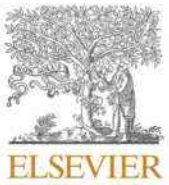
Intelligence 107 (2024) 101871



Contents lists available at ScienceDirect

Intelligence

journal homepage: www.elsevier.com/locate/intell

DNA and IQ: Big deal or much ado about nothing? – A meta-analysis

Florence A.R. Oxley [a,b,*], Kirsty Wilding [a], Sophie von Stumm [a]

[a] Department of Education, University of York, York YO10 5DD, UK
[b] Department of Language and Linguistic Science, University of York, York YO10 5DD, UK

ARTICLE INFO

Keywords:
Polygenic score
Genomics
Intelligence
Meta-analysis
Systematic review

ABSTRACT

Intelligence is polygenic, highly heritable, and predicts wide-ranging life outcomes. Here, we meta-analysed the predictive validity of polygenic scores for intelligence based on the largest available genome-wide association study (or *GWAS*; Savage et al., 2018) for tested, phenotypic intelligence to date. Across 32 estimates from 9 independent samples, which all came from WEIRD countries and were of European ancestry (N_{total} = 452,864), our meta-analytic estimate for the association between polygenic and phenotypic intelligence was $\rho = 0.245$ ($p < .001$, 95 % CI = 0.184–0.307), an effect of medium size. The meta-analytic estimate varied across samples, studies, and phenotypic measures of intelligence, and even after accounting for these moderators, polygenic score predictions remained significantly heterogenous. Our findings support claims that polygenic predictions of intelligence benefit and advance research but their utility in other contexts is yet to be demonstrated.

1. Introduction

Intelligence – the ability to learn, reason, and solve problems – strongly predicts life outcomes relating to education, occupation, and health and wellbeing (Strenze, 2007; Deary, 2012). Twin and family studies have shown that about half of people's differences in intelligence can be attributed to their genetic differences, with the heritability of intelligence increasing across the lifespan from infancy, to childhood, to adolescence, and adulthood (Haworth et al., 2010; Plomin & Deary, 2015; Polderman et al., 2015). Heritability denotes the proportion of individual differences in a phenotypic trait that can be attributed to people's inherited genetic differences. In the wake of the DNA revolution, it has become possible to identify some of the DNA variants that likely drive the heritability of intelligence using genome-wide association studies (GWAS; Plomin & von Stumm, 2018). GWAS signal the strength with which DNA variants across the genome – so-called single-nucleotide polymorphisms (SNPs) – are associated with phenotypic traits of interest. Trait-associated DNA variants can then be aggregated into polygenic scores that index a person's genetic propensity for that trait. To date, nine GWAS of intelligence have been published, reporting polygenic score predictions of phenotypic intelligence in independent samples (Fig. 1).

Seven GWAS, published between 2008 and 2017, included discovery samples of between 7000 and 112,000 participants (Table S2). The mean weighted prediction estimates of the corresponding polygenic scores accounted for less than 1 % of variance in phenotypic intelligence in independent samples (Table S1). Two further intelligence GWAS with samples >250,000, published in 2018, yielded stronger polygenic score predictions, ranging from 4.37 % (Davies et al., 2018) to 4.81 % (Savage et al., 2018). The most predictive GWAS on intelligence to date (Savage et al., 2018) combined eight samples, which were also included in the eight previous intelligence GWAS (see Fig. 1), along with seven new samples (N_{total} = 269,867). The corresponding polygenic score, hereafter IQ_{2018}, accounted for 4.81 % of variance in phenotypic intelligence across four independent population cohorts, with the GWAS' SNP heritability estimate, which represents the theoretical upper bound of the polygenic score prediction, at 19 % (Savage et al., 2018).

The discrepancy between polygenic score predictions, SNP heritability, and twin heritability is known as the 'missing heritability gap' (Manolio et al., 2009; Plomin & von Stumm, 2018). This gap is expected to narrow in the future when larger discovery samples become available, enabling more powerful GWAS that identify more of the DNA variants, including rare ones, that drive the heritability of intelligence (Plomin & von Stumm, 2018; Young, 2019). For example, a GWAS for years spent in full-time education (Lee et al., 2018; N ~ 1.1million) produced polygenic scores that predicted 14.8 % of the variance in school performance and 9.9 % of the variance in intelligence (Allegrini et al., 2019). The latter estimate is twice as high as the prediction from the IQ_{2018} polygenic scores (Savage et al., 2018). Developments in whole genome-sequencing and genomic structural equation modelling may

* Corresponding author at: Department of Language and Linguistic Science, University of York, York YO10 5DD, UK.
E-mail address: florence.oxley@york.ac.uk (F.A.R. Oxley).

https://doi.org/10.1016/j.intell.2024.101871
Received 18 March 2024; Received in revised form 24 September 2024; Accepted 24 September 2024
Available online 19 October 2024

also be key in resolving missing heritability (Grotzinger et al., 2019: Wainschtein et al., 2022).

1.1. IQ_{2018} – the tool for the job or a tool in need of honing?

Applications of polygenic scores for intelligence are hotly debated. Proponents argue that polygenic scores could (a) help to identify children who might benefit from additional learning support early in life, before problems have manifested; (b) enable parents to make informed choices about their children's development and support needs; (c) inform equitable policymaking in education by explicating the potential benefits of personalising learning; and (d) inform future research through discerning environmental effects that can be interpreted as causal from those that are due to genetic and environmental confounding (cf. 'genetic nuture'; Asbury et al., 2021; Harden, 2021; von Stumm & Plomin, 2021; Wang et al., 2021; Wertz et al., 2019). Opponents argue that the modest effect sizes of polygenic score predictions for behavioural and psychological traits render them useless for effectively identifying and supporting children's differential learning needs (Dale, et al., 2020; Howe et al., 2022; Morris, et al., 2020). This argument gathers weight when comparing polygenic and environmental predictors of children's cognitive development, because environments (e.g., household chaos or families' socioeconomic status) offer, at present, stronger prediction effect sizes for developmental outcomes, even after controlling for the prediction of polygenic scores (e.g., von Stumm et al., 2020; von Stumm et al., 2023). Another reason why DNA-based predictions of intelligence are reluctantly received is their potential to enable discrimination and exclusion for genetic reasons. This risk is exacerbated by the common misperception that inherited genetic differences 'determine' individuals' traits and behaviours. DNA-based predictions are, in fact, probabilistic: they are predictions, rather than causal explanations for individual differences in phenotypic development (Plomin & von Stumm, 2022).

Polygenic prediction of psychological traits is rapidly progressing (Howe et al., 2022; Okbay et al., 2022; Plomin & von Stumm, 2022). Here, we report results from meta-analysis and meta-regression models that evaluated the prediction from polygenic scores based on Savage et al.'s (2018) GWAS for phenotypic intelligence IQ_{2018} and explored effects of factors which may moderate this prediction. Savage et al.'s (2018) GWAS' summary statistics enabled the most predictive polygenic scores for intelligence to date and are likely to influence research in this area until even more powerful polygenic scores for intelligence become available (Martschenko et al., 2024; von Stumm and Plomin, 2021). Evaluating IQ_{2018} polygenic scores' performance across samples is key to ascertaining whether and how polygenic prediction of intelligence can benefit science and society. Finding consistent and meaningful effect sizes across samples would suggest that IQ_{2018} is a valuable tool for research, for example in differentiating genetic and environmental pathways of influence (e.g., Wertz et al., 2023). Sizeable, consistent prediction estimates may also recommend IQ_{2018} for use alongside other tools in applied settings, like helping to identify children with learning difficulties. Such uses would corroborate calls for developing safeguarding guidelines for applied use of polygenic prediction (Lewis & Green, 2021). By contrast, inconsistent, weak predictions would suggest that polygenic scores for intelligence are – at least at present – unlikely to be useful tools in research or elsewhere.

1.2. Open practices statement

We analysed data that we extracted from published articles; requests to access the primary data should be directed to the corresponding authors of the relevant publication/s. Our analysis scripts are openly available at https://osf.io/63zmr/, and our data are reported in Table S4.

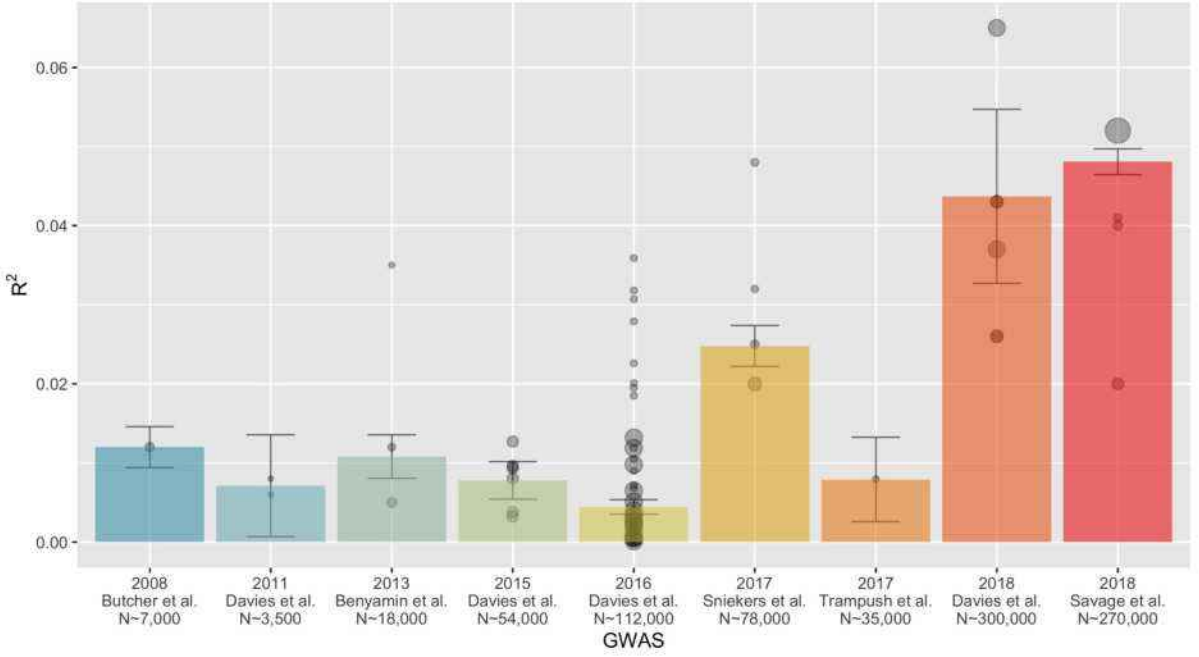


Fig. 1. Polygenic score predictions for phenotypic intelligence across nine GWAS of intelligence (From left to right: Butcher et al., 2008; Davies et al., 2011; Benyamin et al., 2014; Davies et al., 2015; Davies et al., 2016; Sniekers et al., 2017; Trampush et al., 2017; Davies et al., 2018; Savage et al., 2018).
GWAS' year of publication, first author, and discovery sample size are shown along the x-axis. Bars indicate the weighted mean proportion of variance (R^2) in phenotypic intelligence accounted for by the respective GWAS' polygenic scores. Error bars reflect 95 % CIs. Grey circles reflect the highest polygenic score prediction estimate per independent sample and phenotypic intelligence measure, as reported in the respective GWAS publication. Circle size indicates independent sample sizes. For a full breakdown of prediction estimates, measures, and discovery and independent sample sizes for the eight GWAS, see Table S1 (Benyamin et al., 2014; Butcher, Davis, Craig and Plomin, 2008; Davies et al., 2015; Davies et al., 2016; Davies et al., 2011; Sniekers et al., 2017; Trampush et al., 2017).

2. Methods

2.1. Reporting

We report our meta-analysis in compliance with Nosek et al.'s (2015) Transparency and Openness Promotion (TOP) guidelines, Appelbaum et al.'s (2018) Meta-Analytic Reporting Standards (MARS) and Page et al.'s (2021) Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA, see also Moher et al., 2015; Shamseer et al., 2015). The project is pre-registered with the Open Science Framework (https://osf.io/63zmr/).

2.2. Search strategy

Our systematic review identified 11 publications eligible for inclusion, reporting k = 32 prediction estimates across n = 9 independent samples (N_{total} = 452,864). We identified 1703 potentially relevant publications by gathering all citations that Savage et al.'s (2018) GWAS of intelligence had garnered between its publication in June 2018 and May 2023, when we conducted our searches (i.e., forward snowballing). We focused exclusively on Savage et al.'s (2018) GWAS since it yielded the most predictive polygenic scores to date, and subsumed several of the discovery samples used in previous GWAS (see Fig. 1). We identified 1703 publications, using Google Scholar (789 citations), Web of Science (458 citations), and Scopus (456 citations). After exporting these citations to an Endnote 20 library and manually removing 949 duplicates, we screened the remaining 754 unique entries (Fig. S1).

2.3. Screening

We screened these 754 publications using Rayyan – an online tool, designed to support systematic, traceable, reproducible literature screening for single-authored or collaborative reviews (Ouzzani et al., 2016). We included only published, peer-reviewed journal articles reporting original statistical estimates for IQ_{2018} polygenic predictions of phenotypic intelligence, as assessed by validated, reliable psychometric intelligence tests, (e.g., Wechsler Intelligence Scale for Children 4th Edition; Wechsler, 2003). This latter criterion, which all identified publications met, ensured that included estimates were truly comparable (i.e., not apples vs. oranges; Harrer et al., 2021).

The first and second authors independently double-screened 10 % of the publications' abstracts (n = 76), which were selected using the random number generator function (=*RANDBETWEEN(1754)*) in Microsoft Excel (Microsoft Corporation, 2018). Interrater agreement was 97.37 % (n = 74) and the 2.63 % (n = 2) cases of conflict were resolved through discussion and recorded in Rayyan (Ouzzani et al., 2016). Because interrater agreement was >95 %, the first author single-screened all remaining abstracts, of which 38 passed screening. For these, full text articles were downloaded. A further 27 publications were excluded during coding and data extraction because they did not meet our inclusion criteria (Table S3). Table 1 details all publications that were retained for our meta-analysis (all data extracted from these publications are available at https://osf.io/63zmr/).

We note that the UK Biobank samples that were analysed by Li et al. (2020, N=427,306) and Savage et al. (2018, N=195,653) overlap partially. However, we deemed these samples to be sufficiently different to warrant including both for three reasons. First, the two studies selected different subsets of participants from UK Biobank, which differed in their characteristics (e.g., ancestry, relatedness, age, and age range). Second, Li et al. (2020) and Savage et al. (2018) included other, non-overlapping samples (N = 231,653 and N = 74,214, respectively). Third, the studies reported distinct prediction estimates (Li et al. (2020): r = 0.288, R^2 = 8.3 %; Savage et al. (2018): r = 0.221, R^2 = 4.8 %). Further, Li et al.'s (2020) reported effect size is close to the median value of our meta-analytic estimate, which was adjusted for nestedness at the level of samples.

Table 1
Publications included in current meta-analysis.

Publication	Sample	N	Age range (years)
Ajnakina et al. (2022)	ELSA	5,088	50–77
Allegrini et al. (2019)	TEDS	7,026	12-16
Ferraro et al. (2022)	EU-GEI	1,263	18–64
Genç et al. (2021)	FPRUB	518	18–75
Lett et al. (2019)	IMAGEN	1,651	13.54–14.33
Li et al. (2020)	UKB	427,306	40–69
Malanchini et al. (2021)	TEDS	2,780	23.29–26.41
Mitchell et al. (2022)	BLTS	2,335	15.7–28.9
Selzam et al. (2019)	TEDS	3,138	11–12
Tsapanou et al. (2023)	RAN-CR	168	2-17
Yap et al. (2021)	AAB	1,591	21.6-73.7

In order of appearance: ELSA = English Longitudinal Study of Aging; TEDS = Twins Early Development Study; EU-GEI = European Network of National Schizophrenia Networks Studying Gene-Environment Interactions Project; FPRUB = Faculty of Psychology at Ruhr University Bochum sample; IMAGEN = Imaging Genetics Consortium; UKB = UK Biobank; BLTS = Brisbane Longitudinal Twin study; RAN-CR = Reference Ability Neural Network and Cognitive Reserve studies; AAB = Australian Autism Biobank. *N* refers to the samples from which reported estimate was drawn and not the full cohort size.

2.4. Data extraction

We coded and extracted the following data from the eligible publications: i) measure of intelligence used (i.e., the name of psychometric test); ii) sample age/ age range; iii) sample sex distribution; iv) sample ethnicity; v) sample nationality; vi) reported estimate; vii) reported p-value; viii) reported standard error; ix) reported estimate type (e.g., r, β, R^2); and x) statistical analysis conducted (e.g., regression, correlation). All coded and extracted data were reported either within the main text or supplementary information (SI) of the retained publications; in one case, data detailing adult participants' age were obtained by contacting the original studies' authors (Yap et al., 2021; details in Table S4).

2.5. Data harmonisation

Where possible, we extracted estimates as correlation coefficients (k = 5) or beta regression coefficients (k = 17) from models with no other covariates besides age, sex, and principal components (PCs; to adjust for population stratification). Meta-analysing regression betas can be problematic, when predictors' covariates are inter-correlated (Roth et al., 2018). However, this issue does not apply here because the polygenic score covariates of age, sex, and PCs are independent of each other (S1). Where publications reported estimates as R^2 values (k = 10), we transformed these into correlation coefficients using an online effect size converter (https://www.escal.site; Table S4). We then transformed all coefficients to Fisher's Z, in line with meta-analysis modelling conventions (Alexander et al., 1989).

2.6. Meta-analysis

We conducted a multi-level random effects meta-analysis (MREM) to generate a pooled association estimate and to assess the presence, extent, and source of heterogeneity between the studies' reported estimates. A nested design was appropriate to control for non-independence in the data. Seven estimates were extracted from three publications using different subsets of the TEDS sample (Allegrini et al., 2019; Malanchini et al., 2021 and Selzam et al., 2019), and twenty-nine estimates were extracted from eight publications. Of these eight publications, three reported separate estimates for different age groups (Ajnakina et al., 2022; Allegrini et al., 2019; Yap et al., 2021); four for different psychometric tests (Ajnakina et al., 2022; Ferraro et al., 2022; Genç et al., 2021; Yap et al., 2021); and three for different factors of intelligence (Ajnakina et al., 2022; Genç et al., 2021; Malanchini et al., 2021; Mitchell et al., 2022).

Our MREM had three levels: i) participant-level variance; ii) within-sample variance, and iii) between-sample variance. The third level in multi-level meta-analyses is often specified as publication, but here we specified the third level as sample, because this better captured the source and extent of nestedness in our meta-analysis (Harrer et al., 2019). We used the R Studio package *metafor* (R Core Team, 2019; Viechtbauer, 2010) to model our data. We created figures using *metafor* and the *wesanderson* colour palette (Ram & Wickham, 2018). The function *rma.mv()* within *metafor* returns a pooled association estimate, weighted by estimate sample size (Table S4), and calculates Q and σ^2 statistics to assess heterogeneity. We used the *metafor* companion package *dmetar* (Harrer et al., 2019) to generate I^2 statistics. While the Q statistic indicates whether there is significant heterogeneity (Huedo-Medina et al., 2006), it cannot identify its source. We calculated I^2 statistics for each level in the model to estimate the proportion of heterogeneity attributable to each. A higher I^2 statistic at the between-sample level (iii) than at level (i) and (ii), for instance, would indicate that most of the observed heterogeneity resulted from systematic differences between the independent samples. An I^2 statistic of >50 % at any level indicates substantial heterogeneity at that level (Huedo-Medina et al., 2006). The σ^2 statistics in a multi-level meta--analysis replaces the τ^2 statistic in a one-level random effects meta-analysis (Harrer et al., 2019). That is, σ^2 statistics provide a measure of within- and between-cluster variance which, unlike I^2 statistics, is not sensitive to meta-analytic sample size. We ran ANOVAs to compare model fits after restricting variance within- and between-samples to zero in turn.

2.7. Meta-regression

Next, we conducted a meta-regression to explore effects of methodological variable/s that might explain heterogeneity observed in our meta-analysis. Using *metafor*, we built a model including the domain of intelligence assessed, samples' mean age at the time of phenotypic assessment, samples' age range at phenotypic assessment, and samples' nationality. Continuous moderators were centred to facilitate interpreting the intercept. Table S4 and Models 1a-2d (see SI) detail the levels within these moderators.

2.8. Risk of publication bias

We tested for evidence of publication bias in three ways. First, we examined funnel plots to gauge the direction and extent of deviation of each reported estimate from the pooled estimate, before and after controlling for the moderators outlined above. Second, we performed Egger's regression test using estimates' standard errors in a single moderator meta-regression to assess (a)symmetry in the distribution of our included estimates. Third, we conducted a *p*-curve analysis, which visualises the proportion of estimates that passed different *p*-thresholds. A clustering of low *p*-values (i.e., $p \sim 0.01$) indicates that the effect under investigation is truly significant. A clustering of high *p*-values (i.e., $p \sim 0.05$) is suggestive of selective reporting (i.e., *p*-hacking).

3. Results

3.1. Descriptive statistics

Eleven publications met our inclusion criteria, which included k = 32 estimates across nine independent samples, ranging in size from $N = 168$ to $N = 427{,}306$ individuals (N_{total}=452,864; Table S2, Fig. 2). All samples were recruited from WEIRD countries (Western, Educated, Industrialised, Rich, and Democratic) and comprised participants of European ancestry. Participants were between 2 and 77 years old at the time of intelligence assessment, with 96.8 % of participants being adults (aged 18 years or over), and 3.2 % being children (under 18 years of age). Across publications, polygenic score predictions were reported for six

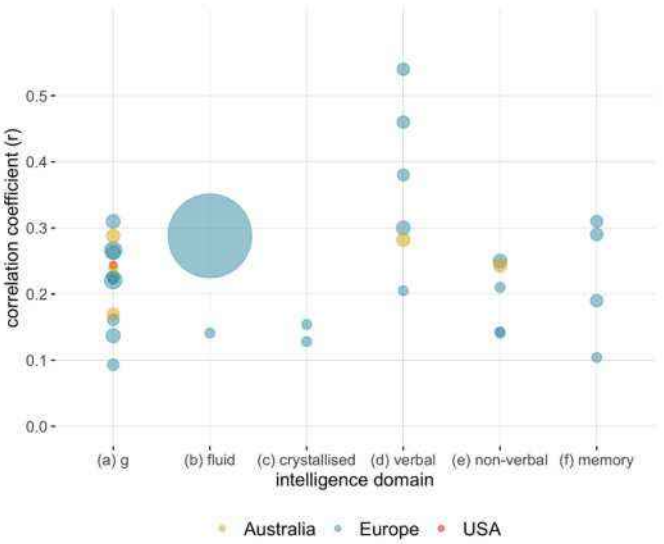


Fig. 2. Polygenic score prediction estimates across samples' countries of origin and intelligence domains.
Correlation coefficients are shown after data harmonisation but before Fisher's z-transformation. The bubble sizes index sample sizes; the bubble colours index samples' nationality.

domains of intelligence, including general intelligence or *g* (k = 13); fluid intelligence (k = 2); crystallised intelligence (k = 2); verbal intelligence (k = 6); non-verbal intelligence (k = 5); and memory (k = 4) (Table S4).

3.2. Multi-level meta-analysis

All included estimates showed significant, positive associations between IQ_{2018} polygenic scores and phenotypic intelligence, ranging from 0.090 to 0.600 after Fisher's z-transformation (Fig. 2). Our multi-level meta-analysis returned a pooled prediction estimate of $\rho = 0.245$ ($p < .001$, 95 % CI = 0.184–0.307), indicating a positive association of medium effect size (Funder & Ozer, 2019; Figs. 3, 4, Table S6).

The *Q*-statistic of 593.95 ($p < .001$) reflected significant heterogeneity between estimates (Table S6). The I^2 statistics suggested that less than half of the observed heterogeneity occurred between samples ($I^2_{Level3} = 44.9$ %), just over half was within samples ($I^2_{Level2} = 52.5$ %), and a much smaller proportion at the participant level ($I^2_{Level1} = 2.6$ %). The estimated variance between samples was $\sigma^2 = 0.005$ and that within samples was $\sigma^2 = 0.006$. ANOVAs indicated significant heterogeneity at both the between- ($p = .002$) and within-sample levels ($p < .001$). Significant heterogeneity at both levels indicates possible moderating effects of between-sample differences (e.g., samples' age/age range, samples' nationality) and within-sample differences (e.g., the domain of phenotypic intelligence investigated in distinct publications using the same sample, Table S4).

The Egger's regression test did not suggest publication bias ($r = -1.127$, $p = .503$; SI, Model 3), although the funnel plot showed a slightly asymmetric distribution (Fig. S3, pane A). Our *p*-curve analysis indicated that included estimates were truly significant (full *p*-curve: $Z = -31.73$, $p < .001$, half *p*-curve: $Z = -30.73$, $p < .001$), and that evidential value was neither inadequate nor absent (full *p*-curve $Z = 23.85$, $p = .999$, half *p*-curve $Z = 25.14$, $p = .999$) (Fig. S2, Table S5). It is therefore unlikely that *p*-hacking influenced the distribution of the included estimates. Instead, it is likely that the observed heterogeneity can be attributed to between- and within-sample differences, which we tested in our meta-regression.

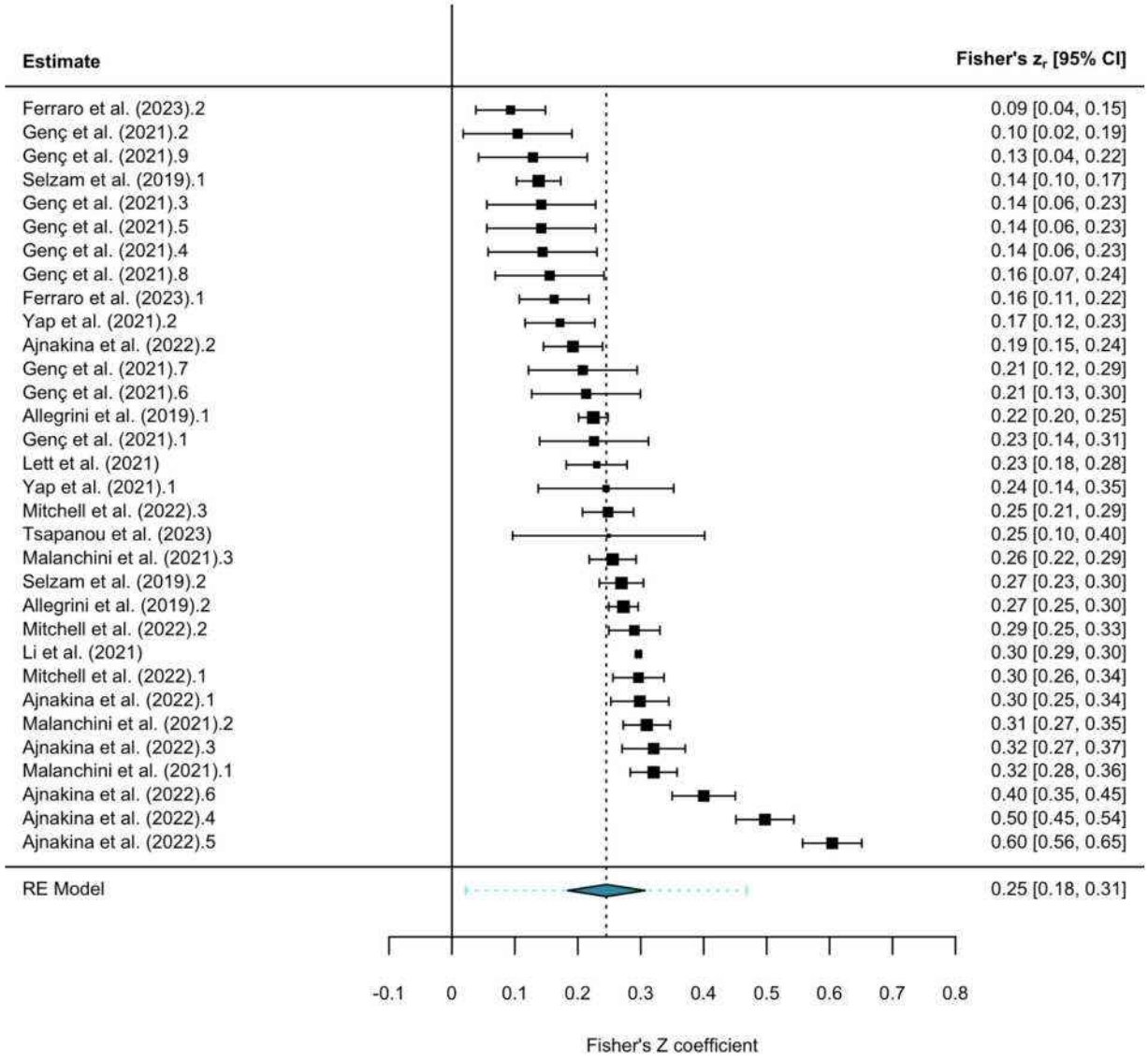


Fig. 3. Forest plot for meta-analysis of IQ_{2018} polygenic predictions of phenotypic intelligence.
Correlation coefficients are shown after data harmonisation and Fisher's *Z*-transformation. Squares represent individual estimates, square size indexes sample size, and horizontal bars indicate 95 % CIs. Included estimates are ordered by effect size (i.e., Fisher's Z). The 'Estimate' column shows the publication from where the estimate was coded (cf. Table S4 for estimate and publication details). Diamond and dotted line indicate pooled effect size with 95 % CI. Estimates falling to the right of the solid vertical line represent positive associations between IQ_{2018} and phenotypic intelligence.

3.3. Multi-level regression

We next built single-moderator multi-level meta-regression models testing for moderating effects of sample mean age at phenotypic assessment, sample age range at phenotypic assessment, sample nationality, and the intelligence domain (SI, Models 2a-d). Only intelligence domain (SI, Model 2a) and sample age range (SI, Model 2b) proved significant, and so these were included in our final Model 2 (see SI), with general intelligence (*g*) (vs the five other intelligence domains) as the reference group. The meta-regression's pooled estimate for general intelligence was $\rho = 0.237$ ($p < .001$, 95 % CI = 0.176–0.298), equivalent to an effect of medium size (Funder & Ozer, 2019, Table 2, Fig. S4).

IQ_{2018} predicted verbal intelligence significantly more strongly than general intelligence (*g*, the reference domain, see Table 2). However, no significant differences in the strengths of associations were seen between general intelligence and the other intelligence domains. Assuming a normal distribution of IQ with a mean of 100 and a SD of 15, a one-SD increase in IQ_{2018} polygenic scores equated to an increase of 3.56 IQ points for general intelligence ($p < .001$), and 4.92 IQ points for verbal intelligence ($p = .029$) in European participants. Sample age range at the time of assessment approached but did not pass the threshold for significance ($p = .066$).

The funnel plot for our meta-regression (Fig. S3, pane B) shows a more symmetrical distribution than that for our meta-analysis (Fig. S3, pane A), indicating that our moderators accounted for some of the heterogeneity. The *Q* statistic was 259.25 ($p < .001$), a reduction in heterogeneity by 56 % compared to the model without moderators. The I^2 statistics for our meta-regression model indicated that, of this remaining heterogeneity, half occurred between samples ($I^2_{Level3} = 49.46$ %), just under half within samples ($I^2_{Level2} = 45.51$ %), and $I^2_{Level1} = 5$ % at the participant level. The estimated variance between and within samples was $\sigma^2 = 0.003$ ($p = .002$ and $p < .001$, respectively). Thus, the intelligence domain tested explained some of the heterogeneity observed in our meta-analysis, but significant unexplained heterogeneity remained, suggesting moderating effects of other factors not explored here.

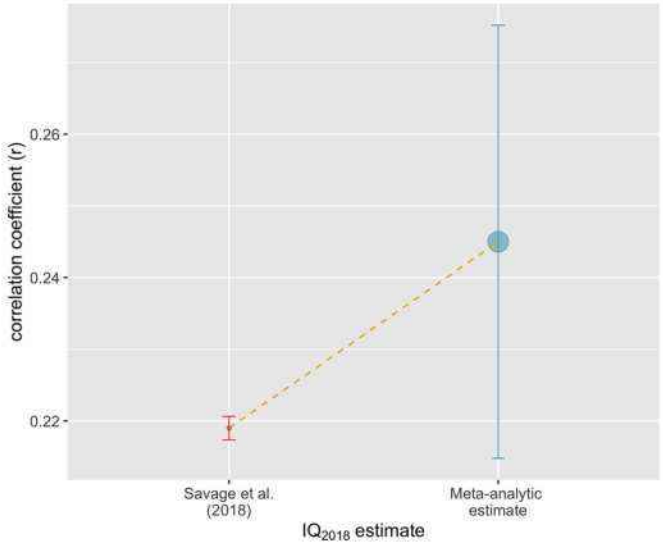


Fig. 4. Polygenic score predictions of phenotypic intelligence comparing Savage et al.'s (2018) estimate and the pooled estimate from the present meta-analysis.
Error bars reflect 95 % CIs. Dot size reflects sample size.

Table 2
Multi-level meta-regression estimates for IQ_{2018} polygenic prediction of phenotypic intelligence.

	estimate	*p-value*	*95 % CI$_{lower}$*	*95 % CI$_{upper}$*
Intercept (g)	0.237	<0.001***	0.176	0.298
Crystallised intelligence	−0.026	0.685	−0.156	0.104
Fluid intelligence	0.013	0.842	−0.116	0.141
Memory	−0.093	0.076	−0.195	0.012
Non-verbal intelligence	0.001	0.985	−0.081	0.083
Verbal intelligence	0.091	0.029*	0.010	0.172
Sample age range	−0.002	0.066	−0.004	0.000

The reference group (intercept) is the meta-regression coefficient for IQ_{2018} polygenic score prediction of general intelligence in European ancestry participants. *** $p \leq .001$; * $p \leq .05$.

4. Discussion

4.1. Contributions and implications

Our findings make three main contributions to the debate surrounding the utility of polygenic scores for intelligence in research, practice, and policy.

First, IQ_{2018} predicted phenotypic intelligence with medium effect size ($\rho = 0.245$), which approximates the pooled estimate reported in the original GWAS (i.e., a weighted mean correlation of $r = 0.219$) and thus, substantiated the robustness of IQ_{2018}'s overall prediction across independent samples. The effect size of our meta-analytic estimate can be interpreted as having explanatory and practical use (Funder & Ozer, 2019) but it does not allow for meaningful statements about individuals (Möttus, 2022). Our pooled estimate varied across studies; there was significant heterogeneity both within and between independent samples. The moderators included in our models accounted for over half this heterogeneity. Specifically, polygenic score predictions varied by domain of intelligence assessed, being significantly stronger for verbal intelligence than for general intelligence (*g*). Translated into IQ point effect sizes, a Standard Deviation increase in polygenic scores was associated with gains of 3.6 IQ points in general intelligence, and of almost 5 IQ points – a third of a Standard Deviation in IQ – in verbal intelligence. Verbal intelligence shows more assortative mating (~0.50) than general (~0.40) or non-verbal intelligence (~0.30), which likely contributes to boosting its heritability relative to that of other intelligence domains (Mascie-Taylor, 1989; Plomin & Deary, 2015). Not finding significant differences in polygenic score predictions for domains other than verbal intelligence suggests that IQ_{2018} is too blunt a predictor to fully discriminate between domain-specific cognitive factors. Future GWAS targeting specific intelligence domains separately could help discern genetically intelligence factors at the domain level (i.e., second stratum of the Cattell-Horn-Carroll hierarchical model; McGrew, 2009), which may be key to creating profiles of cognitive strengths and weaknesses in applied settings, for example for clinical diagnoses or personalising education (Procopio et al., 2022). Although we modelled moderator effects and our meta-analytic sample comprised only WEIRD populations, significant heterogeneity remained, suggesting that other, untested factors may have caused the observed differences in prediction estimates. Here, insufficient data were available to explore other potentially moderating factors like sex, socio-economic status, country of origin, or educational background, which were associated with individual differences in intelligence test scores in previous studies (e.g., Alves et al., 2016; Jiang et al., 2020; Pinto & Kühnel, 2020; Ritchie & Tucker-Drob, 2018; Weiss & Saklofske, 2020). Future research must prioritise elucidating the sources of residual heterogeneity of polygenic score predictions of intelligence before IQ_{2018} can be considered ready for application at the individual level in non-research contexts (e.g., policymaking, clinical practice, personalising education).

Our second finding was that IQ_{2018} predictions of intelligence have only been tested and reported for European ancestry samples from WEIRD countries, confirming that there is a persistent Eurocentric bias in behavioural genetic and genomic research (Mills & Rahal, 2019; Table S4). Arguably due to practical, financial, and ethical issues, only a quarter of the world's population is represented by the samples included in GWAS thus far (Martin et al., 2019). Polygenic scores from European ancestry GWAS discovery samples predict phenotypic outcomes more strongly in independent samples of European than of mixed or non-European ancestry, with the lowest estimates reported for African ancestry samples (e.g., Duncan et al., 2019; Ruan et al., 2022). There is likely also a difference in polygenic score prediction strengths for intelligence between ancestries, suggesting that IQ_{2018} cannot be directly extended to predicting phenotypic traits in mixed or non-European ancestry populations (Henrich et al., 2010). Our meta-analysis underscores the necessity of efforts to address the Eurocentric bias in genomics, some of which are already underway but have yet to come to fruition (Mills & Rahal, 2019).

Our third contribution concerns the 'missing heritability gap' (Manolio et al., 2009; Plomin & von Stumm, 2018). Our meta-analytic estimate suggests that IQ_{2018} accounts for approximately 6 % of variance in intelligence across domains – slightly higher than the 4.81 % of variance in general intelligence that it explained across independent samples in Savage et al.'s (2018) original GWAS. However, our estimate falls far below both the GWAS' SNP heritability estimate for intelligence of 19 % (i.e., the upper bound of the polygenic score prediction; Savage et al., 2018) and the twin and family study heritability estimate for intelligence of about 50 % (Haworth et al., 2010; Polderman et al., 2015). Thus, IQ_{2018} likely only captures a fraction of the inherited DNA variants underpinning the heritability of intelligence. We note that our meta-analysis did not compare between- and within-family polygenic score prediction estimates. Because between-family polygenic score predictions are confounded by gene-environment correlations, they tend to be higher than within-family estimates (e.g., comparing dizygotic twins; Selzam et al., 2019). Within-family polygenic score prediction effects for cognitive traits are, on average, about half the size of those between families, a difference that is largely attributable to environmental factors (e.g., families' socioeconomic status; Okbay et al., 2022; Selzam et al., 2019; Wertz et al., 2023). It is therefore likely that only half of our meta-analytic IQ_{2018} prediction estimate reflects potential causal genetic effects on intelligence that are independent of the environment. IQ_{2018} polygenic scores may be a useful tool for research seeking to pinpoint

environmental influences on intelligence that are independent of genetic confounding without the need to collect data from twins or other family design studies. However, as long as their prediction for intelligence is weaker than and confounded by environmental factors (Protzko et al., 2013; Ritchie & Tucker-Drob, 2018; Selzam et al., 2019), IQ_{2018} polygenic scores are of limited practical use in applied settings, such as personalising education in schools.

4.2. Limitations

The key limitations of our meta-analyses stem from the extant research literature on polygenic predictions of intelligence. First, disentangling gene-environment interplay in intelligence was not possible because the publications that met our inclusion criteria reported between- but not within-family estimates. We therefore could not empirically discern the variance that is likely due to direct, causal genetic influences from the variance resulting from gene-environment correlations (e.g., polygenic score correlations with the rearing environment; see Selzam et al., 2019; Okbay et al., 2022). Second, several publications in our meta-analysis reported only a single estimate for samples comprising individuals of wide ranges of ages. Although intelligence is known to be differentially heritable across the lifespan (Haworth et al., 2010; Plomin & Deary, 2015), there was insufficient data to compare IQ_{2018}'s predictive validity at different life stages (e.g., childhood, adolescence, adulthood). Since twin studies have shown that intelligence becomes increasingly heritable with age (Haworth et al., 2010; Plomin & Deary, 2015), we might hypothesise that IQ_{2018} predictions are stronger in older individuals – another potential source of the heterogeneity in polygenic score predictions of intelligence.

5. Conclusion

We found that polygenic scores for intelligence – IQ_{2018}, based on Savage et al.'s (2018) GWAS – predicted phenotypic intelligence with medium effect size across 32 estimates from nine independent samples. Our meta-analytic estimate of $\rho = .245$ ($\sim R^2 = 6\%$) was heterogenous across studies. Substantial heterogeneity remained after adjusting for a priori identified moderators (e.g., intelligence domain), suggesting that other, unobserved factors affect IQ_{2018} predictions. We conclude that IQ_{2018} polygenic scores may be useful tools for research seeking to identify the influence of specific environmental factors on intelligence, independent of genetic confounding. At the same time, our findings offer little support for claims of the imminent practical value of IQ_{2018} polygenic scores in policymaking, clinical practice, or parenting and personalising education. Such practical value may, however, be realised in the future, if summary statistics from GWAS with larger discovery samples for differentiated cognitive phenotypes enable creating more powerful polygenic scores. In this case, safeguarding will be needed to ensure the ethical use of DNA-based predictions for intelligence and other phenotypes, to maximise their benefits and minimise their risks.

CRediT authorship contribution statement

Florence A.R. Oxley: Writing – original draft, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Kirsty Wilding:** Writing – review & editing, Validation, Methodology. **Sophie von Stumm:** Writing – review & editing, Validation, Supervision, Methodology, Funding acquisition, Conceptualization.

Declaration of competing interest

None.

Data availability

Our data and code are available at https://osf.io/63zmr/.

Acknowledgements

This work was supported by a Nuffield Foundation grant awarded to SvS (EDO/44110). SvS supported by fellowships from the Jacobs Foundation (2022–2027) and the Paris Institute of Advanced Study (2023–2024) during the writing of this article. KW was supported by a PhD Scholarship (2021–2024) from the Department of Education at the University of York. The data reported in this study are available in the Open Science Framework at https://osf.io/63zmr/.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.intell.2024.101871.

References

Ajnakina, O., Murray, R., Steptoe, A., & Cadar, D. (2022). The long-term effects of a polygenetic predisposition to general cognition on healthy cognitive ageing: Evidence from the English longitudinal study of ageing. *Psychological Medicine, 1–9*. https://doi.org/10.1017/s0033291721004827

Alexander, R. A., & Scozzaro, M. J. (1989). Statistical and empirical examination of the chi-square test for homogeneity of correlations in meta-analysis. *Psychological Bulletin, 106*(2), 329–331. https://doi.org/10.1037/0033-2909.106.2.329

Allegrini, A. G., Selzam, S., Rimfeld, K., von Stumm, S., Pingault, J. B., & Plomin, R. (2019). Genomic prediction of cognitive traits in childhood and adolescence. *Molecular Psychiatry, 24*(6), 819–827. https://doi.org/10.1038/s41380-019-0394-4

Alves, A., Martins, A., & Almeida, L. S. (2016). Interactions between sex, socioeconomic level, and Children's cognitive performance. *Psychological Reports, 118*(2), 471–486. https://doi.org/10.1177/0033294116639428

Appelbaum, M., Cooper, H., Kline, R. B., Mayo-Wilson, E., Nezu, A. M., & Rao, S. M. (2018). Journal article reporting standards for quantitative research in psychology: The APA publications and communications board task force report. *American Psychologist, 73*(1), 3. https://doi.org/10.1037/amp0000191

Asbury, K., McBride, T., & Rimfeld, K. (2021). *Genetics and early intervention*. Early Intervention Foundation.

Benyamin, B., Pourcain, B., Davis, O. S., Davies, G., Hansell, N. K., Brion, M. J., & Visscher, P. M. (2014). Childhood intelligence is heritable, highly polygenic and associated with FNBP1L. *Molecular Psychiatry, 19*(2), 253–258. https://doi.org/10.1038/mp.2012.184

Butcher, L. M., Davis, O. S., Craig, I. W., & Plomin, R. (2008). Genome-wide quantitative trait locus association scan of general cognitive ability using pooled DNA and 500K single nucleotide polymorphism microarrays. *Genes, Brain and Behavior, 7*(4), 435–446. https://doi.org/10.1111/j.1601-183X.2007.00368.x

Dale, P. S., von Stumm, S., Selzam, S., & Hayiou-Thomas, M. E. (2020). Does the inclusion of a genome-wide polygenic score improve early risk prediction for later language and literacy delay? *Journal of Speech, Language, and Hearing Research, 63*(5), 1467–1478. https://doi.org/10.1044/2020_JSLHR-19-00161

Davies, G., Armstrong, N., Bis, J. C., Bressler, J., Chouraki, V., Giddaluru, S., … Deary, I. J. (2015). Genetic contributions to variation in general cognitive function: A meta-analysis of genome-wide association studies in the CHARGE consortium (N= 53 949). *Molecular Psychiatry, 20*(2), 183–192. https://doi.org/10.1038/mp.2014.188

Davies, G., Lam, M., Harris, S. E., Trampush, J. W., Luciano, M., Hill, W. D., … Deary, I. J. (2018). Study of 300,486 individuals identifies 148 independent genetic loci influencing general cognitive function. *Nature Communications, 9*, 2098. https://doi.org/10.1038/s41467-018-04362-x

Davies, G., Marioni, R. E., Liewald, D. C., Hill, W. D., Hagenaars, S. P., Harris, S. E., … Deary, I. J. (2016). Genome-wide association study of cognitive functions and educational attainment in UK biobank (N= 112 151). *Molecular Psychiatry, 21*(6), 758–767. https://doi.org/10.1038/mp.2016.45

Davies, G., Tenesa, A., Payton, A., Yang, J., Harris, S. E., Liewald, D., … Deary, I. J. (2011). Genome-wide association studies establish that human intelligence is highly heritable and polygenic. *Molecular Psychiatry, 16*(10), 996–1005. https://doi.org/10.1038/mp.2011.85

Deary, I. J. (2012). Intelligence. *Annual Review of Psychology, 63*, 453–482. https://doi.org/10.1146/annurev-psych-120710-100353

Duncan, L., Shen, H., Gelaye, B., Meijsen, J., Ressler, K., Feldman, M., Peterson, R., & Domingue, B. (2019). Analysis of polygenic risk score usage and performance in diverse human populations. *Nature Communications, 10*(3328). https://doi.org/10.1038/s41467-019-11112-0

Ferraro, L., Quattrone, D., La Barbera, D., La Cascia, C., Morgan, C., Kirkbride, J., & Velthorst, E. (2022). First-episode psychosis patients who deteriorated in the premorbid period do not have higher polygenic risk scores than others: A cluster analysis of EU-GEI data. *Schizophrenia Bulletin, 49*(1), 218–227. https://doi.org/10.1093/schbul/sbac100

Funder, D. C., & Ozer, D. J. (2019). Evaluating effect size in psychological research: Sense and nonsense. *Advances in Methods and Practices in Psychological Science, 2*(2), 156–168. https://doi.org/10.1177/2515245919847202

Genç, E., Schlüter, C., Fraenz, C., Arning, L., Metzen, D., Nguyen, H. P., … Ocklenburg, S. (2021). Polygenic scores for cognitive abilities and their association with different

aspects of general intelligence—A deep phenotyping approach. *Molecular Neurobiology, 58*(8), 4145–4156. https://doi.org/10.1007/s12035-021-02398-7
Grotzinger, A. D., Rhemtulla, M., de Vlaming, R., Ritchie, S. J., Mallard, T. T., Hill, W. D., … Tucker-Drob, E. M. (2019). Genomic structural equation modelling provides insights into the multivariate genetic architecture of complex traits. *Nature Human Behavior, 3*, 513–525. https://doi.org/10.1038/s41562-019-0566-x
Harden, K. P. (2021). *The genetic lottery: Why DNA matters for social equality*. Princeton University Press.
Harrer, M., Cuijpers, P., Furukawa, T., & Ebert, D. D. (2019). dmetar: Companion R Package For The Guide 'Doing Meta-Analysis in R'. R package version 0.1.0. http://dmetar.protectlab.org/.
Harrer, M., Cuijpers, P., Furukawa, T. A., & Ebert, D. D. (2021). *Doing Meta-analysis with R: A hands-on guide*. Boca Raton, FL and London: Chapman & Hall/CRC Press. https://bookdown.org/MathiasHarrer/Doing_Meta_Analysis_in_R/.
Haworth, C. M. A., Wright, M. J., Luciano, M., Martin, N. G., De Geus, C., Van Beijsterveldt, C. E. M., & Plomin, R. (2010). The heritability of general cognitive ability increases linearly from childhood to young adulthood. *Molecular Psychiatry, 15*, 1112–1120. https://doi.org/10.1038/mp.2009.55
Henrich, J., Heine, S. J., & Norenzayan, A. (2010). The weirdest people in the world? *The Behavioral and Brain Sciences, 33*(2–3), 61–83. https://doi.org/10.1017/S0140525X0999152X
Howe, L. J., Nivard, M. G., Morris, T. T., Hansen, A. F., Rasheed, H., & Cho, Y. (2022). … Giannelis, A. (2022). Within-sibship genome-wide association analyses decrease bias in estimates of direct genetic effects. *Nature Genetics, 54*, 581–592. https://doi.org/10.1038/s41588-022-01062-7
Huedo-Medina, T. B., Sánchez-Meca, J., Marín-Martínez, F., & Botella, J. (2006). Assessing heterogeneity in meta-analysis: Q statistic or I2 index? *Psychological Methods, 11*(2), 193–206. https://doi.org/10.1037/1082-989X.11.2.193
Jiang, R., Calhoun, V. D., Fan, L., Zuo, N., Jung, R., Qi, S., … Sui, J. (2020). Gender differences in connectome-based predictions of individualized intelligence quotient and sub-domain scores. *Cerebral Cortex, 30*(3), 888–900. https://doi.org/10.1093/cercor/bhz134
Lee, J. J., Wedow, R., Okbay, A., Kong, E., Maghzian, O., Zacher, M., … Cesarini, D. (2018). Gene discovery and polygenic prediction from a genome-wide association study of educational attainment in 1.1 million individuals. *Nature Genetics, 50*(8), 1112–1121. https://doi.org/10.1038/s41588-018-0147-3
Lett, T. A., Vogel, B. O., Ripke, S., Wackerhagen, C., Erk, S., Awasthi, S., … Desrivières, S. (2019). Cortical surfaces mediate the relationship between polygenic scores for intelligence and general intelligence. *Cerebral Cortex, 30*(4), 2708–2719. https://doi.org/10.1093/cercor/bhz270
Lewis, A. C. F., & Green, R. C. (2021). Polygenic risk scores in the clinic: New perspectives needed on familiar ethical issues. *Genome Medicine, 13*(14). https://doi.org/10.1186/s13073-021-00829-7
Li, L., Pang, S., Zeng, L., Güldener, U., & Schunkert, H. (2020). Genetically determined intelligence and coronary artery disease risk. *Clinical Research in Cardiology, 110*(2), 211–219. https://doi.org/10.1007/s00392-020-01721-x
Malanchini, M., Rimfeld, K., Gidziela, A., Cheesman, R., Allegrini, A. G., Shakeshaft, N., … Plomin, R. (2021). Pathfinder: A gamified measure to integrate general cognitive ability into the biological, medical, and behavioural sciences. *Molecular Psychiatry, 26*(12), 7823–7837. https://doi.org/10.1038/s41380-021-01300-0
Manolio, T. A., Collins, F. S., Cox, N. J., Goldstein, D. B., Hindorff, L. A., Hunter, D. J., … Visscher, P. M. (2009). Finding the missing heritability of complex diseases. *Nature, 461*(7265), 747–753. https://doi.org/10.1038/nature08494
Martin, A. R., Kanai, M., Kamatani, Y., Okada, Y., Neale, B., & Daly, M. J. (2019). Clinical use of current polygenic risk scores may exacerbate health disparities. *Nature Genetics, 51*, 584–591. https://doi.org/10.1038/s41588-019-0379-x
Martschenko, D. O., Matthews, L. J., & Sabatello, M. (2024). Social and behavioral genomics: What does it mean for pediatrics? *The Journal of Pediatrics, 264*, Article 113735. https://doi.org/10.1016/j.jpeds.2023.113735
Mascie-Taylor, C. G. N. (1989). Spouse similarity for IQ and personality and convergence. *Behaviour Genetics, 19*, 223–227.
McGrew, K. S. (2009). CHC theory and the human cognitive abilities project: Standing on the shoulders of the giants of psychometric intelligence research. *Intelligence, 37*(1), 1–10. https://doi.org/10.1016/j.intell.2008.08.004
Microsoft Corporation. (2018). Microsoft Excel. Retrieved from https://office.microsoft.com/excel.
Mills, M. C., & Rahal, C. (2019). A scientometric review of genome-wide association studies. *Nature Communications, 2*, 9. https://doi.org/10.1038/s42003-018-0261-x
Mitchell, B. L., Hansell, N. K., McAloney, K., Martin, N. G., Wright, M. J., Renteria, M. E., & Grasby, K. L. (2022). Polygenic influences associated with adolescent cognitive skills. *Intelligence, 94*, Article 101680. https://doi.org/10.1016/j.intell.2022.101680
Moher, D., Shamseer, L., Clarke, M., Ghersi, D., Liberati, A., Petticrew, M., & Stewart, L. A. (2015). Preferred reporting items for systematic review and meta-analysis protocols (PRISMA-P) 2015 statement. *Systematic Reviews, 4*(1), 1–9. https://doi.org/10.1186/2046-4053-4-1
Morris, T. T., Davies, N. M., & Smith, G. D. (2020). Can education be personalised using pupils' genetic data? *eLife, 9*, Article e49962. https://doi.org/10.7554/eLife.49962
Mõttus, R. (2022). What correlations mean for individual people: A tutorial for researchers, students and the public. *Personality Science, 3*. https://doi.org/10.5964/ps.7467
Nosek, B. A., Alter, G., Banks, G. C., Borsboom, D., Bowman, S. D., Breckler, S. J., … Yarkoni, T. (2015). Promoting an open research culture. *Science, 348*(6242), 1422–1425. https://doi.org/10.1126/science.aab2374
Okbay, A., Wu, Y., Wang, N., Jayashankar, H., Bennett, M., Nehzati, S. M., … Young, A. I. (2022). Polygenic prediction of educational attainment within and between families from genome-wide association analyses in 3 million individuals. *Nature Genetics, 54*, 437–449. https://doi.org/10.1038/s41588-022-01016-z
Ouzzani, M., Hammady, H., Fedorowicz, Z., & Elmagarmid, A. (2016). Rayyan – A web and mobile app for systematic reviews. *Systematic Reviews, 5*(210). https://doi.org/10.1186/s13643-016-0384-4
Page, M. J., McKenzie, J. E., Bossuyt, P. M., Boutron, I., Hoffmann, T. C., Mulrow, C. D., et al. (2021). The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. *British Medical Journal, 372*(71), 1–9. https://doi.org/10.1136/bmj.n71
Pinto, C., & Kühnel, A. (2020). *The Role of Education by Intelligence Testing*. https://doi.org/10.13140/RG.2.2.25010.22725
Plomin, R., & Deary, I. J. (2015). Genetics and intelligence differences: Five special findings. *Molecular Psychiatry, 20*(1), 98–108. https://doi.org/10.1038/mp.2014.105
Plomin, R., & von Stumm, S. (2018). The new genetics of intelligence. *Nature Reviews Genetics, 19*(3), 148–159. https://doi.org/10.1038/nrg.2017.104
Plomin, R., & von Stumm, S. (2022). Polygenic scores: Prediction versus explanation. *Molecular Psychiatry, 27*, 49–52. https://doi.org/10.1038/s41380-021-01348-y
Polderman, T. J., Benyamin, B., De Leeuw, C. A., Sullivan, P. F., Van Bochoven, A., Visscger, P. M., & Posthuman, D. (2015). Meta-analysis of the heritability of human traits based on fifty years of twin studies. *Nature Genetics, 47*(7), 702–709. https://doi.org/10.1038/ng.3285
Procopio, F., Zhou, Q., Wang, Z., Gidziela, A., Rimfeld, K., Malanchini, M., & Plomin, R. (2022). The genetics of specific cognitive abilities. *Intelligence, 95*, Article 101689. https://doi.org/10.1016/j.intell.2022.101689
Protzko, J., Aronson, J., & Blair, C. (2013). How to make a Young child smarter. *Perspectives on Psychological Science, 8*(1), 25–40. https://doi.org/10.1177/1745691612462585
R Core Team. (2019). *R: A language and environment for statistical computing*. Vienna, Austria: R Foundation for Statistical Computing. https://www.R-project.org/.
Ram, K., & Wickham, H. (2018). wesanderson: A Wes Anderson Palette Generator. R package version 0.3.6. https://CRAN.R-project.org/package=wesanderson.
Ritchie, S. J., & Tucker-Drob, E. M. (2018). How much does education improve intelligence? A meta-analysis. *Psychological Science, 29*(8), 1358–1369. https://doi.org/10.1177/0956797618774253
Roth, P. L., Le, H., Oh, I. S., Van Iddekinge, C. H., & Bobko, P. (2018). Using beta coefficients to impute missing correlations in meta-analysis research: Reasons for caution. *Journal of Applied Psychology, 103*(6), 644. https://doi.org/10.1037/apl0000293
Ruan, Y., Lin, Y.-F., Feng, Y.-C. A., Chen, C.-Y., Lam, M., Guo, Z., … Ge, T. (2022). Improving polygenic prediction in ancestrally diverse populations. *Nature Genetics, 54*, 573–580. https://doi.org/10.1038/s41588-022-01054-7
Savage, J. E., Jansen, P. R., Stringer, S., Watanabe, K., Bryois, J., De Leeuw, C. A., … Posthuma, D. (2018). Genome-wide association meta-analysis in 269,867 individuals identifies new genetic and functional links to intelligence. *Nature Genetics, 50*(7), 912–919. https://doi.org/10.1038/s41588-018-0152-6
Selzam, S., Ritchie, S. J., Pingault, J.-B., Reynolds, C. A., O'Reilly, P. F., & Plomin, R. (2019). Comparing within- and between-family polygenic score prediction. *The American Journal of Human Genetics, 105*(2), 351–363. https://doi.org/10.1016/j.ajhg.2019.06.006
Shamseer, L., Moher, D., Clarke, M., Ghersi, D., Liberati, A., Petticrew, M., … Stewart, L. A. (2015). Preferred reporting items for systematic review and meta-analysis protocols (PRISMA-P) 2015: Elaboration and explanation. *British Medical Journal, 349*, Article g7647. https://doi.org/10.1136/bmj.g7647
Sniekers, S., Stringer, S., Watanabe, K., Jansen, P. R., Coleman, J. R., Krapohl, E., … Posthuma, D. (2017). Genome-wide association meta-analysis of 78,308 individuals identifies new loci and genes influencing human intelligence. *Nature Genetics, 49*(7), 1107–1112. https://doi.org/10.1038/ng.3869
Strenze, T. (2007). Intelligence and socioeconomic success: A meta-analytic review of longitudinal research. *Intelligence, 35*(5), 401–442. https://doi.org/10.1016/j.intell.2006.09.004
von Stumm, S., Kandaswamy, R., & Maxwell, J. (2023). Gene-environment interplay in early life cognitive development. *Intelligence, 98*, Article 101748. https://doi.org/10.1016/j.intell.2023.101748
von Stumm, S., & Plomin, R. (2021). Using DNA to predict intelligence. *Intelligence, 86*, Article 101530. https://doi.org/10.1016/j.intell.2021.101530
von Stumm, S., Smith-Woolley, E., Ayorech, Z., McMillan, A., Rimfeld, K., Dale, P. S., & Plomin, R. (2020). Predicting educational achievement from genomic measures and socioeconomic status. *Developmental Science, 23*(3), Article e12925. doi:10.1111.desc.12925.
Trampush, J. W., Yang, M. L. Z., Yu, J., Knowles, E., Davies, G., Liewald, D. C., … Lencz, T. (2017). GWAS meta-analysis reveals novel loci and genetic correlates for general cognitive function: A report from the COGENT consortium. *Molecular Psychiatry, 22*(3), 336–345. https://doi.org/10.1038/mp.2016.244
Tsapanou, A., Mourtzi, N., Gu, Y., Habeck, C., Belsky, D. W., & Stern, Y. (2023). Polygenic indices for cognition in healthy aging; the role of brain measures. *Neuroimage: Reports, 3*(1), 100153. https://doi.org/10.1016/j.ynirp.2022.100153
Viechtbauer, W. (2010). Conducting meta-analyses in R with the *metafor* package. *Journal of Statistical Software, 36*(3), 1–48. https://doi.org/10.18637/jss.v036.i03
Wainschtein, P., Jain, D., Zheng, Z., Aslibekyan, S., Becker, D., Bi, W., … Visscher, P. M. (2022). Assessing the contribution of rare variants to complex trait heritability from whole-genome sequence data. *Nature Genetics, 54*(3), 263–273. https://doi.org/10.1038/s41588-021-00997-7
Wang, B., Baldwin, J. R., Schoeler, T., Cheesman, R., Barkhuizen, W., Dudbridge, F., & Pingault, J. (2021). Robust genetic nurture effects on education: A systematic review

and meta-analysis based on 38,654 families across 8 cohorts. *American Journal of Human Genetics, 108*(9), 1780–1791. https://doi.org/10.1016/j.ajhg.2021.07.010

Wechsler, D. (2003). *Wechsler intelligence scale for children – Fourth edition (WISC-IV)*. Texas, PsychCorp: San Antonia.

Weiss, L. G., & Saklofske, D. H. (2020). Mediators of IQ test score differences across racial and ethnic groups: The case for environmental and social justice. *Personality and Individual Differences, 161*, Article 109962. https://doi.org/10.1016/j.paid.2020.109962

Wertz, J., Moffitt, T. E., Agnew-Blais, J., Arseneault, L., Belsky, D. W., Corcoran, D. L., … Caspi, A. (2019). Using DNA from mothers and children to study parental investment in children's educational attainment. *Child Development, 91*(5), 1745–1761. https://doi.org/10.1111/cdev.13329

Wertz, J., Moffitt, T. E., Arseneault, L., Barnes, J. C., Boivin, M., Corcoran, D. L., … Caspi, A. (2023). Genetic associations with parental investment from conception to wealth inheritance in six cohorts. *Nature Human Behaviour, 7*(8), 1388–1401. https://doi.org/10.1038/s41562-023-01618-5

Yap, C. X., Alvares, G. A., Henders, A. K., Lin, T., Wallace, L., Farrelly, A., … Lawson, L. (2021). Analysis of common genetic variation and rare CNVs in the Australian autism biobank. *Molecular Autism, 12*(1). https://doi.org/10.1186/s13229-020-00407-5

Young, A. I. (2019). Solving the missing heritability problem. *PLoS Genetics, 15*(6), Article e1008222. https://doi.org/10.1371/journal.pgen.1008222