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Evidence of correlations between human partners based on systematic reviews and meta-analyses of 22 traits and UK Biobank analysis of 133 traits

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Positive correlations between mates can increase trait variation and prevalence, as well as bias estimates from genetically informed study designs. While past studies of similarity between human mating partners have largely found evidence of positive correlations, to our knowledge, no formal meta-analysis has examined human partner correlations across multiple categories of traits. Thus, we conducted systematic reviews and random-effects meta-analyses of human male–female partner correlations across 22 traits commonly studied by psychologists, economists, sociologists, anthropologists, epidemiologists and geneticists. Using ScienceDirect, PubMed and Google Scholar, we incorporated 480 partner correlations from 199 peer-reviewed studies of co-parents, engaged pairs, married pairs and/or cohabitating pairs that were published on or before 16 August 2022. We also calculated 133 trait correlations using up to 79,074 male–female couples in the UK Biobank (UKB). Estimates of the 22 mean meta-analysed correlations ranged from r_{meta} = 0.08 (adjusted 95% CI = 0.03, 0.13) for extraversion to r_{meta} = 0.58 (adjusted 95% CI = 0.50, 0.64) for political values, with funnel plots showing little evidence of publication bias across traits. The 133 UKB correlations ranged from r_{UKB} = −0.18 (adjusted 95% CI = −0.20, −0.16) for chronotype (being a 'morning' or 'evening' person) to r_{UKB} = 0.87 (adjusted 95% CI = 0.86, 0.87) for birth year. Across analyses, political and religious attitudes, educational attainment and some substance use traits showed the highest correlations, while psychological (that is, psychiatric/personality) and anthropometric traits generally yielded lower but positive correlations. We observed high levels of between-sample heterogeneity for most meta-analysed traits, probably because of both systematic differences between samples and true differences in partner correlations across populations.

Phenotypic resemblance between human mates, spouses and co-parents (hereafter, 'partners') is a widely studied area of research with applications to an array of disciplines. Quantitative and behavioural geneticists are interested in the magnitudes and mechanisms underlying partner correlations because both can affect genetic and environmental parameters and estimates of interest, informing how model-derived estimates should be interpreted[1–4]. Sociologists, anthropologists, economists and other social scientists have studied partner

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correlations to gain insight into trends in societal values[5], the labour market[6,7], socioeconomic inequalities[5–7], relationship satisfaction and divorce rates[8–13]. Understanding patterns of (dis)similarity between mates thus provides useful context for both sociocultural and biological phenomena as well as their intersection, and can shed light on the forces shaping human mating trends.

Contrary to the maxim 'opposites attract', non-zero phenotypic correlations between human[8,14–32] and non-human[33] mates and partners—a phenomenon referred to as 'assortative mating' (AM) when the similarity is present at the beginning of the relationship—are overwhelmingly in the positive direction, with only a handful of examples of significant negative partner correlations ('disassortative mating') reported in the literature[9,15,19,31,33–40]. Several potential mechanisms leading to partner resemblance in humans have been described. Phenotypic homogamy (also called 'primary phenotypic assortative mating') occurs when partners match directly on the trait of interest[41]. While phenotypic homogamy is often conceptualized as partners simply preferring similarity, it can also arise from indirect selection (for example, in parent-arranged marriages) or when partnerships form within strata that are associated with trait values (for example, partner correlations for educational attainment arising as an indirect consequence of mate choice occurring within a profession). Social homogamy, on the other hand, results when partners assort on non-heritable aspects of a trait[29,42]. This can occur through several different processes, such as when romantic/marital partnerships form within strata that are heavily determined by social factors (for example, social networks) that are not genetically correlated with the focal trait. Genetic homogamy describes the opposite phenomenon: when partners assort on heritable aspects of a trait. Genetic homogamy may occur when there is phenotypic homogamy on a trait that is genetically but not environmentally correlated with the trait of interest[41,43], resulting in a genetic correlation between partners that is higher than would be expected given the phenotypic correlation for the focal trait. For example, there is evidence of genetic homogamy for educational attainment[43] in the UK Biobank (UKB), suggesting that partners in this sample are assorting heavily upon a different trait (such as intelligence quotient score) that is more genetically than environmentally correlated with education level. Finally, convergence occurs when partners become more similar over time[14,19], either due to direct (reciprocal or one-way) phenotypic influences or to the mutual influence of environmental factors shared between partners. Unlike social/phenotypic/genetic homogamy, convergence is not considered a type of AM because it is not a consequence of initial matching (or assortment). Importantly, the processes that lead to partner correlations need not be mutually exclusive, as multiple mechanisms may work in concert to produce an observed correlation between partners for a given trait. For example, it is possible that a mixture of social homogamy and phenotypic homogamy for a given trait is more common than 'pure' social homogamy or 'pure' phenotypic homogamy. Similarly, it is possible that individuals with a high environmental load for a given trait may preferentially mate with individuals that have a high genetic load for the same trait, forming a gene–environment correlation between partners that would probably appear as phenotypic homogamy.

When occurring on heritable traits, phenotypic and genetic homogamy increase correlations between and within causal genetic variants, which in turn increase both the genetic covariance between relatives and the trait's genetic and phenotypic variation. Such an increase in variation could manifest as increased prevalence rates of dichotomous traits such as psychiatric disorders[29,44], although this effect would only be pronounced under certain conditions (for example, for rare, highly heritable traits undergoing strong phenotypic AM[29]). Failing to account for phenotypic and genetic homogamy can also lead to biases in association statistics from genome-wide association studies[45], in heritability and genetic correlation estimates based on twin/family designs and single nucleotide polymorphisms[46,47] and in the strength of estimated causal associations from Mendelian randomization studies[48]. Similarly, despite having no genetic consequences, convergence and social homogamy may still increase the environmental and phenotypic variance of a trait and the covariance between relatives to the extent that the trait is shaped by non-genetic parental influences; as a result, these non-genetic mechanisms could still ostensibly affect the prevalence of dichotomous traits. Understanding the magnitude of partner correlations across a wide variety of traits is therefore important for setting expectations of what analyses are at risk of bias due to the influences of homogamy and for guiding efforts to uncover the mechanisms that underlie these correlations.

In addition to contributing to knowledge around the architecture and aetiology of traits, research on shared disease/disorder risk factors and health-related behaviours can also have public health implications for screening and intervention practices[16,38,49,50]. For example, research on the nature of partner correlations for behavioural similarities could potentially inform how partners can effectively make lifestyle changes together[17,51], particularly if and when partner resemblance is due to mutual influences within the relationship. Furthermore, partner correlations can lead to the concentration of certain traits (for example, education or income) within particular social groups, potentially exacerbating existing social inequalities[8]. Characterizing patterns of mate similarity, particularly with regard to how they differ across populations, is thus of great sociological relevance.

In this Article, we meta-analyse and compare estimates of 480 male–female partner correlations (taken from 199 independent studies) on 22 traits frequently investigated in this research arena. In addition, we calculate correlations across 133 traits using up to 79,074 male–female partner dyads from the UKB sample. These results can help shed light on contemporary human mating and relationship trends, refine the interpretation of heritability estimates, motivate investigation into the various causes of partner correlations across traits, and aid in the choice of design in genetic and non-genetic studies.

Results

Meta-analysis

Our random-effects meta-analytic results include a total of 480 partner correlations across 22 (out of an initial 26) traits for which we were able to find results from at least three samples that satisfied our criteria. The total number of partners for each trait ranged from 2,527 (for generalized anxiety) to 2,727,151 (for diabetes). Supplementary Tables 1 and 2 show all studies that we included in our meta-analysis for continuous and dichotomous traits, respectively, as well as the effect sizes for each sample. For comparability across traits, we focus on Pearson's, Spearman's and tetrachoric correlations, some of which were transformed from the original reported statistic, for continuous, ordinal and dichotomous traits, respectively. Our meta-analyses are based on pooled Fisher *Z*-transformed estimates from the included studies because this transformation yields a sampling distribution that is approximately normal. We then back-transformed the meta-analysed estimates and report correlations along with their adjusted 95% confidence intervals (CIs) (see Table 1).

Figure 1 displays the estimates of the mean meta-analysed correlations for all meta-analysed traits and, where applicable, the correlations for comparable traits in the UKB (see 'Partner correlations in the UKB'), along with the adjusted CIs associated with each trait. The estimates of the mean correlations for the meta-analysed traits (r_{meta}) were greater than zero at the two-tailed, Bonferroni-corrected significance level for 18 traits, with the remaining 4 estimates being based on only 4 samples each. r_{meta} was greater than 0.35 for 9 attitudinal, academic and substance-related traits, ranging from $r_{meta} = 0.38$ for smoking initiation to $r_{meta} = 0.58$ for political values. It is important to note that despite being associated with relatively large point estimates,

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Table 1 | Results for the random-effects meta-analyses of mating partner pairs across 22 traits

Trait	*r* (CI)	*k* (*k*$_s$)	*N*	*P* value	I^2 total (within-, between-)	τ (within-, between-)	Prediction interval
EA[12,13,15,34,58,61,67,81–115]	0.55 (0.50, 0.60)	86 (42)	1,911,720	<0.0001	0.99 (0.40, 0.59)	0.094, 0.114	(0.31, 0.72)
IQ score[9,22,100,116–125]	0.44 (0.23, 0.61)	17 (13)	5,672	<0.0001	0.94 (0,0.94)	0, 0.223	(−0.03, 0.74)
Political values[9,13,35,86,126–132]	0.58 (0.50, 0.64)	12 (11)	11,658	<0.0001	0.80 (0.11, 0.69)	0.028, 0.068	(0.45, 0.68)
Religiosity[9,13,126,130,132–134]	0.56 (0.25, 0.77)	8 (7)	6,269	0.0022	0.94 (0, 0.94)	0, 0.203	(0.12, 0.82)
PAU[28,31,135,136]	0.28 (−0.21, 0.66)	4	9,751	0.2480	0.62	0.079	(−0.01, 0.53)
Drinking[12,19,49,103,137–141]	0.42 (0.07, 0.68)	9	7,055	0.0187	0.98	0.255	(−0.17, 0.79)
Smk. cessation[16,51,142,143]	0.54 (−0.28, 0.91)	4	3,613	0.1530	0.84	0.161	(0.02, 0.83)
Smk. initiation[12,16,19,38,49,51,137,140,142–146]	0.38 (0.23, 0.53)	16 (13)	118,856	<0.0001	0.96 (0.14, 0.82)	0.054, 0.130	(0.09, 0.61)
Smk. quantity[15,147–151]	0.29 (0.07, 0.68)	9 (6)	12,072	0.0002	0.82 (0.58, 0.24)	0.056, 0.036	(0.13, 0.44)
Smk. status[16,19,25,38,58,142,143,145,146,149,151–160]	0.49 (0.38, 0.59)	27 (20)	244,597	<0.0001	0.98 (0.24, 0.74)	0.089, 0.157	(0.15, 0.72)
SUD[72,161,162]	0.43 (−0.73, 0.95)	4 (3)	1,534,416	>0.999	1.00 (0, 1.0)	0, 0.223	(−0.36, 0.86)
Agree.[9,10,34,36,127,163–169]	0.11 (0.03, 0.20)	17 (12)	19,471	0.0039	0.82 (0.22, 0.60)	0.036, 0.059	(−0.04, 0.26)
Consc.[9,10,34,36,127,163–169]	0.16 (0.06, 0.25)	18 (12)	19,930	<0.0001	0.85 (0, 0.85)	0, 0.078	(−0.02, 0.32)
Extrav.[9,10,13,19,22,34–36,86,116,126,127,133,148,163–178]	0.08 (0.03, 0.13)	40 (30)	32,729	<0.0001	0.79 (0.50, 0.29)	0.056, 0.043	(−0.07, 0.22)
Neurot.[9,10,13,19,22,34–36,127,133,148,163–171,173–182]	0.11 (0.08, 0.15)	39 (30)	35,706	<0.0001	0.58 (0.52, 0.06)	0.039, 0.013	(0.03, 0.19)
Open.[9,10,34,36,127,163,165,167,169,181,183]	0.21 (0.10, 0.31)	16 (11)	19,377	<0.0001	0.89 (0.06, 0.83)	0.022, 0.083	(0.02, 0.38)
BMI[12,38,49,50,81,86,110,113,115,137,142,146,156,178,184–209]	0.16 (0.11, 0.21)	57 (40)	123,881	<0.0001	0.94 (0.28, 0.66)	0.049, 0.075	(−0.02, 0.33)
Height[19,84,86,110,115,120,141,178,186,187,197,198,204–244]	0.24 (0.20, 0.28)	68 (53)	293,461	<0.0001	0.97 (0.61, 0.36)	0.080, 0.061	(0.04, 0.42)
WHR[49,185,189,196,203,245]	0.17 (0.03, 0.29)	6	6,055	0.0184	0.59	0.043	(0.04, 0.28)
Depression[27,28,31,72,246]	0.15 (0.03, 0.28)	7 (5)	1,483,486	0.0198	0.96 (0.01, 0.95)	0.005, 0.039	(0.04, 0.26)
Diabetes[16,27,39,72,146,203,247–250]	0.15 (0.04, 0.26)	12 (10)	2,727,151	0.0046	0.99 (0.09, 0.90)	0.023, 0.087	(−0.02, 0.31)
Gen. anx.[28,30,31]	0.17 (−0.45, 0.68)	4 (3)	2,527	>0.999	0.54 (0.54, 0)	0.098, 0	(−0.20, 0.50)

Citations after each trait refer to each study that was meta-analysed for that trait. *k* is the number of samples meta-analysed, where all samples for a specific trait were determined to be distinct; *k*$_s$ is the number of published studies from which the samples were taken; *r*(d.f. = *k*−1) is the estimate of the mean meta-analysed random-effects partner correlation (Pearson's *r* for continuous traits; tetrachoric *r* for dichotomous traits); CI is Bonferroni adjusted for 22 tests; *N* is the number of total partner pairs meta-analysed; *P* value is based on two-sided tests, Bonferroni adjusted for 22 tests; I^2 is the Higgins and Thompson's I^2 statistic, a measure of between-/within-study heterogeneity, where overall I^2 is the proportion of variance in the meta-analysis that is not due to sampling error; τ is the estimated standard deviation of the true effect size (when applicable, heterogeneity values are given for both the within-study and between-study levels, respectively); Prediction interval is the interval in which the correlations for future meta-analyses of the trait in question are expected to fall. PAU, problematic alcohol use; Drinking, drinking quantity; Smk., smoking; SUD, substance use disorder; Agree., agreeableness; Consc., conscientiousness; Extrav., extraversion, Neurot., neuroticism; Open., openness to experience; WHR, waist-to-hip ratio; Gen. anx., generalized anxiety.

3 of the 7 substance use traits we meta-analysed did not achieve Bonferroni-corrected significance. Estimates of mean meta-analysed correlations for anthropometric traits and (non-substance related) disorder traits were all low to moderate ($0.15 \leq r_{meta} \leq 0.24$), although the estimate for generalized anxiety ($r_{meta} = 0.17$, $P_{adj} > 0.999$) was not significant. The three lowest estimates we found were for the Big Five personality traits extraversion ($r_{meta} = 0.08$), neuroticism ($r_{meta} = 0.11$) and agreeableness ($r_{meta} = 0.11$). Point estimates for conscientiousness ($r_{meta} = 0.16$) and openness to experience ($r_{meta} = 0.21$) were slightly higher (see Table 1 for Bonferroni-adjusted two-tailed *P* values, Bonferroni-adjusted 95% CIs and total sample sizes associated with each meta-analysed trait).

In addition, Table 1 summarizes heterogeneity estimates for each meta-analysis as well as prediction intervals of future studies' effect sizes. For traits meta-analysed using random-effects models with two levels, we calculated a single Higgins and Thompson's I^2, which estimates the proportion of variation resulting from heterogeneity across a trait's study-/sample-level effect sizes that is not attributable to sampling error. For traits meta-analysed using models with three levels (those for which at least one publication reported two or more effect sizes), Table 1 also shows two I^2 values: one representing the proportion of variance resulting from heterogeneity within-study and one representing the proportion of variance resulting from between-study heterogeneity, which sum to the total I^2 (see 'Meta-analytic method')[52]. The median within-study I^2 for three-level analyses was 0.18, while the median between-study I^2 was 0.715. Partitioning within- vs between-study I^2 estimates can yield unstable results, especially for meta-analyses that only involve a small number of effect sizes within a common study, and thus should be interpreted with caution. Across our 22 meta-analyses, the median total Higgins and Thompson I^2 statistic was 0.915, reflecting very high rates of heterogeneity in partner correlations not due to sampling error.

In general, high I^2 values may reflect not only high between-sample heterogeneity in estimated effect sizes but also low within-sample estimation error. Thus, the high I^2 values we observed may in part be due to the high precision of estimates afforded by the large number of participants comprising many of the samples included in our analysis. To this end, we also report an alternative metric of heterogeneity, τ, that estimates the standard deviation of the true effect size and is unaffected by the precision of individual estimates. The median overall τ was 0.090 and ranged from 0.039 (depression) to 0.255 (drinking). τ tended to be positively associated with r_{meta}, with the average ratio of τ to r_{meta} (the coefficient of variation) being 0.373 (s.d. = 0.180) across traits.

Overall, our results show that partner correlations are characterized by substantial heterogeneity across samples. Given that traits measured objectively (for example, height, body mass index,

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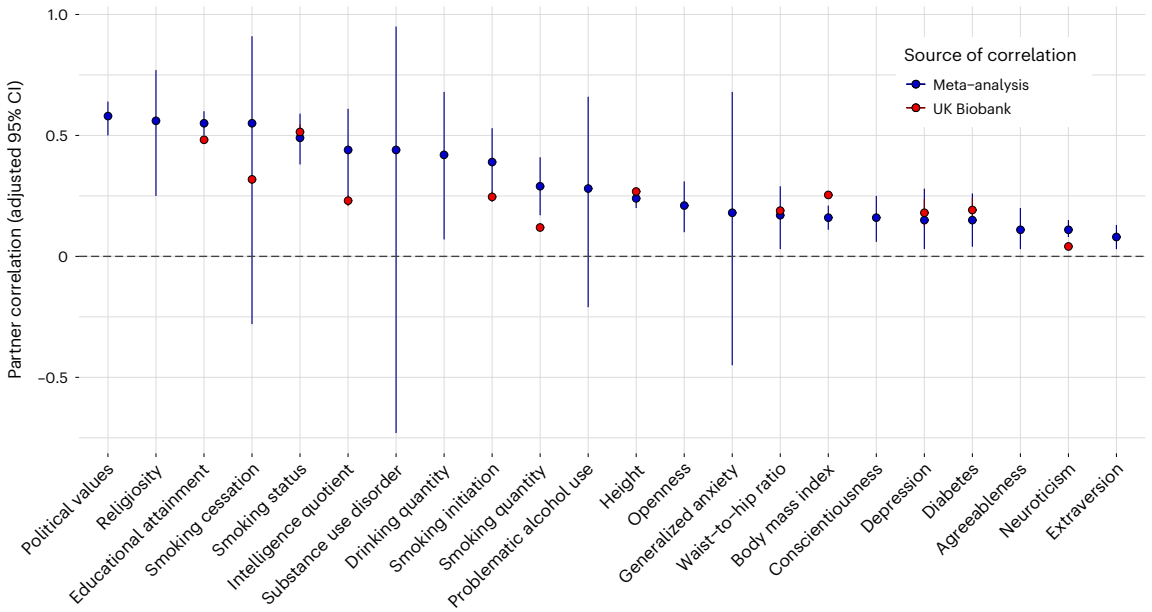

Fig. 1 | Point estimates of the mean meta-analysed random-effects partner correlations and UKB partner correlations for comparable traits. The dark blue points represent the random-effects estimates of the mean meta-analysed correlations for partners, while the red points on the same vertical axis represent the point estimates of the partner correlations for a comparable trait in the UKB, where applicable. To account for multiple testing, meta-analysed and UKB correlations are shown with Bonferroni-adjusted 95% CIs (adjusting for 22 and 133 traits, respectively). Table 1 and Supplementary Table 4 include the precise sample size and point estimate/adjusted confidence interval/adjusted *P* value and so on for each of these traits in the meta-analysis and UKB, respectively.

waist-to-hip ratio) were also associated with considerable heterogeneity, our observed I^2 and τ estimates suggest that there are substantial differences in true partner correlations for a given trait across populations differentiated by location, time and/or culture.

Finally, we produced funnel plots for each meta-analysed trait to assess publication bias. Funnel plots demonstrating a large negative regression slope, particularly those with several studies clustering in the bottom right but not the bottom left, are indicative of publication bias. Overall, there were no obvious patterns of asymmetry across traits (Extended Data Fig. 1a–v). The clearest trend across most of the funnel plots was the large number of points outside the expected triangular region, again reflecting the high heterogeneity in correlations observed across samples.

Partner correlations in the UKB

We investigated correlations for an initial 140 traits using up to 79,074 pairs of UKB participants inferred to be opposite-sex partners (see 'Analysis of partner correlations in the UKB'). Seven (5%) of these traits are not included in our primary analyses, in Fig. 2, or in Extended Data Fig. 2 because of low sample size or low endorsement rate. However, results for all 140 traits are presented in Supplementary Table 4, which includes Bonferroni-adjusted two-tailed *P* values and Bonferroni-adjusted 95% CIs, as well as summary statistics, cell frequencies and prevalence rates (for dichotomous traits), corresponding UKB field IDs and trait descriptions. Of the 133 adequately powered UKB traits presented in Fig. 2 and Extended Data Fig. 2 (including 59 continuous, 24 ordinal and 50 dichotomous traits estimated using Pearson's, Spearman's and tetrachoric correlations, respectively), 118 partner correlations (88.72%) were significant after correction for multiple testing and 15 (11.28%) were not. Of the 118 partner correlations that were significant at the Bonferroni-corrected level, 3 (2.54%) were negative and 115 traits (97.46%) were positive. The mean partner correlation was 0.19 and correlations ranged from $r_{UKB} = -0.18$ (chronotype, or being an 'evening person' vs being a 'morning person') to 0.87 (year of birth).

Twelve of the traits that we analysed in the UKB were also meta-analysed (see Fig. 1). Pearson and tetrachoric correlations for all but one (cigarettes per day, not restricted to ever-smokers) of these 12 overlapping UKB traits fell within the prediction intervals (distinct from the CIs) of corresponding traits from our meta-analyses; in addition, the Spearman correlation for non-restricted cigarettes per day in the UKB, as well as both correlational effect sizes for cigarettes per day when restricted to ever-smokers, fell in the prediction interval for smoking quantity in the meta-analysis. Across both analyses, there were notable overall findings: for example, estimates of educational attainment and current smoking status ranked among the highest correlations, while estimates of partner correlations for neuroticism were uniformly low across the two analyses. In addition, correlational estimates for some traits were notably different across the meta-analysis and UKB, as evidenced by the non-overlapping CIs for a minority of trait pairings in Fig. 1. Because overlapping CIs for the remaining trait pairings do not necessarily indicate a lack of significant difference, we performed a paired test comparing the two groups of correlations across all 12 traits but found no significant difference: $t(11) = -1.724$, $P = 0.113$, mean difference = −0.053; 95% CI: −0.121, 0.015; $V = 23$, $P = 0.233$.

Next, we compared correlations for groups of traits within the UKB. We regressed the UKB partner correlation point estimates on the category of trait (a factor with six levels: anthropometric, health-related, psychological, behavioural, demographic/family and substance use) for the 133 adequately powered UKB traits, controlling for how the correlations were estimated (Pearson's, Spearman's or tetrachoric); we used weighted least squares (weights = $\frac{1}{\text{s.e.}(r_{UKB})^2}$) for our estimation because the Levene homogeneity of variance test suggested violation of the equal variances assumption across trait categories when controlling for correlation type ($F_{(5,127)} = 9.75$, $P = 6 \times 10^{-8}$). There were significant differences between mean correlations depending on the category of trait ($F_{(5,125)} = 28.20$, $P < 2 \times 10^{-16}$, $\eta_p^2 = 0.530$). In a follow-up analysis, we found that traits that were anthropometric

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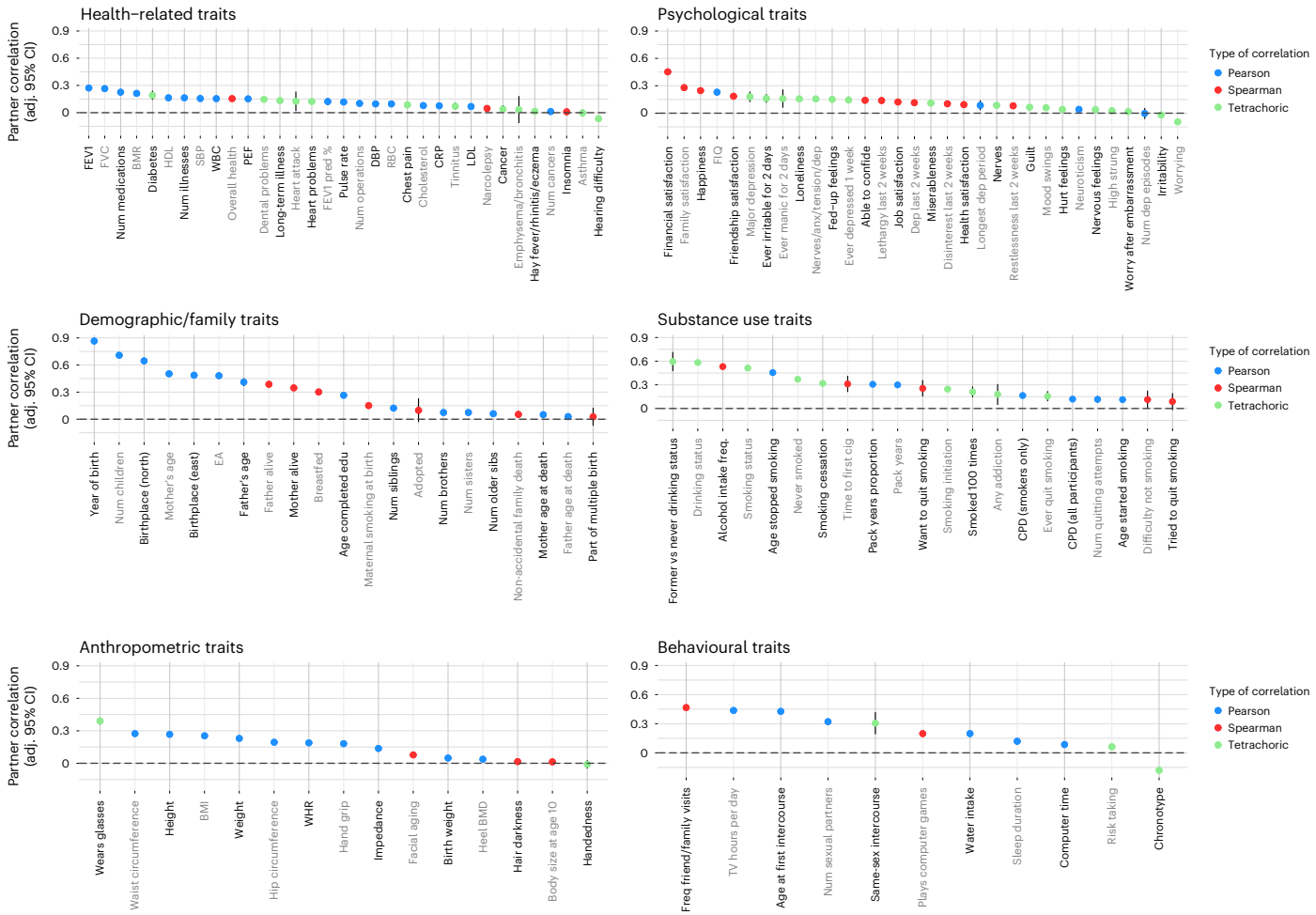

Fig. 2 | The UKB partner correlation point estimates for 133 traits grouped by category. The point estimates on the *y* axis represent the estimated partner correlation, along with Bonferroni-adjusted 95% CIs (adjusting for 133 traits), for the corresponding trait on the *x* axis. Estimates are based on up to 79,074 pairs; Supplementary Table 4 includes the precise sample size/point estimate for each trait along with the Bonferroni-adjusted *P* values associated with the adjusted 95% CIs depicted in this figure. Traits are grouped into six categories: health-related, psychological, demographic/family, substance use, anthropometric and behavioural. Points representing partner correlations for continuous traits (Pearson correlations) are blue; points representing partner correlations for ordinally coded traits (Spearman correlations) are red; points representing partner correlations for dichotomously coded traits (tetrachoric correlations) are light green. Num Dep Episodes, number of depressive episodes; Heel BMD, heel bone mineral density (in the form of a *t*-score); LDL, direct low-density lipoprotein cholesterol; CRP, C-reactive protein; RBC, red blood cell (erythrocyte) count; DBP, diastolic blood pressure; CPD (all participants), cigarettes per day (includes current, former and never smokers); FEV1 pred %, forced expiratory volume in 1 second (FEV1), predicted percentage; PEF, peak expiratory flow; WBC, white blood cell (leucocyte) count; SBP, systolic blood pressure; HDL, high-density lipoprotein cholesterol; CPD (smokers only), cigarettes per day (restricted to current or former smokers); WHR, waist-to-hip ratio; BMR, basal metabolic rate; FIQ, fluid intelligence quotient; FVC, forced vital capacity; Time to First Cig, time to first cigarette.

($\bar{r}_{UKB}$ = 0.15, s.e. = 0.031), health-related ($\bar{r}_{UKB}$ = 0.11, s.e. = 0.013) or psychological ($\bar{r}_{UKB}$ = 0.12, s.e. = 0.018) yielded significantly lower partner correlations on average than traits related to behaviour ($\bar{r}_{UKB}$ = 0.22, s.e. = 0.059), demography/family ($\bar{r}_{UKB}$ = 0.29, s.e. = 0.055) or substance use ($\bar{r}_{UKB}$ = 0.29, s.e. = 0.036) ($t(131) = 6.94$, $P = 1.61 \times 10^{-10}$, Cohen's $d = 0.88$; 95% CI: 0.53, 1.20). Controlling for category of trait, magnitude of partner correlations did not differ based on correlation type ($F_{(2,125)} = 2.38$, $P = 0.096$, $\eta_p^2 = 0.037$).

Several of the highest positive zero-order UKB partner correlations for demographic variables are unsurprising given that individuals tend to marry and enter into long-term relationships with others who are of similar ages and from nearby geographic locations (for example, r_{UKB} for north coordinate of birthplace = 0.65) and because romantic partners often share children with one another (r_{UKB} for number of children = 0.71). More interestingly, partner correlations for several substance use traits were also very high. For adequately powered substance use traits, we calculated the highest correlation for former vs never drinking status (among only current non-drinkers, whether or not a person is a previous or never drinker: r_{UKB} = 0.60), followed by drinking status (whether or not a person currently drinks alcohol: r_{UKB} = 0.58). We evaluated smoking behaviour with multiple measures and, of these, found the highest correlations for smoking status (r_{UKB} = 0.51) and the age that individuals stopped smoking (restricted to former regular smokers; r_{UKB} = 0.46), and the lowest correlation for presence and duration of quitting attempts (measured ordinally) in current regular smokers (r_{UKB} = 0.09, P_{adj} = 0.569). While we were underpowered in the UKB to examine concordance for addictions to specific narcotic or prescribed substances, addiction to 'any substance or behaviour' showed a significant correlation (r_{UKB} = 0.18, P_{adj} = 0.000199), suggesting a modest positive relationship between partners for general addiction.

As expected, educational attainment between partners in the UKB (measured as level of academic qualification/degree achieved) yielded

a fairly large correlation (r_{UKB} = 0.48). Fluid intelligence quotient (IQ) score, evaluated using a brief 13-item test, showed a somewhat modest correlation, at r_{UKB} = 0.23.

Consistent with the results from our meta-analyses, anthropometric, health-related and psychological (that is, personality and psychiatric) traits generally showed low to moderate correlations, with some of these traits showing no significant correlation between partners. Correlations for height, waist-to-hip ratio and body mass index were r_{UKB} = 0.27, r_{UKB} = 0.19 and r_{UKB} = 0.25, respectively. Partner correlation for overall neuroticism score was low but significant (r_{UKB} = 0.04, $P_{adj} < 2.66 \times 10^{-14}$) and partner correlations for the 12 dichotomous (yes/no) items comprising the neuroticism scale ranged from −0.09 to 0.16 (see Supplementary Table 4). Furthermore, 3 dichotomous depression traits that we analysed in the UKB all yielded modest correlations that were similar to one another: r_{UKB} = 0.15–0.18.

We additionally explored multiple traits that did not overlap with those we examined in our meta-analysis. The largest significant negative correlation across all UKB traits was for chronotype, which was dichotomized to indicate whether an individual is a 'morning person' or an 'evening person' (r_{UKB} = −0.18). Furthermore, we found moderate correlations for traits related to sexuality (age of first intercourse: r_{UKB} = 0.43; number of lifetime sexual partners: r_{UKB} = 0.32; ever had same-sex intercourse: r_{UKB} = 0.31). Finally, general happiness (r_{UKB} = 0.25) and measures of satisfaction with various life aspects (financial situation: r_{UKB} = 0.45, family relationships: r_{UKB} = 0.28, friendships: r_{UKB} = 0.19, job r_{UKB} = 0.12 and health r_{UKB} = 0.09) showed small to moderate positive partner correlations.

Discussion

In this study, we collated and synthesized results from a large number of studies on same-trait correlations in opposite-sex/gender couples, spousal pairs and co-parents (collectively, 'partners') to provide a better understanding of the degree to which partners correlate across traits and the degree of between-sample heterogeneity in those correlations. We also analysed partner correlation for 133 traits in putative partners from the UKB.

We found widespread evidence of positive correlations across traits, with significant variation across traits with respect to degree of partner similarity. Across both the meta-analyses and UKB raw data analyses, correlations for educational attainment, substance use measures, attitudinal traits and behavioural traits were often moderate and relatively higher, whereas correlations for anthropometric, psychological (that is, psychiatric/personality) and health-related traits were typically low to moderate. In our meta-analysis, we calculated the highest mean meta-analysed partner correlations for political and religious values, educational attainment, IQ score and some substance use traits, although a few meta-analyses in the latter category produced non-significant estimates. Although estimates for other traits were smaller, 18 of 22 traits reached Bonferroni-level significance. Likewise, partner correlations for the large majority (88.72%) of traits we examined in the UKB reached Bonferroni-corrected significance. Overall, educational attainment and certain substance use traits also yielded among the highest correlations in the UKB. While point estimates for comparable traits across our two analyses were not significantly different overall, not every UKB estimate was identical to what we observed in the corresponding meta-analysis. This is not surprising given that our meta-analysis revealed strong evidence for between-sample heterogeneity (see below) and given that the UKB probably differed in multiple ways from the typical sample included in our meta-analysis; for example, individuals in the UKB are of a relatively narrow age range, reside in the United Kingdom and differ as a whole from the general population with respect to several demographic and lifestyle variables (as discussed below in the limitations).

In addition to finding widespread positive associations in mating pairs across traits, our meta-analysis found evidence of substantial within-trait heterogeneity across different samples. Because of this large degree of heterogeneity and because we performed random (rather than fixed) effects meta-analyses, the results presented here should not be interpreted as estimates of a single true partner correlation for a given trait, but rather as estimates of the typical level of partner similarity across many possible levels that might be observed in different populations. Our results suggest that much of the observed between-study and between-sample heterogeneity is due to true differences that exist across populations (cultures, time periods and so on) from which our samples were drawn. This is sensible given that resemblance between partners can result from personal preferences, social stratification and/or couple dynamics that are unlikely to be consistent across different cultural contexts for a given trait. However, owing to insufficient diversity in nationality (see limitations) within the samples we included in most of our meta-analyses and to the small number of samples in some of our meta-analyses, we were unable to meaningfully evaluate whether there were significant differences in partner correlations across even broad geographical subdivisions. We were also unable to formally assess changes in partner resemblance over time or control for age in the meta-analyses because publication year served as an inaccurate measure of the year/age at which partners entered into the relationship. Furthermore, although we excluded samples with known biologically related pairs from our meta-analysis, it is possible that varying degrees of unknown or unreported relatedness in couples (consanguinity) across samples could have contributed to heterogeneity.

In addition to the possible causes described above, the heterogeneity in effect size estimates may be partially due to differences in how constructs were assessed across studies and samples (for example, differences in the measurement batteries used or differences in measure interpretation). Potentially consistent with this, we observed that the reported prevalence rates of dichotomous traits were highly heterogeneous across supposedly non-ascertained samples, which may have contributed to the heterogeneity we observed in our correlation coefficients (two identical odds ratios translate into different tetrachoric correlations if the prevalence rates in the samples differ). Nevertheless, we observed high levels of heterogeneity in correlations for traits such as height and body mass index that are not dichotomous and that are measured in standardized ways, suggesting that measurement invariance and ascertainment are unlikely to be complete explanations. More thoroughly examining sources of heterogeneity in partner correlations across samples remains an important direction for future studies.

There are several implications for the ubiquitous evidence of partner similarity we documented across traits in our meta-analysis. First, partner similarity due to phenotypic or genetic homogamy within a population can bias estimates of genetic correlations[53], latent variances from twin/family studies[21,47,54], Mendelian randomization[48] and SNP heritability[46]. To the degree that observed levels of partner similarity are due to these mating mechanisms, our results suggest that for many traits, some degree of bias due to AM may exist in the aforementioned estimates and designs. Second, genetically informed designs that attempt to model AM often must assume that the degree of partner resemblance has been stable across time and place; our observations of high between-sample heterogeneity call this assumption into question. Such heterogeneity has complex implications with respect to various heritability estimates and dating and marriage trends, as well as long-term implications for genetic parameters such as stabilization of genotypic frequencies (genetic (dis)equilibrium). Circumventing the caveats resulting from such trends is increasingly possible in genetic designs; for example, the method structural equation modelling-polygenic score (SEM-PGS) can account for various modes of AM even if equilibrium has not been reached for the trait of interest[55]. Third, partner similarity has the capacity to increase prevalence of disorders and other dichotomous traits through increasing

phenotypic variance via increase in environmental/genetic variance, although the precise consequences of partner similarity will depend on which mechanism(s) is/are at play, the magnitude of resemblance and the heritability of the focal trait. There is evidence demonstrating that although the resulting increase in prevalence for common traits may be weak, phenotypic AM for a rare, highly heritable trait could substantially increase the trait's prevalence (with estimates for some trait increases reaching 1.5-fold over one generation and 2.7-fold over ten generations)[29]. Finally, partner correlations have social and interpersonal implications that are relevant to outcomes of mating partners regardless of whether they procreate. For example, there is longitudinal evidence that smokers with a smoking partner are less likely to quit smoking[51], while other findings suggest that being married is associated with reduction in drinking quantity among men but increased drinking quantity among women, with divorce showing the opposite trend[56]. Thus, knowledge of patterns in partner correlations offers an important understanding of the context shaping complex human traits and refines our interpretations of existing results.

While the current paper included many samples with varying characteristics, there are several further research topics related to human mating relationships that we did not directly assess. For example, past research has found that partner similarity for variables such as academic performance/aspirations, relationship involvement[57], political/religious beliefs[34], mental distress, exercise, drinking and smoking[58] may be predictive of relationship longevity. In the same vein, several papers have suggested that similarity on personality traits (for example, happiness) predicts satisfaction and/or relationship longevity[59–62], while others have found that partners' individual characteristics (as opposed to their degree of similarity) are stronger predictors[8,9,63,64]. Finally, some research comparing partner similarity in male–female, female–female and male–male couples has pointed to differences across groups for certain demographic, anthropometric and health-related traits[65–67]. In light of these potential differences, researchers should further explore how sex/gender make-up interacts with partner resemblance in co-parents to affect rearing environments, as well as how sexual and romantic orientation may be associated with different patterns of similarity within partnerships. These avenues of inquiry have applications that extend beyond the downstream genetic and environmental consequences of AM among procreating pairs and may further help illuminate how social forces shape human relationships.

The current study had limitations that we were not able to address. First, the vast majority of samples in our meta-analyses were drawn from Europe and the United States, with a smaller number coming from East Asia; samples residing in Sub-Saharan Africa, South Asia, Latin America and the Caribbean, Southwest Asia and Northern Africa, and Oceania (the last of which is notably less populous than the other regions) were each present in only a handful of studies. Additionally, participants in the UKB were all between the ages of 40 and 69 when originally recruited, are disproportionately healthy, are less likely to live in socioeconomically deprived areas and are less likely to smoke and drink on a daily basis. Typically, such restriction of range should diminish the magnitude of correlations towards zero and the fact that both members of each putative UKB couple (rather than only one) came from this sample may have further exacerbated any effects of such ascertainment on partner similarity. Furthermore, a large majority of those in the UKB are of European ancestry, which is not reflective of the overall human population[68]; relatedly, we were not able to include cross-ancestry pairs in the UKB because of how relatedness was assessed in this sample (see 'Analysis of partner correlations in the UKB'), which may have had an effect on partner correlations for some traits. In addition, despite comprising a non-negligible share of couples and parents throughout time, partners and co-parents who did not mutually select each other, either because of cultural norms around marriage or because of coercion, are unlikely to be proportionally reflected in recent volunteer samples. For the above reasons, findings from both our meta-analysis and partners in the UKB are unlikely to be generalizable to all human populations and time periods. Next, there was a broad range of total sample sizes used to calculate partner correlations across traits, suggesting some degree of imbalance in power across trait analyses. Finally, the results of the present study must be interpreted as cross-sectional estimates of resemblance within dyadic partnerships, and hence are not directly informative with respect to selection mechanisms, longitudinal trends across generations or trends relating to partner similarity across multiple (concurrent or sequential) partnerships within a single individual. Future studies will be necessary to understand the specific processes underlying partner similarity (that is, phenotypic/social/genetic homogamy and convergence), including how these processes have changed over time.

In summary, we have conducted a raw data analysis and a set of meta-analyses of human partner correlations, with the former drawing from over 79,000 putative partner pairs and the latter being based on estimates from millions of partner pairs and over a century of research. Across both our meta-analyses and our study of partners in the UKB, we found the highest estimates of similarity for traits related to substance use, educational attainment and attitudes/behaviours, with smaller correlations for personality, psychological, anthropometric and disorder traits. We also observed high levels of heterogeneity in point estimates across studies for most traits investigated, suggesting that partner resemblance may differ across populations. Research into the resemblance between partners and co-parents provides valuable information about the nature of relationships and cultural norms around human mating, as well as information pertinent to the aetiological roots of disorders and to the interpretation of results from genetically informed designs.

Methods

Literature search for the meta-analysis

For the meta-analytic portion of this paper, we conducted a systematic review of English-language studies that examined concordance rates in partners for the same or very similar complex traits. All included studies were published in peer-reviewed journals on or before 16 August 2022, with no lower limit on when the study could be published and with studies from books being excluded. The analysis was not preregistered and no protocol was prepared as this was not a hypothesis-driven study.

We selected an initial 26 traits on which to conduct our meta-analysis. While partner concordance has been analysed for many traits, we focused on those most studied in the literature as well as some less commonly studied dichotomous traits. First, we searched for words pertaining to the traits of interest (except for height; see below and 'Inclusion and exclusion criteria') in conjunction with 13 terms pertaining to partner correlations and AM (see Supplementary Table 6 for exact search terms used) in the search engines PubMed (using the RISmed package[69] in Rstudio v.1.4.17.17) and ScienceDirect. In addition, Google Scholar was used for the original submission of this manuscript. Although the latter search engine was not used for the most recent literature review, studies that we recorded in the first iteration of our study were preserved even if they did not appear in the corresponding PubMed or ScienceDirect search. We excluded educational attainment (EA) and body mass index (BMI) from our PubMed search because we had already obtained large sample sizes using only ScienceDirect (1,911,720 partner pairs from 86 samples for EA and 123,881 partner pairs from 57 samples for BMI).

Three authors then worked independently to evaluate whether each paper met the relevant criteria for meta-analysis and then manually collected data from relevant publications. One author then double-checked that each report determined to be applicable was indeed eligible for inclusion; next, all relevant findings from eligible reports were recorded in Supplementary Table 1 or 2 and included for meta-analysis. Supplementary Fig. 1a–v display forest plots for every

trait; each includes every correlation, as presented in Supplementary Table 1 or 2, associated with each included sample, with publications ordered by year and colour coded by geographic region(s) of the sample. Citations that were collated for assessment but that did not meet inclusion criteria are shown in Supplementary Table 3 (except for a small number of studies corresponding to excluded traits, which were included in Supplementary Table 2; see 'Dichotomous traits'), along with the reasons for each exclusion.

For each of the traits that we meta-analysed, Supplementary Fig. 2 presents the number of results for each set of search terms and the number of studies included and excluded for each trait, grouped by reason(s) for exclusion, in accordance with PRISMA guidelines[70,71].

Inclusion and exclusion criteria

Our analysis was restricted to studies of co-parents, engaged pairs, married pairs and/or cohabitating pairs, with some studies containing a small number of divorced couples. We refer to the couples or co-parenting dyads as 'partners' and refer to the female/woman vs male/man designations of individuals as 'sex/gender' unless referring to a sample that specifically uses one of these terms. Samples of only or predominantly same-sex/gender partners were also excluded (although a small fraction of some samples were probably composed of such couples by chance); we believe these samples warrant separate analysis because same- and opposite-sex/gender partners have shown different patterns of similarity for some traits[66,67] (see Discussion) and because there is less data on the former. For traits such as personality, we required that measures be self-reported, but we permitted case statuses of psychiatric traits reported by family members or partners due to the low numbers of applicable studies for such traits. Except for studies that ascertained partners for the trait of interest, we excluded studies in which partners were ascertained in a way that might have affected the magnitude of concordance for the trait of interest (for example, concordance for depression in parents of depressed children). We limited our analysis to studies with sample sizes equal to or greater than 100 partner pairs. When sample sizes were reported along with percentages for each cell of a contingency table, we inferred the sample size of each cell by multiplying the percentages by n. When studies reported partner concordance at different waves over time in the same sample, we reported the results from the first wave. For studies conducted in or after 2000, if the n for a sample was neither provided nor inferable, or if we discovered an inconsistency/point of confusion, we contacted the corresponding author(s) for clarification. In total, we contacted authors from nine different studies and five responded. Although we initially gathered data on rare psychiatric disorders, we did not formally meta-analyse the tetrachoric correlations for these traits because too few studies met our inclusion criteria (see Supplementary Tables 2 and 3).

For consistency, when reported concordance rates were stratified by a variable (for example, zygosity of a twin sample), we included results for each stratum as a separate entry. We calculated effect sizes from the data reported in primary studies rather than from other meta-analyses or reviews. However, because partner correlations for height have already been meta-analysed extensively[20], we analysed only samples included in ref. 20 for height in place of conducting a literature review for this trait, pooling results the same way that we did for other continuous traits after eliminating studies that met our exclusion criteria.

When we determined that samples for a given trait in multiple studies overlapped or were likely to overlap on the basis of information provided in the publication, we only used the study that had the largest sample size or that we otherwise determined to be more appropriate. We also excluded samples taken from the UKB because we calculated partner correlations in this sample ourselves as a separate analysis. Finally, we restricted our meta-analysis to traits for which there were at least three samples that met our criteria.

For continuous and ordinal traits, we only included Pearson/interclass correlations and Spearman rank correlations. When a study presented cross-partner contingency tables for each level of an ordinal trait but did not report a concordance statistic, we directly calculated the Spearman rank correlation and its corresponding standard error. Because most studies reported them, we analysed raw correlations when studies reported both the raw correlation and the partial correlation(s) controlling for covariates. However, we included some correlations that controlled for age when the raw correlation was not available because age was a common covariate; in addition, we made occasional exceptions for types of adjustments (for example, whether the participant was a twin) that attempted to correct for heterogeneity induced by the sampling or measurement scheme used in the study (see Supplementary Tables 1 and 2 for covariates associated with each sample). In every other case, we excluded studies that did not report raw partner correlations.

Dichotomous traits. Within the context of ascertained studies that used probands and controls rather than randomly sampling from a population, we excluded those involving probands taken from a clinical setting (a hospital or other treatment facility) when the case status of their partners was determined by a different set of criteria (for example, achieving a certain score on a diagnostic measure but not necessarily having a history of treatment). Furthermore, results from simulation showed that mixing male and female probands with highly discrepant prevalence rates would lead to unacceptable levels of bias. As a result, ascertained studies that did not label discordant partners on the basis of sex/gender and for which such information was not otherwise inferable were eliminated if there was a greater than ~2-fold difference in male and female prevalence rates. Because of possible differences in the strength of calculated concordance based on an all-male proband sample versus that based on an all-female proband sample, we excluded studies that only included single-sex/gender probands. However, when data were available for both a female proband and a male proband (only a single study[72]), estimates based on both probands (female and male) were included as separate results.

We also restricted our meta-analysis to studies with expected contingency table cell frequencies of five or greater for all cells and observed cell frequencies of greater than zero. Four of the traits in our supplementary tables posed a problem because they are rare (bipolar disorder and schizophrenia) or have not been studied in sufficiently large samples (panic disorder and phobia), resulting in underpowered contingency tables in many instances. As a result, there were not enough studies meeting our inclusion criteria to justify formally meta-analysing these four traits, although we show results from studies on these traits that mostly met our criteria in Supplementary Table 2.

We required that either odds ratios (OR), risk ratios (RR), phi coefficients (Φ), contingency tables (from which an OR/RR and tetrachoric correlation can be calculated) or tetrachoric correlations (if the study was not ascertained; see 'Effect size conversion') be reported for dichotomous traits because these effect sizes can be pooled together. No studies that met our inclusion criteria only reported RR and so we do not discuss them further. All effect sizes for dichotomous traits that were not already in the form of a tetrachoric correlation were then converted to a tetrachoric correlation (see 'Effect size conversion').

Effect size conversion

To make estimates across continuous and dichotomous traits more comparable, we converted all effect sizes for studies examining dichotomous traits to tetrachoric correlations if the study was not ascertained and did not report one. For non-ascertained studies that reported contingency tables, we simply calculated the corresponding tetrachoric correlation using the polychor() function from the 'polycor' package[73] in Rstudio v.1.4.17.17.

If the contingency table was unknown but the OR was reported, we first inferred the contingency table using an R function described in the supplementary methods of ref. 29 (which the authors provided to us)

and then used the polychor() function to convert it to a tetrachoric correlation. If the values of the contingency table cells were unknown but the Φ was available, we used the phi2tetra() function from the 'psych' package[74] in Rstudio to convert it to a tetrachoric correlation. Both conversions required inputting male and female prevalence rates, which we based on published male and female prevalence rates for the trait or, if provided or inferable in the study and the sample was not ascertained, the prevalence rates indicated in the study. When gathering prevalence rates from outside sources, we selected those reporting on samples that matched the partner correlation sample as closely as possible with respect to nationality, year and so on. Sample/population prevalence rates for dichotomous traits are reported in Supplementary Table 2 for each applicable meta-analysed sample.

For ascertained studies, it was necessary to use the aforementioned R function from ref. 29, regardless of whether the contingency table was reported, to produce an expected population (non-ascertained) contingency table on the basis of the OR and the population prevalence before converting to a tetrachoric correlation (as described above). This correction was necessary because the case-to-control ratio in an ascertained sample was usually higher than in the general population, and hence simply deriving the correlation on the basis of the uncorrected contingency table would lead to a biased estimate.

Meta-analytic method

We conducted two- or three-level (see below) random-effects meta-analyses on the basis of Fisher *Z*-transformed correlations and their respective variances; we reversed this transformation when reporting final results for ease of interpretation. Note that evaluating within-sample/study normality was not of primary concern in our meta-analysis because of the large minimum sample size we required ($N = 100$ pairs; see 'Inclusion and exclusion criteria'); in addition, any between-sample/study non-normality was unlikely to drastically affect point estimates[75].

We used two-level meta-analysis when none of the publications for a given trait reported multiple effect sizes and used three-level meta-analysis when at least one publication for a given trait reported multiple effect sizes. Two-level random-effects meta-analyses allow for the estimation of heterogeneity due to sampling variance versus that due to true effect size differences between populations from which the samples were drawn. Three-level meta-analyses further break down the true effect size differences to capture heterogeneity resulting from variation between studies versus heterogeneity resulting from variation within studies[52]. Partner correlations estimated in the same study could be more similar due to, for example, measures being taken at the same time or in the same culture, or due to the use of common instruments, even if the samples in a study were collected independently.

We utilized restricted maximum likelihood for both two-level and three-level meta-analyses. For the two-level meta-analyses, we used the rma.uni() function in the 'metafor' package[76] in Rstudio v.1.4.17.17 to specify two levels (standard random-effects modelling). For three-level analyses, we used the rma.mv() function, within which we set individual effect sizes (the *Z*-transformed partner correlations in different samples) to be nested within individual studies for the 'random' argument. For both two-level and three-level meta-analyses, we set the test of the coefficient to follow a *t*-distribution and report two-tailed *P* values that were Bonferroni adjusted for multiple testing, as well as 95% CIs that were Bonferroni adjusted for multiple testing.

For continuous traits, we specified the effect sizes as Fisher *Z*-transformed Pearson or Spearman correlations and specified variances as $\frac{1}{(n-3)}$ and $\frac{1+\frac{r^2}{2}}{n-3}$, respectively[77]. For dichotomous traits, we converted the ascertained contingency table to a tetrachoric correlation corrected for ascertainment, as described above, and used the Fisher *Z*-transformed tetrachoric correlations for pooling in the meta-analyses. We used polychor() to calculate the untransformed standard error ($s.e._{ut}$) for non-ascertained studies examining dichotomous traits. For ascertained studies examining dichotomous traits, we estimated $s.e._{ut}$ by creating 1,000 bootstrapped contingency tables, each of size *n* (the number of partner pairs) and sampling from the study's (raw, ascertained) contingency table with replacement and then estimating the standard deviation of the 1,000 bootstrapped tetrachoric correlations arising from the contingency tables. We then transformed all $s.e._{ut}$ for tetrachoric correlations using the delta method[78], wherein the standard error of the *Z*-transformed tetrachoric correlation is equivalent to $\sqrt{\frac{s.e._{ut}^2}{(1-r^2)^2}}$ and used the square of these transformed standard errors for the variance argument in the rma.uni/rma.mv function. For reporting purposes, all meta-analysed point estimates were back-transformed to their original format (Pearson's correlation or tetrachoric correlation), which were recorded alongside their respective Bonferroni-adjusted 95% CIs.

We estimated two sets of heterogeneity parameters, τ and I^2, at the between-study level and, when applicable, within-study level. τ estimates the standard deviation of the true effect size at a given level. The total Higgins and Thompson's I^2 represents the percentage/proportion of variance not due to sampling error and is the sum of I^2 at the between-study level and I^2 at the within-study level. The heterogeneity statistics are detailed in Table 1. We created funnel plots (Extended Data Fig. 1a–v), which plot Fisher *Z*-transformed correlations on the *x* axis against their standard errors on the *y* axis, to visually inspect whether there was evidence for asymmetry—a potential indicator of publication bias.

Analysis of partner correlations in the UKB

In addition to our meta-analyses, we analysed correlations between inferred male–female partners in the UKB (Application Number 16651) for an initial 140 ordinal, dichotomous and continuous traits using R v.4.2.2 and the University of Colorado Boulder computing cluster resources. We detected putative partners using an approach similar to that of past studies on partner correlations/AM in the UKB[44,53]. Beginning with 502,414 individuals, we first restricted our sample to individuals who reported living with a 'Husband, wife, or partner' and who did not report living with an unrelated roommate (based on data-field 6141), leaving 359,189 individuals. Using a co-location variable that was previously provided by the UKB, we subsequently limited our sample to pairs of individuals who were living at the same address at the time of recruitment, which narrowed the pool down to 200,707 participants. Although the co-location variable is no longer available from the UKB, the code used for detecting putative mate pairs and conducting the UKB analyses is available at https://github.com/JaredBalbona/UKB-AM-MetaAnalysis. Afterwards, using a series of genomic relationship matrices, we removed all pairs of related individuals ($\hat{\pi} \geq 0.05$), leaving 175,250 individuals; importantly, because genomic relationship matrices were calculated within ancestry groups, all cross-ancestry pairs were also removed at this stage. We then kept only pairs who were concordant for the number of people in their household, whether they own or rent their property and their Townsend Deprivation Index (based on data-fields 709, 680 and 189, respectively), leaving 160,738 participants. Same-sex partners (data-field 31) were then removed, leaving 159,998 participants. Because most discrepancies in self-reported household income (data-field 738) were due either to the pairs choosing two adjacent options (for example, one individual choosing '18,000 to 30,999' and the other '31,000 to 51,999') or to non-responses, we removed only pairs whose reported household incomes differed by more than one category, leaving 158,350 individuals. Finally, pairs with age differences of greater than 20 years were removed to minimize the possibility of including stepchild/stepparent pairs, leaving a final sample of 158,148 individuals comprising 79,074 putative mating pairs.

In some cases, we slightly altered the nature of traits by combining information from multiple traits or changing trait coding schemes

(see Supplementary Table 4 for traits that were altered and the UKB descriptions of each trait we included). In line with our meta-analytic methods, we used responses from the initial wave when responses were collected at multiple times for the same trait. For each continuous trait, we eliminated partners containing at least one member with an absolute *z*-score greater than 4 to reduce the number of invalid responses and then calculated both a Pearson correlation and a Spearman rank correlation to provide two different estimates in the event that unusually distributed data biased correlations. For zero-inflated data, both Pearson and Spearman rank correlations provide a slight underestimate of the true underlying correlation of two continuous measures where values below some threshold provide an observation of zero, but Spearman correlations tend to be less biased[79]. For dichotomous traits, we calculated tetrachoric correlations and odds ratios, and for ordinal traits, we calculated only Spearman rank correlations. In addition to the zero-order correlations for UKB partners, we also present partial correlations in Supplementary Table 4 that control for year of birth, whether an individual was born in the British Isles and each individual's first 10 ancestral principal components. To avoid the re-introduction of covariate effects[80], we ranked our ordinal data before residualizing. Of the initial 140 traits, 6 dichotomous traits did not have expected cell frequencies of 5 or greater, and 1 continuous trait was underpowered because of low sample size. These traits were therefore included in Supplementary Table 4 but not in Fig. 2 or Extended Data Fig. 2, where we visualize the point estimates and adjusted CIs for the remaining 133 traits.

Notably, our subsample contains substantially more partners than certain previous research that used inferred partners in the UKB; for example, despite using similar procedures and criteria to detect partners, ref. 44 detected only 18,984 pairs within the UKB. The discrepancy between these two sample sizes can be attributed to several causes, including our sample containing partners of non-European descent, our approach not requiring perfect matches on household income and our use of the co-location variable. In addition, ref. 44 required that partners be concordant for the number of smokers in their household—a question only asked of non-smokers in the UKB, thus eliminating all pairs that contain one or more current smokers. To evaluate the consequences of our criteria discrepancies, we compared our estimates to those reported in ref. 44 using an independent samples *t*-test for all continuous traits that we examined. For each of these traits, we first calculated r_1', the Fisher-transformed partner correlation for n_1 available pairs from the UKB dataset of ref. 44 and r_2', the Fisher-transformed partner correlation for the same trait in the n_2 available pairs unique to our dataset; we then divided the difference between r_1' and r_2' by $\sqrt{\frac{1}{n_1-3}+\frac{1}{n_2-3}}$, which is the square-root of the sum of their estimated variances. As shown in Supplementary Table 5, the subsamples do not differ substantially for the majority of continuous traits analysed in both studies, despite more conservative partner detection criteria in ref. 44.

Reporting summary

Further information on research design is available in the Nature Portfolio Reporting Summary linked to this article.

Data availability

Studies included in the meta-analysis are listed in Supplementary Tables 1 and 2 and cited in Table 1, as well as in the supplementary note; studies excluded from the meta-analysis are listed in Supplementary Table 3. Raw data from the UK Biobank are not publicly available, but summary statistics for most traits are available on the UK Biobank website at https://biobank.ndph.ox.ac.uk/showcase/search.cgi. Note that there is no longer a Field ID corresponding to the co-location variable in the UKB and that the putative partner dataset we created cannot be publicly shared. As such, our UKB partner dataset cannot be directly used/recreated at this time. However, combinations of other variables (for example, inverse distance to the nearest major road) can potentially be used as proxies for co-location[53], in conjunction with the code we have made available, to estimate partner pairs.

Code availability

The code for the UKB analyses can be found at https://github.com/JaredBalbona/UKB-AM-MetaAnalysis.

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Author contributions

T.B.H. contributed to study design, statistical analyses, manuscript writing/editing, assessment of meta-analysed studies, and creation of figures and tables; J.V.B. contributed to study design, statistical analyses, manuscript writing/editing, assessment of meta-analysed studies, and creation of figures and tables; K.N.P. contributed to manuscript editing, assessment of meta-analysed studies, and creation of tables; M.C.K. contributed to study design, statistical analyses, manuscript writing/editing, assessment of meta-analysed studies, and creation of tables.

Competing interests

The authors declare no competing interests.

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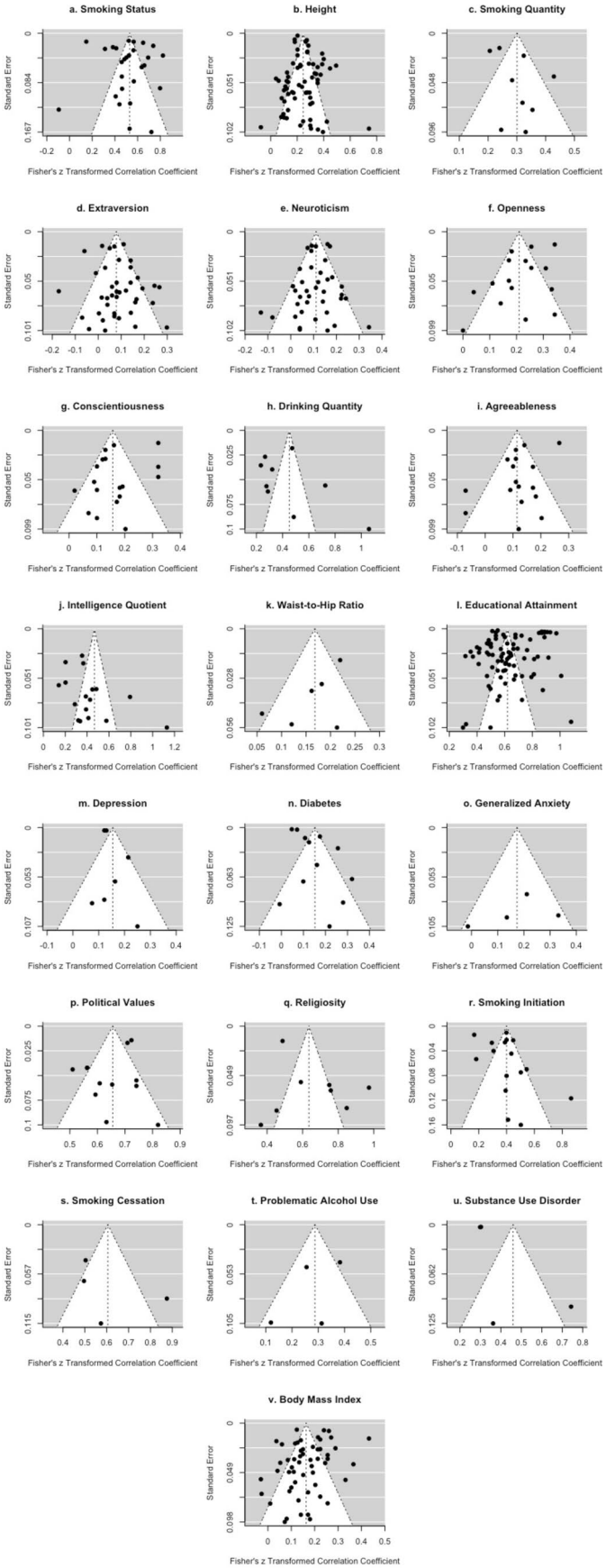

Extended Data Fig. 1 | See next page for caption.

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Extended Data Fig. 1 | Funnel plots for each meta-analysed trait. The funnel plots in Extended Data Fig. 1a Smoking Status, Extended Data Fig. 1b Height, Extended Data Fig. 1c Smoking Quantity, Extended Data Fig. 1d Extraversion, Extended Data Fig. 1e Neuroticism, Extended Data Fig. 1f Openness, Extended Data Fig. 1g Conscientiousness, Extended Data Fig. 1h Drinking Quantity, Extended Data Fig. 1i Agreeableness, Extended Data Fig. 1j Intelligence Quotient, Extended Data Fig. 1k Waist-to-Hip Ratio, Extended Data Fig. 1l Educational Attainment, Extended Data Fig. 1m Depression, Extended Data Fig. 1n Diabetes, Extended Data Fig. 1o Generalized Anxiety, Extended Data Fig. 1p Political Values, Extended Data Fig. 1q Religiosity, Extended Data Fig. 1r Smoking Initiation, Extended Data Fig. 1s Smoking Cessation, Extended Data Fig. 1t Problematic Alcohol Use, Extended Data Fig. 1u Substance Use Disorder, and Extended Data Fig. 1v Body Mass Index are designed to assess possible publication bias for each meta-analysis. Here, the Fisher Z-transformed correlations are plotted against their respective standard errors. For dichotomous traits, standard error was estimated using the delta method (see main text).

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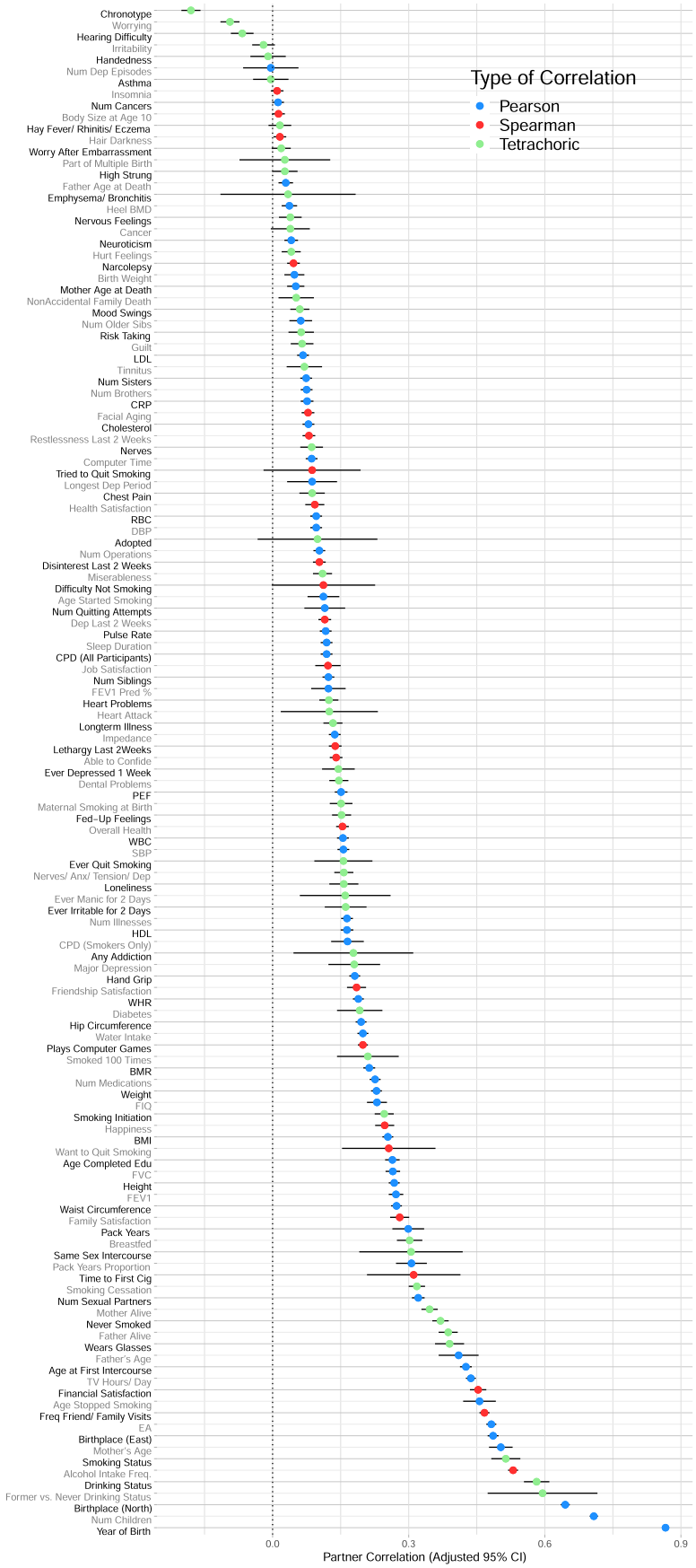

Extended Data Fig. 2 | See next page for caption.

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Extended Data Fig. 2 | Partner correlations and Bonferroni-Adjusted 95% confidence intervals for 133 traits in the UK Biobank. The visualized traits represent partner correlations for all of the adequately-powered UK Biobank traits (out of an original 140). Each estimate is color-coded by the correlation type—Pearson (in blue), Spearman (in red), and Tetrachoric (in green), used for continuous, ordinal, and binary traits, respectively—with the lines depicting the Bonferroni-adjusted 95% confidence interval for each trait. Estimates are based on up to 79,074 pairs; Supplementary Table 4 includes the precise sample size for each trait along with the Bonferroni-adjusted *p*-values associated with the adjusted 95% confidence intervals depicted in this figure. See main text for description of specific analyses. Num Dep Episodes = Number of Depressive Episodes; Heel BMD = Heel Bone Mineral Density (in the form of a t-score); LDL = Direct Low-density Lipoprotein Cholesterol, CRP = C-reactive Protein; RBC = Red Blood Cell (Erythrocyte) Count; DBP = Diastolic Blood Pressure; CPD (All Participants) = Cigarettes per Day (Includes Current, Former, and Never Smokers); FEV1 Pred % = Forced Expiratory Volume in 1-Second (FEV1), Predicted Percentage; PEF = Peak Expiratory Flow; WBC = White Blood Cell (Leukocyte) Count; SBP = Systolic Blood Pressure; HDL = High-density Lipoprotein Cholesterol; CPD (Smokers Only) = Cigarettes per Day (Restricted to Current or Former Smokers); WHR = Waist-to-hip Ratio; BMR = Basal Metabolic Rate; FIQ = Fluid Intelligence Quotient; BMI = Body Mass Index; FVC = Forced Vital Capacity; Time to First Cig = Time to First Cigarette; EA = Educational Attainment.

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Software and code

Policy information about availability of computer code

Data collection	The package "RISMed" (version 2.3.0) (Kovalchik, S. RISmed: Download Content from NCBI Databases. (2021). https://cran.r-project.org/web/packages/RISmed/index.html) was used for data collection within the PubMed search engine for our meta-analyses. No software was used for collecting data from ScienceDirect. Three authors worked independently to evaluate whether each paper met the relevant criteria for meta-analysis and then manually collected data from relevant publications. The raw data for the UKB analysis was obtained via Application Number 16651.
Data analysis	R Studio (version 1.4.1717) and/or R (version 4.2.2) were used for all analyses. We used the packages "psych" (version 2.2.9), "polycor" (version 0.8-0), "dplyr" (version 1.0.10), "metafor" (version 3.8-1), and "RISmed" (version 2.3.0) in R and RStudio. The code for the UKB analyses can be found at: https://github.com/JaredBalbona/UKB-AM-MetaAnalysis. Note that there is no longer a Field ID corresponding to the co-location variable in the UKB, so our UKB partner dataset cannot be directly recreated. This work also used resources from the University of Colorado Boulder Research Computing Group.

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Studies included in the meta-analysis are listed in Supplementary Tables 1 and 2 and cited in Table 1, as well as in the supplementary note; studies excluded from the meta-analysis are listed in Supplementary Table 3. Raw data from the UK Biobank is not publicly available, but summary statistics for most traits are available on the UK Biobank website: https://biobank.ndph.ox.ac.uk/showcase/search.cgi. Note that there is no longer a Field ID corresponding to the co-location variable in the UKB and that the putative partner dataset we created cannot be publicly shared. As such, our UKB partner dataset cannot be directly used/recreated at this time. However, combinations of other variables (e.g., inverse distance to the nearest major road) can potentially be used as proxies for co-location (e.g., see Border et al., 2022, as cited in the text)—in conjunction with the code we have made available—to estimate partner pairs.

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Study description	Our study was a systematic review and quantitative meta-analysis of studies measuring magnitudes of partner concordance for 22 complex traits and a quantitative raw data analysis of putative partner pairs for 133 complex traits in the UK Biobank
Research sample	For the raw data analysis portion of our study, we used the UK Biobank (UKB). The UKB dataset is one of the largest and most detailed datasets in the world, containing extensive health-related information on over 500,000 individuals who were recruited from across the United Kingdom when between the ages of 40 and 69. While the sample is somewhat representative of the UK population, it does skew toward white-identifying individuals of European ancestry (94% in the UKB vs. 87% in the UK as a whole) and toward individuals with higher levels of health, education, and socioeconomic status (which we address in our manuscript). The meta-analysis portion of our study was collected from English language studies from around the world and gathered using PubMed and ScienceDirect search results (more details below). Additionally, Google Scholar was used for the original submission of this manuscript. Though the latter search engine was not used for the most recent literature review, studies that we recorded in the first iteration of our study were preserved even if they did not appear in the corresponding PubMed or ScienceDirect search.
Sampling strategy	For the meta-analysis, all samples included at least 100 pairs of predominately opposite-sex/gender co-habitating, engaged, and married partners, as well as co-parents. The pairs came from a range of backgrounds and could be of any age, race, ethnicity, or nationality, though samples most frequently came from the United States and Europe. Three authors worked independently to determine whether each paper met the relevant criteria for inclusion (described in the manuscript). Information on all included samples is described in Supplementary Tables 1 and 2 and all excluded studies are listed in Supplementary Table 3. Across all traits, samples from the UK Biobank included up to 79,074 putative opposite-sex partner pairs, as this was the total number available in the UKB who met our inclusion criteria (see below). The code for the UKB analyses can be found at: https://github.com/JaredBalbona/UKB-AM-MetaAnalysis.
Data collection	As all of our data were archival/secondary, we did not directly collect any data for this study.
Timing	All included studies were published in peer-reviewed journals on or before August 16, 2022, with no lower limit on when the study could be published. Initial data collection for the meta-analyses in Google Scholar began in October 2019. Then there was a gap in data collection between January 2022 and July 2022, at which point data collection for PubMed and ScienceDirect began. Final data collection for meta-analyses ended on October 2022. For our UK Biobank analyses, data were based on each individuals' characteristics at baseline; UKB data was retrieved for analysis on September 2022.
Data exclusions	For the meta-analysis, samples comprised entirely of same-sex/gender partners were excluded, as we believe these samples warrant separate analysis (for reasons described in our manuscript). For traits such as personality, we required that measures be self-reported, but we permitted case statuses of psychiatric traits reported by family members or partners due to the low numbers of applicable studies for such traits. Except for studies that ascertained partners for the trait of interest, we excluded studies in which partners were ascertained in a way that might have affected the magnitude of concordance for the trait of interest (e.g., concordance for depression in parents of depressed children). We limited our analysis to studies with sample sizes equal to or greater than 100 partner pairs. When samples for a given trait in multiple studies overlapped or were likely to overlap based on information provided in the publication, we only used the study that had the largest sample size or that we otherwise determined to be more appropriate. We also excluded samples taken from the UK Biobank (UKB) because we calculated partner correlations in this sample ourselves as a separate analysis. Additionally, we restricted our meta-analyses to traits for which there were at least three samples that met our criteria. Within the context of ascertained studies (i.e., those that used probands and controls rather than randomly sampling from a population), we excluded those involving probands taken from a clinical setting (i.e., a hospital or other

	treatment facility) when the case status of their partners was determined by a different set of criteria (i.e., achieving a certain score on a diagnostic measure but not necessarily having a history of treatment). Relatedly, ascertained studies that did not label discordant partners based on sex/gender and for which such information was not otherwise inferable were eliminated if there was a greater than ~two-fold difference in male and female prevalence rates. Because of possible differences in the strength of calculated concordance based on an all-male proband sample versus that based on an all-female proband sample, we excluded studies that only included single-sex/gender probands. We also restricted our meta-analyses of dichotomous traits to studies with expected contingency table cell frequencies of five or greater for all cells and observed cell frequencies greater than zero. For the UK Biobank analyses, we excluded individuals who did not report living with a "husband, wife, or partner" and who did not report living with an unrelated roommate. Beginning with this subset of pairs, we subsequently excluded those that reported living at different addresses, had a genetic relatedness coefficient of greater than .05, or that were discordant for the number of people in their household, whether they own or rent their property, and their Townsend Deprivation Index. Because genomic relationship matrices were calculated within ancestry groups, cross-ancestry pairs could not be included. We then removed pairs who were severely discordant for their household income. Finally, we removed same-sex pairs and pairs with age differences of greater than 20 years. Ultimately, this reduced our initial sample of 502,414 individuals to 158,194.
Non-participation	No participants were involved in this study.
Randomization	Participants were not allocated into experimental groups.

Reporting for specific materials, systems and methods

We require information from authors about some types of materials, experimental systems and methods used in many studies. Here, indicate whether each material, system or method listed is relevant to your study. If you are not sure if a list item applies to your research, read the appropriate section before selecting a response.

Materials & experimental systems

n/a	Involved in the study
☒	☐ Antibodies
☒	☐ Eukaryotic cell lines
☒	☐ Palaeontology and archaeology
☒	☐ Animals and other organisms
☒	☐ Human research participants
☒	☐ Clinical data
☒	☐ Dual use research of concern

Methods

n/a	Involved in the study
☒	☐ ChIP-seq
☒	☐ Flow cytometry
☒	☐ MRI-based neuroimaging