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SPECIAL ISSUE

Nature vs. nurture is nonsense: On the necessity of an integrated genetic, social, developmental, and personality psychology

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The field of behavioural genetics unambiguously demonstrates that heritable individual differences exist and are important in explaining human behaviour. Despite this, some psychological perspectives ignore this research. If we wish to comprehensively understand the impact of parenting, the environment, or any social factor, however, we must engage with genetics. In this article, I review research that reveals that genes affect not only our personalities, but the way that we understand and react to the social world. Studies further reveal that notable life events are in part explained by genetic variance. I detail how this could be the case through active, evocative, and passive genetic correlations, and go on to argue that all complex psychological traits are likely the result of multifaceted gene by environment interactions. A mistaken belief that genetic influence implies genetic essentialism, and is therefore tantamount to prejudice, is raised as possible reason why heritability is often ignored in the social sciences. The article concludes with practical suggestions for how we can embrace behavioural genetics as our methods struggle to match the divine complexity of human existence.

KEYWORDS
attitudes—beliefs and values, behavior genetics, biological correlates, development, parent–child interactions, social psychology

1 | INTRODUCTION

> "…all traits are the same: they all require an interaction of genes and environment to develop, and that's that." (Turkheimer, 2011, p. 826)

In the present article, I make the case that any psychological perspective that ignores heritable individual differences ignores: (a) reality, (b) an exciting opportunity for vital scientific progress into understanding people, and (c) the beauty and intricacy of human diversity. As a classically trained social psychologist, my passion and background centre on the power of the situation to influence people, and the way in which our social world shapes us as individuals. This approach is important, but often overlooks or underplays the role that heritable individual differences might play in determining human behaviour; but it should not.

I am not the first person to make this case. Hans Eysenck (1980) made the same point when he stated: "(t)here is little doubt that the major need at the moment is for an integration of social and biological factors, and a study of their joint effects on human conduct" (p. 125). Almost 20 years on the need for, and lack of, integration, was still evident. In 1999, Peter Singer asked why we ignore Darwinian principles and heredity in the social sciences, and issued an urgent recommendation that this exclusion be corrected. Twenty years have passed again, and we have over half a century of highly replicated and consistent twin studies (refer to Plomin, DeFries, Knopik, & Neiderhiser, 2016, for a review), but heritable individual differences are still overlooked in much of psychology.

With the mapping of the human genome completed in the last 15 years, and subsequent genome wide association studies, it seems likely that blindness to the impact of

genetic influences on human psychology will soon no longer be defensible or even feasible. We are each bundles of genes, reacting and changing in response to the environment, and the sooner that we accept this and move beyond the tired nature vs. nurture debate into a fully integrated biopsychosocial understanding of people, the sooner we can move towards truth. In the present article, I aim to provide an accessible review of relevant behavioural genetics studies for those in psychology who have not fully considered the role that genes might play in explaining that which they research. As will be seen in this review, many of the phenomena that we understand as socially determined are tied to heritable factors. I will argue that reluctance to engage with genetic research in the social sciences is in part due to its association with prejudice. Finally, suggestions will be made for how we might better incorporate genetic research into psychological research in the future.

WHAT IS ALREADY KNOWN ABOUT THIS TOPIC

- Social factors shape the way that we think, feel, and behave
- Heritable individual differences exist, and are important in shaping human experience
- The study of environmental influence on human behavior is often divorced from behavioural genetic research, and vice versa

WHAT THIS TOPIC ADDS

- A concise review of research showing that many factors that we understand as "environmental" are partially shaped by heritable factors
- A discussion of how all complex human traits are likely the result of multifaceted gene x environment interactions
- A summary of how we might better incorporate insights from behavioural genetics into psychological science without increasing genetic essentialism.

2 | THE FIRST LAW OF BEHAVIOURAL GENETICS

> "The nature-nurture debate is over. The bottom line is that everything is heritable." (Turkheimer, 2000, p. 160).

Heritability refers to the proportion of population variation in a trait that can be accounted for by genetic variation among individuals. The classical twin study is perhaps the prototype of a great natural experiment, allowing us to estimate heritability of human traits. Identical or monozygotic (MZ; from a single zygote) twins share, on average, 100% of their genetic material. On the other hand, nonidentical or dizygotic (DZ; from two zygotes) twins share approximately 50% of their genetic material. Consequently, if a trait is completely heritable, we would expect MZ twins to correspond perfectly (i.e., have a correlation of 1.0), whereas DZ twins would have a correspondence of half the size (i.e., a correlation of 0.5). Like MZ twin pairs, DZ twin pairs share 100% of their shared environment (e.g., the nation, state, and suburb that they live in, socioeconomic status, and family environment). If this shared environment is 100% responsible for any trait, then we would expect both pairs of twins to show perfect concordance in that trait. Finally, there is a nonshared environment. These are the experiences that happen to one twin but not another. For example, one twin (but not another) may break a leg or have a disastrous love affair. In addition, individual twins will be treated differently by teachers, peers, parents, and so forth. Were nonshared or unique experiences fully responsible for variation in any trait, then we would not expect any concordance between either MZ twins or DZ twins (i.e., each twin pair's scores on a particular trait would correlate at 0). Put very simply, a genetic effect is identified when there is a larger correlation in a trait between monozygotic twins compared to dizygotic twins. An effect of shared environment is found when both MZ and DZ twins have similarly strong correlations. The effect of unique experience is indicated through the degree of nonconcordance (lack of similarity) in both MZ and DZ twin pairs.[1]

Almost 30 years ago, Turkheimer and Gottesman (1991) laid out what they describe as the first, and primary law of behavioural genetics (see also Turkheimer, 2000): that *all* human behavioural traits are heritable. This is a bold statement that warrants interrogation. A good place to start is with a meta-analysis conducted by Polderman et al. (2015). They set out to look at the heritability of human traits as estimated through 50 years of twin studies, analysing data from 2,748 publications, examining 17,804 traits, and drawing on data from 14,558,903 twin pairs. Traits examined included those that we traditionally think of as "biological"; including height, eyesight, hearing, and cardiovascular function. Critically, the authors also investigated traits that we might typically think of as "psychological"; including personality, temperament, and conduct problems. On average, around 50% of the population variance in any given trait was accounted for by genetic factors, and the remainder by environment (primarily the nonshared environment, reflecting unique experiences) and error.

Polderman et al. (2015) identified 1,774 analyses of personality traits suitable for inclusion in their meta-analysis (refer to supplementary material in Polderman et al., 2015). Estimates for temperament and personality closely reflected the overall result: approximately 50% of the variance in measured traits could be accounted for by genetic factors, and the remainder by nonshared environment (i.e., unique

experiences) and error (see also Vukasović & Bratko, 2015). To give the context to the size of the effect, personality is estimated to be similarly heritable to sexual orientation, and only slightly less heritable than weight (Turkheimer, 2011).[2]

However, Turkheimer (Turkheimer, 2000; Turkheimer & Gottesman, 1996) did not just state that basic personality traits (in the narrow sense of Big Five traits) were heritable—he claimed that *all* traits are heritable, including many that fall within a broader, more encompassing understanding of personality. In line with this proposition, political conservatism, social dominance orientation, right-wing authoritarianism, and a tendency to feel and express prejudice to outgroups have all been shown to be heritable (for an overview see Barlow, Sherlock, & Zietsch, 2017). One study found that 47% of the variance in religious leisure time interests (e.g., interest in attending religious services) and 41% of the variance in religious occupational interests (e.g., interest in being a missionary, priest or rabbi) were heritable (Waller, Kojetin, Bouchard Jr, Lykken, & Tellegen, 1990). Attitudes towards pyjama parties are heritable, as are attitudes towards nudist camps, censorship, computerised music, and wearing conventional clothes (Martin et al., 1986). Even a tendency to fill out the very surveys we rely on as researchers is strongly heritable (Littvay, Popa, & Fazekas, 2013; Thompson, Zhang, & Arvey, 2011). The research supports Turkheimer's claim that all human traits are heritable.

3 | THE SHARED ENVIRONMENT

Even though the exact percentages of heritability detailed above should not be overinterpreted (they will change depending on variance in the environment and genotype; see Turkheimer, 2011), a replicated heritability estimate of "0" on a trait would be meaningful—it would indicate that in this particular environment, zero variation in this particular trait could be accounted for by genes. Although few studies of any trait have resulted in a heritability estimate of "0," a large number of studies suggest that the impact of *shared* environment is negligible or nonexistent (see Polderman et al., 2015; Turkheimer, 2000; Turkheimer & Gottesman, 1996; Turkheimer & Waldron, 2000). The shared environment reflects broad commonly experienced variables such as the nation, suburb, and house twins live in, or the school they go to. It also encompasses all the shared aspects of social influence including, for example, exposure to societal norms through the news and social media. Critically, it also incorporates shared experiences in the home environment, including parenting.

From Freud onwards, psychologists have assumed that parents are one of the most critical factors shaping children's psychology. Attachment style is largely understood to be a response to parenting, and in particular, the consistency and availability of the mother (Ainsworth, Blehar, Waters, & Wall, 2015; Bowlby, 2008). Children's attachment style is also theorised to be intimately associated with the health and happiness of their future relationships and lives in general (Ainsworth, 1989; Ainsworth et al., 2015; Bowlby, 2008; Waldinger & Schulz, 2016). While parents are understood to vary in the way that they treat individual children within the same family (Belsky, 1984), there is also an assumption that effective parenting can be quantified, taught, and practiced (Baumrind, 1991; Dinkmeyer & McKay, 1989; and there is some evidence that it can be taught, see Sandler, Schoenfelder, Wolchik, & MacKinnon, 2011). If there are specific parenting (or home, or societal) factors that vary meaningfully between twin pairs, and reliably cause some pairs to be more extraverted than others, for example, or antisocial, or politically conservative, then we would expect the results from twin studies to reveal a substantial portion of variance in that trait to be accounted for by the shared environment.

In sharp contrast to this possibility, in Polderman and colleagues' meta-analysis (Polderman et al., 2015), results were consistent with a model whereby shared environment accounted for *little to none* of the variance in temperament and personality. That is, there does not appear to be any consistent parenting or family or social factor influencing the extent to which people are agreeable, or open to experience, after taking into account their genes. Indeed, multiple genetic studies suggest that the shared environment only weakly accounts for variance in attachment styles when genes are taken into account, if it does at all (Brussoni, Jang, Livesley, & MacBeth, 2000; Fearon, Shmueli-Goetz, Viding, Fonagy, & Plomin, 2014; Picardi, Fagnani, Nisticò, & Stazi, 2011). Despite this, attachment studies rarely acknowledge the possibility of a genetic confound, let alone account for it (see Sherlock & Zietsch, 2018, for commentary).

It is not just attachment that is largely unexplained by shared environment in twin studies—several other psychological traits show the same pattern (refer to Plomin, 1989; Plomin & Daniels, 1987; Plomin et al., 2016; Turkheimer & Waldron, 2000). Turkheimer (2000) posits that the ubiquity of this finding leads to a second law of behavioural genetics: that the effect of being raised in the same family (or same environment) is smaller than the effect of genes. This might surprise most people: Parents themselves see their child-rearing style as vitally important in the development of their children's academic, social, and personality outcomes (Chao, 1996; Himelstein, Graham, & Weiner, 1991; Respler-Herman, Mowder, Yasik, & Shamah, 2012). Furthermore, how we remember our childhood and parents predicts a wide range of life outcomes, from physical and mental health through to incarceration (Bellis, Lowey, Leckenby, Hughes, & Harrison, 2013; Dalton III, Frick-Horbury, & Kitzmann, 2006; Kelley et al., 2005).

The apparent impotence of the shared environment is theoretically, and perhaps personally, challenging. Twins' communal environment is one big, objective chunk of shared nurture, and yet it appears to account for little variance in human psychology. To some, these findings may seem so

implausible that they call twin studies in general into question. A number of developmental psychologists have countered with the argument that parents treat each individual child so differently as to mask any shared effect of parenting (although this is unlikely; see Harris, 1995, 2011). Another option is to take this finding at face value, and conclude that shared environmental influences are simply not important when it comes to how people develop (a relatively popular approach; see Harris, 1995, 2011; Plomin, 1989; Plomin & Bergeman, 1991; Plomin & Daniels, 1987; Plomin & Rende, 1991). In fact, Plomin & Rende (1991; p. 180) have provocatively stated: "(w)hat runs in families is DNA, not experiences shared in the home." From this perspective, you could plop children into any kind of family and have no measurable impact on their eventual outcomes. An alternative, albeit more complex, approach to the problem is to consider objective vs. effective environments (Goldsmith, 1993).

4 | OBJECTIVE VERSUS EFFECTIVE ENVIRONMENTS

Objective environments are shared environments that may be observed by researchers (e.g., having parents that go through a divorce, being born into an affluent suburb, or living in the outback) (Goldsmith, 1993). *Effective* environments are defined by their outcome—what environments lead to (e.g., two different environments can be *effectively* shared if they both result in mental illness). In twin studies, there are many environments that are *objectively* shared by twins or siblings; but these environmental factors' impact on behaviour will only be accounted for as shared variance to the extent that they make twins more like one another (after all, similarity between both MZ and DZ twins is how the impact of the shared environment is estimated in twin studies). If any of these environmental factors has a strong causal influence on a trait for only one twin, or impact twins in opposite directions, all the variance would fall into the non-shared pool. In short, objectively shared experiences will only be quantified as shared in twin studies to the extent that they are *effectively* shared (Goldsmith, 1993; Turkheimer & Waldron, 2000). Given this, it is possible that what happens in the shared environment (e.g., in the home, suburb, school, and nation) may be very meaningful, but its meaningfulness may itself be dependent on how different individuals perceive it.

5 | VARIANCE IN THE ENVIRONMENT CAN BE EXPLAINED BY GENETIC FACTORS

A large body of research suggests that objective environments are less associated with personal outcomes than perceived environments. Indeed, many psychologists have long recognised different people interpret and respond to the same stimuli in different ways (e.g., Allport, 1937, 1961; Bem & Allen, 1974; Funder, 2006; Mischel, 1977). For instance, correlations between parents', childrens', and observers' ratings of parental warmth and negativity are only weakly to moderately correlated (e.g., Feinberg, Neiderhiser, Howe, & Hetherington, 2001). Likewise, perceived and actual social support are often only modestly correlated, and sometimes not at all (Barrera, 1986; Wethington & Kessler, 1986).

The question thus becomes: what makes one person perceive the environment as positive and rich, whereas the next person sees it as negative and barren? Again, an answer is found in genetics (for informative reviews see Kendler & Baker, 2007; Plomin, Reiss, Hetherington, & Howe, 1994; also see varied responses and challenges in commentaries to Plomin et al., 1994). In one study, for example, 800 twin pairs were interviewed via phone about their social support (Kendler, 1997). Twins were asked (at two separate time points, 5 years apart) about 16 indices of social support. These indices loaded on to six factors covering relative and friend problems, relative and friend support, confidants, and social integration. Between 43–75% of variance in stable indices of social support was accounted for by genes. In contrast, shared environment significantly predicted only 2 of the 6 social support measures (20% relative problems and 28% relative support). In this study, twins' shared environment had a much smaller impact on reports of social support relative to genes. Other studies confirm the role that genes play in predicting the extent to which someone reports they have social support from friends, family, and spouses (e.g., Agrawal, Jacobson, Prescott, & Kendler, 2002; Bergeman, Neiderhiser, Pedersen, & Plomin, 2001; Figueredo, Vasquez, Brumbach, & Schneider, 2004; Schnittker, 2008).

Genes may be thought of here as a personalised lens. In line with this idea, attributional style has been shown to be heritable (Lau, Rijsdijk, & Eley, 2006; Zavos, Rijsdijk, Gregory, & Eley, 2010), as has optimism (Schulman, Keith, & Seligman, 1993). To move to a concrete example, people who are more depressed perceive lower social support, with the same genes accounting for variance in both depression and perceived social support (Spotts et al., 2005). Although it is possible that depression drives people away, research also reveals that depressed people see themselves and self-relevant events as more negative than they objectively are (e.g., Gotlib, 1983; Noles, Cash, & Winstead, 1985; Roth & Rehm, 1980). This is a clinical example, but the general principle of genetic perception likely informs an unendingly wide array of predispositions that shape whether we see environmental events as severe, friendly, scary, and so on.

Sometimes it is hard to disentangle the extent to which genes shape perceptions of experiences, or experiences themselves. The patterns reviewed above could reflect genetically driven variance in either perceived or *actual* social

support, or a combination of both. The same is true when we turn to the home environment. In a study of Finnish twins, for example, recollections of maternal abuse, control, responsiveness, and warmth were 25–30% heritable (with estimates slightly lower for fathers' parenting; Harlaar et al., 2008). Identical twins remember being kissed by their parents at a more similar rate than nonidentical twins (Loehlin & Nichols, 1976). Multiple other studies confirm that the same family environments are either objectively different or experienced differently by people in a way that is reliably accounted for (in part) by genes (e.g., Jacobson & Rowe, 1999; Jang, Vernon, Livesley, Stein, & Wolf, 2001; Kendler, 1996; Lichtenstein et al., 2003; Neiderhiser et al., 2004; O'Connor, Hetherington, Reiss, & Plomin, 1995; Plomin et al., 1994; Rende, Slomkowski, Stocker, Fulker, & Plomin, 1992; Rowe, 1983).

This effect of genes on perception of our environment also extends to the school environment. In one study, researchers measured teen twins' "school connectedness." This connectedness was measured via eight items asking students about their general school environment (e.g., whether the school is safe, and teachers treat students fairly) as well as their treatment at school (i.e., whether teachers and friends care about them). The extent to which participants reported school connectedness was highly heritable (45% for girls and 17% for boys) (Jacobson & Rowe, 1999). The bulk of the remainder of the variance was explained by the nonshared environment (i.e., unique experiences and error).

However, it is not just the way that we remember or experience relationships that is heritable. Genes can also play a role in shaping our general life experiences. In one study, 2,315 twin pairs were asked to report on stressful life events that occurred in the past year (Kendler, Neale, Kessler, Heath, & Eaves, 1993). The researchers found that genes accounted for variance in being robbed or assaulted (32.8%) and suffering from financial stress (39%) (Kendler et al., 1993). In another study, 20% of the variance in having experienced an assaultive trauma (e.g., being held captive, beat up, or robbed) was accounted for by genes (Stein, Jang, Taylor, Vernon, & Livesley, 2002). Other studies result in similar estimates of the heritability of life events such as getting fired or made redundant, changing residence, getting in trouble with the law, or experiencing the breakdown of a relationship (e.g., Bemmels, Burt, Legrand, Iacono, & McGue, 2008; Billig, Hershberger, Iacono, & McGue, 1996; Boardman, Alexander, & Stallings, 2011; Distel et al., 2011; Foley, Neale, & Kendler, 1996; Kendler et al., 2010; Middeldorp, Cath, Beem, Willemsen, & Boomsma, 2008; Middeldorp, Cath, Vink, & Boomsma, 2005; Plomin, Lichtenstein, Pedersen, McClearn, & Nesselroade, 1990; Saudino, Pedersen, Lichtenstein, McClearn, & Plomin, 1997; Thapar, Harold, & McGuffin, 1998; Wierzbicki, 1989). Years of education undertaken (Behrman & Taubman, 1989; Schnittker, 2008) and socioeconomic status (Schnittker, 2008) are also heritable.

There is no gene that could directly affect whether someone is robbed, or whether someone gets teased at school; an individual's genes directly affect (well, make up) them, not their environment. Consequently, it is sensible to ask how actual environments might appear heritable. Several answers are found in work looking at gene–environment correlations: the extent to which variance in an environmental factor overlaps with genetic variance in a trait. Three types of possible genetic correlations (active, evocative, and passive) are detailed below.

6 | ACTIVE GENE–ENVIRONMENT CORRELATIONS

People are not passive recipients of the environment. Instead, they can choose to create, seek out, and transform environments as a function of their preferences, personality, interests, or needs (for an overview from a personality perspective, see Furr & Funder, 2018). This is a process often referred to as *niche building* (Caspi, Roberts, & Shiner, 2005; Plomin, DeFries, & Loehlin, 1977; Scarr & McCartney, 1983). From this perspective, sociable people might seek out multiple social connections, or work to deepen existing relationships, and connect with those around them. On the other hand, impulsive people may put themselves in situations that are stimulating and unpredictable; situations that may increase the likelihood of being involved in violence. Indeed, research shows that personality influences what situations people choose to put themselves into (e.g., Diener, Larsen, & Emmons, 1984; Emmons, Diener, & Larsen, 1986; Ickes, Snyder, & Garcia, 1997). Through niche building, then, genes partly "cause" environment through self-selection. Once in the preferred environment, the environment in turn may reinforce the particular trait or tendency that led to its selection (for the interesting possibility that this is particularly the case for highly heritable attitudes, see: Crelia & Tesser, 1996; Tesser & Crelia, 1994; Tesser, Whitaker, Martin, & Ward, 1998).

7 | EVOCATIVE GENE–ENVIRONMENT CORRELATIONS

Another way that genes may affect environments is through individual differences eliciting different kinds of environmental responses (for a personality perspective, see Furr & Funder, 2018). That is, an inherited trait or tendency itself gives rise to a particular environmental response. This contention has been examined within marriages. There is evidence to suggest that the personalities of wives in turn shape the tone of the marriage; genetic variance in wives' personalities predicts both husbands' and wives' evaluations of the

relationship (Spotts et al., 2004, 2005; Spotts, Prescott, & Kendler, 2006). Mothers' negativity and control of children also appear to arise in response to genetic variance in children's behaviour (Neiderhiser et al., 2004). Meta-analysis confirms the idea that parents respond to the genotype of their child (Avinun & Knafo, 2014). None of this should come as a surprise—different people, through their behaviour and demeanour, evoke different behavioural responses from those around them.

8 | PASSIVE GENE–ENVIRONMENT CORRELATIONS

Gene–environment correlations can also occur through an inactive process, such that parents and children share the same genes that predispose to both a parental environment and a child's response. For example, the same genes that predispose to maternal depression may predispose to child depression. In this case, although the environmental factor of having a mother with depression would predict child depression, a twin study would suggest that it is genes shaping depression for both parent and child, separate from any shared experience. This pattern is referred to as passive genetic correlation (Jaffee & Price, 2007; Plomin et al., 1977; Scarr & McCartney, 1983);

9 | TO UNDERSTAND SOCIAL INFLUENCE WE NEED TO UNDERSTAND HERITABILITY (AND VICE VERSA)

To understand the impact of the environment, then we need to take genes into account. We are not carbon copies of one another; as seen above we seek out, shape, and interpret our environments, and respond to them in unique ways. Consequently, in the future, it is likely we will come to recognise that most responses to the environment are influenced by genes. I propose that there is a reason that we often see little impact of shared environment, but it is not that shared environments are unimportant in the development of human personality or behaviour. Rather, I suggest that genes play a vital role in determining whether and how objective environments become effective environments.

Genes only exist within environments, and we are studying genetic effects within particular environments. It is sensible to then question the extent to which any effect we are looking at is in fact a nature/nurture interaction (rather than just nature main effects + nurture main effects).[3] A conceptual gene x environment interaction posits that the effect of a given environment depends on one's genetic makeup, and the effect of a genetic predisposition on any trait or outcome depends on one's environment (see Sauce & Matzel, 2018, for a discussion of this possibility as it relates to IQ). The presence of such interactions also might go some way to resolving the common finding that shared environment accounts for little variance in any complex trait; simulations suggest that the impact of environment will be underestimated in twin studies when there are gene x environment interactions (Turkheimer & Gottesman, 1996).

The idea that the person cannot be separated from the situation, or vice versa, is consistent with Lewin's (1951) classic theorising on the relationship between personality, behaviour, and the environment, and more recently Funder's (2006) *situation construal model* of personality development. It should also be noted that while the study of gene × environment interactions is currently underdeveloped, there have been concerted efforts to look at how different people respond to different environments from personality (e.g., Diener et al., 1984; E. T. Higgins, 1990), organisational (e.g., O'Reilly III, Chatman, & Caldwell, 1991), and social (e.g., Pryor, LaVite, & Stoller, 1993) psychological perspectives.

Gene x environment interactions would similarly lend themselves to empirical examination *if* a) there were any single genetic predictor for any complex human trait, or b) any single environmental predictor for any complex human trait. Instead, however, complex human traits are polygenic, influenced by many (perhaps thousands) of genetic variants. Likewise, such traits are likely polyenvironmental, influenced by many (perhaps thousands) of cumulative and interacting environmental variants. To really look at gene x environment interactions, then we need understand what genes predispose to any given behaviour. At present, however, this task is impossible. Single gene studies routinely fail to replicate, as do single gene x environment interactions (Duncan & Keller, 2011). Even genome wide association studies cannot currently identify the exact combination of genes that might be responsible for any complex human trait.

This complexity and uncertainty might explain, in part, why we as psychologists often ignore heritability, at best keeping it in the back of our minds (if not our articles). Our avoidance is pragmatic—we do not know what to do with the information. This point is important, and I address it further below. Pragmatics aside, however, a more potent objection to genetic research likely lies in its controversial history (see Barlow et al., 2017), and concerns about social consequences of accepting such views (see Barlow et al., 2017; Turkheimer, 2011, for discussion).

10 | GENETIC EXPLANATIONS OF BEHAVIOUR HAVE BEEN LINKED TO PREJUDICE

Psychological essentialism is the idea that surface characteristics of any person or object are seen to be underpinned by unifying "essences"; for example, two entities (e.g., a cat and dog) can have relatively similar surface features

(e.g., ears, tail, paws, fur) but be understood to have fundamentally different "essences" (e.g., cat vs dog essence; Medin & Ortony, 1989). A person or entity's essence is typically understood to be fixed, natural, discrete, homogenous, immutable, and fundamental (Dar-Nimrod & Heine, 2011; Haslam & Whelan, 2008; Medin & Ortony, 1989). Genetic essentialism is the tendency to attribute differences between people and groups solely to their genes, with genes forming the essences of people (Dar-Nimrod & Heine, 2011).

Above I have described work showing that multiple complex human traits *are* in part genetic. It turns out that being exposed to this information itself, however, has a marked environmental impact on how we think, feel, and behave. In their excellent review of the impact of genetic essentialism on social attitudes and behaviour, Dar-Nimrod and Heine (2011) show that reading genetic information can lead to racism, sexism, and pessimism (as is the case for psychological essentialism more generally; Bastian & Haslam, 2008; Haslam, Bastian, Bain, & Kashima, 2006; Haslam & Whelan, 2008).

As a first example, a belief in behavioural genetics (indexed by agreement with statements such as: "I think genetic predispositions have little influence on a person's personality characteristics" (reverse scored) and "I am convinced that very few behavioural traits of humans can be traced back to their genes" (reverse scored)) is related to racial stereotyping, sexism, and prejudice (Bastian & Haslam, 2006; Keller, 2005). To put it another way, denial of a genetic basis for human psychology is associated with *low* levels of racism and sexism, whereas acceptance of the kinds of findings reviewed in this article is associated with increased racism and sexism. Moving specifically to the genetics of race, experimental studies reveal that those exposed to arguments about the biological and genetic basis of race respond with greater ingroup bias (Keller, 2005), racism (Condit, Parrott, Bates, Bevan, & Achter, 2004), and avoidance of other-race friendships and interactions (Williams & Eberhardt, 2008). Similarly, exposure to genetic explanations of gender increase men's and women's acceptance of gender inequality (Morton, Postmes, Haslam, & Hornsey, 2009), and endorsement of restrictive gender stereotypes (Brescoll & LaFrance, 2004). Furthermore, although people with mental disorders are "blamed" less for those disorders when people believe that they are biologically determined, those who take a biological approach to mental disorders are more pessimistic about recovery for the mentally ill, and more fearful and avoidant of them (Haslam & Kvaale, 2015; Kvaale, Haslam, & Gottdiener, 2013).

Two things thus appear true: (a) genes play a substantial role in shaping human behaviour, and (b) people who believe they play a substantial role in shaping differences between groups tend to hold more prejudiced attitudes (but not towards sexual minorities; see Haslam & Levy, 2006), and believe that personal and social change are less possible. These two true things sit uncomfortably together, and I suggest that many of us outside of behavioural genetics simply resolve this discomfort by accepting the latter truth, and rejecting the former. Countering a social wrong (e.g., prejudice) with a lie (e.g., "personality does not have a genetic basis") is attractive in its simplicity. It is a concrete answer to a difficult problem, and it shuts down potential debates that could increase, rather than decrease, prejudice. But there is that pesky problem of the lie—in a world of post truth politicians it is vital that we are not post-truth scientists (K. Higgins, 2016). K. Higgins (2016) states: "Scientists and philosophers should be shocked by the idea of post-truth, and they should speak up when scientific findings are ignored by those in power or treated as mere matters of faith. Scientists must keep reminding society of the importance of the social mission of science—to provide the best information possible as the basis for public policy. And they should publicly affirm the intellectual virtues that they so effectively model: critical thinking, sustained inquiry and revision of beliefs on the basis of evidence."

11 | IMPLICATIONS AND FUTURE DIRECTIONS

Challenging a post-truth world not only involves bringing an evidence base to public policy, but also confronting our own biases, and grappling with difficult and conflicting information. Vitally, we must revise our beliefs (and theories and methods) on the basis of new evidence. Trust in science only works to the extent that we are telling the truth, and in the face of the available evidence we must counter the negative effects of genetic information using truth.

If we take seriously the notion that a right to equal opportunity is predicated on there being negligible genetic differences between people psychologically, then we are effectively acknowledging a goal and defeating it in the same breath. But we do not have to. I would argue that the social goal of providing rich, fulfilling, and supportive environments to all people should not be contingent upon insisting that people are identical. Equal opportunity should be afforded to all, irrespective of the fact that different individuals within the "all" meet that opportunity with varying traits, interests, and inclinations.

Genetic literacy seems a vital first step for overcoming both essentialist biases and reactance to behavioural genetics. For example, high heritability estimates do not mean that something is "fixed" or unaffected by the environment. Heritability estimates at any one point in time depend on both the variability of the environment and genotype (Turkheimer, 2011). Take, for instance, the fact that in the Western world people have been getting taller as nutrition and health care improves, and yet in a snapshot study height is estimated to be mainly heritable and only weakly

environmental (Silventoinen et al., 2003). The high heritability estimate in no way indicates that height is unaffected by the environment. Instead, this estimate simply suggests that within the current environmental constraints, whereby most twins have access to adequate nutrition and healthcare, variation in height is primarily driven by genes, rather than smaller between-environment differences (Turkheimer, 2011). With no variability in a meaningful environment, all variance will appear genetic, and conversely, holding genotype constant, all variance will appear environmental. All this to say that heritability estimates tell us whether something has a genetic component or not within a current environment, and should not be interpreted to infer that something is fixed or not (Turkheimer, 2011; Turkheimer & Waldron, 2000).

Mass media coverage of genetic information typically acknowledges none of this nuance, and instead has often been deterministic (Condit, Ofulue, & Sheedy, 1998), a problem that even extends to biology textbooks (Dos Santos, Joaquim, & El-Hani, 2012). To date, efforts to reduce genetic determinism have yielded mixed results (see Heine, Dar-Nimrod, Cheung, & Proulx, 2017, for a review). Emphasising that genes work together with the environment to shape human outcomes, for example, in some cases appears to slightly reduce deterministic thinking (Walker & Read, 2002), and in others does not (Boysen & Gabreski, 2012). There is evidence, however, that people can respond to news articles about genetics in nonbiased and nondeterministic ways (Condit, 1999). Furthermore, people who have higher levels of education, or have higher levels of genetic knowledge, tend to show weaker genetic essentialist biases (refer to Heine et al., 2017). Consequently, ignoring genetics in our articles and courses may be contributing to the very genetic determinism that we wish to avoid. Conversely, acknowledging ourselves as biological and social creatures may even help with achieving social equality in the long run; we need to know about the biology of human nature to evaluate influential neo-Darwinian ideas and debunk them when they are faulty (Singer, 1999). One simple way to bring psychology and behavioural genetics closer to reconciliation, therefore, might be to arm ourselves with genetic knowledge, and communicate this to our students and readers.

Psychologists who look at social influence and communication are also well placed to deploy their theories to explain how best to convey genetic information. As stated above, simply conveying genetic information while acknowledging the environment does not seem to be enough (Heine et al., 2017). Hornsey and Fielding (2017) argue that people's understanding and acceptance of scientific information depends on their intuitions, wants and needs. Specifically, they propose that people engage in motivated reasoning (Kunda, 1990), and accept information that aligns with their pre-existing beliefs. Hornsey and Fielding (2017) make the case that foundational "attitude roots" underlie rejection or acceptance of scientific information. These are: worldviews, conspiratorial ideation, vested interests, personal identity expression (that may be personality based), social identity needs, and fears and phobias. In their article, they draw on decades of social psychological work on persuasion to suggest that rather than simply bombarding people with information, change in attitudes needs to be created by working with (rather than against) these attitude roots. As it stands, people's genetic essentialist biases appear relatively resistant to change (Heine et al., 2017). It could be, however, that rather than a typical study manipulation, presenting nuanced genetic and environmental information to people over a longer period of time in a way that aligns with their social and personal identity needs (e.g., perhaps by appealing to a person's desire to appear accurate and nonbiased), is effective in getting people to accept genetic information without increasing genetic essentialist driven prejudice.

Another practical thing that we can do is engage with genetic information in our own research. Returning to the pragmatic issue of complexity, if we cannot identify exact alleles (genetic variants) associated with behaviours, we cannot reliably test for gene × environment interactions, and if real life gene × environmental interactions are both polygenic and polyenvironmental, the task of reliably quantifying meaningful explanatory associations is currently difficult. Turkheimer and Waldron (2000) respond to this concern by stating:

"The limitations of our existing social scientific methodologies ought not to provoke us to wish that human behaviour were simpler than we know it to be; instead they should provoke us to search for methodologies that are adequate to the task of understanding the exquisite complexity of human development." (Turkheimer & Waldron, 2000, p. 93)

Adequately engaging with genetic research presents the next big challenge for our field. As our methods struggle to decipher the exquisite complexity referred to by Turkheimer and Waldron (2000), however, psychologists who study environmental and social factors need to take the currently available insights from behaviour genetics seriously. This may involve testing objective environmental predictors against perceived environmental predictors of outcomes, and openly acknowledging that there is robust evidence that such perceptions reflect both the individual and their environment (and neither in isolation). Behaviour genetics also makes us critically think about the tenuous separation between individual differences and social variables, at least as we currently measure them (for an overview of similar discussions in personality psychology, and potential approaches to the problem, see Furr & Funder, 2018). Perceived quality of parenting, social attitudes, and stressful life events are no less genetic than personality, which in turn is no less environmental than many of these classic social and developmental psychological variables. Finally, we may also wish to open communication with behavioural geneticists who may work

with us to test environmental vs. genetic predictors of twins' psychological outcomes, or collaborate with us in the future to test for gene x environment interactions.

12 | CONCLUSION

There are big questions in psychology concerning the extent to which we live lives determined by our biology and environment, connecting to age-old questions about agency and free will (Bandura, 2006, 2009; Baumeister, 2008; Sappington, 1990). These questions cannot be understood without considering genes, and their answers may have practical use, fundamentally changing how we understand the world and interact with it. A psychology that cares about scientific truth is one that must fully engage with behaviour genetics, accept temporary uncertainty about how genes and environment work together to shape all human thought, feeling, and behaviour, and work vigorously to update its priors. Recognising the existence of genetic influences will not dilute the power and potency of social influence, parenting, or the environment, but add nuance to understanding their effects on our behaviour, our experiences, and our personalities.

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NOTES

[1]Twin studies themselves rely on MZ and DZ twins having equally similar shared environments (the *equal environments assumption*). MZ twins, however, typically report that they are treated more similarly by others relative to DZ twins (although this appears to be a response to, rather than precursor of, similarity; Lytton, 1977). They also socialise together more frequently than DZ twin pairs (Kendler & Gardner, 1998). These differences, however, do not account for twin resemblance in depression, anxiety, or panic disorders. Genetic similarity, on the other hand, does (Kendler & Gardner, 1998; see also Kendler, Neale, Kessler, Heath, & Eaves, 1994). Furthermore, some examinations of personality concordance in twins have found magnitude of the correlation between MZ twin pairs raised together is similar to the correlation between MZ twin pairs reared apart (Tellegen et al., 1988)

[2]Relying on *none* of the assumptions of twin studies, work using genome wide complex trait analysis has estimated that common single nucleotide polymorphisms can account for 15.2% of the variance in neuroticism (Realo et al., 2017), 25% of the variance in bipolar disorder (Lee et al., 2013), 31–41% of the variance in working memory (Vogler et al., 2014), 12% of extraversion (Vinkhuyzen et al., 2012), and 8.7% of the variance in major depression (Wray et al., 2018).

[3]Another type of statistical gene x environment interaction has been widely studied, testing whether different traits are more or less heritable in different environments. The interested reader may look to an excellent review on these gene x environment interactions (Rutter, Moffitt, & Caspi, 2006), and as well as recent work from genome wide association studies (Rimfeld et al., 2018).

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