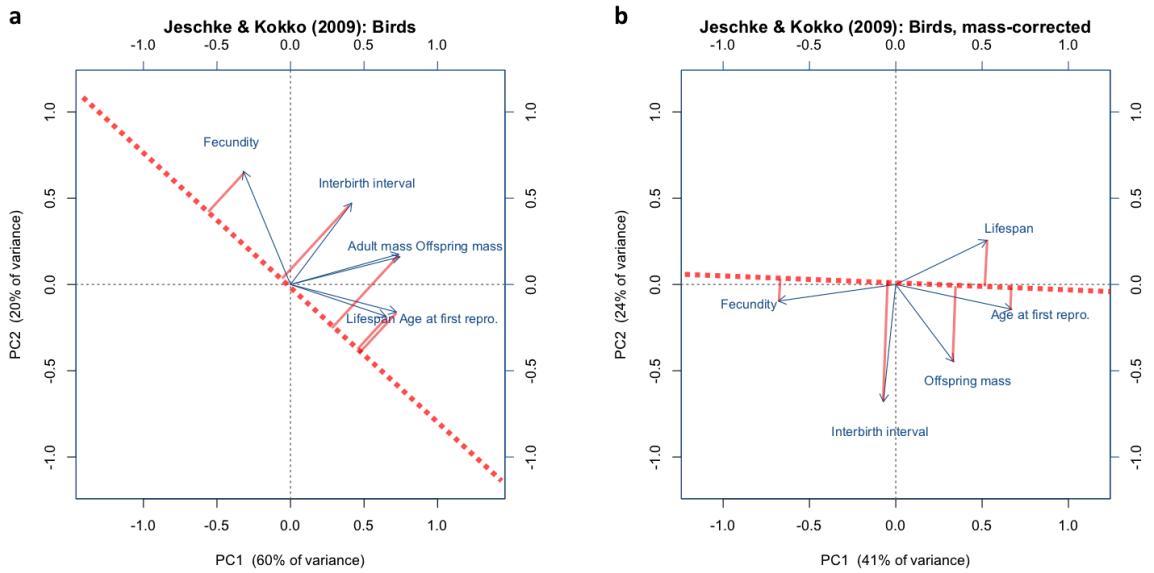

Figure S1.4. Unrotated loading plots of bird life histories based on Jeschke & Kokko (2009), with rotated axes superimposed in red. The new axes are rotated so as to align with fertility (here "fecundity") and age at first reproduction, and describe a fast-slow continuum very similar to the one observed in mammals. While the original solutions in (a) and (b) look superficially different, the rotated axes are almost identical, as can be seen from the loadings of the variables on the new axes (solid red lines).

In sum, researchers interested in life history variation should be careful about using standard rotation methods. These methods are designed to approximate a simple structure, and are unable to recover general axes of variation such as the fast-slow continuum. Standard orthogonal rotations such as varimax are probably best avoided in this context; an alternative approach is to use oblique rotations (e.g., oblimin, geomin, promax) to obtain correlated components, then subject those components to further dimension reduction. For example, when I applied an oblique rotation to the data in Bielby et al. (2007), the correlation between the two rotated components was .65 in the uncorrected solution and .32 in the mass-corrected solution (Del Giudice, 2014). However, the first unrotated component is usually a good description of the fast-slow continuum when the latter accounts for a large proportion of the variance. That said, it is always a good idea to inspect loading plots to detect atypical solutions like the one in Figure S1.3e. This is even more crucial when PC1 and PC2 account for similar proportions of variance, as is generally the case in mass-corrected datasets; the resulting solutions tend to be less stable, and the unrotated components may not align with the meaningful axes of variation in the data.

Since all the possible rotations are mathematically equivalent and the standard "simple structure" is not appropriate in this context, an interesting option is to anchor the solution to a biologically relevant criterion. For example, Oli (2004) showed that the ratio of fertility rate to age at first reproduction (F/α) is a meaningful summary of a species' position on the fast-slow continuum in mammals (most likely, the same applies to birds; see Jeschke & Koko, 2009; Section 2.1). This suggests that, in ambiguous situations like the one in Figure S1.4, researchers could rotate the first component so that it aligns with age at first reproduction and fertility rate, or another theoretically justified set of variables (for example by maximizing the sum of the squared loadings of those variables). If the anchoring variables are chosen sensibly, the rotated solution has a good chance of being more interpretable and biologically meaningful than the unrotated one.

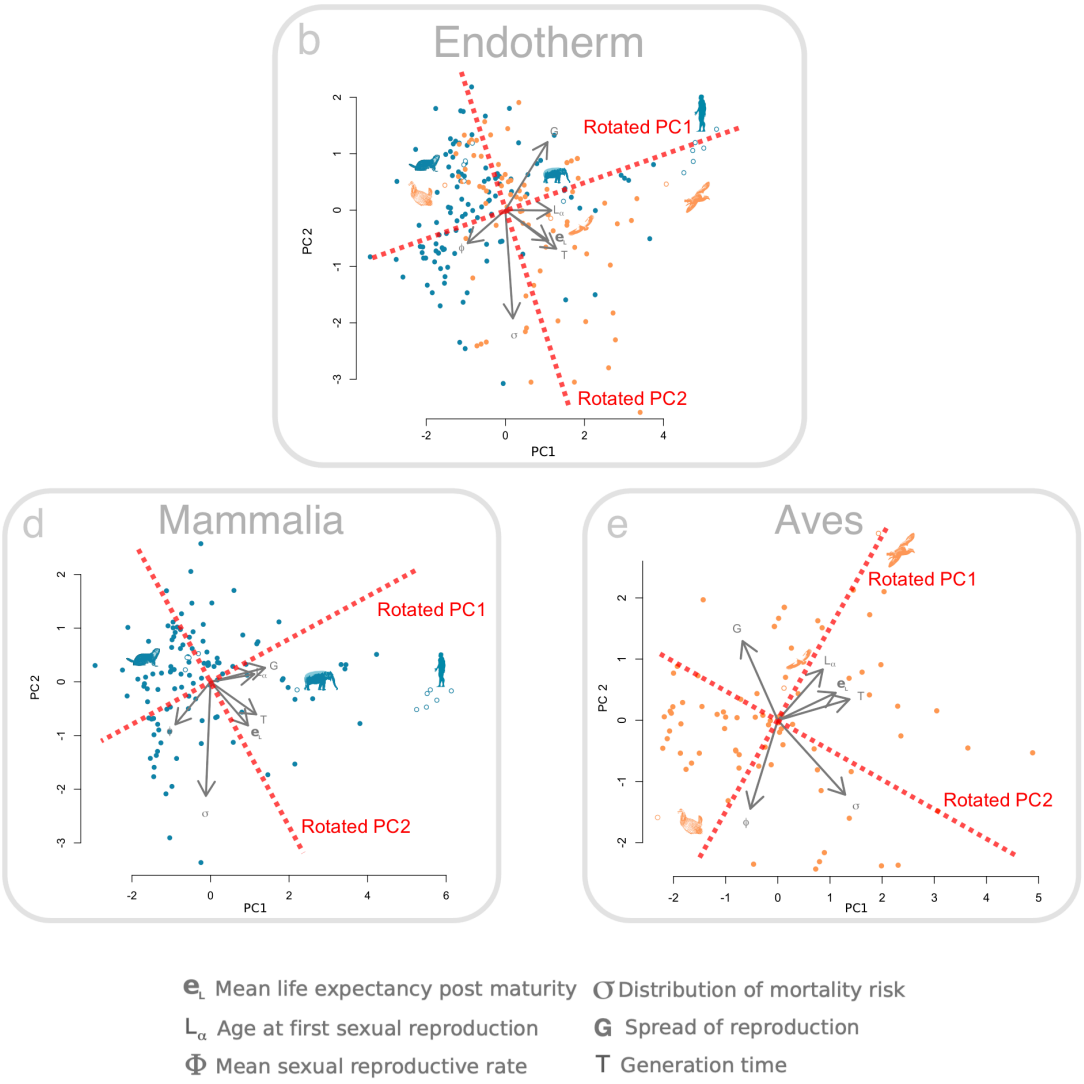

Figure S1.5. PCA plots from Supplementary Figure 2 in Healy et al. (2019; mass- and phylogeny-corrected), with rotated axes superimposed in red. The components have been rotated so that PC1 aligns with age at first reproduction and fertility rate (see Oli, 2004). Adapted with permission from Healy et al. (2019).

To further illustrate this approach, Figure S1.5 shows PCA plots from the recent large-scale study by Healy et al. (2019). Consistent with the classic fast-slow continuum, the first unrotated component (PC1) summarizes variation in age at first reproduction, life expectancy, and generation time across a wide range of species. However, reproductive rate shows the expected negative loading on PC1 only in endotherms and mammals, but not in birds (and ectotherms; not shown here). This and similar findings led the authors to conclude that the fast-slow continuum is only partially supported across taxonomic groups. An alternative possibility is that PCA failed to precisely identify the biologically meaningful axes of variation in these datasets, perhaps owing to the particular mix of species and/or variables included in the analyses. Note that the data were corrected for body mass and phylogeny; in birds, the resulting PC1 and PC2 account for similar proportions of variance (46% and 24%, respectively). Rotating the axes so that PC1 aligns with fertility rate and age at first reproduction (e.g., the superimposed red axes in Figure S1.5) yields a more interpretable solution. The rotated PC1 describes a classic fast-slow

continuum in endotherms, birds, and (somewhat less clearly) mammals. Taken together, the new solutions show that the apparent discrepancy between fast-slow continua in endotherms and birds reflects an artifact of rotation, not a true taxonomic difference.

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S2. Balancing selection, temporal fluctuations, and the genetics of individual differences

The assumption that individual differences in life history-related traits can be maintained by balancing selection has been criticized because of its apparent inconsistency with the genomic data. Specifically, critics argue that balancing selection should lead to an overwhelming contribution from alleles of high frequency (Verweij et al., 2012; Zietsch & Sidari, 2020) and/or large phenotypic effect (Penke et al., 2007). In contrast, the bulk of variation in polygenic traits is due to common variants of small effect and rare mutations/structural variants. More generally, genomic studies have found relatively few loci matching the expected signatures of balancing selection (but see Bitarello et al., 2018); thus, many authors regard balancing selection as marginal or limited to special gene classes (e.g., immune-related genes; Sella & Barton, 2019).

There are several problems with this view (see also Penke & Jokela, 2016). Statistical tests of balancing selection have important limitations, including low detection power and high error rates; more importantly, they can only detect long-term instances of balancing selection that may be the exception rather than the rule (Fijarczyk & Babik, 2015). The "signatures" of balancing selection in the genome emerge on different time scales, and only if the same alleles are maintained at equilibrium for many generations. In humans, an excess of high-frequency variants is only expected to become apparent after hundreds of thousands of years of consistent selection on the same alleles (that is, a number of generations of about 0.4 times the effective population size; see Fijarczyk & Babik, 2015). In view of the rapid evolution and striking ecological expansion of humans over the past tens of thousands of years, this kind of scenario seems unlikely. The process is even slower—and leaves even weaker traces—when balancing selection involves antagonistic effects, a plausible scenario for life history trade-offs (Connallon & Clark, 2013; Fijarczyk & Babik, 2015). When traits are highly polygenic and dominated by small effects, adaptation typically takes place via "soft" or incomplete sweeps, and subtle changes in allele frequency across multiple genes (*polygenic adaptation*; see Hermisson & Pennings, 2005, 2017; Messer et al., 2016; Pritchard et al., 2010). Under these conditions, balancing selection should often proceed via transient episodes on the background of a shifting genetic architecture, with few instances of the long-term equilibria envisioned by classic models. The signatures of this kind of process are exceedingly hard to differentiate from those of recent directional selection or even neutrality, which probably explains the dearth of genomic findings (Fijarczyk & Babik, 2015; Vitti et al., 2013; Yeaman, 2015).

Similarly, the expectation that alleles maintained by balancing selection should have large phenotypic effects (e.g., Penke et al., 2007) depends on the unrealistic modeling assumption that a single gene can take full control of expression level of a trait (e.g., Bürger, 2002; Kopp & Hermisson, 2006). Of course, such a trait would be extremely fragile against mutations and other disturbances, and it is highly unlikely that most behavioral and physiological mechanisms—with their multiple layers of redundancy, extensive feedback regulation, and modular organization—would ever evolve in this way (see Del Giudice, 2012).

Historically, the skepticism about balancing selection has been even stronger for hypotheses that involve fluctuations over time (see Messer et al., 2016). Early models of variable selection indicated that temporal fluctuations are generally unable to maintain genetic variation in a population, in contrast with spatial variation (e.g., Frank & Slatkin, 1990; Hedrick et al., 1976; Hedrick, 1986). However, this initial finding has been overturned by a new generation of models. If traits are highly polygenic and subject to recurrent mutations, temporal fluctuations

can be effective at maintaining variation (Bürger & Gimelfarb, 2002). Even more importantly, the early models assumed discrete and nonoverlapping generations; as a result, the entire population is exposed to negative selection at the same time and genetic variation gets rapidly depleted. But in species with overlapping generations (including humans) and/or maternal effects that buffer juveniles from temporary negative selection, polymorphisms can be easily maintained as the environment fluctuates ("storage effects;" Bertram & Masel, 2019; Ellner & Hairston, 1994; Ellner, 1996; Ellner & Sasaki, 1996; Hedrick, 1995; Yamamichi & Hoso, 2016). *Dominance reversal* is another potential mechanism that increases the effectiveness of fluctuating selection, and may plausibly occur in the context of life history trade-offs (Bertram & Masel, 2019; see Connallon & Chenoweth, 2019). The distribution of environmental states over time is also important: for example, some models suggest that fluctuations maintain variation at a larger number of loci if they follow a heavy-tailed distribution (Ellner & Sasaki, 1996). Note that these facilitating factors have been identified in theoretical models, but there is still no empirical evidence regarding their role (or lack thereof) in our species.

In sum, the genetic architecture of life history-related traits in humans and other animals is compatible with a mixture of mutation-selection balance and balancing selection (e.g., Charlesworth, 2015). Contrary to widespread assumptions, balancing selection can be sustained by temporal variation, and in realistic conditions does not necessarily result in a large contribution of intermediate-frequency alleles (whether of small or large phenotypic effect). Some recent studies have found cues of long-term balancing selection across the human genome (e.g., Bitarello et al., 2018); but there are reasons to believe that many if not most instances of balancing selection have left subtle, ambiguous traces that cannot be reliably detected using current genomic tools.

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S3. Trade-offs and life history-related traits in humans

Maturation timing and the current-future reproduction trade-off

The human life history literature has devoted a great deal of attention to the timing of sexual maturation (e.g., Belsky et al., 1991; Belsky, 2012; James & Ellis, 2013). Maturation timing is theoretically important as a mediator of trade-off between somatic versus reproductive effort and current versus future reproduction; and there are genetic as well as phenotypic correlations between puberty, age of sexual debut, age of first birth, and (female) fertility (e.g., Barban et al., 2016; Briley et al., 2017; Dunbar et al., 2008; Ibitoye et al., 2017; Lawn et al., 2019; Tropf et al., 2015; Udry & Cliquet, 1982). However, the net effect of puberty timing on fertility is limited, and the link between maturation and the start of reproduction can be weakened by various social factors (e.g., marriage practices; Borgerhoff Mulder, 1989; Udry & Cliquet, 1982). Maturation is also strongly affected by nutrition, with the result that allocations to current versus future reproduction are confounded with adaptive responses to improved physical condition and energy availability (e.g., Kyweluk et al., 2018; Stearns & Rodrigues, 2020). Even if maturation timing is not the main mediator of reproductive allocations in humans, it can play other roles in a broader life history perspective; for example, a slow and protracted developmental schedule can favor the acquisition of knowledge and skills—forms of "embodied capital" that pay off later in life (Berger-Tal et al., 2014; Eliassen et al., 2007; Kaplan et al., 2000).

More generally, our species has a long reproductive window, considerable flexibility in the timing of reproduction, and an extended period of care and provision for dependent offspring. These factors converge in reducing the relative importance of the current-future reproduction trade-off—particularly regarding the timing of sexual maturation—and increasing that of other trade-offs involving mating, parenting, and offspring quality (see below). This applies particularly to men, who are not limited by the physiological constraints of pregnancy, lactation, and menopause, and whose reproductive success depends more critically on the age of their partners than on their own (Borgerhoff Mulder & Ross, 2019; Ponzi et al., 2020). Especially in growing populations, an earlier start of reproduction can boost a woman's fitness (Jones & Bird, 2014), and may not entail sacrificing the quality of parental investment if she can count on a committed partner and/or support from kin. In total, the links between early maturation and other traits related to "fast" strategies should be stronger in females than in males; but even in females, correlations are likely to be small, owing to the partially stochastic nature of maturational events, the confounding effects of condition and nutrition, and the flexibility of human reproduction.

The centrality of the mating-parenting trade-off

The remarkable intensity of parental investment in our species suggests that quality-quantity trade-offs should play a major role in human life history strategies. The evidence, however, is contradictory and hard to interpret (Lawson & Borgerhoff Mulder, 2016). There are several reasons for this. First, most studies have used child survival as the metric of quality (see Lawson & Borgerhoff Mulder, 2016), but humans can contribute to the eventual reproductive success of their offspring by other means—for example by transmitting material resources, knowledge, status, and social networks (Jones, 2015). Second, and regardless of the definition of quality, parents with better health and more resources can typically afford to have more children; at least in traditional societies, this could easily mask the existence of a trade-off (Lawson &

Borgerhoff Mulder, 2016). Third, when population size is not stable (e.g., in a growing population), fitness does not just depend on the number of children but also on the timing of childbearing; when timing is accounted for, trade-offs involving fertility become more apparent (Jones & Bird, 2014). Finally, children are not just a cost on parents, but can actively contribute to childcare and other subsistence activities, thus lessening the strength of the trade-off (Lawson & Borgerhoff Mulder, 2016).

In our species, the quality-quantity trade-off overlaps to a large extent with that between mating and parenting effort, which in turn is intensified by the long duration of juvenile dependency (Borgerhoff Mulder & Ross, 2019; Lawson & Borgerhoff Mulder, 2016; Winking & Koster, 2015). Stereotypes of mating- versus parenting-oriented men are salient and easily recognized across cultures (Kruger et al., 2003, 2015). I have argued that the mating-parenting trade-off occupies a central place in the structure of the human fast-slow continuum—especially in men, who are less constrained by the timing of reproduction, and can potentially sire many offspring with little or no parental investment (see Copping & Richardson, 2020; Del Giudice, 2018). As noted above, even differences in maturation timing may partly reflect allocations to embodied capital in view of later competition for status and mating.

Multiple pathways to mating and parenting

In most animals, the main currencies of parental effort are energy/nutrient transfer and protection from danger. As noted earlier, humans can transfer multiple kinds of resources to their offspring; particularly since the invention of agriculture, people can enhance the fitness of their children—and often their grandchildren—by endowing them with land, cattle, money, and other forms of wealth (Borgerhoff Mulder et al., 2019; Jones, 2015). This opens up an alternative pathway to parental effort: especially for men, investment in parenting may involve the accumulation and transmission of wealth in addition to (or in place of) direct caregiving and provisioning.

Another important innovation in our species is the evolution of dual status hierarchies based on *dominance* and *prestige* (Henrich & Gil-White, 2001). The relevant point is that status and influence can be gained not only with the threat of physical force, but also with the possession of valued skills and knowledge. Again, this multiplies the pathways to mating success—particularly for men, who benefit more consistently from status and wealth (Nettle & Pollet, 2008; Pérusse, 1993; Stulp et al., 2016; von Rueden & Jaeggi, 2016; Winegard et al., 2018). Alternatively (or concurrently), the material benefits of high status can be transferred to one's family and offspring and thus channeled into indirect parenting effort.

A final aspect to consider is the nature of human courtship and mate choice. In our species, mate choice is not unidirectional but has a marked reciprocal component (Stewart-Williams & Thomas, 2013). Both sexes court potential partners by displaying desirable qualities, including intelligence and creativity (e.g., Conroy-Beam et al., 2019; Gabora & Kaufman, 2010; Winegard et al., 2018). At the same time, it is important to keep in mind that, in most societies, mates are not chosen freely but under various degrees of parental influence or outright control (Apostolou, 2010, 2017). Success in courtship can be achieved by appealing not just to the preferences of the partners themselves, but also to those of their parents—who are likely to place more value on traits that advertise cooperation and reliable parenting (see Apostolou, 2017; Buunk et al., 2008).

In sum, the mating-parenting tradeoff in humans can take a variety of forms. Allocations to mating and parenting can be realized through multiple pathways, which in turn should reward different combinations of cognitive and behavioral traits. For this reason, the fast-slow continuum is unlikely to be associated with a unitary set of traits; instead, fast and slow strategies may comprise a range of variants of "profiles," with similar implications for basic trade-offs but distinct psychological mediators (Del Giudice, 2018; Figure S3.1).

Mapping human life history-related traits

In order to use the fast-slow continuum as a heuristic for individual differences, it is useful to distinguish between basic life history traits and the broader suite of behavioral, physiological, and morphological phenotypes that contribute to determining them (Section 3.2). I suggested the label "life history-related" for traits that (a) are 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; and (c) plausibly contribute to mediating those allocations, or function as proxies of traits that do. Validating putative life history-related traits with survival and reproduction data is fraught with difficulties, especially in post-demographic transition societies with easy access to contraception, abortion, and modern medicine. For example, as contraception breaks the link between mating and reproduction (e.g., Pérusse, 1993), people with a strong desire for sexual variety may postpone or forgo reproduction, increasing the relative fertility of those with traits that promote long-term commitment and a desire for children (e.g., Woodley of Menie et al., 2017). But even in traditional societies, fitness is not just determined by the number of children or the number of mates: the timing of reproduction can be critical when populations are growing or shrinking, and the quality of mates can be just as important as their number (see Borgerhoff Mulder & Ross, 2019; Jones & Bird, 2014). An underappreciated challenge of doing research on college students is that few of them have children: some psychological aspects of the mating-parenting trade-off may not be fully expressed until people become parents and experience the constraints imposed by childbearing and/or caregiving, on top of the physiological changes associated with parenthood. Studies based on college students are likely to underestimate the strength of the trade-off (see e.g., Kruger, 2017). The validation of life history-related traits is a truly intricate task, and the existing data are still tentative in many ways.

In some research contexts, one may want to select a subset of traits that can serve as "markers" of fast versus slow strategies. In a recent book on evolutionary psychopathology (Del Giudice, 2018), I proposed the following as "core" behavioral markers of life history strategies: conscientiousness and honesty-humility; impulsivity, present vs. future orientation, sensation seeking, and risk-taking; timing of sexual development; restricted vs. unrestricted sociosexuality; long-term mating orientation; stable vs. unstable romantic attachments; exploitative vs. cooperative attitudes; and sensitivity to sexual/moral disgust. For a full exposition see Del Giudice (2018). These traits are meant to be used together as convergent but fallible markers of life history strategy. Empirical correlations involving some of these traits can be moderated by various factors (e.g., attractiveness, nutrition; Copping et al., 2014; James & Ellis, 2013), and may differ somewhat between alternative profiles.

This list of markers is deliberately selective, since many aspects of personality and individual differences have complex or less than straightforward links with life history trade-offs. For example, extraversion and openness to experience are not included in the list because their facets show contrasting relations with mating behaviors and other life history-related traits (e.g., Holtzman & Strube, 2013; Manson, 2017). The general factor of personality (GFP) is also

excluded, partly because of its low resolution and partly because it is strongly contaminated by evaluative biases (see Davies et al., 2015; Dunkel et al., 2016). Agreeableness is generally associated with "slow" traits and outcomes, but the model I propose includes a slow life history profile with moderate/low agreeableness. While the list includes conscientiousness, the role of this broad personality trait should be reconsidered and defined more precisely. Measures of conscientiousness often yield contradictory or paradoxical findings, because they include elements of performance (e.g., succeeding in being orderly and punctual) that are subject to evaluative distortions (e.g., perfectionism) and may not track motivation very closely (e.g., Mike et al., 2018; Mõttus et al., 2018).

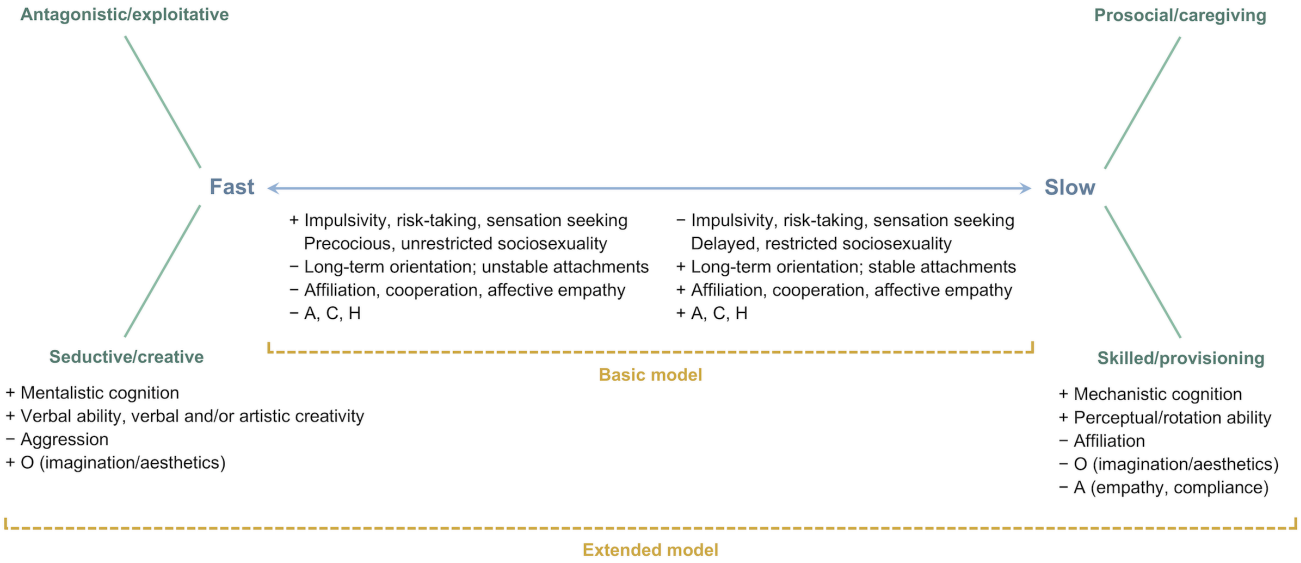

Figure S3.1. An extended model of life history-related traits in humans (adapted with permission from Del Giudice, 2018). The basic model only distinguishes between fast and slow strategies; the extended model postulates the existence of alternative profiles defined by specific clusters of behavioral and cognitive traits. A = agreeableness; C = conscientiousness; H = honesty-humility; O = openness to experience.

By way of illustration, Figure S3.1 shows my recent proposal for an extended model of life history-related traits in humans, based on the idea of multiple profiles. In addition to the clusters of traits envisioned by the standard or "basic" model that informs the current literature (here labeled *prosocial/caregiving* and *antagonistic/exploitative*), I described two additional profiles: a *creative/seductive* profile associated with narcissistic and psychotic-like traits, and a male-biased *skilled/provisioning* profile associated with autistic-like traits (for details see Del Giudice, 2018). If the model is broadly correct, the profiles it describes cannot be identified by standard factor-analytic methods. Popular "life history" questionnaires such as the *Mini-K* (Figueredo et al., 2006) mostly reflect a combination of agreeableness, conscientiousness, extraversion, and—to a smaller extent—neuroticism (Manson, 2017; Olderbak et al., 2014), and correlate weakly with indicators of mating effort (Copping et al., 2017). According to the model in Figure S3.1, these scales map on the fast-slow continuum in a partial and imperfect fashion; for example, one would expect them to misclassify people who match the skilled/provisioning profile as "fast" strategists because of their comparatively low agreeableness and sociability (see Del Giudice, 2018).

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S4. Reply to Zietsch & Sidari (2020)

This is a point-by-point reply to the extended critique of the fast-slow paradigm in humans by Zietsch & Sidari (2020), henceforth Z&S. Instead of repeating the arguments presented in the main text, I briefly summarize them and point to the relevant sections. The aim of this supplement is to organize the material in a convenient format, and clarify the points of agreement and disagreement with Z&S.

Z&S, Section 2: The fast-slow continuum applied to inter-individual trait covariation within human populations

Z&S: *The r-K (or fast-slow) continuum fell out of favor in biology […] modern biology research that invokes the term 'life history theory' rarely adopts the fast-slow framework.*

Reply: The fast-slow continuum is a robust empirical pattern (Section 2.1), and the term is routinely used to describe patterns of life history variation across species (for recent examples see Bakewell et al., 2020; Healy, 2019; Salguero-Gómez, 2017). The *r/K* framework is a specific theoretical approach that has been used to explain the fast-slow continuum. While *r/K* models have been out of favor for some time since the 1980s, they have experienced a resurgence in recent years (Section 2.2).

Z&S: *The empirical evidence for a unitary fast-slow continuum is mixed when looking across species within clades and controlling for body size.*

Reply: This is a common misconception, largely based on the comparative studies by Bielby et al. (2007) and Jeschke & Kokko (2009). In fact, much of the apparent instability of the fast-slow continuum is an artifact of axis rotation in PCA (Section 2.1; supplementary material S1). When the data of these papers are properly reanalyzed, the fast-slow continuum turns out to be robust to mass correction, and much less variable across species than often believed (see supplementary material S1 for details).

The word "unitary" might be taken to mean that, for the fast-slow continuum to be valid, the structure of life history strategies must be one-dimensional. But it has been clear from the beginning that there are other dimensions besides fast vs. slow, and that at least two axes are needed to describe broad patterns of variation in animals and plants (Section 2.1).

Z&S, Section 3: Species differ largely because of selection, whereas individuals differ largely because of inheritance

Z&S: *Within populations as well, individuals differ in part because of genetic differences […], but there is no equivalent (i.e. Darwinian) process that tailors varying individual genotypes to individuals' varying personal environments. […] There is no equivalent evolutionary process creating inter-individual trait covariation. Selection and evolution can lead to phenotypic plasticity and adaptive calibration of individuals' traits to their personal environments […]; but Darwinian phenotype-environment matching at the species level and plasticity at the individual level are completely different processes and may or may not lead to equivalent predictions regarding trait covariation.*

Reply: Z&S are right to stress that individual differences are not like species/population differences, and are generated by different processes. Still, the two levels of organization may be functionally connected, so that individual variation within a population mirrors in important ways the patterns of variation between populations. I proposed the term "ecological gambit" for the working assumption that this is the case (Section 3.1). Of course, the assumption may or may not be warranted in any given case, and needs to be tested rather than simply taken for granted. In the context of life history strategies, basic trade-offs offer a possible functional link between population and individual differences (Section 3.1).

Z&S, Section 4: Exceptions to Mendel's law of independent assortment do not mean inter-individual trait covariation can be explained by Darwinian selection for trait clusters

Z&S: *Intense correlational selection can in principle generate inter-individual trait covariation by generating transient covariation between genetic variants at different loci […] However, even if such selection applied to human life history traits (for which there is no evidence), we show with empirical simulations (Supplementary Material) that the trait covariation it created would be weak and temporary, immediately eliminated once the correlational selection is relaxed. We further show that the greater the number of genetic variants underlying the traits, the weaker the temporary trait correlation.*

Reply: Z&S are right to point out that correlational selection *per se* is ephemeral, and linkage disequilibrium is not enough to produce robust patterns of covariation. However, persistent correlational selection may favor the evolution of pleiotropic effects that generate the same patterns, and pleiotropic effects can be robust and long-lasting (see Peiman & Robinson, 2017; Roff & Fairbairn, 2007). The same applies to fluctuating selection that acts simultaneously on multiple traits (Pavličev et al., 2011; Section 3.3).

Z&S: *Quantitative traits are underlain by variation at thousands of loci across the genome, so there is no reason to expect physical linkage to cause such traits to covary in adaptively helpful ways.*

Reply: To my knowledge, physical linkage has not been proposed as an explanation of covariation among life history-related traits in humans.

Z&S: *Covariation between variants at different loci can be generated by non-random mating […] However, the presence or absence of this covariation across loci does not pertain to whether the relevant traits "do or do not work together to serve their multiple adaptive functions" (Figueredo et al., 2013), and cross-trait assortative mating does not parallel evolutionary processes that create inter-species trait covariation.*

Reply: This point is broadly correct. At the same time, it seems to imply that assortative mating *only* occurs for phenotypic condition/quality. But if there is also assortative mating for alternative life history strategies and/or life history-related traits (e.g., some aspects of personality), mating patterns can play a role in maintaining trait covariation.

Z&S: *Another way genetic variation in different traits can cluster is through pleiotropy, whereby the same genetic variant affects variation in multiple different traits. […] Non-zero mutational effects tend to be both pleiotropic and deleterious […] since they are random alterations to a complex, integrated design. This property of mutations would bias genetic correlations towards being directionally concordant with respect to overall quality or condition*

(creating a quality factor) […]. Fig. 1, which shows genetic correlations among varied human traits relating to physical and mental robustness, is consistent with such a tendency.

Reply: The role of deleterious mutation is clearly important, and I agree that it has not received the attention it deserves in human research based on the fast-slow paradigm (Section 3.3). The genetic correlation matrix shown in Z&S' Figure 1 contains measures of well-being and physical health, plus neuroticism, intelligence, physical traits such as stature and BMI, and three psychiatric disorders (depression, schizophrenia, and ADHD). The matrix makes the point that physical and mental conditions share common genetic sources that may be summarized by a general "quality" factor; but its relevance to life history strategies is unclear. While some authors have proposed a general factor of physical and mental health ("covitality") as a component of slow life histories (Figueredo et al., 2004, 2007), this is by no means a universal assumption in the field. In fact, the relations between mutation load, health, and life history allocations are likely more complex, as the same authors have acknowledged in other publications (Sefcek & Figueredo, 2010). For a detailed account of these relations in the domain of mental health, see Del Giudice (2018).

Z&S: *Nor should we assume the same pattern across species, because: 1) the rationale for directional pleiotropy of mutational effects within species does not apply to inter-species trait covariation; 2) genetic variants causing inter-individual trait (co)variation need not be the same as those causing inter-species trait (co)variation; and 3) selection on different traits varies across species (which is largely why species themselves vary).*

Reply: These points are all correct; however, the *mechanisms* that generate pleiotropic effects (e.g., hormones) can be highly conserved across species, and have the potential to link within- and between-species patterns of variation, particularly among closely related species (Section 3.1). Cross-level similarity cannot be simply assumed (which is why the ecological gambit is a gambit), but there may be reasons that make it more or less plausible in any given case (Section 3.1).

Z&S: *Directional pleiotropy as described above is consistent with a range of observed trait covariances in humans: for example, the positive manifold in diverse cognitive abilities […]; the positive genetic correlation between most psychiatric disorders (including those that are hypothesised to be at opposite ends of the fast-slow spectrum, such as schizophrenia and autism; Del Giudice, 2014)*

Reply: I agree with Z&S that directional pleiotropy of the kind they describe contributes to the risk for many mental disorders, and to the pattern of positive correlations that gives rise to the so-called "p factor" of psychopathology. At the same time, I have argued that the p factor is not a unitary construct but a composite of three functionally distinct and largely separable sources of variation: (1) risk for "fast spectrum" disorders, that is, disorders associated with fast strategies and their phenotypic correlates; (2) risk for "defense activation" disorders (depression, generalized anxiety, panic, phobias, PTSD, and a subtype of OCD); and (3) low cognitive ability, which is strongly influenced by mutation load and, as shown by Z&S in their Figure 1, correlates with health measures along a general "quality" dimension (Del Giudice, 2018). This alternative model accounts for the observed large-scale structure of psychopathology, and correctly predicts that factor analysis will recover a seemingly unitary p factor (see Del Giudice, 2016a, 2018).

The example of autism vs. schizophrenia deserves a closer look. As noted by Z&S, there is a small positive genetic correlation between autism and schizophrenia (e.g., Grove et al., 2019; Warrier et al., 2019). But while schizophrenia is negatively associated with IQ at the genetic and phenotypic level, polygenic scores for autism show a weak *positive* correlation with IQ (Clarke et al., 2016; Hagenaars et al., 2016; Grove et al., 2019). The genetic association with risk-taking is positive for schizophrenia but negative for autism (Linnér et al., 2018); there is also some evidence that polygenic scores for autism and schizophrenia tend to predict age at first intercourse and first birth in opposite directions—earlier for schizophrenia, later for autism (Ni et al., 2019; note that this study found a mix of significant and non-significant results using different methods, and indications of a U-shaped relation between schizophrenia polygenic scores and age at first birth). Of note, genetic risk for schizophrenia has been found to predict larger numbers of sexual partners (Lawn et al., 2019). Because of their overall correlation, both autism and schizophrenia load on a GWAS-based genetic p factor; but if two components are extracted instead of just one, autism and schizophrenia end up loading on different components (Selzam et al., 2018). This pattern mirrors the phenotypic distribution of autistic-like and schizotypal traits, and is compatible with a diametrical model (see Del Giudice et al., 2014).

Taken together, these findings clearly indicate that directional pleiotropy is only part of the story. I have argued that a life history perspective can help make sense of the empirical literature, in view of the strong phenotypic and genetic heterogeneity of autism (Warrier et al., 2019). Specifically, I have suggested that "autism spectrum disorders" comprise two main functionally independent subtypes: a (mostly) high-functioning subtype, with a large contribution of common genetic variants and a specific cognitive/behavioral profile associated with slow strategies; and a (mostly) low-functioning subtype with a high risk of intellectual disability, a large contribution of rare/*de novo* mutations, and no apparent link with life history strategies (Del Giudice, 2018). This distinction is consistent with the genetic evidence on the role of mutations in autism (Gardner et al., 2019; Iossifov et al., 2014, 2015; Ronemus et al., 2014), and is supported by convergent epidemiological data on paternal/maternal age, socioeconomic status, and so forth (details in Del Giudice, 2018, Ch. 10).

If this hypothesis is correct, genetic correlations based on a unitary diagnosis of "autism spectrum disorder" reflect a mixture of functionally distinct conditions and symptom dimensions, and may hide as much as they reveal. For example, the shared role of deleterious alleles in the development of schizophrenia and (low-functioning) autism may mask the existence of negative relations between specific components/subtypes of the two disorders.

On a related note, the major impact of deleterious alleles on schizophrenia risk does not preclude a role for balancing selection (Keller, 2018), and is consistent with a life history/sexual selection account of this disorder (see Del Giudice, 2017, 2018). (Note that schizophrenia is also heterogeneous, and there are still some unanswered questions regarding its overlap with autism; see Del Giudice, 2018, Ch. 8.) This example illustrates that it is entirely possible to reconcile the idea of a fast-slow continuum with the existence of a pervasive dimension of genetic quality, and that the fast-slow distinction can be used with more nuance than implied by Z&S.

Z&S: *Some have claimed that genetic correlation among human life history traits 'supports the hypothesis that Life History Strategy is predominantly under the control of regulatory genes that coordinate the expression of an entire array of life history traits' (Figueredo, Vásquez, Brumbach, & Schneider, 2004). But genetic correlation does not imply any such thing. A genetic correlation between two traits could reflect one heritable trait directly influencing the other, or one heritable trait influencing environmental conditions that in turn influence the other trait, or both traits being influenced by a third heritable trait, or the traits being positively or negatively linked by shared developmental processes, among various other causal possibilities.*

Reply: This argument is technically correct, but ignores what we already know about the *actual* mechanisms of trait covariation in humans and other animals. For example, there is ample evidence that interlinked endocrine systems such as the HPA and HPG axes regulate key life history allocations (to survival, growth, reproduction, mating, parenting, and so forth) and coordinate the development and expression of multiple traits (Section 3.3). As Z&S note, a genetic correlation does not imply a particular mechanism of covariation, but the idea that life history(-related) traits are coordinated by pleiotropic regulatory mechanisms is highly plausible. This does not imply the absence of the other causal pathways listed by Z&S.

Z&S: *the genetic architecture of quantitative traits, which is absent any large-effect, pleiotropic 'genetic switches', is incompatible with the existence of regulatory genes that coordinate the expression of an entire array of life history traits (Penke, Denissen, & Miller, 2007, p. 568).*

Reply: Here, Z&S seem to assume that regulatory genes can only act in isolation as large-effect "switches." But trait coordination is typically mediated by complex regulatory mechanisms (e.g., endocrine systems), whose functioning parameters are themselves highly polygenic (Section 3.3). In other words, there is no contradiction between the idea of pleiotropic regulatory genes and the empirically observed architecture of quantitative traits. Z&S cite Penke et al. (2007), who based their own argument on the classic notion that balancing selection can only produce intermediate-frequency alleles of large effect. This idea has been overturned in the recent theoretical literature, as discussed in Section 3.3 and acknowledged by Penke and Jokela (2016) in their update of the 2007 paper (more details in the supplementary material S2).

Z&S, Section 5: Claims regarding correspondence between inter-species and inter-individual trait covariation are usually not based on cogent theory

Z&S: *Those who have used observations or theory from inter-species trait covariation to explain (or predict) inter-individual trait covariation have usually not specified why they should correspond. […] To our knowledge the life history literature in humans is absent any explicit description of a Darwinian process that should align human trait covariation with inter-species trait covariation. […] A recent systematic review showed that the few pertinent formal models do not provide consistent or unique predictions regarding inter-individual covariation among life history and other traits (Mathot & Frankenhuis, 2018), leaving the pace-of-life perspective without a clear theoretical basis.*

Reply: Z&S are right to point out that the link between the species and individual level of variation has been simply assumed, or justified with generic arguments (but see Wright et al., 2019). This is a major limitation of the fast-slow paradigm and a crucial topic for research, including formal modeling. In Section 3.1 I have tried to clarify the arguments in favor of cross-level similarity, and explicitly noted that the assumption of similarity is in fact a theoretical "gambit" that may or may not succeed in any given case.

Z&S: *Wright, Bolstad, Araya-Ajoy, and Dingemanse (2019) proposed density-dependent fluctuating selection as a mechanism that might align inter-species and inter-individual trait covariation. […] Its applicability to human trait covariation is doubtful though: modelling predicts that trait variation maintained by fluctuating selection will be explained disproportionately by alleles of intermediate frequency […], whereas the genetic architecture of*

human quantitative traits that have been examined exhibits the opposite tendency, i.e. disproportionate contribution of rare alleles.

Reply: The model by Wright et al. (2019) is an interesting attempt to explicitly link between- and within-species variation in life history strategies. The particular explanation proposed by Wright et al. (fluctuating density-dependent selection) may or may not prove correct in the case of humans; however, Z&S' argument against it rests on the outdated assumption that fluctuating selection (a specific kind of balancing selection) necessarily leads to an overwhelming contribution of common alleles to the genetic architecture of a trait. I discuss why this assumption is not supported by current theoretical models in Section 3.3 and the supplementary material S2.

Z&S, Section 6: Genetic coadaptation vs. adapted developmental plasticity

Z&S: *A possibility is that scholars reading this might accept that genetic coadaptation does not viably align inter-species and inter-individual trait covariation, while still maintaining that these types of covariation are aligned by species-typical adaptations that tailor individuals' traits to the environments in which those individuals developed.*

Reply: Given that genetic coadaptation is in fact quite plausible (see above), there is no need to discard it as an explanation. Moreover, the phenotypic effects of genetic and environmental variation may often align, owing to the fact that they are both channeled through the same coordination mechanisms (Section 3.3). This possibility is broadly consistent with the fact that phenotypic and genetic correlations between the same traits tend to have the same sign and a similar magnitude. (In biology, this correspondence is known as "Cheverud's conjecture;" see Section 3.3.)

Z&S: *A large proportion of variation in life history and related traits is attributable to genetic variation among individuals, and little is attributable to variation in the shared environment (i.e. the developmental home environment shared within twin pairs, including socio-economic status, parenting style, father absence, risky upbringing). […] The remainder of the variation in such traits tends to be mainly accounted for by residual factors, which include measurement error and random or idiosyncratic effects (biological or environmental) unshared by twin pairs. […] Reported associations between developmental environment and adult traits are rarely controlled for genetic confounders […] and when they are controlled the associations are often weaker or null. In light of these observations, a perspective focussed on adapted responses to early environmental conditions does not seem promising as a broad framework for explaining human trait covariation.*

Reply: Z&S make two important points about the small size of shared environmental effects in behavior genetic studies, and the lack of genetic control in most developmental research. These findings challenge to the role of developmental plasticity in the development of individual differences, including life history-related traits. In principle, there are ways to reconcile a degree of plasticity with the behavior genetic evidence (e.g., Del Giudice, 2015, 2016b; Section 3.3), but current ideas only scratch the surface and many crucial questions remain unanswered. We also urgently need to more information about the predictive value of early life factors, which in turn depends on the statistical properties of the environment (Section 3.3; Frankenhuis et al., 2019).

Z&S: *Another problem with this perspective is that its most central hypothesis, that an adapted response to harsh environments (e.g. higher mortality and resource stress) should be to activate a faster life history strategy […] is not justified by formal evolutionary modelling (Baldini, 2015). […] For example, depending on how population density affects population fertility and how environmental harshness is defined (e.g. mortality rate, or effectiveness of investments in survival, growth, or reproduction), harsh environments are often predicted to lead to* slower *not faster life histories (Baldini, 2015). Further, the optimal strategies often comprise some 'slow' features and some 'fast' features (Baldini, 2015), contrary to the idea that trait covariation should cohere around a unitary fast-slow continuum.*

Reply: I agree with Z&S that there is a need for more formal models of life history strategies, especially at the within-population level. To be fair, the role of density-dependence has been addressed by some authors in this area (e.g., Ellis et al., 2009), even if it has not been emphasized in later research. On the other hand, it is important to acknowledge the limitations of Baldini's (2015) models. These models contain some implausible assumptions and a non-standard definition of mortality. Baldini's key results critically depend on these questionable aspects of the models (see Section 3.1; André & Rousset, 2020).

Z&S, Section 7: Empirical evidence does not support inter-individual trait covariation being organised around a fast-slow continuum in humans or in other species

Z&S: *empirical support is weak for the hypothesis that inter-individual covariation of physiological, behavioural, and life history traits cohere around a fast-slow continuum (a hypothesis that derives from observations of inter-species trait covariation). Observed covariation of self-reported life history indicators in humans does not fit a model involving a single fast-slow dimension.*

Reply: This is a crucial point that needs to be addressed on two levels. First and most important, the idea that individual differences are partly organized along a fast-slow continuum does *not* imply that the pattern of covariation among traits should be adequately described by a single factor. There are several reasons for this, including the existence of other functional axes of variation (which are also observed at the species level; see Section 2.1), the confounding effects of condition/quality, and the fact that many traits show nonlinear or interactive relations with life history outcomes (Section 3.4). Second, many commonly used inventories include putative indicators that have not been validated against life history variables and/or are not well suited as indicators (see Copping et al., 2017; Richardson et al., 2017).

Z&S: *More fundamentally, it is difficult to assess what human trait covariation is in line with a fast- slow continuum and what is not, because a trait's directionality as 'fast' or 'slow' is often inferred from the direction of its observed correlation with other supposed fast-slow traits or factors. For example, neuroticism is characterised by worry, self-doubt, and caution, and is accordingly associated with low-risk taking […] But in exploratory factor analyses, neuroticism actually loads* negatively *on a 'K-factor' (i.e. tends to correlate positively with 'fast' traits); so high neuroticism is then hypothesised to indicate a fast life history strategy and its factor loading taken as supporting evidence (e.g. Figueredo et al., 2007; Richardson et al., 2017). This kind of circularity makes the theory nearly unfalsifiable and the existing evidence hard to evaluate.*

Reply: I agree with the concern raised by Z&S; circularity and lack of external validation are important limitations of certain approaches to the fast-slow continuum in humans.

Z&S: *Evidence from a meta-analysis of empirical data in the animal literature is strikingly unsupportive of the hypothesis that inter-individual covariation of traits should cohere around a fast-slow continuum (Royauté, Berdal, Garrison, & Dochtermann, 2018). The mean correlation among traits expected to positively covary was 0.06; within vertebrates it was 0.02. […] Overall this meta-analysis seems to us a clear disconfirmation of the fast-slow continuum as a general organising principle for inter-individual trait covariation.*

Reply: Z&S are right to point out that the meta-analysis by Royauté et al. (2018) yielded largely negative findings. It is also important to understand *what* exactly has been disconfirmed, namely, the specific list of predictions advanced by Réale et al. (2010) about the behavioral and physiological correlates of fast vs. slow strategies. As I discuss in Section 3.2., those predictions were admittedly tentative, and the authors did not expect them to apply widely across species. Unfortunately, Réale et al.'s list has been reified and applied automatically, without testing its assumptions or adapting it to the ecology of different species (see also Del Giudice, 2018). This has been a major problem in the POLS literature, compounded by the questionable validity of standard behavioral measures (Royauté et al., 2018; see also Beckmann & Biro, 2013; Carter et al., 2012). Hopefully, this meta-analysis will prompt researchers to improve their methodology and develop more sophisticated predictions.

Z&S, Section 8: Trade-offs

Z&S: *most of the work on non-human animals that invokes the term 'life history theory' does not involve the concept of fast and slow strategies. Instead it tends to emphasise specific trade-offs in how individuals allocate limited energy/resources to different aspects of their life histories […] In this approach it is not generally argued that covariation between different life history traits should be understood per Darwinian principles, such that traits correlate because they 'work well together'; rather the trait correlations are thought to result from specific trade-offs that are due to fundamental limitations of an individual's resources such as energy or time.*

Reply: Contrary to what Z&S seem to imply here, the fact that fundamental constraints on energy and time drive life history trade-offs is not an alternative to the notion that traits covary because they "work well together;" in fact, constraints are what shapes the logic of adaptive trait coordination. Trade-offs force organisms to make "decisions" within constraints; the implementation of those decisions requires the coordination of behavior, physiology, and morphology in ways that "work well together," in the sense that they tend to bring about adaptive outcomes without wasting precious resources (Section 3.2). To avoid confusion, it may be useful to distinguish between classic life history traits such as longevity and fertility—which are the outcomes of the organism's allocations—and the life history-related traits (behavioral, physiological, morphological) that mediate those allocations (Section 3.2).

Z&S: *It is problematic, though, to assume that trade-offs within individuals should cause corresponding trait covariation across individuals […] individuals differ in the amount of bioenergetic resources they have or can acquire (e.g. due to variation in mutation load or favourability of their environment), and so the covariation between traits that functionally trade-off within an individual can covary positively across individuals, depending on the means and variances in resource acquisition vs. allocation of those resources.*

Reply: This is an important point that has been noted repeatedly in the theoretical literature (e.g., Rezinck et al., 2000; Roff & Fairbairn, 2007; Wilson, 2014), but not consistently applied to life history variation in humans and other animals. Clearly, the existence of individual

differences in condition/resources can mask the functional structure of trade-offs. However, this is not an insurmountable problem as much as an opportunity for progress: in many cases, it should be possible to control for the effects of individual condition and recover a clearer picture of the underlying trade-offs (Section 3.4; for an empirical example see McLean et al., 2019).

Z&S: *Further, Houle (1991) showed, under the assumption that genetic variation in fitness-relevant traits is maintained by mutation-selection balance, that inter-individual genetic covariation of fitness-relevant traits depends on the underlying functional architecture of the loci that affect the traits—in particular, the relative number of loci involved in acquiring versus allocating resources. That is, 'studies estimating G [genetic correlations among traits] do not test for the existence of life-history tradeoffs' (Houle, 1991, p. 630).*

Reply: Z&S are correct, and their point is a reminder that genetic correlations cannot be simply taken at face value as reflections of the underlying functional relations (Section 3.4). However, the situation is not fundamentally different at the genetic vs. phenotypic level: in both cases, the effects of individual condition/quality can be accounted for (at least in principle) so as to reveal the underlying trade-offs (e.g., McLean et al., 2019; Wilson, 2014).

Overall summary: Z&S are right to point out the difference between the level of population/species differences and that of individual variation. However, there can be functional links between the two levels (e.g., basic trade-offs) that justify the ecological gambit as a working assumption. Another important contribution of Z&S is to highlight the role of mutations and broad "quality" factors. But while those factors may potentially mask the functional structure of trade-offs at the phenotypic and genetic level, they can be accounted for within a more comprehensive model. Moreover, genetic quality alone is clearly insufficient to explain the totality of the evidence, for example regarding the functional basis of autism vs. schizophrenia. I argue that Z&S are wrong to discount the importance of phenotypic integration and dismiss the role of balancing selection and pleiotropic regulatory mechanisms. However, they raise important questions about the evidence for adaptive plasticity, the empirical support for trait covariation, and the methodological and conceptual problems of popular measurement approaches.

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