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2 **A comprehensive meta-analysis of human assortative mating in 22 complex traits**

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4 Tanya B Horwitz^{1,2*} and Matthew C Keller^{1,2*}

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6 ¹Institute for Behavioral Genetics, University of Colorado Boulder, Boulder, CO, United States

7 of America.

8 ²Department of Psychology and Neuroscience, University of Colorado Boulder, Boulder, CO,

9 United States of America.

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12 **Abstract**

13 Assortative mating (AM) occurs when the correlation for a trait between mates is larger than
14 would be expected by chance. AM can increase the genetic and environmental variation of traits,
15 can increase the prevalence of disorders in a population, and can bias estimates in genetically
16 informed designs. In this study, we conducted the largest set of meta-analyses on human AM
17 published to date. Across 22 traits, meta-analyzed correlations ranged from $r = .08$ to $r = .58$,
18 with social attitude, substance use, and cognitive traits showing the highest correlations and
19 personality, disorder, and biometrical traits generally yielding smaller but still positive and
20 nominally significant ($p < .05$) correlations. We observed high between-study heterogeneity for
21 most traits, which could have been the result of phenotypic measurement differences between
22 samples and/or differences in the degree of AM across time or cultures.

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30 A comprehensive meta-analysis of human assortative mating in 22 complex traits

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32 Assortative mating (AM) is the phenomenon whereby individuals with similar trait

33 values mate with one another at levels higher than expected by chance¹. Contrary to the maxim

34 “opposites attract,” nonzero phenotypic correlations between human^{2–21} and nonhuman¹ mates

35 are overwhelmingly in the positive direction, with only a handful of examples of disassortative

36 mating, or negative mate correlations, reported in the literature^{1,4,8,20,22–29}. Several potential

37 mechanisms of AM in humans have been described, although they are not mutually exclusive

38 because multiple mechanisms can simultaneously be responsible for observed correlations.

39 Phenotypic homogamy (also known as primary phenotypic assortment) occurs when mates

40 match directly on the trait of interest³⁰. While phenotypic homogamy is often conceptualized as

41 mates actively preferring similarity, this type of homogamy can also be a function of indirect

42 selection, such as when mates are chosen from among strata that are partially determined by

43 individuals’ phenotypic values (e.g., AM for educational attainment arising as an indirect

44 consequence of mate choice occurring within job occupations). Social homogamy, on the other

45 hand, occurs when individuals match within strata that are determined by non-heritable

46 background social factors^{18,31}, such as within social class in cultures where class is not

47 genetically influenced. At the other end of the spectrum, genetic homogamy is the mechanism

48 whereby mates correlate more genetically than phenotypically for a trait; this can occur when

49 there is phenotypic homogamy on a trait that is more correlated genetically than environmentally

50 with the trait of interest^{30,32}. Finally, convergence occurs when mates become more similar over

51 time^{3,8}, either due to direct (reciprocal or one-way) phenotypic influences on one another or to

52 the mutual influence of shared environmental factors.

53 Social scientists and quantitative geneticists care about the mechanisms and the strength
54 of AM because both influence parameters of interest and impact how various estimates in the
55 literature should be interpreted. Phenotypic and genetic homogamy on heritable traits increase
56 correlations between and within causal loci, which in turn increases the genetic covariance
57 between relatives and the trait's phenotypic and genetic variation. Such an increase in variation
58 could manifest as increased prevalence rates of dichotomous traits such as psychiatric
59 disorders^{18,33}, although this effect should only be pronounced in rare, highly heritable disorders
60 under strong AM¹⁸. Social homogamy can also increase trait variation when parental phenotypic
61 values for sociocultural traits are inherited by offspring via vertical transmission³⁴. Failing to
62 account for AM can lead to biases in estimates from genetically informed designs, including the
63 association statistics from genome-wide association studies³⁵, heritability estimates from
64 twin/family designs and from single nucleotide polymorphisms³⁶, and the strength of estimated
65 causal associations in Mendelian randomization studies³⁷.

66 Given that the genetic consequences of AM and the impacts of not accounting for it in
67 certain genetically informed designs are non-negligible, it is important to understand the strength
68 of AM for traits commonly investigated in human genetics. The strength and breadth of AM is
69 also of interest to investigators of human mating in psychology, sociology, and economics.
70 While many studies have reported estimates of AM in humans, we are aware of no study that has
71 meta-analyzed AM on a large number of phenotypically diverse traits. In the current report, we
72 use stringent methodology to meta-analyze and compare partner correlations for 22 commonly
73 investigated complex traits. These results are the most comprehensive set of meta-analyses on
74 human AM to date, and should shed light on contemporary human mating trends, help with the

75 interpretation of heritability estimates, motivate studies into the various causes of AM across
76 traits, and aid in the choice of design in genetic studies.

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78 **Results**

79 *Meta-analysis*

80 We meta-analyzed partner concordance rates for 22 traits. While AM has been analyzed
81 for hundreds of traits, we focused on those most studied in the AM literature as well as some less
82 commonly studied dichotomous traits that have important health implications. The total number
83 of partner pairs for each trait ranged from 2,270 (for drinking quantity) to 1,533,956 (for
84 substance use disorder); effective sample sizes for dichotomous traits (see *Methods*) ranged from
85 721 (for alcohol use disorder) to 241,817 (for substance use disorder). Supplementary Tables S1
86 and S2 show all studies that we included in our meta-analysis for continuous and dichotomous
87 traits, respectively, as well as the effect sizes for each sample. For comparability across traits, we
88 focus here on Pearson and tetrachoric correlations for continuous and dichotomous traits,
89 respectively. Supplementary Table S2 also includes an alternative metric of partner concordance
90 for dichotomous traits, the odds ratio (OR), which is the odds of a participant possessing a trait
91 given that their partner has it divided by the odds of a participant possessing the trait given that
92 their partner does not have it. Supplementary Table S3 lists studies excluded from our meta-
93 analysis along with the reasons for their exclusion.

94 Fig. 1 displays the meta-analyzed random effects correlations for all traits along with
95 their 95% confidence intervals. The meta-analyzed correlations were greater than zero at the
96 nominal significance level ($p < .05$) for all traits. The point estimates for fourteen traits were also

97 significant at the Bonferroni-corrected ($p < .05/22 = 0.00227$) significance level. Cognitive and
98 social attitude traits showed the highest correlations ($.39 \leq r_{meta} \leq .58$); personality,
99 anthropometric traits, substance use disorders, and other disorders showed the lowest ($.08 \leq r_{meta}$
100 $\leq .29$); and correlations for non-pathological substance use traits typically lay between these two
101 sets ($.24 \leq r_{meta} \leq .54$) (see Table 1). Fig. S1 displays forest plots for all the traits we analyzed
102 with publications ordered by year and color-coded by region. The meta-analyzed fixed effects
103 results for each trait (Fig. S2) were qualitatively similar to the random effect results. Fig. S5
104 shows the number of studies included and excluded for each trait.

105 Table 2 summarizes each trait's heterogeneity estimates and the prediction intervals of
106 future studies' effect sizes. We quantified heterogeneity using the Higgins & Thompson's I^2
107 metric, which represents the percentage of variance resulting from between-study heterogeneity
108 in effect sizes rather than within-study sampling error³⁸. Higgins and Thompson (2002)³⁹
109 classified I^2 values of 25%, 50%, and 75% as low, medium, and high heterogeneity, respectively.
110 Across traits in our 22 meta-analyses, the median Higgins & Thompson I^2 statistic was 87.5%,
111 reflecting very high heterogeneity in AM estimates for most traits. However, a high I^2 reflects not
112 only high between-study heterogeneity in estimated effect sizes but also low within-study
113 heterogeneity due to highly precise estimates of individual studies. Thus, these high I^2 values
114 may in part be due to the high precision of estimates afforded by the large sample sizes of many
115 of the studies included in our analyses. An alternative metric of heterogeneity that is unaffected
116 by the precision of estimates of individual studies, τ^2 , represents the estimated variance of the
117 true effect size under a random effects model. The estimated standard deviations of true effects
118 (τ) were large relative to the meta-analyzed correlation values for many traits. The median
119 coefficient of variation $\left(\frac{\tau}{r_{meta}}\right)$ was .41, and the coefficient of variation was above .50 for

120 intelligence quotient (IQ), drinking quantity, agreeableness, conscientiousness, extraversion,
121 body mass index (BMI), and generalized anxiety disorder (GAD). However, for some traits, such
122 as EA ($r_{meta} = .53 \pm \tau = .10$), political values ($r_{meta} = .58 \pm .08$), and depression ($r_{meta} = .14 \pm$
123 $.02$), the estimated standard deviation of true effects was not very large compared to the meta-
124 analyzed estimate. Overall, our results suggest that AM is characterized by substantial
125 differences in the strength of true effect across populations differentiated by place or time.

126 For each trait, we also created Graphic Display of Heterogeneity (GOSH) plots (Fig.
127 S4)⁴⁰, which are scatterplots of the meta-analyzed correlations for all possible 2^{k-1} combinations
128 of k studies of size 2 through k (up to 1 million combinations) on the x-axis and the I^2 values of
129 these combinations on the y-axis. Two or more distinct clusters anywhere in the plot may
130 indicate subpopulations that differ in their average effect size⁴⁰, although a smear of points along
131 the bottom of GOSH plots is caused by two or more study results that happen to be similar
132 (thereby producing I^2 values near 0) and is typically not of interest. For most traits plotted in Fig.
133 S4, there are no obvious clusters. However, for IQ and conscientiousness, there do appear to be
134 two clusters, one made up of study combinations that have higher heterogeneity and higher
135 average correlations, and another with lower heterogeneity and lower average correlations. The
136 two clusters in the GOSH plot for IQ may have resulted from an outlier reported in a 1938 study
137 that found a partner correlation of $.81^{41}$, which is substantially greater than the meta-analyzed
138 estimate we report for this trait.

139 Because AM studies ostensibly focus more on effect size than hypothesis testing, we
140 expected that publication bias was unlikely to be a major factor for the study results we meta-
141 analyzed. Nevertheless, we created funnel plots (Fig. S3), which plot study effect size (Fisher Z
142 transformed correlations here) on the x-axis against standard error on the y-axis, to visually

143 inspect whether there was evidence for asymmetry, a potential indicator of publication bias.
144 Overall, there was no obvious asymmetry across the funnel plots. Only for IQ and drinking
145 quantity did it appear that there may be a systematic bias of larger studies having smaller effect
146 sizes, but both were based on 10 or fewer studies, which can lead to apparent asymmetry by
147 chance^{38,42}. The more obvious pattern observed in most funnel plots was the large number of
148 points that were outside the expected triangular region, again reflecting the high heterogeneity in
149 correlations observed across studies.

150

151 **Discussion**

152 In this study, we collated and synthesized the results from a large number of studies on
153 human AM to provide a better understanding of which traits mates assort on and how strong the
154 assortment is. To our knowledge, this is the largest and most comprehensive set of meta-analyses
155 on human AM to date. We found the highest levels of AM for political and religious values,
156 educational attainment, IQ, and some substance use traits; partner correlations for other traits
157 were smaller. Nevertheless, we found nominally significant ($p < .05$) evidence for AM for every
158 trait investigated. More than half of the meta-analyzed correlations were also significant at the
159 Bonferroni-corrected level. Whether these correlations are due to convergence or to initial
160 nonrandom mating based on phenotypic, social, or genetic homogamy remains to be determined,
161 though some research has attempted to investigate which of these mechanisms is responsible for
162 observed AM for particular traits.

163 The two social attitude traits that we examined—political attitudes and religiosity—
164 showed the highest levels of AM of all the traits we assessed. For these traits, we examined
165 continuous measures of attitudes toward political issues and self-report of multiple religious

166 ideas/practices. Interestingly, despite clear geographical stratification of religious and voting
167 trends apparent in countries such as the United States, most studies to date investigating the
168 cause of mate similarity on political and religious attitudes have suggested that the data is most
169 consistent with phenotypic rather than social homogamy, and there is no compelling evidence of
170 substantial convergence for either trait^{4,43-46}. This may be relevant to current events because, to
171 the degree that social attitudes are genetically or socially heritable, AM on them may contribute
172 to heightened political and cultural polarization.

173 We also found a high partner correlations for educational attainment (EA) ($r_{\text{meta}} = .53$),
174 and only one sample⁴⁷ out of 27 reported a correlation under .30. Thus, there is consistent
175 evidence for strong AM on EA across recent decades and across cultures in which the trait has
176 been studied. Robinson *et al.* (2017)³² found that the implied phenotypic correlation for EA
177 between partners in the UK Biobank, extrapolated from the observed correlation between
178 partners' trait-associated loci, was .65. This value was substantially larger than the phenotypic
179 correlation they observed for EA in the same sample and exceeds the upper limit of our
180 confidence interval for the meta-analyzed EA partner correlation. This suggests that AM for EA
181 is consistent with genetic homogamy, and that mates may be assorting on some trait that is more
182 genetically than environmentally correlated with EA. Contrary to Robinson *et al.*'s (2017)³²
183 finding, Torvik *et al.* (2022)⁴⁸ did not find evidence for genetic homogamy in educational
184 attainment in a sample of partners, siblings, and in-laws in Norway. Instead, they found evidence
185 that AM on EA was due to a mix of both social homogamy and phenotypic homogamy. Whether
186 this discrepancy is due to differences in EA AM between Norway and the UK or to differences
187 in sample characteristics (e.g., ascertainment) is an open question.

188 The meta-analyzed partner correlation coefficients for substance use/abuse traits ranged
189 from $r_{\text{meta}} = .24$ to $r_{\text{meta}} = .54$. Interestingly, some (but not all^{49,50}) studies that have examined
190 mechanisms of assortment in drinking and smoking have reported evidence of convergence for
191 these behaviors^{6,8,12,51}, making these traits amongst the only ones to show support for
192 convergence in the literature.

193 We observed substantial between-study heterogeneity in partner correlations for most
194 traits. A large degree of between-study heterogeneity would certainly be problematic in fixed
195 effects meta-analyses that assume a single underlying effect. However, even for random effects
196 meta-analyses, which are viewed as more appropriate when heterogeneity is present, high levels
197 of heterogeneity suggest caution should be used in interpretation of results. Random effects
198 meta-analyses assume an underlying (normal) distribution of true effects across the studies'
199 sampled populations, and the meta-analytic result is the estimated mean of those true effects.
200 Thus, the estimates we present here cannot be interpreted as estimates of a single true level of
201 AM for a given trait, but rather estimates of the typical level of AM across many possible levels
202 that might be observed at different times or locations.

203 There are several possible causes of the high levels of heterogeneity in AM we observed
204 across studies within the same trait. Most obviously, it is possible that the true degree of AM
205 varied across populations due to cultural differences in mating systems or preferences. This
206 seems plausible; AM involves mate preferences, social stratification, and/or couple dynamics,
207 and so it is unlikely to be consistent across different cultural contexts. Differences in population
208 size, mobility, and/or education across populations may impact the pool of a person's potential
209 mates and thereby the degree to which preferences can be acted on. However, there was
210 insufficient cultural diversity within traits to test whether there were significant differences in

211 partner concordance across cultures. Similarly, we determined that publication year was too
212 coarse a metric of the year in which mates were married, and too many studies failed to report
213 sufficient information for us to formally assess changes in AM over time.

214 It is also possible that some of the heterogeneity in AM effect sizes was due to
215 differences in how constructs were measured across studies—for example, differences in the
216 measurement batteries used, differences in participants' interpretations of battery items, or
217 differences in the clinical thresholds employed. Potentially consistent with this possibility, we
218 observed that the prevalence rates of dichotomous traits varied greatly in supposedly non-
219 ascertained samples, which may have contributed to the heterogeneity we observed in our
220 correlation coefficients. Nevertheless, we observed high levels of heterogeneity even for traits—
221 such as height and BMI—measured in standardized ways, suggesting that differences in how the
222 constructs were measured is unlikely to be a complete explanation. Finally, it is possible that
223 publication bias led to heterogeneity, particularly if studies that found AM results that were
224 substantially different from those already published in the literature were more likely to be
225 submitted and published—a kind of "novelty bias." However, it is also possible that a
226 "conformity bias" exists in the opposite direction and has led to downwardly biased estimates of
227 heterogeneity. While we could not test and therefore cannot rule out either possibility, we find
228 them unlikely given that the incentives for both seem dubious.

229 Although we initially gathered data on AM for rare psychiatric disorders, we did not
230 formally meta-analyze the tetrachoric correlations for these traits because too few studies met
231 our inclusion criteria as a result of unspecified sample sizes, the use of longitudinal rather than
232 cross-sectional measurements of concordance, and small expected cell frequencies (see
233 Supplementary Table S2 and S3). Nevertheless, studies that have provided robust estimates of

234 partner concordance for psychiatric disorders have suggested low to moderate AM, both within
235 and across disorders^{18,21,52,53}. For example, based on data from Swedish population registers that
236 included more than 700,000 unique cases—originally analyzed by Nordsletten *et al.* (2016)⁵⁴--
237 Peyrot *et al.* (2016)¹⁸ estimated ascertainment-corrected tetrachoric correlation coefficients of .26
238 for schizophrenia, .10 for bipolar disorder, .28 for autism spectrum disorder, and .31 for
239 attention-deficit/hyperactivity disorder.

240 There are several implications for the consistent evidence of AM across traits we
241 documented in this meta-analysis. First, as noted above, AM can increase the genetic variance and
242 the prevalence of a disorder. Although the increase in prevalence for common disorders may not
243 be large (e.g., ~10%), the levels of AM observed for rare traits of high heritability, such as autism,
244 could lead to a ~1.5-fold prevalence increase after one generation, and an even higher increase
245 (~2.4-fold) over many generations¹⁸. Second, AM can create biases in estimates of interest in
246 genetically informative designs, such as estimates based on twin studies^{10,54}, genome-wide
247 association studies (GWAS)³⁵, Mendelian randomization³⁷, and SNP-heritability³⁶. Finally, to the
248 degree that the heterogeneity in AM we observed was due to true differences in the strength of
249 AM rather than differences in measurement, our estimates of the strength of AM may not
250 generalize to other populations. While estimates for some traits, such as height, were based on a
251 geographically and ethnically diverse set of samples, most of the samples included in our meta-
252 analyses were drawn from Europe, North America, and Australia, and Asia. For example, all
253 estimates of AM for religiosity came from samples in the United States.

254 In summary, we conducted the largest and most comprehensive set of meta-analyses of
255 human AM to date. Our estimates were based on nearly a century of research and millions of
256 partner pairs. We found high partner correlations for traits related to substance use, IQ, EA, and

257 social attitudes, and smaller but nominally significant ($p < .05$) correlations for personality,
258 anthropometric, and disorder traits. However, we also observed high levels of heterogeneity in
259 AM estimates across studies for most traits investigated, suggesting that AM may differ across
260 time or place and that a single estimate of AM cannot typically be assumed for a given trait
261 across populations.

262

263 **Methods**

264 *Inclusion and exclusion criteria*

265 We conducted a systematic review of English-language studies that examined AM based
266 on partners' continuous and dichotomous self-reports on the same complex traits. All included
267 studies were published in peer-reviewed journals on or before December 22, 2021. To conduct
268 this review, we searched for words pertaining to the traits of interest in conjunction with the
269 terms *assortative mating*, *assortative marriage*, *partner concordance*, *partner correlation*,
270 *nonrandom mating*, *homogamy*, *marital resemblance*, and *marital homophily* in Google Scholar,
271 and we checked relevant papers cited in these studies for adherence to our criteria. We restricted
272 our analysis to studies of opposite-sex co-parents, engaged pairs, married pairs, and/or
273 cohabitating pairs (referred to as "partners" hereafter), with a few studies containing a small
274 number of divorced couples; we excluded same-sex partners because same-sex and opposite-sex
275 pairs show different patterns of assortment for some traits^{55,56}, because there is less data on the
276 former, and because same-sex assortment does not have the same implications for genetic
277 studies. With the exception of studies that intentionally ascertained partners for the trait of
278 interest, we excluded studies in which pairs had a characteristic that deviated from the norm in
279 the general population in a way that might have affected the magnitude of concordance (e.g., a

280 sample of only adoptive parents was excluded), and we only included studies where the sample
281 size was reported or could be inferred. For example, if only percentages were reported for each
282 cell of a contingency table, the sample size of each cell could be inferred as the percentage
283 multiplied by N .

284 We restricted our analysis to studies with sample sizes greater than 100. For dichotomous
285 traits, we restricted our analysis to studies with expected contingency table cell frequencies of
286 five or greater and observed cell frequencies greater than zero. When the samples in multiple
287 studies that were appropriate for our meta-analysis overlapped or were likely to have overlapped
288 based on information provided in the publication, we only used the study with the largest sample
289 size. We calculated effect sizes from the data reported in primary studies rather than relying on
290 effect size estimates from other published meta-analyses. If a study reported partner concordance
291 rates for multiple independent samples, each was included as a separate entry. When studies
292 reported partner correlation at different waves, we reported the results from the first wave.

293 When studies reported both the raw correlation and the partial correlation(s) controlling
294 for covariates (such as age), we included the raw correlation for consistency across studies. For
295 studies that only reported partial correlations, we used the estimate with the fewest number of
296 covariates. For ordinal and continuous traits, studies typically reported Spearman's rho or
297 Pearson's r but at times reported polychoric correlations. We excluded polychoric correlations
298 reported for such traits in order to avoid pooling two classes of correlation for the same meta-
299 analyzed effect size. Because polychoric correlations occurred rarely, we do not anticipate a
300 large loss of power as a result. Because AM for height has already been meta-analyzed
301 extensively by Stulp *et al.* (2017)⁹, we re-analyzed studies from the paper's supplement in the

302 same way we analyzed other continuous traits, after eliminating studies from this meta-analysis
303 in accordance with our exclusion criteria. Finally, we restricted our meta-analysis to traits for
304 which there were at least three samples that met our criteria.

305 Dichotomous traits

306 For dichotomous traits, we primarily considered studies that examined pairs in non-
307 ascertained community samples or national registers as well as those from samples that
308 ascertained probands. Most ascertained studies were ultimately excluded because probands were
309 typically in clinical settings (e.g., hospitalized), whereas partners of probands with the disorder
310 typically were not. Although such ascertainment can be dealt with if all the applicable
311 populations' (i.e. inpatient, outpatient, and those who have never received treatment) prevalence
312 rates are known, it was typically impossible to know all of these rates. We eliminated any
313 ascertained studies in which there was a >2 -fold difference in male and female prevalence if
314 there was not enough information to divide discordant couples based on sex. Simulation results
315 suggested that mixing individuals of different sexes when prevalence rates were more discrepant
316 than this would lead to unacceptable levels of bias. Because of possible differences in the
317 strength of AM implied from concordance of male probands versus that implied from female
318 probands, we excluded studies that only included single-sex probands. When both male and
319 female proband data was available (only a single study⁵²), estimates based on each proband
320 (female and male) were included as separate results.

321 We only used cross-sectional measures of partner concordance and therefore excluded
322 studies that used longitudinal metrics such as morbidity risks⁵⁷, hazard ratios, and incidence
323 ratios. We required that either odds ratios (ORs), risk ratios (RR), phi coefficients (Φ),
324 contingency tables, or—if the study was not ascertained (see below)—tetrachoric correlations,

325 were reported for dichotomous traits. Concordance rates captured by any of the first four of these
326 measures were then converted to tetrachoric correlations for consistency. When the contingency
327 table was unknown but the OR was reported, we first inferred the contingency table using an R
328 function described in the supplementary methods of Peyrot *et al.* (2016)¹⁸ (provided to us by the
329 authors) and then estimated the tetrachoric correlation. When the contingency table was
330 provided, we calculated the OR and tetrachoric correlation (using the polychoric() function from
331 the “polycor” package⁵⁸) in R ourselves, and thus the effect size we used in our analysis was
332 sometimes different than that reported in the original study. When the contingency table was
333 unknown but Φ was reported, Φ was converted to a tetrachoric correlation using the phi2tetra()
334 function from the “psych” package⁵⁹ in R. The prevalence rates for each sex used for these
335 conversions (from Φ and the OR) are reported in Supplementary Table S2. No studies that we
336 included in our final analysis reported an RR.

337 For studies where probands were ascertained, we used the OR, which is not influenced by
338 ascertainment, along with estimates of sex-specific prevalence rates from the country or region
339 the sample came from, to calculate tetrachoric correlations. To do this, we used the
340 aforementioned R function provided to us by Peyrot and colleagues, which produces the
341 population (non-ascertained) contingency table that is implied given the observed OR in the
342 ascertained sample and the assumed population prevalence in each sex. We then used this
343 implied contingency table to estimate the underlying (non-ascertained) tetrachoric correlation in
344 the population. This correction is necessary because the liability in the ascertained sample, where
345 the case to control ratio is usually higher than that in the population, is different than the liability
346 distribution in the population, which would lead to upwardly biased estimates if the tetrachoric
347 correlation was estimated based on just the sample contingency table.

348 We used the `metacor()` function from the “meta” package in R⁶⁰ to conduct both random
349 and fixed effects meta-analyses using inverse-variance weighting of the Fisher z transformed
350 correlations. For continuous traits, we used the Knapp-Hartung adjustment^{61,62} to calculate the
351 variance of point estimates and restricted maximum-likelihood (REML) to estimate τ^2 , the
352 variance of the true overall effect size under random effects^{63,64}. For binary traits, we used the
353 Paule-Mandel estimator⁶⁵ to estimate τ^2 and applied the Knapp-Hartung adjustment^{61,62} to our
354 calculation of the variance of the point estimate. We conducted a Monte Carlo analysis to
355 determine how best to pool information for different studies in a meta-analysis. While the “true”
356 base spousal correlation varied across simulated meta-analyses, the population-level spousal
357 correlation across “studies” within the same meta-analysis was consistent (in order to establish a
358 true rate of spousal concordance against which to compare our point estimates). However,
359 prevalence rates were allowed to vary across populations in the same simulated meta-analysis
360 (see Supplementary Table S4 for the results of each method used in conjunction with various
361 parameter estimates). We found that calculating tetrachoric correlations for each sample and then
362 meta-analyzing them provided more accurate point estimates than pooling contingency tables
363 and then calculating tetrachoric correlations. Thus, we followed this procedure for binary traits
364 throughout. The `metacor()` function internally calculates the expected variance of correlations
365 based on sample sizes and assumes they are Pearson correlations, which would be incorrect for
366 tetrachoric correlations. Thus, we needed to input effective (rather than actual) sample sizes for
367 tetrachoric correlations. For non-ascertained studies, we estimated the effective sample sizes by
368 using the standard error calculated in the `polychor()` package and solving for n in the equation
369 $SE(r) = \sqrt{\frac{(1-r^2)}{(n-2)}}$. For ascertained studies examining dichotomous traits, we created bootstrapped
370 contingency tables, each of size n (the number of partners) and sampled from the study’s (raw,

371 ascertained) contingency table with replacement. We followed the procedure described above to
372 convert the ascertained contingency table to a tetrachoric correlation corrected for ascertainment.
373 We repeated this process 1,000 times, calculated the standard error by estimating the standard
374 deviation of the 1,000 bootstrapped tetrachoric correlations, and used this standard error to
375 calculate the effective sample size as described above.

376 Four of the traits in our supplementary tables—bipolar disorder, schizophrenia, panic
377 disorder, and phobia—posed a problem because they were rare (bipolar disorder and
378 schizophrenia) or have not been studied in sufficiently large samples (panic disorder and phobia).
379 This resulted in contingency tables with zero frequency cells or with expected cell frequencies
380 that were less than five. As a result, there was not a sufficient number of studies meeting our
381 inclusion criteria to justify formally meta-analyzing these four traits, though we included the
382 results from studies that otherwise met our criteria for these traits in Supplementary Table S2.

383

384 **Data availability**

385 Studies included in the meta-analysis are listed in Supplementary Tables S1 and S2, and studies
386 excluded from the meta-analysis are listed in Supplementary Table S3.

387

388 **Code availability**

389 The code for the analyses and simulations is available from the authors upon request.

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530 **Author contributions statement**

531 TBH contributed to study design, statistical analyses, manuscript writing, collection of studies to
532 be meta-analyzed, simulation, and creation of all figures and tables; MCK contributed to study
533 design, statistical analyses, manuscript writing, and simulation.

534

535 **Additional information**

536 The authors declare no competing interests.

537

| Trait | <i>r</i> [CI] | <i>K</i> | <i>N</i> | <i>Effective N</i> | <i>p</i>-value |
|--------------------|----------------------|-----------------|-----------------|---------------------------|-----------------------|
| EA | .53 [.49; .56] | 27 | 230,915 | NA | < .0001 |
| IQ | .39 [.21; .54] | 10 | 2,561 | NA | .0012 |
| Political values | .58 [.53; .63] | 9 | 10,694 | NA | < .0001 |
| Religiosity | .57 [.37; .72] | 5 | 5,750 | NA | .0024 |
| AUD | .24 [.09; .38] | 3 | 5,162 | 721 | .0221 |
| Drinking quantity | .41 [.11; .64] | 6 | 2,270 | NA | .0178 |
| Smoking cessation | .54 [.31; .72] | 4 | 3,613 | 1,426 | .0066 |
| Smoking initiation | .37 [.30; .43] | 12 | 87,253 | 13,469 | < .0001 |
| Smoking quantity | .24 [.14; .34] | 6 | 4,701 | NA | .0020 |
| Smoking status | .46 [.35; .56] | 15 | 168,404 | 20, 584 | < .0001 |
| SUD | .29 [.29; .30] | 3 | 1,533,956 | 241,817 | < .0001 |
| Agreeableness | .11 [.05; .18] | 11 | 10,347 | NA | .0035 |
| Conscientiousness | .16 [.10; .23] | 11 | 10,347 | NA | .0003 |
| Extraversion | .08 [.05; .11] | 29 | 22,483 | NA | < .0001 |
| Neuroticism | .10 [.07; .13] | 30 | 23,154 | NA | < .0001 |
| Openness | .21 [.14; .28] | 11 | 10,483 | NA | < .0001 |
| Body mass index | .16 [.12; .19] | 31 | 131,079 | NA | < .0001 |
| Height | .23 [.21; .26] | 74 | 299,763 | NA | < .0001 |
| Waist-to-hip ratio | .16 [.08; .24] | 5 | 83,630 | NA | .0050 |
| Depression | .14 [.11; .17] | 7 | 1,483,486 | 211,154 | < .0001 |
| Diabetes | .15 [.07; .23] | 7 | 178,522 | 17,530 | .0038 |

| | | | | | |
|-----|----------------|---|---------|-------|---------|
| GAD | .14 [.04; .24] | 6 | 116,911 | 5,284 | .018638 |
|-----|----------------|---|---------|-------|---------|

539

540 **Table 1.** r = meta-analyzed random effects spousal correlation (Pearson's r for continuous
 541 traits; tetrachoric r for dichotomous traits), CI = confidence interval, K = number of samples
 542 meta-analyzed, N = number of total spouse pairs meta-analyzed; EA = educational
 543 attainment, IQ = intelligence quotient, AUD = alcohol use disorder, SUD = substance use
 544 disorder, GAD = generalized anxiety disorder; $Effective\ N = \frac{1 - r^2}{se^2} + 2$ (rearranged from the
 545 formula for the standard error estimate).

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548

| Trait | I^2 [CI] | τ | τ^2 [CI] | Prediction Interval |
|--------------------|----------------|--------|-------------------------|---------------------|
| EA | 93% [91%; 94%] | .100 | 0.0100 [0.0058; 0.0238] | [0.3568; 0.6607] |
| IQ | 91% [86%; 95%] | .260 | 0.0675 [0.0288; 0.2524] | [-0.2220; 0.7772] |
| Political values | 80% [62%; 89%] | .082 | 0.0067 [0.0018; 0.0343] | [0.4256; 0.7014] |
| Religiosity | 95% [91%; 97%] | .204 | 0.0417 [0.0128; 0.3736] | [-0.0662; 0.8782] |
| AUD | 0% [0%; 90%] | .000 | 0 [0.0000; 0.3788] | [-0.2221; 0.6153] |
| Drinking quantity | 92% [86%; 96%] | .294 | 0.0862 [0.0301; 0.5821] | [-0.4228; 0.8671] |
| Smoking cessation | 90% [77%; 96%] | .169 | 0.0285 [0.0069; 0.4410] | [-0.2102; 0.8928] |
| Smoking initiation | 95% [93%; 97%] | .104 | 0.0108 [0.0046; 0.0355] | [0.1408; 0.5587] |
| Smoking quantity | 68% [24%; 87%] | .084 | 0.0070 [0.0006; 0.0642] | [-0.0103; 0.4700] |
| Smoking status | 98% [98%; 99%] | .227 | 0.0517 [0.0247; 0.1400] | [-0.0095; 0.7651] |
| SUD | 0% [0%; 90%] | .000 | 0 [0.0000; 0.0404] | [0.2722; 0.3119] |

| | | | | |
|--------------------|----------------|------|-------------------------|-------------------|
| Agreeableness | 88% [80%; 93%] | .086 | 0.0074 [0.0022; 0.0278] | [-0.0908; 0.3108] |
| Conscientiousness | 90% [84%; 94%] | .093 | 0.0087 [0.0028; 0.0266] | [-0.0564; 0.3698] |
| Extraversion | 68% [54%; 79%] | .068 | 0.0046 [0.0017; 0.0117] | [-0.0625; 0.2198] |
| Neuroticism | 58% [37%; 72%] | .040 | 0.0016 [0.0004; 0.0073] | [0.0142; 0.1845] |
| Openness | 87% [78%; 92%] | .090 | 0.0081 [0.0027; 0.0345] | [-0.0070; 0.4027] |
| Body mass index | 96% [95%; 97%] | .086 | 0.0074 [0.0038; 0.0129] | [-0.0205; 0.3267] |
| Height | 91% [89%; 92%] | .098 | 0.0096 [0.0069; 0.0167] | [0.0408; 0.4091] |
| Waist-to-hip ratio | 68% [18%; 88%] | .052 | 0.0027 [0.0001; 0.0380] | [-0.0265; 0.3380] |
| Depression | 55% [0%; 81%] | .022 | 0.0005 [0.0000; 0.0085] | [0.0728; 0.2052] |
| Diabetes | 78% [55%; 90%] | .072 | 0.0052 [0.0005; 0.0445] | [-0.0531; 0.3391] |
| GAD | 51% [0%; 80%] | .076 | 0.0058 [0.0000; 0.0734] | [-0.0987; 0.3607] |

549

550

Table 2. Heterogeneity statistics for each trait's meta-analysis. CI = confidence interval, I^2 =

551

Higgins & Thompson's I^2 statistic, a measure of between-study heterogeneity, τ = the estimated

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standard deviation of the true effect size, τ^2 = the estimated variance of the true effect size; EA =

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educational attainment, IQ = intelligence quotient, AUD = alcohol use disorder, SUD = substance

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use disorder, GAD = generalized anxiety disorder.

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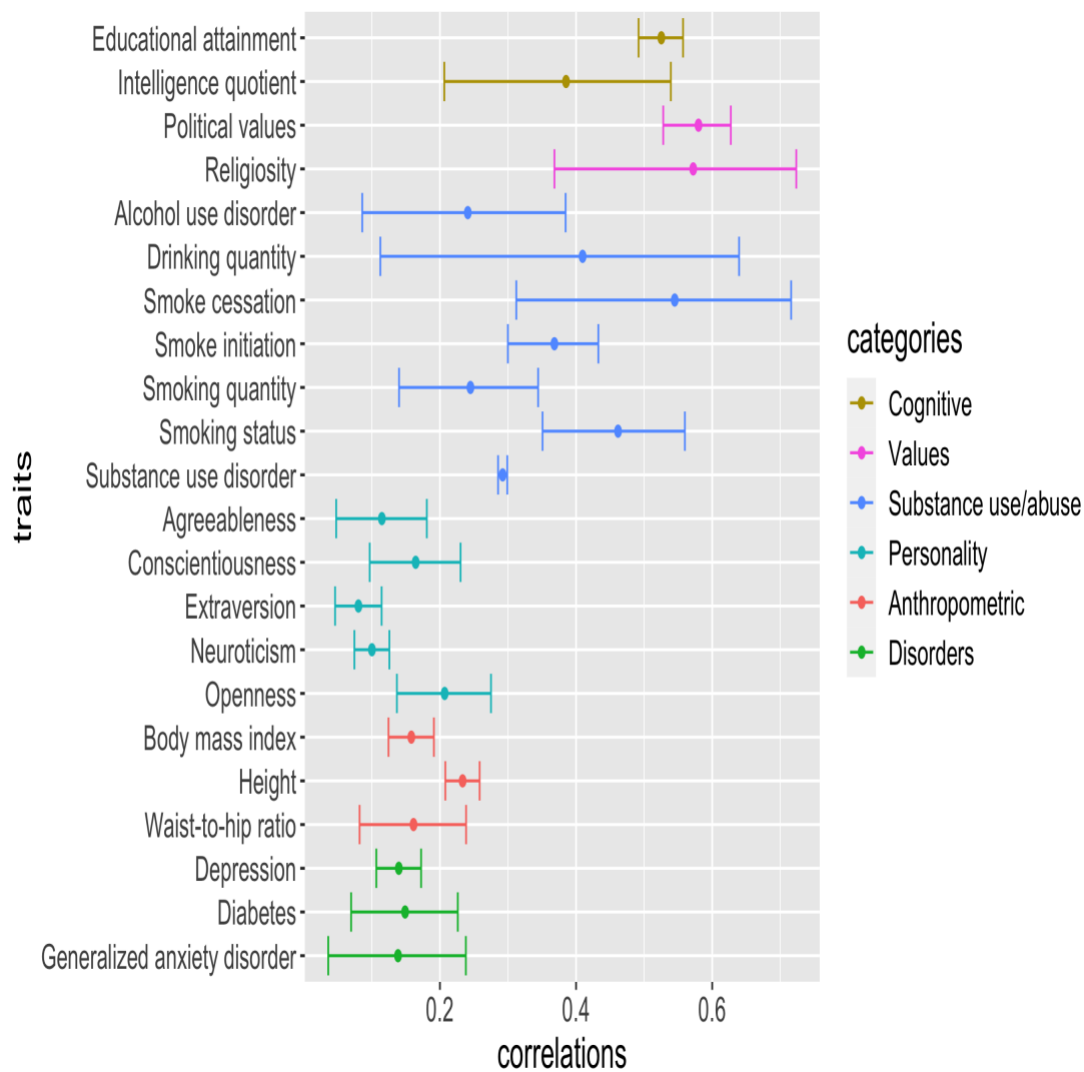
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Figure 1



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564

The meta-analyzed random effects spousal correlations and 95% confidence intervals for each trait.