

## Review

## The polygenic and poly-environmental nature of personality

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Although personality differences are supposed to arise from both genetic and environmental sources, neither single genes nor specific environmental factors have yet been identified to robustly explain considerable variance. Merging relevant puzzle pieces from the current state of research, this review provides new insights into genetic and environmental personality differences. First, the nature of personality is apparently polygenic and poly-environmental, with various small, rare, and interdependent genetic and environmental factors. Second, self-ratings on broad trait dimensions as the most common operationalization of personality might mask actual genetic and environmental effects. Third, genetic and environmental factors are also interwoven and interact in multiple, complex, and probably very individual ways. Future studies should address these aspects through multi-perspective approaches and multimodal designs.

### Addresses

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## Introduction

Personality traits are defined as person characteristics that are inferred from relatively stable and consistent patterns of individual behaviors, thoughts, feelings, and motivations [1]. Understanding how and to what extent personality differences are rooted in genetic differences, shaped by individual environments, and evolved from a complex interplay of genetic and environmental factors is one of the fundamental research goals of differential and personality psychology. A comprehensive meta-analysis on almost 18,000 complex human traits from >2500 twin research publications between 1958 and

2012 yielded that genetically identical monozygotic (MZ) twins are generally more similar than dizygotic (DZ) twins [2], suggesting the relevance of genetic factors. More specifically, 568 traits were treated as descriptions of temperament and personality functions. For these traits, the overall *heritability* was  $h^2 = .47$ , which means that 47% of personality differences in human traits are due to genetic variance. The remaining variance was mainly attributable to individual-specific environmental influences not shared by twins, whereas environmental influences shared by twins reared together were rather negligible. Thus, while genetic factors are responsible for both similarities and differences within families, environmental factors primarily contribute to the reinforcement of personality differences within and between families. In other words, family similarity is primarily due to genetics and the environment appears to be the most important driver for individual uniqueness.

Although it is undisputed today that both genetic and environmental differences are responsible for personality variance [3,4], no single gene has yet been identified that could reliably explain at least a small part ( $\geq 1\%$ ) of the considerable heritability found in twin studies [5,6]. Similarly, influences of single life events or other measured environmental factors, such as family constellation, parental behavior, and social interactions, do not account for a substantial portion of environmental personality differences [7\*\*<sup>8</sup>]. Thus, it has yet to be understood how genetic and environmental factors separately, commonly, or interactively contribute to personality differences. In this article, we review recent research providing new insights into relevant puzzle pieces for understanding genetic and environmental sources of personality differences. Key findings from twin research and family studies, molecular genetic studies, multimodal studies and lifespan research are therefore presented, revealing the specifics and the interplay of genetic and environmental factors.

## How do genetic factors contribute to personality differences?

Complex human traits are generally considered *polygenic* [9]: They can be influenced by several hundred genetic variants, including protein-coding and noncoding regulatory regions of the human DNA. Genome-wide association studies (GWAS) can examine hundreds to

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millions of smallest units in the genome that can vary among humans: single nucleotide polymorphisms (SNPs). Earlier GWAS failed to find robust and replicable trait-SNP associations [10–12], but the number of significant associations increased as a function of sample size [5,13,14], although the effect sizes remained very small. This is consistent with a polygenic model of multiple genetic variants, each of which has a small effect. Although effects of specific SNPs on personality traits are small, it is possible to estimate the joint effect of numerous involved SNPs on trait differences between unrelated individuals. This so-called SNP-heritability ranges from 9% to 18% for Big Five personality traits (Table 1).

The gap between the SNP-heritability and the average heritability obtained from twin studies is known as *missing heritability* [15], which may have several reasons. On the one hand, SNP-heritability estimates may represent lower bounds of genetic contributions to personality differences, as they only take into account the additive contributions of SNPs [6,16]. However, the genetic similarity of genetically identical twins can be due to both additive genetic influences ( $a^2$ ) and nonadditive genetic effects due to allelic dominance deviations or multiple gene–gene interactions ( $na^2$ ). The latter can explain why twin studies usually yield higher heritability estimates than non-twin family and adoption studies ( $h_{\text{Twin}}^2 = a^2 + na^2 = .47$  vs.  $h_{\text{Family}}^2 = a^2 = .22$ ), because nonadditive genetic influences cannot contribute to the similarity between parents and their biological offspring or most other relatives [3,4,17\*]. Therefore, a substantial proportion of the genetic contribution to personality differences may be nonadditive. The still lower SNP-heritability obtained from samples of unrelated individuals (on average  $h_{\text{SNP}}^2 = .13$ ) as compared to the heritability obtained from family studies may additionally be due to the fact that SNP-based estimates are typically limited to common genetic variants (frequency  $\geq 1\%$ ), whereas family-based heritability also includes rare genetic variants (frequency  $< 1\%$ ). Some studies reported first hints on rare genetic variants accounting for additional variance in the most frequently studied personality trait Neuroticism (i.e., negative emotionality or emotional instability) [18,19\*]: Rare genetic variants explained about 7% to 19% of the variance (on average  $h_{\text{RARE}}^2 \approx .13$ ), which can explain the gap between  $h_{\text{Family}}^2$  and  $h_{\text{SNP}}^2$  (because  $h_{\text{Family}}^2 = a^2 = h_{\text{SNP}}^2 + h_{\text{RARE}}^2$ ).

On the other hand, heritability estimates obtained from twin studies may be overestimated to the extent that specific underlying assumptions are violated [20]. First, if environmental factors cause genetically identical MZ twins to be more similar than DZ twins (i.e., violation of the equal environment assumption), higher MZ twin correlations compared to those of DZ twins would not

reflect their higher genetic overlap. Studies on twins growing up apart have indeed shown somewhat, but only slightly, lower heritability estimates for personality traits [3,4]. Second, classical twin models do not allow estimates of nonadditive genetic effects in the presence of shared environmental influences, and vice versa. If both are relevant sources of variance, but only one source can be considered in the model, their respective contributions would be underestimated, while the additive genetic variance and thus the heritability would be overestimated. In extended twin family studies (ETFS) that include additional family members, this problem can be overcome to some extent, and some lower heritability estimates for personality traits have indeed been found [21,22,23\*]. Regarding overall heritability estimates, however, deviations from classical twin models were small. For instance, a large-scale ETFS of Neuroticism yielded an overall heritability of 47%, including 27% due to additive genetic factors and 20% due to nonadditive genetic sources [21]. Overall, previous research suggests that around 40–50% of personality differences are genetic, with additive and nonadditive factors explaining an almost equal share (see Figure 1).

## How do environmental factors contribute to personality differences?

Studies of extended twin families, particularly those that include both parents, children and partners of twins, also provide less biased estimates of environmental trait differences [23\*,24]. Such an ETFS [21] found that shared environmental influences explained 13% of Neuroticism variance ( $se^2 = .13$ ) in 18-year-old or younger individuals, but not in older ones. This indicates effects of a shared household when the siblings still live with their parents. A recent ETFS [25\*] yielded equally small but significant environmental contributions to the similarity of twins reared together for both Big Five and HEXACO personality traits, alongside additive and nonadditive genetic factors (see Table 1). These shared environmental influences were primarily twin-specific, suggesting that they reflect age-associated influences that contribute to personality similarity of same-age siblings growing up together. It is not yet clear whether these are common peer influences, common developmental transitions, simply twin-specific interactions, or other shared influences.

Across all the studies and traits examined, environmental influences primarily affect differences between family members and thus individualization ( $nse^2 = .38 - .63$ , see Table 1). Even after correction for random error of measurement, environmental influences not shared by twins or other family members represent the primary environmental source of personality variance (Figure 1) [25–28]. However, it is still unclear what exactly these sources represent. Large-scale studies and meta-analyses of possible

**Table 1**  
**Genetic and environmental contributions to the variance of primarily self-reported personality traits.**

Study	Traits	Design/ method	Rater	N/k	Family correlations			$h^2$	$a^2$	$n^2$	$se^2$	$nse^2$
					MZT	DZT	NTS					
<b>Meta-analyses, population-based, and cross-national studies of twin and family data</b>												
Johnson et al. (2008) [3]	Diverse temperament and personality traits	>90 % twin	>90 % self	N > 660,000	0.45	0.21	0.15	0.45	0.39	0.06	?	0.55
	Neuroticism	>90 % twin	>80 % self	N = 212,710	0.43	0.18	0.15	0.43	0.30	0.13	?	0.57
	Extraversion	>90 % twin	>80 % self	N = 114,112	0.50	0.17	0.19	0.17	0.50	0.18	0.32	?
	Openness	Twin	>80 % self	N = 17,206	0.47	0.23	—	—	0.47	0.44	0.03	?
	Agreeableness	Twin	>80 % self	N = 15,450	0.39	0.20	—	—	0.38	0.38	?	0.01
	Conscientiousness	>95 % twin	>80 % self	N = 17,418	0.46	0.19	0.20	—	0.46	0.30	0.16	?
Polderman et al. (2015) [2]	Temperament and personality functions	Twin	—	N > 1.2 Mio.	0.47	0.23	—	—	0.47	0.44	0.03	0.00
Vukasović & Bratko (2015) [4]	PEN, NPC & FFM/B5	Twin	Self	k = 38	—	—	—	—	0.47	0.22	0.25	—
	PEN, FFM/B5	Family	Self	k = 16	—	—	—	—	0.22	0.22	—	—
Kandler et al. (2021) [35]	Honesty-Humility	Twin	Self	N = 7026	0.37	0.20	—	—	0.34	0.34	?	0.03
	Emotionality	Twin	Self	N = 7026	0.44	0.15	—	—	0.44	0.16	0.28	?
	Extraversion	Twin	Self	N = 7026	0.53	0.22	—	—	0.53	0.36	0.17	?
	Agreeableness	Twin	Self	N = 7026	0.41	0.12	—	—	0.41	0.09	0.32	?
	Conscientiousness	Twin	Self	N = 7026	0.40	0.13	—	—	0.40	0.12	0.28	?
Möttus et al. (2025) [17]	Openness	Twin	Self	N = 7026	0.62	0.30	—	—	0.62	0.58	0.04	?
	Neuroticism	Family	Self	N = 32,004	—	—	0.13	0.14	0.26	0.26	—	?
	Extraversion	Family	Self	N = 32,004	—	—	0.12	0.14	0.26	0.26	—	?
	Openness	Family	Self	N = 32,004	—	—	0.20	0.19	0.40	0.40	—	?
	Agreeableness	Family	Self	N = 32,004	—	—	0.11	0.14	0.26	0.26	—	?
	Conscientiousness	Family	Self	N = 32,004	—	—	0.13	0.14	0.26	0.26	—	?
<b>Extended twin family studies (ETFS)</b>												
Boomstra et al. (2018) [21]	Neuroticism	ETFS	Self	N = 31,152	0.53 <sup>a</sup>	0.26 <sup>a</sup>	0.20	0.19 <sup>a</sup>	0.47	0.27	0.20	0.13 <sup>a</sup>
	FFM/B5	ETFS	Self	N = 1817	0.52	0.20	0.15	0.47	0.27	0.20	0.00	0.53
Kandler et al. (2024) [25]	HEXACO	ETFS	Self	N = 1971	0.53	0.26	—	0.17 <sup>b</sup>	0.39	0.24	0.15	0.46
<b>Meta-analyses of genome-wide association studies (GWAS)</b>												
Lo et al. (2017) [13]	Neuroticism	LDSC	Self	N = 59,206	—	—	—	—	0.12	0.12	?	—
	Extraversion	LDSC	Self	N = 59,226	—	—	—	—	0.18	0.18	?	—
	Openness	LDSC	Self	N = 59,206	—	—	—	—	0.12	0.12	?	—
	Agreeableness	LDSC	Self	N = 59,176	—	—	—	—	0.10	0.10	?	—
	Conscientiousness	LDSC	Self	N = 59,176	—	—	—	—	0.09	0.09	?	—

(continued on next page)

Study	Traits	Design/ method	Rater	N/k	Family correlations			$h^2$	$a^2$	$nse^2$	$nse^2$
					MZT	DZT	NTS				
Schwaba et al. (2025) [14]	Neuroticism	LDSC	Self	N = 1,136,711	—	—	—	0.12	0.12	?	—
	Extraversion	LDSC	Self	N = 662,617	—	—	—	0.16	0.16	?	—
	Openness	LDSC	Self	N = 611,985	—	—	—	0.15	0.15	?	—
	Agreeableness	LDSC	Self	N = 611,037	—	—	—	0.11	0.11	?	—
	Conscientiousness	LDSC	Self	N = 641,167	—	—	—	0.11	0.11	?	—

Note. N/k: number of individuals/studies; MZT: monozygotic twins; DZT: dizygotic twins; NTS: non-twin siblings; P-O: parents-offspring;  $h^2$ : heritability (i.e., the overall genetic contribution to trait differences);  $nse^2$ : variance component due to additive genetic factors;  $nse^2$ : variance component due to nonadditive genetic factors;  $nse^2$ : variance component due to environmental influences shared by twins/within families;  $nse^2$ : variance component due to individually unique environmental influences (not shared within families); PEN: Eysenck's 3-dimensional personality model (Psychoticism, Extraversion, Neuroticism); NPC: Tellegen's 3-dimensional personality model (Negative Emotionality, Positive Emotionality, Constraint); FFM/B5: Five-factor model/Big Five of personality (Neuroticism, Extraversion, Openness, Agreeableness, Conscientiousness); HEXACO: Six-factor personality model (Honesty, Humility, Emotionality, Extraversion, Agreeableness, Conscientiousness); LDSC: linkage disequilibrium score regression method; —: not reported, available, or computable; ?: nonadditive genetic and shared environmental influences are unknown or assumed to be negligible.

a Estimates for twins aged 18 years or younger.

b Estimates for twin-parents and twin-offspring dyads.

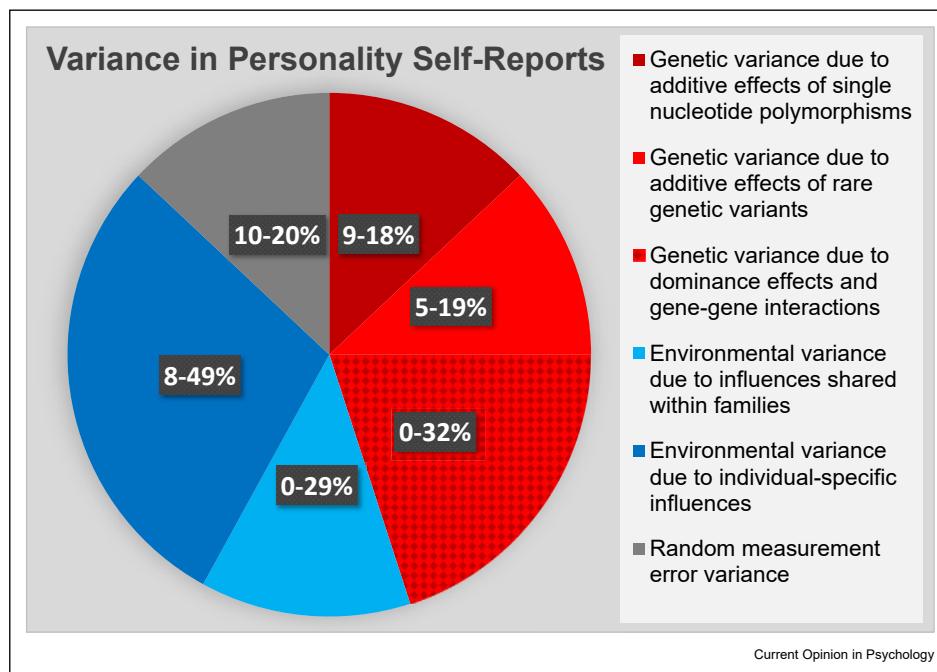
environmental factors, such as non-normative life events (i.e., accidents or lottery wins), partner effects, differential parental treatment, or normative life transitions, did not reveal substantive or replicable effects on personality differences [7\*\*,8,29]. Researchers suppose that personality differences are *poly-environmental*, as a parallelism to them being polygenic [30]. In other words, a single environmental factor may not account for a substantial variance component, whereas aggregates of numerous specific experiences, each with a very small effect size, may explain more [8,31,32].

As with genetic effects, there is no reason to assume that environmental effects are only additive. They may interact in multiple, complex, and very individual ways. The same event could be experienced very differently by different individuals (or different experiences could have the same effect on different people) depending on individual situations, contextual circumstances, or other life experiences. For instance, influences of negative life events on emotionality can be buffered by positive experiences [33,34]. Such additive and interactive effects of multiple individual experiences could act primarily as unsystematic, idiosyncratic, and random individualizing influences [29].

### Do genetic and environmental contributions to personality differences vary across different personality characteristics and measures?

Personality traits are usually measured with self-reports on a limited set of three, five, or six broad descriptive trait dimensions with differing labels (see Table 1). Previous research found little evidence of differences between these personality traits regarding genetic and environmental contributions [3,4,27,35]. By contrast, some studies suggest that important genetic and environmental personality differences occur at the level of more specific facets and nuances which may be obscured by aggregating them into more abstract trait dimensions [36–38]. Moreover, personality characteristics in the broader sense, such as interests, values, goals, self-schemas, attachment and coping styles, tend to show on average a lower heritability and different environmental contributions [25\*,39].

If the way how individuals experience the world and see or evaluate themselves is heritable, it is not surprising that self-assessments themselves are partly heritable. As a consequence, the heritability of personality traits based on self-reports would be overestimated. There are only a few twin and family studies based on data from multiple raters for personality traits [17,40,41]. These studies suggest that family similarities based on informant-ratings are lower compared to self-reports (Table 2), which indicates genetic influences that contribute specifically to self-ratings. Latent trait scores indicated by self-reports and reports from independent

**Figure 1**

Current summarizing knowledge from the findings of Vukasović & Bratko (2015), Lo et al. (2017), Boomsma et al. (2018), Hill et al. (2018), Kandler et al. (2024), Wu et al. (2024), and Schwaba et al. (2025) (see also [Table 1](#)).

**Table 2**

**Genetic and environmental contributions to the variance of personality traits based on different measures.**

Study	Traits	Design/ method	Measure	N individuals	Familial similarity				$h^2$	$a^2$	$na^2$	$se^2$	$nse^2$
					MZT	DZT	NTS	P-O					
Riemann et al.(1997) [41]	FFM/B5	Twin	Self-report	1928	0.52	0.23	—	—	0.52	0.40	0.12	?	0.48
	FFM/B5	Twin	ind. inf. Rep.	1928	0.40	0.18	—	—	0.40	0.32	0.08	?	0.60
	FFM/B5	Twin	Com. Trait	1928	0.71	0.29	—	—	0.71	0.44	0.27	?	0.29
Borkenau et al.(2001) [40]	FFM/B5	Twin	VBR1	600	0.53	0.31	—	—	0.44	0.44	?	0.09	0.47
	FFM/B5	Twin	VBR2	600	0.52	0.34	—	—	0.36	0.36	?	0.16	0.48
	FFM/B5	Twin	Com. Trait	600	0.66	0.43	—	—	0.46	0.46	?	0.20	0.34
Möttus et al.(2025) [17]	FFM/B5	Family	Self-report	1820	—	—	0.13	0.18	0.32	0.32	—	?	—
	FFM/B5	Family	ind. inf. Rep.	1820	—	—	0.10	0.09	0.20	0.20	—	?	—
	FFM/B5	Family	Com. Trait	1820	—	—	0.18	0.25	0.44	0.44	—	?	—

*Note.* MZT: monozygotic twins; DZT: dizygotic twins; NTS: nontwin siblings; P-O: parents-offspring;  $h^2$ : heritability (i.e., the overall genetic contribution to trait differences);  $a^2$ : variance component due to additive genetic factors;  $na^2$ : variance component due to nonadditive genetic factors;  $se^2$ : variance component due to environmental influences shared by twins/within families;  $nse^2$ : variance component due to individually unique environmental influences (not shared within families); FFM/B5: Five-factor model/Big Five of personality (Neuroticism, Extraversion, Openness, Agreeableness, Conscientiousness); ind. inf. rep.: independent informant report; com. trait: more accurate common trait measure across single methods of measurement; VBR1 and VBR2: video-based observer ratings 1 (odd settings) and 2 (even settings); —: not reported, available, or computable; ?: nonadditive genetic and shared environmental influences are unknown or assumed to be negligible.

but well-informed others may reduce individual rater biases and thus provide more accurate reflections of true trait differences [42,43]. Genetically informative studies using those multi-rater scores typically yield

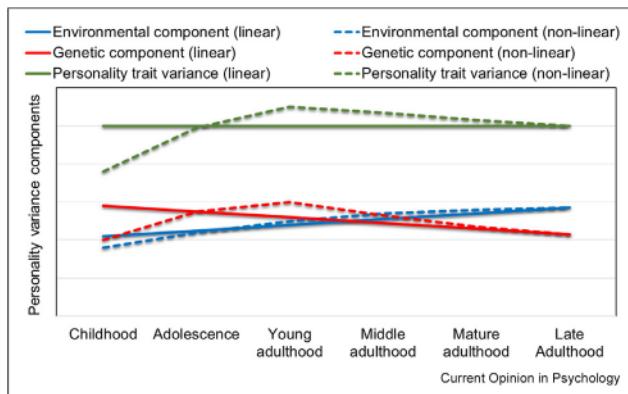
higher heritability estimates [17,40,41,44]. To decipher the polygenic and poly-environmental sources of personality traits beyond rater-specific influences, future studies should not solely rely on self-reporting.

## How are genetic and environmental factors intertwined and interdependent?

The outlined concept of polygenic and poly-environmental personality differences can explain apparent inconsistencies in previous research. However, genetic and environmental factors do not have independent effects. They can transact and interact in various ways leading to non-random gene-environment correlations and interdependencies in which the effects of genetic and environmental factors can depend on each other [30,45,46]. As a special case of gene-environment interaction, environments may change the methylation status of DNA sequences, known as environmentally driven epigenetic regulation. This in turn can change the genetic sensitivity to these or other environmental influences. Although direct evidence for gene-environment correlations and interactions influencing personality differences are sparse and primarily limited to Neuroticism [47,48], indirect evidence comes from longitudinal and multi-cohort twin studies (partly also based on multiple raters). These studies suggest that the genetic contribution tends to be high and even increases from childhood to young adulthood [49,50], reaches a plateau in young adulthood, and decreases again during adulthood (Figure 2) [26,27,35].

As children grow up, they become increasingly independent, which is accompanied by a shift from a more passive and static role to more active and dynamic transactions between them, including their heritable personality traits, and their environment. Furthermore, interactions between genetically determined personality differences and environmental influences shared by siblings raised within the same household are more plausible during the first developmental years. If these interplays are not directly estimated, they are confounded with estimates of genetic differences

**Figure 2**



How genetic and environmental personality differences may roughly shift across the lifespan due to gene-environment interactions and transactions as well as epigenetic drift based on current empirical knowledge (see Text for more details).

[30,45,49], which then increase with age during childhood and adolescence. When emerging adults leave their parental home and go their own ways, the probability of environmental conditions shared by siblings reared together declines, whereas the probability of individualizing contextual circumstances increases. Thus, potential interaction effects between individuals' heritable traits and individual-specific environmental conditions are more probable. These would be confounded with estimates of nonshared environmental influences, which then increase across adulthood. Furthermore, individuals mature continuously through the experiences they have in the course of their lives which could induce changes in the DNA-methylation status and thus may even individualize them epigenetically [51,52].

## Conclusion

To comprehensively understand the genetic and environmental sources of personality differences, the complex interplay within and between genetic and environmental factors needs to be considered. This fundamental research goal should prospectively be approached from multiple perspectives and multiple measures beyond self-reports. Future research should realize study designs that are able to tackle the polygenic and poly-environmental nature and the complexity of gene-environment interplays throughout the entire lifespan.

## Credit author statement

Christian Kandler: Conceptualization; Investigation; Project administration; Writing – original draft.

Jana Instinske: Visualization; Writing – review & editing.

## Declaration of competing interest

All authors declare that they have no conflicts of interest.

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- \* of special interest
  - \*\* of outstanding interest
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## Further information on references of particular interest

7. This preregistered meta-analysis summarized the most recent available evidence on personality change in response to life events using data from 44 studies, including 89 samples with a total of 121,187 participants. The authors reported some reliable and specific albeit relatively small effects of life events on personality differences.
14. The authors synthesized genotypic data from 46 cohorts covering about 600,000 to 1,140,000 unrelated participants of broadly European and African ancestry and about 56,000 related participants to perform the largest genome-wide association study (GWAS) and family GWAS of the Big Five personality traits at the time of writing this paper. They identified 14 times more SNPs for Big Five personality traits compared to previous studies.
17. This population-based multi-rater study on the similarity of parent-offspring, sibling-sibling, and second-degree relative pairs (32,004 self-reports and 2258 informant-reports) yielded family-based heritability estimates of about 40% for personality traits after correction for attenuation due to random error of measurement and nonrandom rater specificity.
19. In this study, large-scale exome analysis of 394,005 white British individuals from the UK Biobank were performed. They identified 14 genes, of which rare variants can account for up to 7.3% of the variance in Neuroticism.
23. In this study, differences between extended twin family models and classical twin models were analysed regarding error-adjusted estimates of genetic and environmental contributions to variance in 93 personality-relevant constructs. Extended twin family models yielded on average significantly lower heritability estimates and larger environmental differences.
25. This extended twin family study on 638 twin pairs, 787 parent-offspring dyads, and 325 spouses/partners tested differences between different concepts of personality characteristics regarding environmental sensitivity. They found lower environmental influences on differences in Big Five and HEXACO personality traits compared to other constructs, such as interests, value priorities, self-schemas, and religiousness.