Accepted Manuscript

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Brendan P. Zietsch, Morgan J. Sidari

PII: S1090-5138(18)30284-8
DOI: https://doi.org/10.1016/j.evolhumbehav.2019.05.007
Reference: ENS 6274

To appear in: *Evolution and Human Behavior*

Received date: 24 September 2018
Revised date: 21 May 2019
Accepted date: 23 May 2019

Please cite this article as: B.P. Zietsch and M.J. Sidari, A critique of life history approaches to human trait covariation, Evolution and Human Behavior, https://doi.org/10.1016/j.evolhumbehav.2019.05.007

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Brendan P. Zietsch and Morgan J. Sidari

School of Psychology, University of Queensland, Australia

Email: zietsch@psy.uq.edu.au

Word count: 4777

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Abstract

Covariation of life history traits across species may be organised on a 'fast-slow' continuum. A burgeoning literature in psychology and social science argues that trait covariation should be similarly organised across individuals within human populations. Here we describe why extrapolating from inter-species to inter-individual trait covariation is not generally appropriate. The process that genetically tailors species to their environments (i.e. Darwinian evolution) is fundamentally different from processes that tailor individuals to their environments (e.g. developmental plasticity), so their outcomes in terms of trait covariation need not be parallel or even related. We discuss why correlational selection, physical linkage, pleiotropy, and non-random mating do not substantively affect this claim in the context of complex human traits. We also discuss life history trade-offs and their relation to inter-individual trait covariation. We conclude that researchers should avoid hypotheses and explanations that assume trait covariation will correspond across and within species, unless they can mount a theoretically coherent argument to support this claim in the context of their research question.

Keywords: Life history theory; *r*/*K* selection; fast and slow strategies; fitness epistasis; quantitative genetics; tradeoffs; pace-of-life syndromes; covariance

Understanding trait covariation is of major interest in many fields, including psychology. Personality psychology concerns the patterns of trait covariation that give rise to what we see as broad personality dimensions (e.g. the Big Five, HEXACO), and the covariation of these dimensions with other behaviours and life outcomes. Similarly, the positive covariation between different cognitive abilities, summarised by *g*, has been a central focus of intelligence research, and the study of psychopathology involves organising different pathological traits into covarying clusters called disorders.

Life history theory has been promoted as a unifying framework for understanding trait variation and covariation in the social sciences (e.g. Hertler et al. 2018). The 'theory' as it is generally practiced in work on humans (see Nettle and Frankenhuis 2019) is based on the idea that observed patterns of trait covariation across species (Pianka 1970), and the Darwinian principles thought to underlie those patterns, can be extrapolated to trait covariation across individuals within human populations. The problem with this idea is that the processes that create inter-species and inter-individual trait covariation are fundamentally different, and so the two types of covariation need not be parallel or even related. In this article, we argue that this problem renders many of these life history accounts at best underspecified, and at worst implausible.

1. The fast-slow continuum applied to inter-individual trait covariation within human populations

Early in the development of life history theory, researchers emphasised the observation that, across species, life history traits tend to covary along a single continuum: smaller species tend also to be faster to mature, have more offspring in which they invest less, and be shorter-lived (i.e. a cluster of "*r*-selected" or "fast" characteristics), whereas larger species also tend to be slower to mature, have fewer offspring in which they invest more

heavily, and are longer-lived (i.e. a cluster of "*K*-selected" or "slow" characteristics) (MacArthur and Wilson 1967; Pianka 1970). Across species, *K*-selected and *r*-selected characteristics can be reasoned to cluster for good adaptive reasons (though see Stearns (1977)): for example, variable, unpredictable environments involving random mortality threats might favour fast development and reproduction if it is maladaptive to develop slowly when there is high risk of dying before reproducing, whereas more stable, predictable environments allow greater somatic investment and therefore slower development, larger body size, and later reproduction with greater investment (Pianka 1970). Note that although these trait clusters would evolve in response to aspects of the environment, they depend on genetic differences among the species, since evolution occurs through genetic changes. The *r*-*K* (or fast-slow) continuum fell out of favour in the biology literature due to theoretical and empirical inadequacies (Stearns 1977; Stearns 1984) – for example, *K* (unlike *r*) cannot be realistically expressed as a function of life history traits (Stearns 1977), and the empirical evidence for a unitary fast-slow continuum is mixed when looking across species within clades and controlling for body size (e.g. Stearns 1977; Stearns 1983, 1984; Promislow and Harvey 1990; Oli 2004; Bielby et al. 2007; Salguero-Gómez et al. 2016). Modern biology research that invokes the term 'life history theory' rarely adopts the fast-slow framework (Nettle and Frankenhuis 2019).

However, a burgeoning body of evolutionarily informed literature in the social sciences applies the fast-slow continuum to explaining inter-individual trait covariation in humans, whereby it is proposed that "natural selection acts to combine psychosocial traits into meaningful functional composites" (Figueredo et al. 2005). As well as extrapolating from inter-species to inter-individual patterns of covariation, the fast-slow continuum is extended from traditional life history traits (pertaining to growth, reproduction, and lifespan) to a wide range of physical and psychological traits. Thus scores of papers have used such a

framework in explaining covariation of morphology (Mascaro et al. 2013), physical and behavioural development (Ellis et al. 2009), longevity and intelligence (Rushton 2004), mating strategy (Belsky et al. 1991; Jonason and Kavanagh 2010), altruism (Figueredo et al. 2007), personality (Jonason et al. 2010), and psychopathology (Del Giudice 2014; Hurst and Kavanagh 2017). Whether via genetic coadaptation or correlated plastic responses to childhood environment, or often both, these individual differences are claimed to intercorrelate to produce an overarching "life history strategy", which varies on a fast-slow continuum like that arguably observed across species (Rushton 1985; Figueredo et al. 2005; Figueredo et al. 2013). This extrapolation of inter-species trait covariation to inter-individual trait covariation is unjustified and has led to a large body of literature that is based on a flawed theoretical premise.

2. Species differ largely because of selection, whereas individuals differ largely because of inheritance

In large part, a species is the way it is (and is different from other species) because of a process over many generations of selection for genes that confer greater inclusive fitness in environments the species occupies. This process of adaptation genetically tailors each species to its environment – we describe this process and its underlying principles as Darwinian, for brevity. Within populations as well, individuals differ in part because of genetic differences (Polderman et al. 2015), but there is no *equivalent* (i.e. Darwinian) process that tailors varying individual genotypes to individuals' varying personal environments. Take body height as a simple example: humans are taller than rabbits because of their different genes, and individual humans are different heights largely because of their different genes (variation in human height is around 80% heritable within populations; McEvoy and Visscher 2009;

Wainschtein et al. 2019)[1]. But the processes that create genetic height differences between rabbits and humans (i.e. Darwinian evolution) are nothing like the processes that create genetic height differences between individual humans. The fact I (the first author) am taller than a rabbit is primarily due to our different histories of Darwinian evolution over countless generations of different selective pressures; the fact I am taller than my friend or my brother has little (or nothing, in the case of my brother) to do with our different histories of Darwinian evolution, but rather my chance inheritance of more "tall alleles" compared to them. As with all individuals of European ancestry, many of my friends and I share nearly the same set of ancestors who were alive 1,000 years ago (and likely many ancestors more recent than that) (Ralph and Coop 2013) – a very short period in evolutionary time. (Note that recent rapid increase in average height is not thought to be substantially due to selection; Tarka et al. 2015; Berg et al. 2018). Therefore, the reason my friends and I are of different heights is largely independent of our different histories of Darwinian evolution. We are different heights mostly for the same reason my brother and I (who share all our ancestors) are of different heights – that is, the random shuffling process of Mendelian segregation during meiosis gave us different combinations of our ancestors' genetic material. Environmental effects on inter-individual differences in height have no direct parallels in terms of inter-species differences either: the reasons humans are taller than rabbits are not environmental to any significant degree. Overall, this example highlights why we cannot straightforwardly transfer the same evolutionary principles from the explanation of human-rabbit differences to human-human differences[2].

[1] For virtually all complex traits, both physical and behavioural, variation between individuals is substantially due to genetic differences. Turkheimer, E. 2000. Three laws of behavior genetics and what they mean. *Current Directions in Psychological Science* 9:160-164.

[2] NB: Throughout, in referring to inter-individual trait covariation we mean among individuals within populations, not across separate populations within a species. In principle, the latter could be subject to the same Darwinian processes that influence trait covariation across species.

The same argument applies to trait covariation. If environments that tend to favour large bodies also tend to favour high parental investment, then genes controlling the two traits will come to covary across species (Pianka 1970). 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 (Fusco and Minelli 2010); 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 (Baldini 2015). Similarly, there are different consequences at the species and individual levels in situations where selection on one trait depends on the level of another trait. If genes for high parental investment are advantageous in a large species and disadvantageous in a small species, genes for large body size and genes for high parental investment will tend to go together across species, and likewise genes for small body size and low parental investment. But there is no equivalent evolutionary process by which variation in different traits within a population can be "combined into trait clusters that may be differentially selected based on how well they do or do not work together to serve their multiple adaptive functions" (Figueredo et al. 2013). Darwinian evolution cannot combine "tall alleles" with "high parental investment alleles", and "short alleles" with "low parental investment alleles", within a sexually reproducing population in the same way as it can across species, because of Mendel's Law of Independent Assortment: segregating alleles are transmitted to offspring independently of each other (with exceptions discussed in the following section), so allele configurations are not inherited. Darwinian evolution can tailor allele combinations across species but not across individuals because species are reproductively isolated whereas individuals are not.

3. Exceptions to Mendel's Law of Independent Assortment do not mean inter-individual trait covariation can be explained by Darwinian selection for trait clusters

3.1 Correlational selection

Correlational selection is selection for optimal trait combinations. Intense correlational selection can in principle generate inter-individual trait covariation by generating transient covariation between genetic variants at different loci (genomic locations) (Sinervo and Svensson 2002; Rohlfs et al. 2010). 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. Life history traits are quantitative traits, which as a rule are underlain by thousands of tiny-effect variants at loci spread out across the genome (Chabris et al. 2015; Goddard et al. 2016). With this highly polygenic architecture, even sustained and extreme correlational selection on life history traits would have a negligible effect on their covariation.

3.2 Physical linkage

Variants at loci nearby on the same chromosome often covary (i.e. are in linkage disequilibrium) due to incomplete recombination during meiosis (The International HapMap Consortium et al. 2005). As mentioned, though, 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.

3.3 Non-random mating

Covariation between variants at different loci can be generated by non-random mating (Fisher 1918; Yengo et al. 2018). For example if tall men tend to mate with intelligent

women (or vice versa), the variants influencing the two traits will tend to covary in the population (which appears to be the case; Keller et al. 2013). 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.

3.4 Pleiotropy

Another way genetic variation in different traits can cluster is through pleiotropy, whereby the same genetic variant affects variation in multiple different traits. Pleiotropy in loci underlying quantitative traits appears common (Pickrell et al. 2016). Pleiotropy can generate genetic and phenotypic covariation between traits, but it need not – effects of positive and negative pleiotropy across multiple loci can cancel out, as they would tend to if the direction of pleiotropy was random. But the directions of pleiotropic effects are probably not random. First, non-zero mutational effects tend to be both pleiotropic and deleterious (Houle et al. 1994; Wang et al. 2010; Tenaillon 2014), 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) (Houle 2000)[3]. Figure 1, which shows genetic correlations among varied human traits relating to physical and mental robustness, is consistent with such a tendency (see also Hagenaars et al. 2016). This pattern is also consistent with models of the evolution of pleiotropy, which predict that traits that have been subject to concordant directional selection will correlate positively (and that those that have been subject to opposite directional selection will correlate negatively) (Pavličev and Cheverud 2015; Melo et al.

[3] We might call it a 'fitness factor', but modern medicine, contraception, family planning, and IVF have divorced reproductive success in contemporary Western populations from typical indicators of quality or condition. Perusse, D. 1993. Cultural and reproductive success in industrial societies - Testing the relationship at the proximate and ultimate levels. *Behavioral and Brain Sciences* 16:267-283.

2016). Assortative mating for quality would also predict such a pattern (though via linkage disequilibrium rather than pleiotropy) because it would entail positive cross-trait assortment for traits that contribute to quality (per Sect. 3.3 above). These explanations are not mutually exclusive.

Note that this pattern of directional pleiotropic effects on traits does not reflect "how well they do or do not work together to serve their multiple adaptive functions" (Figueredo et al. 2013); for example, poor mental and physical health do not work well together. 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).

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 (Carrol 1993); 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) (Lee et al. 2013); and the tendency for positively valued personality traits to correlate positively (Musek 2007) (though this could alternatively be an artefact of response biases; Irwing 2013). How broad we might expect the pleiotropy to be depends how modular human development is, which is not well understood. In general, though, pleiotropy is likely greater within developmental modules than between (Wang et al. 2010; Melo et al. 2016). Establishing a clear overview of the pattern of genetic correlations among human traits will be helped by large, genotyped samples like the UK Biobank. However, it should be noted that most of these genotype data do not efficiently capture rare variants (e.g. minor allele frequency < .01). Rare variants appear

disproportionately influential (compared with common variants) in human complex traits (Verweij et al. 2012; Hill et al. 2018; Wainschtein et al. 2019), consistent with maintenance of genetic variation by mutation-selection balance (Eyre-Walker 2010) and inconsistent with balancing selection (Turelli and Barton 2004; Bubb et al. 2006; Verweij et al. 2012). Genetic correlations based on these data (e.g. Figure 1) may therefore underrepresent the degree of directional pleiotropy, because variants with pleiotropic deleterious effects are more likely to remain rare in the population (due to greater selection against them) compared with variants exhibiting no pleiotropy or antagonistic pleiotropy (i.e. a mix of beneficial and harmful effects).

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 et al. 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. Further, 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 et al. 2007, p. 568).

4. Claims regarding correspondence between inter-species and inter-individual trait covariation are usually not based on cogent theory

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. For example, in testing the covariation between testicle size and parental investment within humans, a prediction they derived from life history theory, Mascaro et al. (2013) said only: "Although life history theory is traditionally invoked to explain differences between species, it has also been applied to explain individual differences within a species, including humans (Figueredo et al. 2005; Apicella and Marlowe 2007)." In the Figueredo et al. (2005) paper cited, the authors wrote: "In this view [i.e. applying life history theory to human trait covariation], natural selection acts to combine psychosocial traits into meaningful functional composites." The authors did not explain *how* natural selection would do this, but credited Rushton (1985) with originating the idea. Rushton (1985), in turn, wrote:

> "Sociobiologists focus primarily on the evolutionary origins of between-species differences. Yet, clearly, the theory of evolution requires that there be a genetic basis to the within-species differences in the behaviours studied (Plomin, DeFries, & McClearn, 1980). The question thus arises as to whether the *r*/*K* continuum also applies to within-species differences."

This quote involves a misunderstanding and then a non sequitur. First, the theory of evolution does not in fact require there to *be* (as opposed to *have been* at some point in the past) a genetic basis to within-species differences, though in any case all quantitative human traits are partly heritable (Polderman et al. 2015). Second, it is unclear why "the question thus arises" regarding within-species differences. Nowhere in this foundational paper did Rushton identify a Darwinian process that should align inter-species and inter-individual trait covariation. 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.

In the animal literature, a parallel body of work (see Montiglio et al. 2018 for a review) likewise proposes that inter-species trait covariation corresponds to inter-individual covariation of physiological, behavioural, and life history traits along a fast-slow continuum (referred to as pace-of-life syndromes; Réale et al. 2010). Originally, pace-of-life syndromes

were proposed to result from genetic coadaptation through correlational selection (Réale et al. 2010), which we have shown could not substantively explain covariation of quantitative traits (Supplementary Material). Developmental plasticity was later added as an additional explanation (Dammhahn et al. 2018). 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 and Frankenhuis 2018), leaving the pace-of-life perspective without a clear theoretical basis. Even more recently, Wright et al. (2019) proposed density-dependent fluctuating selection as a mechanism that might align inter-species and inter-individual trait covariation. The proposal involves a host of untested assumptions and no formal modelling, but it is nonetheless welcome in its recognition of the need to identify a theoretical basis for proposals regarding alignment of trait covariation across levels. 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 (Turelli and Barton 2004), whereas the genetic architecture of human quantitative traits that have been examined exhibits the opposite tendency, i.e. disproportionate contribution of rare alleles (Verweij et al. 2012; Hill et al. 2018; Wainschtein et al. 2019).

5. Genetic coadaptation vs. adapted developmental plasticity

As mentioned, the foundational application of the fast-slow continuum to inter-individual trait covariation (Rushton 1985) retained the genetic coadaptation rationale of the original theory (Pianka 1970). Many scholars subsequently working in the area have adopted this perspective (e.g. Ellis 1987; Figueredo et al. 2005; Giosan 2006; Gladden et al. 2009; Figueredo et al. 2013; Luoto et al. 2018) or simply cite Rushton or Figueredo and colleagues for why life history theory should be applicable in this way (e.g. Jonason et al. 2010; Mascaro

et al. 2013). Other scholars in the area invoke both genetic factors and phenotypic plasticity in claiming that life history theory can be applied to variation within species (e.g. Ellis et al. 2009; Del Giudice et al. 2015), e.g. "we extend LH theory to the study of individual differences, recognizing that adaptive variation in LH strategy, both between and within species, is generated by a combination of evolved genetic diversity and phenotypic plasticity in response to environmental influences" (Ellis et al. 2009). Others have emphasised mostly evolved responses (e.g. acceleration of physical and sociosexual development) to early environmental conditions (e.g. mortality rates, socioeconomic deprivation, parental harshness, father absence) (e.g. Belsky et al. 1991; Chisholm et al. 1993; Maner et al. 2017). 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. There are several problems with this perspective.

First, 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 socioeconomic status, parenting style, father absence, risky upbringing). Heritability estimates from twin studies range from around 20 to 60% for age at menarche, age at first birth, age of menopause, number of children, number of sexual partners, sociosexuality, extra-pair mating, and the Big Five personality traits (Jang et al. 1996; Bailey et al. 2000; Kirk et al. 2001; Zietsch et al. 2008; Zietsch et al. 2014; Zietsch et al. 2015). Indeed, a factor designed to capture fast-slow life history 'strategy' has itself been estimated at 65% heritability (Figueredo et al. 2004). 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. In contrast, in twin studies the shared environment has not been consistently estimated to account for a substantial proportion of variation in any of the aforementioned traits. In particular, shared environmental influences were estimated to account for zero variation in the fast-slow life history factor (Figueredo et al. 2004). Likewise, covariation between such traits is estimated to be substantially genetic and not substantially attributable to the shared environment (Kirk et al. 2001; Rowe 2002; Figueredo et al. 2004; Figueredo and Rushton 2009; Zietsch et al. 2010). Reported associations between developmental environment and adult traits are rarely controlled for genetic confounders (e.g. in twin-family designs) (McAdams et al. 2014; Zietsch 2016; Barbaro et al. 2017; Sherlock and Zietsch 2017b; Sherlock and Zietsch 2017a; Xu et al. 2017), and when they are controlled the associations are often weaker or null (Mendle et al. 2006; Mendle et al. 2009; McAdams et al. 2014; though see Tither and Ellis 2008). 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 (Zietsch 2016).

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 (Belsky et al. 1991; Frankenhuis et al. 2016), is not justified by formal evolutionary modelling (Baldini 2015). First, an individual's optimal response to a harsh environment within a large heterogeneous environment (which is what an adapted plastic response would be designed to achieve) is not generally the same as the optimal strategy of a population uniformly inhabiting the same harsh environment (which is what genetic adaptation of a species is designed to achieve). Second, in both cases the optimal strategies for harsh environments depend on parameters that are poorly understood in real populations. 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. Note that some researchers use environmental 'harshness' in reference to 'extrinsic mortality' that is unrelated to characteristics of the individual, whereas in Baldini's model 'mortality' can depend on parameters of the individual (whether or not it does depends on the value of another parameter that specifies the effectiveness of investments in mortality reduction). In reality, mortality that is unrelated to characteristics of the individual is probably rare (Abrams 1993).

Other scholars have not posited that traits within human populations are "mutually coadapted" (Figueredo and Rushton 2009) or that they involve specially adapted responses to early environmental conditions, but rather that they covary because of individuals' "contextually appropriate responses to structural and ecological factors" (Pepper and Nettle 2017a). These contextually appropriate responses are proposed to arise through proximate processes such as reasoning, learning, and following norms, while being ultimately explained by Darwinian principles. In particular, Pepper and Nettle (2017a) explained the covariation of behavioural traits associated with low socioeconomic status using evolutionary models (overlapping with life history theory) that "embody principles that were originally used to understand the selective forces leading to the evolution of traits over generations. However, the same principles can be applied to enhance our understanding of how behaviour is shaped by people's environments within their lifetimes." (p. 6). Sherlock and Zietsch (2017a) noted that this theoretical premise is flawed because there is no process equivalent to Darwinian evolution that differentially tailors each individual within a species to its personal environment. In response, Pepper and Nettle (2017b) disagreed by citing the example that

"Individuals growing up in Hungary acquire the Hungarian language and become skilled at driving on the right-hand side of the road, whereas individuals growing up in England do not." (p. 50). This argument misses the point while also illustrating the problem perfectly: although humans can speak and drive thanks to evolved capacities (e.g. specialised and/or domain-general learning mechanisms), it would be useless and misleading to use Darwinian principles to explain variation or covariation in which language and side-of-the-road individuals use.

Various processes can tailor individuals to their environments (or coordinate variation in different traits) – as Pepper and Nettle (2017a; 2017b) note – but these processes (e.g. learning, reasoning, following norms) tailor individuals to their personal environments according to very different sets of principles from those by which Darwinian evolution tailors different sexually reproducing species to their environments (e.g. differential reproduction, inheritance, mutation, selection, speciation). To understand how learning, reasoning, norm-following, etc., generate trait covariation, we need to use the principles of those processes (e.g. regarding conditioning, reward and punishment, logical thinking, overimitation, etc.). Despite the fact that these processes are products of Darwinian evolution, it is a category mistake to haphazardly apply Darwinian principles that explain inter-species trait covariation to inter-individual trait covariation.

6. Empirical evidence does not support inter-individual trait covariation being organised around a fast-slow continuum in humans or in other species

Given that theory provides no clear reason to expect that inter-individual trait covariation should generally correspond to inter-species trait variation, it is not surprising that 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 (Copping et al. 2014; Copping et al. 2017; Richardson et al. 2017). 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 (Nicholson et al. 2005) – i.e. seemingly the opposite of a fast life history strategy, which is hypothesised to be characterised by high risk-taking (Figueredo et al. 2005), boldness, and low stress-system reactivity (Réale et al. 2010). 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.

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 et al. 2018). The mean correlation among traits expected to positively covary was 0.06; within vertebrates it was 0.02. Within females, the mean correlation was directionally opposite to predictions (-0.16), while within males it was null (0.01). In samples with males and females pooled, potentially susceptible to confounding by sex, the correlation was 0.12. 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.

7. Trade-offs

Most of the work on human trait covariation that invokes life history theory (and thus most of our criticism) is based on the organisation of trait covariation around a fast-slow continuum (Nettle and Frankenhuis 2019), often interpreted to reflect adaptively coordinated life history 'strategies'. In contrast, as Nettle and Frankenhuis point out, 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 (e.g. growth, sexual maturation, reproduction, parental investment, and lifespan) (e.g. Lande 1982; Reznick 1985; Sinervo and Svensson 1998; Roff and Fairbairn 2007). 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 indivdiual's resources such as energy or time.

It is problematic, though, to assume that trade-offs within individuals should cause corresponding trait covariation across individuals (Fisher et al. 2018). To take an everyday example cited in Fisher et al. (2018): individual typists must trade-off speed and accuracy (the faster someone types the more errors she tends to make), causing negative covariation between speed and accuracy within-individuals; but across individuals there is wide variation in overall typing ability, so faster typers tend to be also more accurate, i.e. positive inter-individual correlation between speed and accuracy. In the same way as they vary in typing ability, 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 (Noordwijk and Jong 1986). 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).

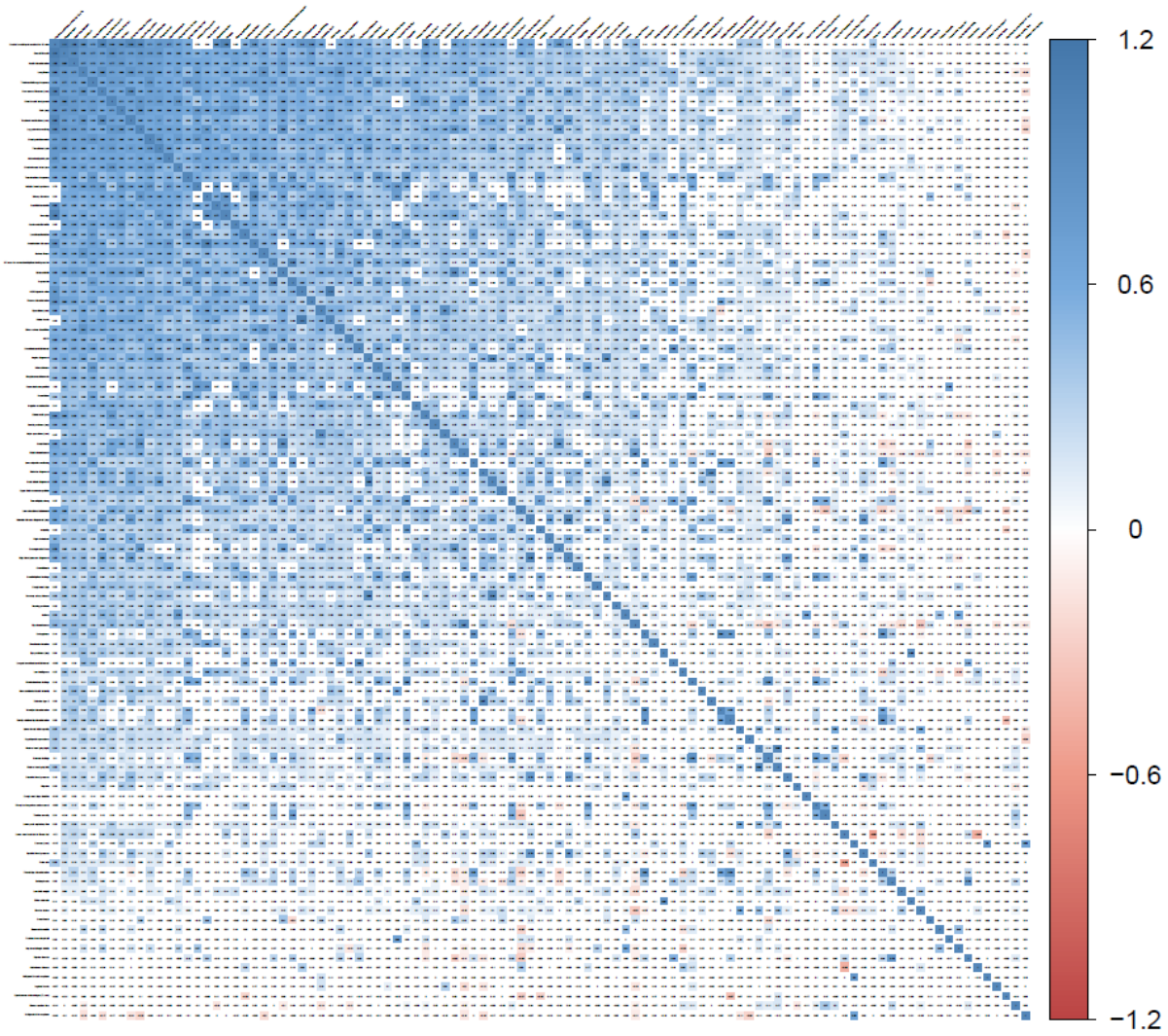
8. Conclusion

Even if it is true that inter-species trait covariation is organised along a fast-slow continuum, there is no clear reason to expect that inter-individual trait covariation should be similarly organised within species. Accordingly, empirical evidence for the latter is weak in humans and disconfirmatory in other species overall. In short, there is little justification for the fast-slow continuum as a framework for understanding human trait covariation. Another life history approach, more common in biology, emphasises specific functional trade-offs among life history traits; but these trade-offs usually relate to resource limitations within an individual and do not straightforwardly predict trait covariation across individuals. A more careful examination of whether the requirements of inter-species or intra-individual models are met when applied to inter-individual covariation could help identify the untenable nature of certain inter-individual models, and could focus research on those tenable models that do emerge.

Acknowledgements

B. P. Z. is supported by the Australian Research Council (FT160100298). Thanks to David Lewis, Jonathan Redshaw, Adam Bulley, Ruben Arslan, Bill von Hippel, Fiona Barlow,

Aaron Lukaszewski, George Richardson, Gert Stulp, Willem Frankenhuis, Daniel Nettle, and several anonymous reviewers for comments and discussion. Thanks to Abdel Abdellaoui for help with Figure 1.



Genomewide association study (GWAS) summary statistics are drawn from: ADHD (Demontis et al. 2019); Alzheimer's disease (Lambert et al. 2013); Bipolar disorder (Ruderfer et al. 2014); Coronary heart disease (Nikpay et al. 2015); Type 2 diabetes (Morris et al. 2012); Educational attainment (Okbay et al. 2016b); Height (Wood et al. 2014); Intelligence (Sniekers et al. 2017); Schizophrenia (Ripke et al. 2014); Subjective wellbeing (Okbay et al. 2016a); All other traits: http://www.nealelab.is/blog/2017/7/19/rapid-gwas-of-thousands-of-phenotypes-for-337000-samples-in-the-uk-biobank

Figure 1.

Genetic correlation matrix of UK Biobank traits and psychiatric disorders that reflect physical or mental robustness. Genetic correlations are estimated using LD score regression (Bulik-Sullivan et al. 2015) based on summary statistics UK Biobank data from up to 337,000 genotyped individuals and from published genomewide association studies (GWASs), listed below. All traits are scored in the same direction with respect to overall quality: for ease of labelling the variables, many of which are disorders, greater scores reflect lower robustness.

Traits are ordered – left to right, top to bottom – by first principle component order. Blue squares represent significant positive genetic correlations and red squares significant negative genetic correlations, the darker the colour the stronger the correlation. The matrix is far more blue than red, which suggests genetic correlations among traits tend to be directionally concordant with respect to overall quality or condition.

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