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Are heritable individual differences just genetic noise? What the architecture of quantitative traits says about their evolution☆

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ABSTRACT

The evolution of heritable individual differences (for example in personality, cognition, and the risk for psychopathology) is the subject of a long-running debate between proponents of adaptive and non-adaptive explanations. Newly available genomic data show that most quantitative traits conform to what I label the "default genetic architecture," characterized by extreme polygenicity with contributions from both common and rare variants, with large-effect variants that tend to be rarer and younger than small-effect ones. Furthermore, targeted tests of balancing selection return largely null or negative results. These findings indicate widespread purifying selection at the genetic level; they have led some scholars to argue that heritable individual differences are essentially non-adaptive or maladaptive, and that evolutionary hypotheses that invoke balancing selection are inconsistent with the data. Here I show that this strong interpretation is not warranted. I distinguish between four questions about the evolution of heritable individual differences, and explain why the data do not support sweeping inferences about their adaptive function (or lack thereof). I also discuss why tests of balancing selection are much less informative than is often believed. While the pervasive role of purifying selection is beyond dispute, the default architecture of complex traits is potentially compatible with a broad range of evolutionary scenarios, including scenarios in which heritable individual differences can be adaptive and functional rather than just manifestations of neutral/maladaptive noise.

1. Introduction

As a rule, quantitative traits in humans show sizable amounts of heritable variation (Polderman et al., 2015). This is true whether the traits in question are physical like height and waist-hip ratio; developmental like puberty timing; psychological like intelligence and personality; or indices of the risk for various medical and psychiatric disorders. The ubiquity of heritable differences raises fundamental questions about the evolutionary reasons for the maintenance of genetic variation within human populations. In the evolutionary behavioral sciences (particularly evolutionary psychology and psychiatry), these questions have sparked a long-running debate on the nature of heritable individual differences—a debate that is taking a new turn as we move through the "genomic era."

In a landmark paper published in 1990—at the time when the results of twin and adoption studies were starting to make waves through the social sciences—Tooby and Cosmides (1990) argued that heritable differences in psychological traits are unlikely to be adaptive, and more likely to represent neutral or maladaptive noise, perhaps the byproduct of pathogen-driven selection for sheer biochemical diversity. At the same time, there is considerable evidence that, at least in certain domains such as personality, different levels of a trait yield different profiles of costs and benefits, with consequences on all sorts of fitness-relevant outcomes (see e.g., Nettle, 2006, 2011); and in fact, Tooby and Cosmides suggested that adaptive psychological differences can arise through *adaptive plasticity* in response to both environmental conditions and a person's physical characteristics (e.g., height, strength, attractiveness).

In the wake of Tooby and Cosmides' paper, the adaptive view of heritable individual differences was defended by other scholars, notably Wilson (1994) and Bailey (1997, 1998). There are a number of ways in which heritable individual differences in a quantitative trait can be locally adaptive. To begin with, different levels of a trait yield about the same fitness—either because the associated costs and benefits balance out (see Nettle, 2006, 2011), because higher vs. lower levels are optimal in different social/ecological niches, or because being different from others lessens the competition for social or material resources, with a net relative benefit over a range of phenotypes (on this last point see

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Figueredo et al., 2005). As a result, the fitness function associated with the trait is characterized by a flattened plateau or by multiple peaks instead of a single peak. This kind of scenario is often equated with "neutrality," but this is misleading: even in presence of a broad plateau, extreme high and/or low levels of the trait can still be maladaptive and selected against—the trait *as a whole* does not become neutral to selection just because it lacks a single sharp optimum (more on this below).

In addition, optimal trait levels may change over time and across different places, niches, etc., in ways that make the fitness function vary instead of remaining constant. A variable fitness function means that a certain trait level can be adaptive at some times or places, but become maladaptive at others. (Once again, certain phenotypes may lie outside the adaptive range of the trait and be *always* selected against.) Variable selection is especially significant because of its ability to maintain genetic variation via *balancing selection* (see below).[1] As noted, these two possibilities are not mutually exclusive; for example, different levels of a trait with countervailing costs and benefits may yield a similar fitness in "average" environmental conditions, but shifts toward more extreme environments may temporarily favor a higher or lower level.

Keller and Miller (2006) advanced an argument with respect to psychopathology that converges on the idea that individual differences are generally maladaptive. They argued that the genetic variants underlying the risk for common, heritable, and harmful mental disorders such as schizophrenia are most likely under *negative* or *purifying selection*, and are maintained in the population by the balance between the onset of new mutations and their elimination by natural selection (*mutation-selection balance*, more accurately described as *mutation-selection-drift* because random genetic drift can increase the frequency of mildly deleterious alleles; see Sella & Barton, 2019). Penke, Denissen, and Miller (2007) proposed the compromise view that, while genetic variation in mental disorders and cognitive ability is largely explained by mutation-selection-drift dynamics, balancing selection is a plausible explanation for heritable differences in personality (see also Penke & Jokela, 2016). Over the years, the debate has continued with other contributions, including Miller (2011), Del Giudice (2012, 2020), Gangestad (2011), and Maestripieri and Boutwell (2022).

1.1. Individual differences in the genomic era

In the past ten years, significant advancements in understanding the genetic architecture of complex traits have been made, thanks to the availability of large-scale datasets and the growing sophistication of genome-wide association studies (GWAS). Zietsch, de Candia, and Keller (2015) used then-novel genomic data on schizophrenia to argue that the genetic architecture of this disorder (see below) is consistent with a history of purifying selection against variants that increase the risk for schizophrenia; in this scenario, heritable differences are maintained by mutation-selection-drift. Keller (2018) expanded this argument with additional data, recognizing the possibility that, even under widespread purifying selection, some schizophrenia risk alleles may have been maintained through balancing selection—potentially due to their role in enhancing creativity and increasing mating success (see Del Giudice, 2017, 2018). However, he suggested that a more significant factor may be the near-neutrality of certain alleles, allowing them to drift to high frequencies due to pleiotropic effects that partially or fully counterbalance the negative fitness consequences associated with increased schizophrenia risk.

In a recent paper, Zietsch (2024) took this line of reasoning one step further. He reviewed accumulating evidence that a broad range of complex traits besides schizophrenia—spanning the gamut of morphology, physiology, cognition, personality, and disease risk—share a common genetic architecture characterized by four key features:

- *Extreme polygenicity*: thousands of loci contribute to heritable individual differences on the trait, but each specific variant accounts for only a tiny fraction of the total heritability.
- *Both common and rare variants contribute* to heritable individual differences. The rarity vs. commonness of a variant is quantified by the corresponding *minor allele frequency* (MAF), i.e., the frequency of the rarer allele at a polymorphic locus in the genome.
- There is a *negative association between the effect of a variant on the trait and the corresponding MAF*. This means that, on average, rarer variants have larger effect sizes than more common ones.
- *Younger variants explain more heritability (per variant) than older ones.* That is, variants that arose more recently in the population (counting from the initial mutation event) tend to show larger effect sizes.

Indeed, this combination of features is so ubiquitous and uniform across traits that it has become the de facto default expectation in genomic studies. Hence, I propose that it can be usefully labeled the *default genetic architecture* of complex traits. Following Zietsch et al. (2015) and Keller (2018), Zietsch (2024) argued that a default architecture is consistent with pervasive mutation-selection-drift dynamics driven by directional selection (higher or lower values of the trait are consistently adaptive) or stabilizing selection (there is an optimal intermediate value of the trait that is maximally adaptive), but inconsistent with either neutrality or balancing selection. For example, if increased risk for schizophrenia is associated with reduced fitness, purifying selection will quickly act against large-effect (i.e., highly deleterious) variants and keep them rare until they are eliminated; while small-effect (i.e., slightly deleterious) variants will often drift to higher frequencies and persist for longer in the gene pool. This inference was reinforced by the results of large-scale tests designed to detect specific patterns of selection at individual loci, which found ample evidence of purifying selection across many traits but no or even negative evidence of balancing selection (Abraham, LaBella, Capra, & Rokas, 2022; see also Gazal et al., 2018; O'Connor et al., 2019; Schoech et al., 2019; Zeng et al., 2021).

Zietsch (2024) concluded that these findings have profound implications for the evolutionary behavioral sciences. In his words, "using balancing selection as an explanation for personality variation flies in the face of the available evidence" (p. 7); hence we must accept that, in the vast majority of cases, heritable individual differences are not adaptive but represent genetic noise that is either nearly neutral or maladaptive, and is maintained by the constant influx of mutations and drift in finite populations. If this view is correct, it could mean that most of the existing theories on the evolution of heritable differences in personality, life history strategies, and the risk for certain mental disorders (see e.g., Del Giudice, 2018, 2020; Nettle, 2006, 2011) are empirically untenable and should be discarded:

> "Adopting this picture—mutation-selection-drift balance—may require a shift in perspective for many evolutionary social scientists, who tend to be strongly adaptationist in their thinking. Instead of asking what is the adaptive function of individuals differences […] the new perspective might involve a default view that trait variation is either roughly neutral or maladaptive" (Zietsch, 2024, p. 8).

Around the same time, other evolutionary scholars independently made similar points in conference talks (Keller, 2024; Penke, 2024), even if not as forcefully. Clearly, this interpretation of the genomic data has weighty implications and a persuasive, seemingly compelling logic. But is it justified? In the remainder of this paper, I show that the logic does not hold up on closer scrutiny, and explain why the data do not support sweeping inferences about the adaptive function (or lack thereof) of individual differences. Among other things, I explain why balancing selection likely contributes to maintain genetic variation across species,

[1] Note that the hypothesis advanced by Tooby and Cosmides (1990) implies a form of balancing selection driven by pathogens—only directed toward biochemical processes (against uniformity) and with behavioral manifestations as mere byproducts.

and why genomic tests of balancing selection are much less informative than many presently believe. While the pervasive role of purifying selection and mutation-selection-drift is beyond dispute, the default architecture of complex traits is potentially compatible with a broad range of evolutionary scenarios, including plausible scenarios in which heritable individual differences can be adaptive and functional. Simply stated: there is nothing in the genomic data that should compel evolutionary behavioral scientists to stop asking questions about the function of individual differences, or to adopt a default "null model" that equates heritable variation with neutral/maladaptive noise.

Note that my goal is *not* to compare alternative hypotheses on the evolution of individual differences in specific traits, or review the growing empirical literature on the genetic basis of those traits and their contributions to fitness. These tasks would require their own dedicated papers. My goal is to address a general argument against the existence of adaptive heritable differences that has been gaining traction in recent years; this argument moves from certain empirical patterns found in genomic studies, but ultimately rests on a set of theoretical assumptions about the evolutionary meaning of those patterns and the inferences that can be drawn from them. Hence, this paper focuses mainly on theoretical issues, although I cite key empirical findings to illustrate the concepts I discuss.

1.2. Four questions about heritable individual differences

Before moving ahead, it is necessary to disentangle four basic questions that underlie the debate around individual differences. These questions are logically and theoretically distinct but often get mixed up by scholars on both sides, leading to confusion and incorrect generalizations. For a given quantitative trait, we can ask:

1. *Have individual differences in the trait been (locally) adaptive? If so, over what range?* This question concerns the adaptive function of individual differences, and the costs and benefits of different trait values in alternative conditions. The issue of range is important, because the full expression range of a trait may include values that are (locally) adaptive, but also values (for example at the high and/or low extreme) that are unconditionally maladaptive regardless of context.
2. *How has selection acted on the trait?* This question concerns the fitness function associated with the trait over its evolutionary history. As I detail below, a trait can be under stabilizing, directional, or disruptive selection, with flatter or more peaked fitness functions; moreover, the fitness function may be constant or vary over time, place, etc.
3. *What processes have maintained genetic variation in the trait?* This question specifically concerns evolutionary processes at the level of the individual loci that contribute to the trait, including the potential role of balancing selection.
4. *What is the genetic architecture of the trait?* This question concerns statistical, aggregate features of the set of loci associated with the trait, such as the degree of polygenicity, the distribution of allele frequencies and effect sizes, and so forth.

The order of the questions follows the flow of causal relations, from question 1 down to question 4: adaptive function determines the characteristics of the fitness function, which in turn shapes evolution at the level of individual loci, which collectively make up the trait's genetic architecture (Fig. 1). But as I discuss in the rest of the paper, these relations are far from simple and straightforward—for example, there is no one-to-one correspondence between the fitness function and selection at individual loci; widely different fitness functions may yield qualitatively similar architectures; and some evolutionary processes that contribute to maintain allelic variation may not leave detectable traces in the genetic architecture.

For this reason, backward-looking inferences (e.g., from question 4 to question 3) are tricky and uncertain, except in special cases; and the uncertainty is amplified when inferences span multiple causal steps (Fig. 1). In particular, inferences that jump from question 4 (about the genetic architecture of a trait) to question 1 (about the adaptive function, or lack thereof, of individual differences) are highly unreliable and should be regarded as tentative at best. In the following sections I detail why this is the case. I start by clarifying the distinction between selection at the level of a trait (*trait level*) and the level of the genetic loci that contribute to that trait (*sequence level*).

2. Selection at the level of traits vs. sequences

2.1. Selection at the trait level

When a trait is *neutral* to selection, all the values of the trait have the same fitness and no value of the trait is favored over another. If instead a quantitative trait is linked to fitness, selection can act on it in three ways, corresponding to three basic kinds of phenotypic fitness functions (Fig. 2; see e.g., Wood & Brodie III, 2016). In *directional selection* (Fig. 2a), higher (or lower) values of the trait have consistently higher expected fitness, so that one extreme of the trait distribution is favored over the rest (e.g., it is adaptive to have a body that is as large as possible). In *disruptive* or *diversifying selection* (Fig. 2b), both high *and* low values enjoy increased fitness while intermediate values are selected against (both smaller and larger bodies are more adaptive than medium-sized ones). In *stabilizing selection* (Fig. 2c), the maximum expected fitness is associated with one or more intermediate values of the trait, and extreme values are selected against (e.g., a medium-sized body is

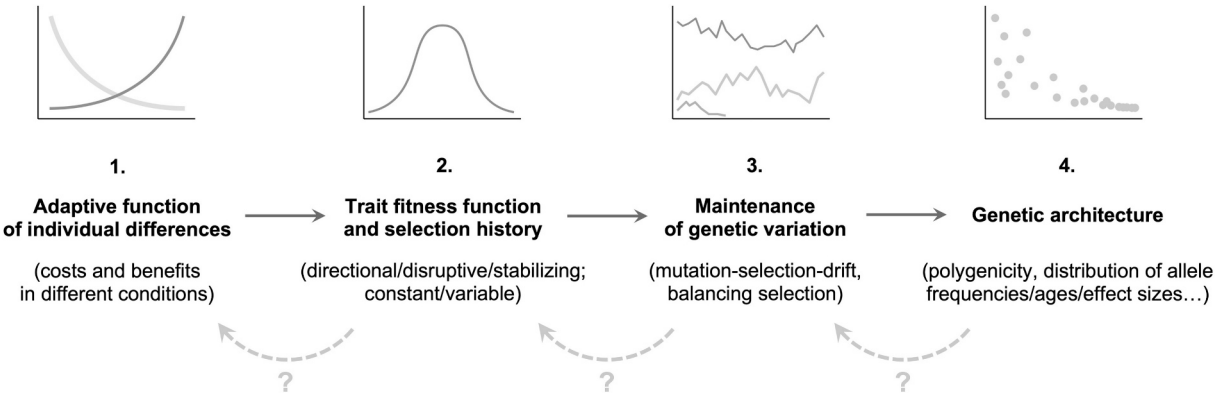

Fig. 1. Four questions about the evolution of heritable individual differences. Each step in the causal chain contributes to determine the outcome of the next (solid arrows). However, backward-looking inferences (dashed arrows) often carry considerable uncertainty, for reasons explained in the main text. Inferences that jump directly from the genetic architecture of a trait (question 4) to the adaptive function of individual differences (question 1) are highly unreliable.

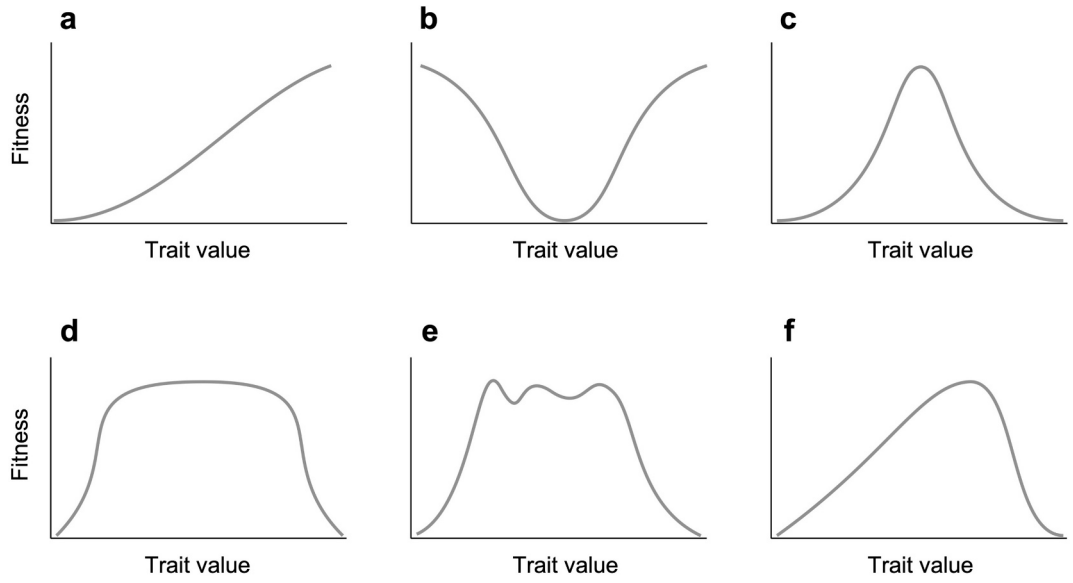

Fig. 2. Examples of fitness functions for a quantitative trait. The top panels illustrate three basic patterns of selection: (a) directional, (b) disruptive, and (c) stabilizing. The bottom panels illustrate three alternative shapes of the fitness function for a trait under overall stabilizing selection: (d) with a plateau, (e) with multiple optima, and (f) "cliff-edged" with asymmetric tails.

most adaptive). A narrower fitness function corresponds to stronger selection against high and low trait values.

While stabilizing selection is usually illustrated and modeled with symmetric, single-peaked functions (as in Fig. 2c), this is not a requirement; it is perfectly possible to have an intermediate region characterized by a plateau or by multiple peaks close to one another (Fig. 2d and e; see for example Haller & Hendry, 2014). These patterns imply that the strength of selection is lessened over a certain range of the trait, but the trait as a whole is not neutral to selection. Also, selection against extreme values does not have to be equally strong in both directions (Fig. 2f; see Nesse, 2004 and Vercken, Wellenreuther, Svensson, & Mauroy, 2012 for the evolutionary implications of "cliff-edged" fitness functions).

Here I use *constant selection* as a shorthand to indicate cases in which a given trait has a history of consistent directional selection (always in the same direction), constant disruptive selection, or stabilizing selection around an effectively unchanging intermediate value. Constant selection regimes can be contrasted with patterns of *variable selection,* in which the fitness function does not remain static but instead varies systematically, for example across time (temporally variable or *fluctuating selection*) and/or space (spatially variable selection). In these scenarios, the fitness function may follow a stabilizing pattern, but with an optimum value of the trait that changes across time/space (e.g., the optimal body size is always intermediate, but larger when food is abundant, smaller when food is scarce); or a directional pattern, with changes in the strength and/or direction of selection (e.g., selection favors the largest bodies in some cases, and the smallest bodies in others). Other less common possibilities exist as well—for instance, selection on a trait may change from stabilizing to disruptive (Siepielski, DiBattista, & Carlson, 2009).

Meta-analyses of data from a variety of species in the wild show that selection often varies in both time and space; what changes more commonly is the strength of selection, but there are also cases in which the direction of selection fluctuates over time (de Villemereuil et al., 2020; Kingsolver, Diamond, Siepielski, & Carlson, 2012; Morrissey & Hadfield, 2012; Mouchet et al., 2021; Siepielski et al., 2013). Note that selection may vary owing to changes in exogenous factors such as temperature or food abundance, but also endogenous population factors such as the sex ratio (Del Giudice, 2012).

Another important pattern of variable selection is *frequency-dependent selection*, in which the fitness of a trait depends on its prevalence in the population. In negative frequency-dependent selection (NFDS), fitness increases as a trait becomes rarer (e.g., the larger the average body size, the more selection favors small bodies, and vice versa); in positive frequency-dependent selection (PFDS), the fitness of a trait increases as it becomes more common. While PFDS tends to push the trait toward one of the extremes, thus making the population more homogeneous, the negative feedback dynamics of NFDS tend to maintain trait diversity. A recent meta-analysis across species has documented widespread frequency-dependent selection, both positive and negative (Gómez-Llano, Bassar, Svensson, Tye, & Siepielski, 2024). Notably, the existence of multiple social/ecological niches within a population can function as a source of frequency-dependent selection (e.g., NFDS if niches have limited capacity and "fill up" as more individuals occupy them; see Hunt & Jaeggi, 2022). Again, frequency-dependent selection can coexist with other selection patterns; for example, Haller and Hendry (2014) explore the interplay of stabilizing selection and NFDS on the same trait, which gives rise to "squashed" stabilizing fitness functions with a flat peak or even multiple peaks.

Under variable selection, individual differences are by definition locally adaptive, at least within a certain range: the same trait value that is selected against at one time and place becomes optimal at another, as the fitness function changes following shifts in ecological conditions and/or in the trait's distribution. Even if some particular trait values (e.g., a body size so small that it is never optimal) may remain consistently maladaptive, variable selection creates the conditions for the emergence of adaptive heritable differences in quantitative traits.

As I noted in the Introduction, the case of constant selection is less straightforward. Under certain fitness functions—such as directional selection without a plateau in the favored direction, or stabilizing selection with a single peak—individual differences are by definition maladaptive, because they always yield a fitness penalty and are selected against irrespective of time, place, and context. In other cases, the trait as a whole is under stabilizing or directional selection because one or both extremes are selected against, but there is a range of trait values with approximately equal fitness (owing to countervailing costs and benefits, multiple niches, etc.) in which individual differences remain adaptive. This is why inferences from question 2 (how has selection acted on the trait?) to question 1 (have individual differences in the trait been adaptive?) are less than straightforward. Knowing that a

trait has been under variable selection means that individual differences have been locally adaptive (but possibly only within a certain range); but simply knowing that a trait has been under constant directional or stabilizing selection does *not* rule out the possibility of adaptive individual differences.

2.2. Different kinds of fitness functions and the role of plasticity

Up to this point, I have talked about "the" fitness function of a trait in the singular; in reality, there are multiple possible fitness functions, depending on what component of the trait is considered in relation to fitness—that is, what kind of values are placed on the horizontal axis in the plots of Fig. 2. When the values are *phenotypic values* of the trait (i.e., the actual body size of individuals), one gets the *phenotypic* fitness function. But if a trait is plastic, phenotypic values are jointly determined by the joint effect of genetic and environmental factors (plus developmental noise). If one is interested in how selection on the trait shapes selection at the level of genes (see below), it is more useful to consider the *genotypic* fitness function, in which phenotypic values are replaced by *genotypic values*—the expected values of the trait based only on the genetic effects, net of environmental influences.[2]

By definition, with a plastic trait the same genotype can express a range of phenotypic values. Assuming that the phenotypic values of the trait are the only cause of differences in fitness (i.e., that the phenotypic fitness function is not confounded by environmental effects; see Morrissey, Kruuk, & Wilson, 2010), plasticity has the effect of flattening the genotypic fitness function, making it wider and less peaked (Frank, 2011). Among other things, this means that plasticity reduces the strength of selection on genotypes. If plasticity is *adaptive*, phenotypic values are matched to the environment (within the limits imposed by the genotype and potential errors) so as to increase the expected fitness; this flattens both the genotypic and phenotypic fitness functions while shifting them upward. In this respect, the impact of adaptive plasticity is similar to that of multiple niches, as both tend to raise and equalize the fitness of different trait values (at least within a certain range). But they do so in very different ways: whereas plasticity changes a genotype's expressed phenotype to match the environment, the existence of multiple niches allows a genotype to find a matching environment *without* having to change its expressed phenotype.

2.3. Selection at the sequence level

Describing selection at the sequence level means summarizing the fate of alleles over the history of a given locus (Hahn, 2018; Hartl & Clark, 2007; Sella & Barton, 2019). A locus is said to be under negative/purifying selection if the vast majority of new mutations that have arisen have been deleterious. Broadly speaking, this is true of virtually any functional region of the genome, and hence it becomes more a matter of degree rather than of presence/absence (e.g., what is the strength of purifying selection at a particular locus?). On the background of pervasive negative selection, *positive selection* is typically used to mean that at least one advantageous mutation at that locus has fixed, or is currently moving toward fixation (see Hahn, 2018). More broadly, one can speak of positive or negative selection on non-neutral alleles; of course, from the perspective of competing alleles positive and negative selection are two sides of the same coin, because if one allele goes to fixation the other(s) get eliminated (see Zietsch, 2024).

Both positive and negative selection tend to reduce allelic diversity (*polymorphism*) in the long run, by eliminating or fixing new mutations. In contrast, balancing selection identifies various selection patterns that tend to maintain polymorphism (Bitarello, Brandt, Meyer, & Andrés, 2023; Fijarczyk & Babik, 2015; Johnson, Tobler, Schmidt, & Huber, 2023; Ruzicka et al., 2025). Classically, balancing selection was restricted to special cases in which selection at a locus can maintain two or more alleles *indefinitely* in a stable equilibrium (at least in principle; in finite populations, drift eventually drives alleles to fixation or loss). I will refer to this kind of scenario as *classical* balancing selection.

Current definitions are typically broader, and include any form of selection that actively maintains genetic variation instead of depleting it (e.g., Fijarczyk & Babik, 2015; Johnson et al., 2023). These patterns include *heterozygote advantage* (the heterozygous genotype is selected for against the homozygous ones) and *antagonistic selection* (an allele has opposite fitness effects in different sexes, at different life stages, and so on). They also include spatially/temporally variable selection and negative frequency-dependent selection—only defined at the level of *alleles* rather than *traits*. This is a crucial distinction that gets easily blurred (see below); at this level of analysis, NFDS means that selection at a locus favors whichever allele is rarer, and temporally/spatially variable selection means that the fitness effect of an allele varies depending on time or place.

It is worth pointing out that the role of fluctuating (temporally variable) selection as a source of balancing selection has been particularly contentious in population genetics, and is still viewed as controversial by some researchers. This is because the early mathematical models—based on the assumption of non-overlapping generations—seemed to indicate that fluctuating selection can maintain a balanced polymorphism indefinitely only in highly restrictive conditions. However, newer models incorporating more realistic features—such as recurrent mutations and overlapping generations (e.g., Bertram & Masel, 2019; Bürger & Gimelfarb, 2002; Ellner, 1996; Gulisija & Kim, 2015; Yamamichi & Hoso, 2017)—show that polymorphisms can be maintained by fluctuating selection much more easily that previously assumed (for reviews, see the supplement of Del Giudice, 2020; see also Johnson et al., 2023; Messer, Ellner, & Hairston Jr, 2016; Ruzicka et al., 2025). Even more importantly, the supposed inability of fluctuating selection to engender classical balancing selection does not mean that it cannot affect the persistence of alleles and their trajectories; it only means that a given polymorphism will not be maintained in the population for very long (Messer et al., 2016).

The scope for balancing selection expands considerably when the relevant alleles exhibit *dominance reversal*, so that they become dominant when beneficial and recessive when deleterious. Evidence of dominance reversal has been found in a number of organisms and traits, and this phenomenon is receiving considerable attention at the moment (Grieshop, Ho, & Kasimatis, 2024; Johnson et al., 2023). Although balancing selection at specific loci is often hard to detect (as I discuss in detail below), data from model species such as *Drosophila* indicate that mutation-selection-drift explains only part of the observed genetic variation in fitness, implying a sizable contribution from balancing selection (Charlesworth, 2015).

2.4. Relations between levels

While there are conceptual analogies between patterns of selection at the trait and sequence levels, it is important to recognize that there is no one-to-one correspondence between the two. Complex traits reflect the joint effect of large numbers of loci, and different loci contributing to the same trait may have different selection histories. For example, stabilizing selection on a trait may involve a mixture of purifying selection at some loci and balancing selection at others (Hahn, 2018; Ruzicka et al., 2025). When a trait is under constant directional selection, one can expect both positive and purifying selection, depending on the direction of the effects associated with the mutations that arise at different loci.

Even more importantly, variable selection on a trait does not rule out purifying selection at some—or many—of the loci involved. Consider a trait under fluctuating selection, where the fitness function is stabilizing

[2] And if the goal is to predict the trait's response to selection, one must specifically consider the *additive* component of genetic effects (see Morrissey et al., 2010). The expected trait values based solely on additive genetic effects are called *breeding values*.

with a variable optimum (i.e., the optimal trait value shifts up and down over time). This is likely to translate into balancing selection at many of the relevant loci. However, large-effect mutations that push the trait outside the range of optimal trait values are unconditionally deleterious, and always selected against. Even when this is not the case, variants with comparatively large effects are more likely to be rapidly lost whenever the trait optimum shifts in the "wrong" direction (Hayward & Sella, 2022). As a result of this blend of balancing and purifying selection, large-effect variants should remain rare (small MAFs) and—on average—younger than small-effect variants. A related implication is that rare variants of large effect will tend to concentrate at the tail(s) of the trait distribution currently disfavored by selection—the upper or lower tail when directional selection favors lower or higher trait values, respectively, or both tails when the fitness function is stabilizing (Souaiaia et al., 2024; see also Koch et al., 2024). In general, most selection regimes—both constant and variable—are going to involve purifying selection at a substantial fraction of the contributing loci, underscoring the ubiquity of this pattern throughout the genome (Sella & Barton, 2019; Wendt et al., 2021; Zeng et al., 2018, 2021). Consistent with this expectation, a new large-scale GWAS found that variants with strong effects on personality traits tend to be selected against, indicating that genetic variation in personality is not solely explained by balancing selection at the sequence level (Schwaba et al., 2025).

The relation between the trait and sequence levels of analysis becomes even more nuanced when the trait in question is the risk for a mental disorder, because the etiology of mental disorders often involves a confluence of qualitatively distinct factors, including aspects of personality and (more or less subtle) impairments in cognitive functionality (see Del Giudice, 2018). For example, the sexual selection model of schizophrenia explicitly postulates an interaction between potentially beneficial, creativity-enhancing dispositions associated with positive schizotypy *and* a high load of harmful, dysfunction-inducing mutations (Shaner, Miller, & Mintz, 2004). Common, mildly deleterious variants that reduce cognitive ability are another likely contributing factor[3] (see Del Giudice, 2017, 2018). Under this model, different categories of loci that contribute to the overall risk for schizophrenia are expected to undergo different and potentially divergent selection histories.

Finally, one should keep in mind that many if not most functional loci contribute to more than one trait (i.e., they are *pleiotropic*), which contributes to create genetic correlations among multiple traits (see Van Rheenen, Peyrot, Schork, Lee, & Wray, 2019). Because of pleiotropy, a trait may appear to be under selection even if has no causal effect on fitness—as long as it shares a genetic basis with one or more traits that do (see e.g., McGuigan, Rowe, & Blows, 2011). Determining whether a trait is under direct or "apparent" selection requires establishing causality, which can be quite challenging without experimental data. However, the distinction is immaterial from the standpoint of the alleles involved; hence, direct and apparent selection lead to similar expectations about the genetic architecture of a trait. Note that extensive pleiotropy among traits further complicates the reconstruction of selection histories: for example, a trait may acquire signatures of stabilizing selection only because another (genetically correlated) trait has evolved under a stabilizing regime.

3. Selection and adaptation in highly polygenic traits

As I noted in the introduction, most quantitative traits of interest to evolutionary behavioral scientists are highly polygenic—indeed, this is a key feature of what I called the default genetic architecture. In a given population at a given time, a trait's polygenicity is the number of loci where functional variation exists and contributes to the trait. Note that the *potential* polygenicity of the same trait is the number of loci in the genome where, *if* a mutation occurred, there would be an effect on that trait; the potential polygenicity of a trait can be orders of magnitude larger than its actual realization in a population.

Classical population genetic models focus on changes in allele frequencies at one or a few loci, and assume that patterns of selection at the target loci do not depend on what happens elsewhere (i.e., in the genetic background). These models predict that adaptive sequence evolution should occur mainly through *selective sweeps*, in which beneficial mutations rapidly fixate in the population, leaving clear and detectable signatures or "footprints" in their vicinity as they rise in frequency. Highly polygenic traits dramatically change the dynamics and predictions of classical models (Barghi, Hermisson, & Schlötterer, 2020; Höllinger, Pennings, & Hermisson, 2019; Láruson, Yeaman, & Lotterhos, 2020; Messer et al., 2016), for two orders of reasons that I now discuss in turn.

3.1. Soft sweeps and polygenic adaptation

To begin with, highly polygenic traits contain a large reservoir of standing genetic variation; this variation can be recruited during selection episodes, yielding "soft sweeps" that do not originate from a single mutation and leave weaker, more diffuse footprints. Moreover, various selection patterns (including balancing selection) may give rise to multiple partial sweeps in which allele frequencies begin to rise, but stop before reaching fixation. In general, high polygenicity means that both the strength of selection and the evolutionary response at each locus tend to be diluted toward neutrality (with the important exception of large-effect deleterious mutations, which are still subject to strong purifying selection). In the limit (when polygenicity is extremely high; Höllinger et al., 2019), adaptive changes in the trait may not involve sweeps at all, but proceed by subtle frequency shifts across many different loci at the same time. The alleles involved may span all starting frequencies, from common to relatively rare. At the level of a single locus, this kind of adaptive response from standing variation (called *polygenic adaptation* or *adaptive tracking*) is virtually indistinguishable from random drift, and can be extremely difficult to detect without genomic data that span multiple generations (Barghi et al., 2020; Messer et al., 2016; Yeaman, 2015).

Depending on the polygenicity of the trait and the nature and intensity of selection, soft and partial sweeps can coexist with polygenic adaptation by small collective shifts (Höllinger et al., 2019). Indeed, polygenic adaptation does not exclude the occurrence of some classical "hard sweeps;" this is especially likely to happen in the early phase after a sudden change in the selection regime (see Barghi et al., 2020; Hayward & Sella, 2022).

The clearest and most striking demonstrations of rapid polygenic adaptation come from genomic studies of insects (such as *Drosophila*), which allow researchers to track environmental shifts, patterns of selection, and changes in allele frequency with high resolution across many successive generations (e.g., Barghi et al., 2019; Bitter et al., 2024; Pfenninger & Foucault, 2022). Notably, some of these studies involve fluctuating selection over time; the systematic back-and-forth of allele frequencies produced by fluctuating selection can be clearly detected in high-resolution time series, but would likely be mistaken for drift in data based on a single snapshot of the population (as in typical human genomic samples) or with a coarse temporal resolution (see Bitter et al.,

[3] This is one reason why, *contra* Zietsch (2024, Footnote 1), evidence of negative selection on common schizophrenia-linked alleles does not falsify the sexual selection hypothesis. The other reason is that positive schizotypy may have been more beneficial ancestrally, with recent selection disfavoring it (on average) over the last 10,000 years or so (e.g., Akbari et al., 2024)—possibly while autistic-like traits were becoming more advantageous after the transition to agriculture (see Del Giudice, 2018). Because some of the variants that increase the risk for autism decrease that of psychosis and vice versa, in a diametrical fashion (see Chen, Li, Lv, & Yue, 2024; Crespi, 2019; Crespi, Stead, & Elliot, 2010; Del Giudice, 2018), recent positive selection on (a subset of) autism-linked variants would imply negative selection on (a subset of) psychosis-linked variants, and vice versa.

2024; Pfenninger & Foucault, 2022).

3.2. Genetic redundancy

Another crucial feature of highly polygenic traits is that the same phenotype (e.g., the same body size) can be produced by many different combinations of alleles residing at many different combinations of loci—a phenomenon known as *genetic redundancy* (Láruson et al., 2020).[4] When redundancy is high, two individuals may reach the same fitness through largely or entirely different genotypes, which become effectively interchangeable. As a result, the response to selection is unlikely to involve the same loci and variants throughout the population. Instead, different sub-populations or even lineages will see the involvement of partly different loci (largely owing to stochastic processes of mutation and drift); moreover, both the strength and the direction of selection on a given allele may depend on the genetic background (i.e., the frequencies of alleles at other loci) specific to that lineage. While the overall response to selection can be effective and potentially very rapid, the genetic basis of adaptation in the population as a whole becomes heterogeneous and transient: no locus makes an important contribution for very long, and the divergent patterns of selection experienced by the same allele on different genetic backgrounds may easily become blurred or muted at the level of the whole population (Barghi et al., 2020; Láruson et al., 2020; Messer et al., 2016). Empirical studies of polygenic adaptation in which the process is repeated in multiple sub-populations find that the response to selection can be quite heterogeneous across replicates, so that the same alleles may contribute in one sub-population but remain irrelevant in another (e.g., Barghi et al., 2019).

3.3. Implications

Sequence evolution in highly polygenic traits diverges dramatically from the predictions of classical models. When polygenic adaptation and redundancy dominate, patterns of positive and balancing selection get diluted and refracted in ways that mask the expected signatures, effectively "covering their tracks" at the level of individual loci. Note that the situation is not exactly the same with respect to purifying selection, since large-effect deleterious mutations are still expected to arise and be quickly eliminated, leaving clear and detectable footprints in the genome. The processes reviewed in this section contribute to further decouple the evolutionary history of individual loci from that of the trait. Most relevant to this paper, a trait can go through a history of long-term variable selection (e.g., NFDS or fluctuating selection) without accumulating detectable signatures of balancing selection at the level of specific loci (Fijarczyk & Babik, 2015; Messer et al., 2016; more on this below), but still show evidence of pervasive purifying selection.

As I noted earlier, adaptation at the sequence level in highly polygenic traits often proceeds via transient, short-lived episodes whereby no locus contributes for a long time. The implications of this dynamics are easy to misconstrue. Notably, Zietsch (2024) criticized an earlier paper of mine (Del Giudice, 2020) for arguing that, in his words, "the transience of episodes of balancing selection in human traits probably explains the lack of evidence of its having shaped genetic variation" (p. 8). He countered:

> "But it is long-term balancing selection that Wright, Bolstad, Araya-Ajoy, and Dingemanse (2019) proposed could align genetic variation in traits along a fast-slow dimension. It does not make sense to claim that *balancing selection is too transient to have left any trace in the genetic variation* of traits but also claim that it has shaped the covariation among traits" (p. 8; emphasis mine).

However, the contradiction is only apparent; this critique rests on a conflation between the trait and sequence levels of analysis, underscored by the ambiguous use of the term "balancing selection" to refer to both. In the cited paper, Wright et al. (2019) hypothesized that life history traits evolve under long-term fluctuating selection (trait level) driven by changes in population density. It is perfectly possible for such a history of variable selection to leave few or no detectable traces of balancing selection at individual loci (sequence level), if they only undergo transient selection episodes and subtle shifts in allele frequencies.

Crucially, variable selection can maintain genetic variation in the trait, even if specific polymorphisms are not maintained indefinitely as in classical balancing selection. We don't live in an equilibrium world; genetic variation that is maintained for a relatively small number of generations (but still longer than expected under neutrality) is just as consequential for the trait *at that point in time* as it would be if it were maintained into the indefinite future (Bitarello et al., 2023). The fact that different loci contribute at different points in time is theoretically important, but immaterial from the standpoint of the trait as a whole.

A final issue to consider in this section is how to think about comparisons between different sources of genetic variation. Given that mutation-selection-drift dynamics are so pervasive, one might ask how much of a trait's genetic variation has been maintained by balancing selection and what is the relative contribution of different evolutionary processes. Estimating the contribution of different processes from empirical data is a complex task, which requires many indirect inferences and triangulation among multiple sources of evidence. (For a detailed illustration, see how Charlesworth, 2015 estimated the genetic variation in fitness due to mutation-selection-drift in *Drosophila*). An alternative approach would be to run forward simulations of a trait's evolution under various relevant scenarios, and obtain hypothetical estimates by comparing the amounts of genetic variation maintained in different conditions.

Whatever approach is used, one tempting mistake to avoid is to compare effects based on the proportion of genetic *variance* they explain. It is vital to realize that this kind of comparison can be quite misleading. Variance is expressed in *squared* units of the phenotype (e.g., square inches or square centimeters for height): this is why it can be conveniently partitioned into components, but also why it does not represent the real-world effects of different factors, which should be measured on the same units as the trait (see e.g., Funder, 2019; Hunter & Schmidt, 2014; Rosenthal & Rubin, 1979). As a result, comparing effects based on how much variance they explain tends to exaggerate the differences among them—often by a large margin. For a realistic comparison, one has to look *not* at the ratio between variances, but at the square root of that ratio (for details and examples see Del Giudice, 2021a). To illustrate: imagine that, for a certain trait, it could be determined that balancing selection explains one fourth of the genetic variance explained by mutation-selection-drift. In real-world units, balancing selection would be *half* as influential as purifying selection. If balancing selection explained only one tenth of the variance explained by mutation-selection-drift, it would be about *one third* as influential in real-world units. Due to the field's reliance on variance components, misinterpretations of the relative importance of effects are rampant in behavior genetics (see Del Giudice, 2021a), so one should be on the lookout for this kind of distortion.

4. The problem with tests of balancing selection

As I noted earlier, some researchers have probed the evolutionary history of complex traits with tests designed to detect balancing selection at individual loci. Most notably, Abraham et al. (2022) applied one such test (the beta statistic; Siewert & Voight, 2020) to sets of single-

[4] Láruson et al. (2020) introduce a distinction between *genotypic redundancy* (based on all possible redundant genotypes that *could* arise by mutation) and *segregating redundancy* (based on the redundant genotypes that are actually present in a given population at a given time). This distinction closely parallels the distinction between the potential and realized polygenicity of a trait.

nucleotide polymorphisms (SNPs) associated with an extensive collection of morphological, developmental, and psychological/psychiatric traits. A large majority of the traits failed to show enrichment for balancing selection; in fact, about half of them showed *negative* enrichment (i.e., the average value of the statistic was lower than expected, based on a set of matched genetic variants). Zietsch (2024) used this result to reinforce the conclusion that variable selection has been unimportant, and that heritable differences are generally non- or maladaptive. However, targeted tests of balancing selection suffer from serious shortcomings that limit their usefulness even in the face of classical balancing selection, and may render them almost uninformative when dealing with highly polygenic traits. Since these shortcomings are not widely understood and recognized (e.g., they were not even mentioned in Zietsch, 2024 or Abraham et al., 2022), I briefly discuss them here.

The main limitations of tests such as the beta statistic (and several others reviewed in Bitarello et al., 2023) have to do with time. Most of these tests look for the expected signatures of classic, long-term balancing selection, such as excess nucleotide diversity around the target locus or an excess of intermediate-frequency alleles in linked loci. The problem is that these signatures require a *very* long time to consolidate—specifically, more than $4N_e$ generations (where N_e is the effective population size. In our species, a generation is about 25 years and $N_e \approx 10{,}000$ over evolutionary time; hence, on a human timescale, $4N_e$ corresponds to roughly one million years (Bitarello et al., 2023). This means that the same alleles have to be maintained in a balanced polymorphism for more than a million years (i.e., longer than the existence of *Homo sapiens* as a species) before they can be detected with any reliability.[5] In fact, a recent simulation study found that a test of long-term balancing selection similar to the beta statistic started to become effective when polymorphisms were $25N_e$ generations old, corresponding to about *six* million years on the human timescale (Soni & Jensen, 2024). It is no wonder that the most robust findings of balancing selection in our species concern immune-linked genes such as those in the human leukocyte antigen (HLA) complex, which contain ancient polymorphisms that go back millions of years and are partly shared with other primates (Leffler et al., 2013; Teixeira et al., 2015).[6]

The main alternative is to employ tests for *recent* balancing selection, which look for patterns of extended linkage disequilibrium around the target locus. However, these patterns are not unique to balancing selection, and are virtually identical to the footprints left by partial sweeps under positive selection (Bitarello et al., 2023; Fijarczyk & Babik, 2015). In simulations, a linkage-based test started to become effective when polymorphisms were younger than 25,000 years; however, distinguishing putative instances of balancing selection from partial sweeps remained challenging (Soni & Jensen, 2024). While researchers are trying out alternative approaches (for example based on neural networks; Isildak, Stella, & Fumagalli, 2021), their empirical success is yet to be demonstrated, and many challenges remain (see Bitarello et al., 2023).

All these results assume classical balancing selection, in which polymorphisms are maintained indefinitely in a balanced equilibrium. But, as discussed in the previous section, balancing selection in highly polygenic traits is more likely to proceed via transient episodes on a shifting, heterogeneous genetic background. In this scenario, polymorphisms are maintained only for a relatively brief time (so that long-term balancing selection does not take place), possibly only in some lineages or sub-populations, and may undergo small frequency changes that are hardly distinguishable from drift. The resulting process does not leave any of the expected signatures of balancing selection (Fijarczyk & Babik, 2015), and is likely to remain more or less undetectable (at least without high-resolution genomic and environmental data collected across multiple generations). In short, when one is dealing with balancing selection in complex traits, absence of evidence is emphatically *not* the same as evidence of absence.

But this is not all. Although classical models (with heterozygote advantage as the prototypical case) predict an excess of intermediate-frequency alleles over the long run, it turns out that not all kinds of balancing selection share this outcome. For example, NFDS with a rare-allele advantage produces an excess of low-frequency variants instead (Bitarello et al., 2023). Most notably, recurrent fluctuating selection tends to produce a pattern that is the exact *opposite* of the standard prediction—namely, a U-shaped frequency spectrum with an excess of both high- and low-frequency alleles at the expense of intermediate frequencies (Huerta-Sanchez, Durrett, & Bustamante, 2008). Moreover, recent simulations show that fluctuating selection at a locus tends to increase nucleotide diversity close to the locus (as expected), but also *reduce* diversity further away, including unlinked regions on other chromosomes. In fact, the genome-wide reduction in diversity outweighs the local increases; in highly polygenic traits, the overall loss of diversity can be substantial even when local allelic fluctuations are small and difficult to detect (Wittmann, Mousset, & Hermisson, 2023; see also Johnson et al., 2023). These effects underscore the shortcomings of standard models and the tests based on them, and may explain why many complex traits show negative enrichment for the "classical" signatures of balancing selection (Abraham et al., 2022).

5. Conclusion

Figuring out the evolution of individual differences is one of the biggest challenges in the evolutionary behavioral sciences. Adaptive hypotheses are relatively easy to generate but hard to substantiate; ultimately, they need to properly account for both the genetic and the phenotypic data. At the same time, the interpretation of genetic results is far from straightforward, and the data are often consistent with multiple evolutionary scenarios. Because of the intricate relations between selection at the trait and sequence levels, the diffuse nature of polygenic adaptation, and the heterogeneity introduced by genetic redundancy, even widely different selection histories are not expected to yield dramatically different genetic architectures in complex traits. Instead, complex traits under selection (i.e., not neutral) tend to converge on the qualitative features that describe what I have called the default genetic architecture: extreme polygenicity with contributions from both common and rare variants, with large-effect variants that tend to be rarer and younger than small-effect ones. These features are shaped by the pervasive, relentless action of purifying selection, largely irrespective of the trait's evolutionary history and the adaptive function (or lack thereof) of individual differences. Observing the default architecture tells us that a trait has not been neutral to selection, but further inferences about the trait's evolutionary history and the adaptiveness of individual differences are not warranted without additional evidence.

While heritable individual differences can be adaptive even in constant environments, hypotheses involving variable selection have rightly attracted a lot of attention (both positive and negative). Ecological and genetic data from other species suggest that variable selection is pervasive, and that balancing selection (even if relatively short-lived at the level of individual variants) can make substantial contributions to genetic variation. At the same time, the genomic traces of variable selection can be difficult or even impossible to detect absent high-resolution, multi-generational data. In particular, commonly used tests of balancing selection suffer from severe limitations even in the classical scenario, and become virtually uninformative for highly redundant traits that evolve mainly by soft/partial sweeps and polygenic adaptation.

[5] Abraham et al. (2022, Table 1) describe the timescale of the beta statistic as "> 10,000 of years," which is a considerable understatement.

[6] Also note that HLA genes do not encode highly polygenic quantitative traits, but discrete combinatorial phenotypes based on a small number of hypervariable loci.

5.1. Other sources of genetic information

While "default" features such as high polygenicity and a negative association between MAF and effect size are of limited inferential value, it is possible that other aspects of a trait's genetic architecture may provide useful information, and help differentiate (at least in part) between different shapes of the past fitness function. For example, the distribution of large-effect variants between trait-increasing and trait-decreasing effect sizes (Koch et al., 2024) and their disproportionate representation at one or both tails of the trait distribution (Souaiaia et al., 2024) have been used to differentiate between directional and stabilizing selection (interestingly enough, the observed pattern for schizophrenia in Koch et al., 2024 was consistent with stabilizing selection, as with the majority of the traits examined in the study). However, the inferences afforded by these methods are relatively crude and low-resolution, and (among other things) do not speak to a trait's history of constant vs. variable selection.

A more promising avenue to look for evidence of variable selection in humans is the use of ancient DNA to track changes in allele frequencies over time. For example, Akbari et al. (2024) found evidence of directional selection trends across the last ~10,000 years for a number of traits (including selection for higher levels of intelligence, and lower risk of schizophrenia and bipolar disorder).[7] In many instances, there was evidence of changes and even reversals in selection coefficients over time, and even the trajectories of polygenic scores for specific traits (with trends smoothed on a timescale of centuries) suggested the presence of marked fluctuations superimposed on longer-term directional trends. Indeed, the authors explicitly noted that selection is unlikely to have been constant throughout the time period investigated. They also stressed that directional polygenic selection over time is compatible with broad-band signals of stabilizing selection, as found for example by Koch et al. (2024); this is because even strong directional selection determines only a small fraction of the total changes in allele frequency observed across the genome (while yielding substantial shifts in the genetic average of the trait).

5.2. What is the right null model? And should we have one?

Even if the default genetic architecture per se is of limited inferential value, one might argue that the kind of scenario in which heritable individual differences are maladaptive, with traits under constant directional selection or stabilizing selection with a single optimum, is the simplest one and should be used as a "null model" in absence of compelling disconfirming evidence. Note that this would be a change in perspective relative to Zietsch (2024), who instead argued that the genomic data (i.e., the default architecture plus negative tests of balancing selection) are downright *inconsistent* with adaptive heritable differences. Still, treating the non-adaptive scenario as the null to be overcome—and, in practice, accepted as true until proven wrong—would place the burden of (dis)proof on adaptive explanations of individual differences. This approach resonates with deep-seated inclinations in population genetics toward mathematically simple, non-adaptive explanations (see e.g., Hahn, 2008; Maynard Smith, 1978; Messer et al., 2016; Pigliucci, 2008; Sarkar, 2015).

Why this approach can be attractive is easy to understand: in general, researchers try to wield Occam's razor to prevent the proliferation of needlessly complex, ad-hoc hypotheses lacking adequate evidence (aka "just-so stories"). To be absolutely clear: while the default genetic architecture should not lead one to automatically *reject* adaptive hypotheses, it also offers no reason to *favor* them. Any specific evolutionary hypothesis must be evaluated on its own merits, preferably based on the convergence between multiple sources of evidence (Schmitt & Pilcher, 2004). Also, hypotheses with more "moving parts" are likely to demand more interlocking pieces of evidence, which may make them harder to test conclusively.

At the same time, the idea of a "null model" is easy to misapply, giving an unjustified advantage to the model or hypothesis perceived as simpler and more economical (see Bausman, 2018; Bausman & Halina, 2018). Simplicity is one scientific virtue among many, and does not trump all the others (see Bausman & Halina, 2018); the fact that a model is simpler according to a certain criterion (e.g., fewer free parameters) does not automatically make it more credible or likely a priori (e.g., Del Giudice, 2021b). Indeed, a given process can be both mathematically simple *and* very unlikely to occur in the real world. Nature does not always follow the simplest and most straightforward path. Consider this: if empirical evidence were to show that variable selection is the rule rather than the exception in natural populations, would it make sense to keep treating constant selection as "true until proven wrong" in any particular case? At what point would it become reasonable to switch to variable selection as the mathematically more complex, but empirically more plausible "null?"

Both Pigliucci (2008) and Hahn (2008) made analogous points concerning the role of selection in population genetics; they also suggested that the asymmetric logic of the null hypothesis should be replaced with a Bayesian approach, in which alternative hypotheses are placed on equal footing and evaluated based on their prior plausibility and supporting evidence. Bausman & Halina, 2018 presented a more detailed argument in the same direction, cautioning researchers against the uncritical adoption of "pseudo-nulls" that are regarded as best explanations because of their simplicity, but without the formal justifications enjoyed by null hypotheses in the domain of statistical testing. Clearly, this is an intricate philosophical and methodological issue that lies beyond the scope of this paper. But we need to be mindful that there *is* an issue here; while a preference for simpler models may be justified (at least in some contexts), it is something that demands justification rather than the self-evidently correct approach to follow.

5.3. Final thoughts

Reports of the death of adaptive individual differences have been greatly exaggerated. However, blanket falsification is not the only way in which genomics can contribute to progress in the evolutionary behavioral sciences. I fully believe that our field will be transformed by the interaction and integration with genetics, and find the prospect very exciting; I also believe that effective integration should go both ways. For example, behavioral scientists will need to better appreciate the pervasive role of maladaptation and mutation-selection-drift dynamics, and learn how to clearly specify the (often non-trivial) genetic implications of their trait-level hypotheses. At the same time, geneticists will have to triangulate their findings with a broader array of non-genomic data, and become more familiar with the hypotheses formulated by behavioral scientists—which can be fairly sophisticated and may not reduce to simple statements about purifying selection, balancing selection, or other sequence-level processes. In fact, a clearer distinction between levels of analysis, and a more nuanced understanding of their relations, are going to be essential preconditions for a successful dialogue between these disciplines. From a methodological standpoint, simulations could be the ideal tool to bridge the gaps between levels and clarify the implications of alternative assumptions and evolutionary scenarios. The interface between evolution and genetics is where the next discoveries are waiting to be made; if controversy and even confrontation end up attracting more researchers to these fascinating

[7] The new findings on schizophrenia reported in Koch et al. (2024) and Akbari et al. (2024) are fascinating but must be interpreted with care, because neither study attempted to distinguish between different functional categories of variants (e.g., variants that contribute to positive schizotypy vs. variants that contribute to reduced cognitive ability). As I noted earlier, different loci that contribute to overall "schizophrenia risk" may have widely different functional roles, and hence widely divergent selection histories (see Del Giudice, 2017, 2018).

topics, the outcome can only be highly adaptive for all parties involved.

CRediT authorship contribution statement

Marco Del Giudice: Writing – review & editing, Writing – original draft, Conceptualization.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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