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Multilevel multiverse meta-analysis indicates lower IQ as a risk factor for physical and mental illness

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Is lower intelligence in early life an overlooked risk factor for later physical and mental illness? Intelligence shapes decision-making, career paths, and other health-relevant factors. However, our understanding of its association with health remains limited because there is no quantitative synthesis of the literature. Here, we conducted a comprehensive systematic review and meta-analysis of associations between intelligence test scores and mental and physical health. We included studies reporting standardized intelligence test scores obtained in childhood, adolescence, or early adulthood (<21 years of age) and their association with later-life health outcomes. We excluded studies limited to clinical populations without healthy controls. Our three-level multiverse analyses of 49 studies ($N > 2{,}900{,}000$) showed a 15-point IQ disadvantage in early life was associated with a 22 percent higher risk of later mental and physical illness (log$HR = 0.20$, 95% CI [0.13, 0.26]). Lower IQ predicted disease risk across various conditions, including schizophrenia, depression, dementia, and diabetes. Notably, the association between IQ and future health diminished with improved healthcare quality and when education was statistically held constant. Nevertheless, a meaningful effect of intelligence remained after adjusting for these variables. Multiple methods for detecting dissemination bias indicated that risk of bias was low. While our summary effect estimates are precise, all included data were collected in highly developed nations. Further, samples were predominantly male, potentially limiting generalizability. We show that lower IQ scores in early life are linked to a higher risk of later physical and mental illness. Improving education and healthcare quality appears as potential measures to address the issue. This research received no specific funding.

Individuals with lower intelligence may face an increased risk of experiencing physical and mental health issues throughout their lives[1]. Lower intelligence (as measured by standardized intelligence tests) has been associated with a higher prevalence of physical health conditions such as arthritis, diabetes, and stroke[2–4], as well as mental health conditions such as depression, bipolar disorder, or schizophrenia[5–7]. This linkage is suspected to be partly rooted in socioeconomic factors correlated with intelligence, like income and education. A higher income often means better healthcare access and higher education typically leads to less physically demanding jobs[8,9]. Health literacy, a product of education, promotes healthier behaviors, including regular physical activity and adherence to medical treatments[10,11]. Notably, however, health literacy has been suspected to be congruent with intelligence, making it a potentially tautological construct[12].

Beyond its associations with specific health behaviors, intelligence plays a pivotal role in various life domains that, in turn, shape overall health trajectories. Intelligence, or cognitive ability, encompasses a wide array of mental skills such as reasoning, planning, and problem-solving[13]. It permeates a person's life on all levels, correlating positively with job performance, financial success[14], academic achievement[15], and even attractiveness[16] but negatively with issues like delinquency and alcoholism[17,18]. Recognized for its predictive power in real-world outcomes, intelligence has been increasingly included in mortality and disease prevention models since the late 1990s, underscoring its importance in understanding population-wide health differentials[8,19,20]. In the field of cognitive epidemiology, the effects of intelligence on health outcomes, including morbidity, mortality, and risk factors like blood pressure, are examined[2,21–25].

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A major challenge in cognitive epidemiology research is to disentangle the effects of intelligence on health outcomes from other contributing factors. Importantly, aging affects both physical health and cognitive abilities, and the common cause hypothesis suggests that a general health decline impacts both, complicating the analysis of their relationship in adults. To avoid these reverse causation issues, especially in studies of older populations, researchers prefer using early-life intelligence test results. Early assessments of cognitive abilities help to more accurately determine their influence on later health outcomes[26], although it must be acknowledged that intelligence could already be affected by life circumstances at a young age.

While most research suggests that higher intelligence is associated with better health outcomes, researchers have indicated that this trend could change direction at the upper end of the intelligence distribution. Some investigations of highly intelligent individuals have found elevated prevalences of certain physical and mental health conditions[27–29], suggesting that high intelligence could be associated with specific challenges. However, many cognitive epidemiology studies are not designed to detect such effects because they often do not apply intelligence tests suitable to differentiate in the high-IQ range[25,30–32]. A recent article based on a large dataset from the UK Biobank did not yield evidence for the nonlinearity hypothesis[33], and a meta-analysis yielded a small, non-significant negative effect size, indicating that gifted individuals were no more likely than the general population to suffer from anxiety and depression[34]. However, the number of available studies scrutinizing this phenomenon is limited and additional targeted investigations of highly intelligent individuals using specialized diagnostic instruments are necessary to draw more robust conclusions.

Understanding the relationship between intelligence and health is important for society and its members. The available evidence suggests that individuals with low intelligence comprise an at-risk group that is vulnerable to illness and consequently may benefit from investments in interventions alleviating the risk[35]. However, despite considerable research advances in the past decades, the intelligence and health association remains elusive because its generality and moderators are poorly understood. Prior meta-analyses have explored the link between intelligence and specific outcomes like mortality, cardiovascular events, stroke risk, and schizophrenia[36–39]. Nevertheless, a systematic account of intelligence as a predictor for health outcomes in general is needed.

Here, we provide a formal systematic review and multiverse meta-analysis of the association of early-life intelligence with later physical and mental illness. This approach allows us to quantitatively examine the generality of the intelligence and health link as well as possible effect differentiation according to moderating variables. Unlike earlier research that concentrated on individual health conditions in isolation, the present research synthesis includes all relevant literature on the intelligence and health link. It thus enables us to draw conclusions about how the association varies across diseases with different causes and susceptibilities to behavioral influences. We are the first to apply a multilevel-multiverse approach (i.e., specification curve and combinatorial meta-analysis) to meta-analytically examine the link between intelligence and physical and mental illnesses as well as the impact of different study methodologies and data analysis methods on this link. This approach enables more reliable conclusions about the universality and robustness of the overall effect sizes compared to traditional meta-analysis. Additionally, we examine the role of further important influences such as access to healthcare and education. We complement these analyses with two interactive, web-based dashboards that allow readers to examine the results in considerable detail (available at https://pietschniglab.univie.ac.at/?page_id=11). Overall, we present a comprehensive, quantitative overview of the intelligence-health link based on data from almost three million individuals.

In summary, we first expected that lower IQ in early life would be associated with an increased risk of developing physical and mental health disorders in later life. Second, we expected that the association would vary by condition. Third, we expected that moderators, such as education, would explain a meaningful portion of the variance in the association.

Methods

This study followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines (PRISMA[40]; checklists available in Supplementary Tables 3 and 4) and was pre-registered (05/19/2023; available at https://aspredicted.org/THW_QG5). We made post-hoc changes to the registered protocol, such as including studies that reported hazard ratios (*HR*s) as effect sizes. Further, we used multilevel models to account for sample overlap, requiring different publication bias methods. Additional moderator analyses were conducted (e.g., assessment age, follow-up time, or healthcare access and quality). A full account of all deviations can be found in Supplementary Methods 2. Because this is a research synthesis, no human or animal subjects were involved in this work. Thus, no approval by an ethics board was required[41].

Literature search

We conducted a systematic review and meta-analysis of all studies reporting associations between early-life intelligence and later-life health outcomes. Searching PubMed, Scopus, and Web of Science, we identified 68,218 potentially relevant records. We used a search term combining "intelligence" and a range of synonyms with "health" and related concepts. To ensure broad coverage, we also included mortality-related terms to identify and include studies addressing both mortality and morbidity. The search queries are available in Supplementary Methods 1. Additionally, we screened the reference lists of one review, presenting the arguably most comprehensive overview of the topic to date, and three meta-analyses of subsets of the literature to identify further includable studies[1,37–39].

Study selection

We used the following inclusion and exclusion criteria. First, studies were required to report standardized intelligence test scores obtained in childhood, adolescence, or early adulthood (<21 years of age) and their association with later-life health outcomes. Second, we only considered studies with clinical health conditions as outcomes, including those that linked physical parameters (e.g., blood pressure) or biomarkers (e.g., blood glucose levels) to clinical diagnoses. Third, we excluded studies estimating intelligence levels from later-life sociodemographic information. Fourth, studies that focused solely on clinical patient cohorts were omitted, but case-control studies were included. Fifth, studies that only reported associations of early-life intelligence with mortality but did not report health outcomes were excluded. Finally, we excluded duplicate publications of identical outcomes based on identical data. Studies were not required to report individual-level data.

Data extraction

Two researchers with previous experience in conducting meta-analyses (JF and SO) independently coded primary studies between June 5, 2023, and October 10, 2023. A random subset (15 percent of the studies) was coded by both researchers. They initially agreed on 96 percent of the coding; discrepancies were later resolved by consulting a third researcher (JP). Besides various standard parameters (publication year, publication type, etc.), we coded characteristics specific to the intelligence-health association (health outcome, source of health outcome, intelligence test used, etc.). Given the controversial claim that the intelligence-health association may vary with a country's distance from the equator[42–44], we aimed to critically evaluate this idea by incorporating the latitudes of the data collection countries. It has also been suspected that the intelligence-health association could be associated with a country's healthcare accessibility[8]. To test this hypothesis, we recorded the Healthcare Access and Quality Index of the data collection countries (HAQ[45,46]).

All coded study characteristics are listed in Supplementary Methods 3; the data and analysis code are available at https://doi.org/10.17605/OSF.IO/ASTHV.

Effect size metrics

We used two effect size metrics in our analysis: hazard ratio (*HR*) and odds ratio (*OR*). *HR*s, used in time-to-event analyses[47], cannot be converted into other metrics[48], so we synthesized *HRs* and *ORs* in two separate branches of analysis. *HR*s and *OR*s are interpreted similarly. In the present context, a one standard deviation decrease in IQ (i.e., 15 points) is associated with a specific risk increase for the respective health condition (e.g., logHR/OR = 0.18, equal to HR/OR = 1.20 or a 20% risk increase).

Effect sizes were obtained from within-study reporting or calculated from group means and standard deviations[49]. When standard errors for *HR*s and *OR*s were unavailable, we computed them based on the confidence intervals of effect sizes[48]. We inverted any effect sizes reported in the reverse direction to maintain a consistent effect size interpretation. We transformed *HR*s and *OR*s using the natural logarithm for data analysis and maintained this logarithmic scale in presenting results and figures, thus ensuring centered values around zero for linear interpretation, in contrast to the curvilinear nature of non-transformed *HR*s or *OR*s[48].

Additional details about effect size calculations are provided in Supplementary Methods 4.

Sample overlap

Several studies used overlapping participant groups, thus violating the independence assumption of standard meta-analytic methods. For example, four included studies reported data from the NLSY 1979 cohort and contributed 18 effect sizes[2,50–52]. To address the non-independence resulting from multiple effect sizes from overlapping samples, we used multilevel models with restricted maximum likelihood and cluster-robust estimators to be able to include all available information[53]. Notably, we assumed effect sizes not to be nested within reports but within participant cohorts. Multilevel I^2 values were calculated according to the approach described by Viechtbauer[54].

Effect size interpretation

Typically, Cohen *d* values of 0.20, 0.50, and 0.80 are interpreted as lower thresholds of small, medium, and large effects[55]. However, given intelligence's impact on countless decisions in every person's life, seemingly small inter-individual differences can compound into large-scale societal consequences. Therefore, we adopt alternative benchmarks with $d = 0.10$ representing a very small but impactful effect over a short period, $d = 0.20$ a small effect with long-term implications, $d = 0.41$ as a moderately-sized effect with immediate practical value, and $d = 0.63$ as a large effect that is "powerful in both the short and the long run"[56], p. 156. These correspond to logHR/logOR values of 0.18, 0.36, 0.74, and 1.14.

Data synthesis

We performed two main analyses for *HR*s and *OR*s. The effect sizes from the included studies were synthesized using multilevel random-effects models with cluster-robust sandwich estimators; this technique is typically applied in data structures with overlapping study samples[57]. Subsequently, we separately examined unadjusted and adjusted effect sizes. The former comprise effect sizes from studies that did not include any covariates in their analytic models apart from age and sex (controlling for age and sex is standard procedure in this field[26]); the latter comprise effect sizes from studies that did include additional covariates (e.g., education or socioeconomic status). Adjusting for covariates can be problematic, as including variables that correlate with intelligence (e.g., education) might remove variance attributable to intelligence and can thus yield underestimated results[58]. In further subgroup analyses, we calculated summary effects according to health conditions for which at least two effect sizes were available. Multilevel mixed-effects meta-regressions were conducted to assess the possible influences of moderators. There has been much debate in the literature about the causal roots and moderating influences underlying the intelligence-health association[8,26,59]. To shed light on these hypotheses, we selected variables that had been previously suspected to influence the intelligence-health association, such as socioeconomic conditions or education[1]. Each moderator was tested individually. All subgroups and moderators are listed in Supplementary Methods 4.

In a meta-analysis, several decisions must be made regarding which data to include and how to analyze them. This "garden of forking paths"[60] can introduce bias, as different choices – such as selecting specific subsets of studies or using a fixed-effect vs. random-effects model – may lead to varying summary effect sizes. To address this, we conducted a multiverse/specification curve analysis. This relatively recent method allows researchers to compute summary effect estimates for many different study subsets. In this framework, a specification refers to a combination of certain study characteristics and analytic choices. For example, one specification might include only studies conducted in Denmark, using health outcomes from national register data, reporting analytic models adjusted for education, and synthesized using maximum-likelihood estimation. Multiverse analysis systematically compares all such combinations, providing a more comprehensive view of how study characteristics and analytic choices influence the results and yielding an aggregate effect size across the specifications[61]. Given overlapping effect sizes in our sample, we adapted our approach for dependent data in a multilevel framework[62], consequently conducting distinct multiverse analyses for both *HRs* and *ORs*. The distribution of the resulting meta-analytic summary effects was evaluated against the null hypothesis of no significant effect using a bootstrap approach[61,62]. For each specification, 1000 random effect sizes were drawn from a normal distribution with a mean of zero and a standard deviation equal to each study's observed standard error. The observed effect sizes were then compared with the range of randomly generated effect sizes.

In combinatorial meta-analyses, the number of models can quickly increase to exceed reasonable computation times (2^{59-1} models for *HR*s and 2^{52-1} models for *OR*s). Thus, we ran a random set of 1,000,000 summary effect estimates. For these analyses, two-level models were used; calculations were carried out separately for *HR*s and *OR*s.

We first visually inspected contour-enhanced color-coded funnel plots to assess potential dissemination bias in multilevel meta-analyses[63]. In the absence of dissemination bias, the studies should be arranged in the shape of a symmetrical, upside-down funnel; an asymmetrical shape indicates potential bias[64]. In addition, we applied multilevel Egger's regression. This method is a formalized test of funnel plot asymmetry adapted for dependent data structures[65]. We further applied an alternative variant of Egger's regression – multilevel meta-regression combined with robust variance estimation using sandwich estimators[65]. Finally, we applied a three-parameter selection model (3PSM). The 3PSM method involves randomly selecting one effect size for each cluster and subsequently computing a weight-function model to the sampled data[65].

For analyses, we used R version 4.2.2[66]. Meta-analytic calculations were performed using *metafor*[67]. Figures and tables were created using *ggplot2*[68], *metaviz*[69], and *rempsyc*[70].

Results

We identified 49 articles comprising 151 effect sizes (79 *HRs*; 72 *ORs*) eligible for inclusion[2–5,7,17,30,31,50–52,71–108]. Notably, the initial study pool of 38,509 studies shrunk considerably because applying our selection criteria led to the exclusion of 98.74 percent of studies. Most studies were ineligible because they did not include a measure of early-life intelligence.

The literature selection process is shown in Fig. 1; the included articles and effects sizes are listed in Supplementary Table 1.

Data from 2,916,312 individuals were included in our analyses (*Mdn* $n = 6923$). Samples were predominantly male, with men outnumbering women at a ratio of about 10:1 ($N_{\text{men}} = 2{,}551{,}980$; $N_{\text{women}} = 332{,}382$). Some studies reported data from overlapping participant cohorts. Thus, the number of independent samples was lower than the number of articles (30 independent samples in 49 articles). Most samples were from the UK (10, including 3 from Scotland and 1 from England), followed by the USA (9 independent samples), Denmark (3), Sweden (3), Israel (2), and with Finland, New Zealand, and Norway each contributing one sample.

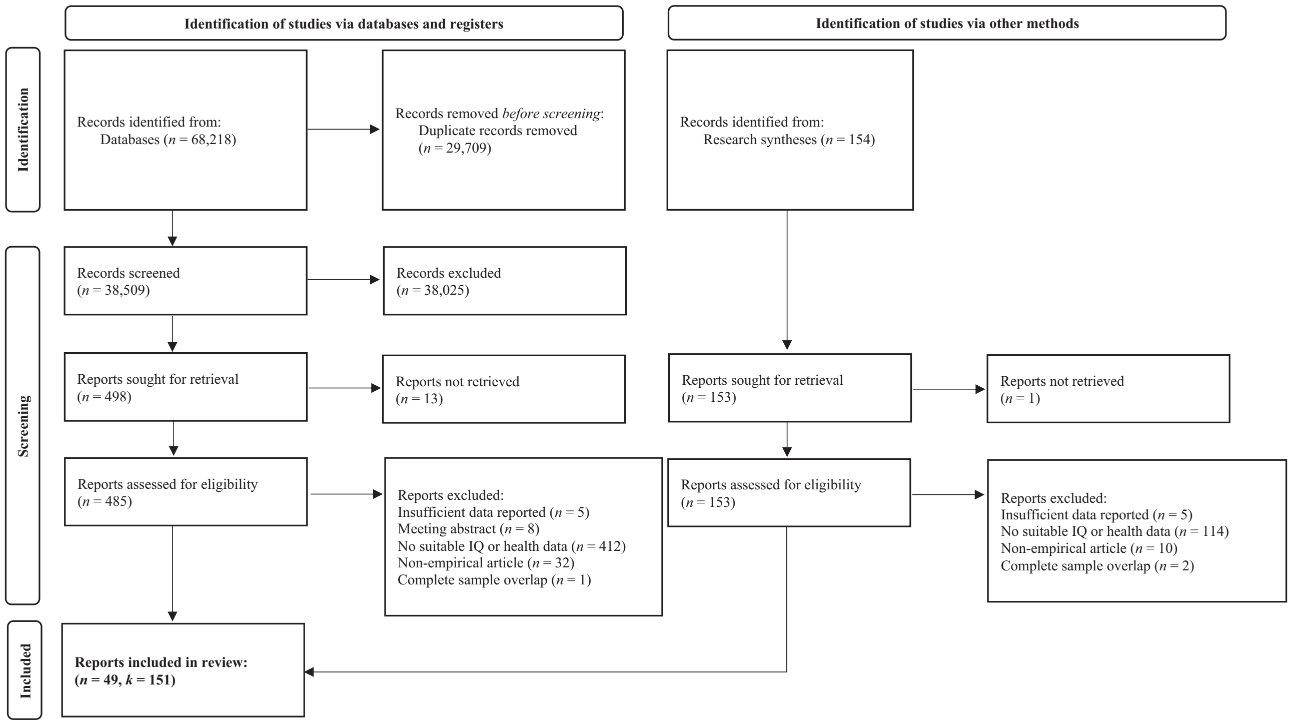

Fig. 1 | PRISMA flowchart of the literature selection process. Illustration of the process of literature search and selection, following the well-established PRISMA guidelines.

The mean age at intelligence assessment was 14.12 years ($SD = 4.38$). Health outcomes were assessed at a mean follow-up time of 34.10 years ($SD = 16.81$) after the intelligence assessment. Almost all samples were from cohort studies, while only one was from a case-control study, and all studies had been published.

We provide further information on the study characteristics (e.g., intelligence measures used in the respective studies, health outcome source, among others) in the meta-analytic dataset, available at https://doi.org/10.17605/OSF.IO/ASTHV.

We ran independent analyses for *HR*s and *OR*s using multilevel meta-analyses with effect sizes nested within participant cohorts. Combining *HR*s, irrespective of health condition, yielded a summary effect of $\log HR = 0.20$, 95% CI [0.13; 0.26], while combining all *OR*s yielded a slightly larger summary effect size with a wider confidence interval, $\log OR = 0.31$, 95% CI [0.17; 0.46]. Thus, with each *SD* disadvantage in intelligence, the risk of developing a given health condition was 22 percent and 36 percent higher, respectively. Note that these analyses included a variety of studies including different covariates in their models (e.g., sex, socioeconomic variables, or education). We ran additional analyses in which we only included unadjusted models (i.e., models that did not report any covariates in addition to age and sex). We observed slightly larger summary effects in these analyses ($\log HR = 0.24$, 95% CI [0.16; 0.32]; $\log OR = 0.38$, 95% CI [0.22; 0.55]). Synthesizing covariate-adjusted models (i.e., models that did include covariates in addition to age and sex, such as education, socioeconomic status, or multiple variables) attenuated the effect sizes in *HR*s, while the model remained statistically significant ($\log HR = 0.17$, 95% CI [0.09; 0.24]). Conversely, the covariate-adjusted model was no longer statistically significant and exhibited wider CIs in *OR*s compared to HRs ($\log OR = 0.08$, 95% CI [−0.13; 0.28]). Figures 2 and 3 show forest plots of the main analyses; Table 1 contains numerical details.

Subgroup analyses according to health condition

We ran multilevel random-effects models within all health conditions for which at least two effect sizes were available. For the full results, see Table 2.

Schizophrenia showed the largest summary effect ($\log OR = 0.70$, 95% CI [0.53; 0.88]), while cancer showed no significant effect ($\log HR = -0.01$, 95% CI [−0.21; 0.20]; $\log OR = -0.06$, 95% CI [−0.20; 0.08]).

Analyses for *HR*s and *OR*s yielded differential effects. For *HR*s, effect sizes ranged from $\log HR = 0.40$, 95% CI [0.37; 0.43] for alcohol- and drug-related disorders to $\log HR = -0.01$, 95% CI [−0.21; 0.20] for cancer. All summary effects showed positive directions, with cancer being the only exception. For *OR*s, effect sizes ranged from $\log OR = 0.70$, 95% CI [0.53; 0.88] for schizophrenia to $\log OR = -0.06$, 95% CI [−0.20; 0.08] for cancer. All summary log*OR*s showed positive directions, except for cancer and, unexpectedly, alcohol- and drug-related disorders which showed inconsistent signs between *HR*s and *OR*s.

Moderator analyses

We examined moderator effects through multilevel mixed-effects meta-regressions (Supplementary Table 2). In *HR*s, effect sizes were significantly differentiated according to data collection country; studies from England yielded a 0.24-point lower estimate compared to Denmark, while studies from Israel yielded a 0.67-point higher estimate. Number of men within samples (henceforth: male percentage), age at intelligence assessment, age at health measurement, follow-up time, latitude, Healthcare Access and Quality Index (HAQ), health outcome source, number of covariates, and covariates, in addition to age and sex, did not significantly influence effect sizes.

In *OR*s, the HAQ showed a significant negative influence on effect sizes; countries with a one-standard-deviation higher HAQ showed a 0.02-point lower effect size. Effects were again observed to be differentiated according to country. Moreover, cohort studies showed smaller effect sizes compared to case-control studies. Studies that examined data from hospitalized patients exhibited larger effect sizes than those based on physical or psychiatric examinations, register data, or self-report data. Each additional covariate in a model was associated with a 0.03-point lower log*OR*. Among specific covariates, models adjusted for either adult socioeconomic status (SES), parental SES, or education showed smaller effect sizes than unadjusted models. Adjusting for education had the most substantial impact on

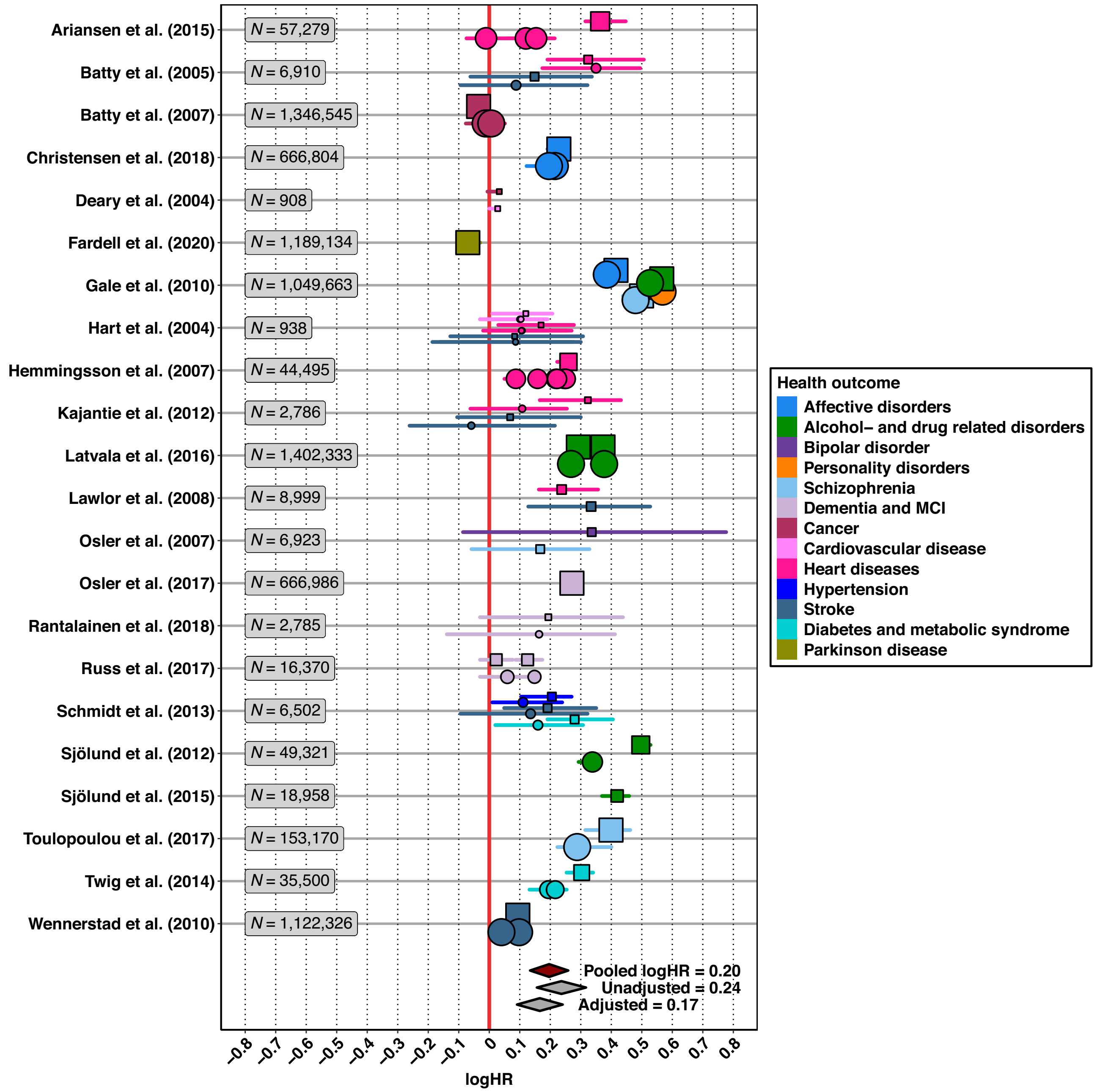

Fig. 2 | Forest plot for the meta-analysis of HRs. Squares represent log*HR*s from unadjusted models, circles represent log*HR*s from adjusted models. Larger symbol sizes indicate higher study precision. The colors of symbols and confidence intervals indicate disease type. log*HR* = 0.00 is provided as reference line. Diamonds indicate summary (i.e., pooled) effects and confidence intervals from multilevel models for the overall model as well as for unadjusted and adjusted effect syntheses.

effect sizes while adjusting for multiple variables did not significantly alter effect sizes. Male percentage, age at intelligence assessment, age at health measurement, follow-up time, latitude, and effect size origin did not account for a meaningful portion of variance among effect sizes.

Publication bias

None of the four methods indicated evidence of publication bias in *HRs* (Egger's multilevel meta-regression: $z = -1.04$, $p = 0.30$; Egger's regression with robust variance estimation: $t = -0.08$, $p = 0.94$; 3PSM: $\chi^2_1 = 1.41$, $p = 0.24$). However, all approaches indicated that the summary effect might be somewhat inflated in *OR*s, as enhanced Egger's multilevel meta-regression ($z = 3.02$, $p = 0.003$), Egger's regression with robust variance estimation ($t = 2.99$, $p = 0.02$), and the 3PSM approach yielded significant results ($\chi^2_1 = 4.62$, $p = 0.03$); the funnel plot suggested a slight overrepresentation of smaller effect sizes in lower-powered studies. However, median power among participant cohorts was high ($HR\ Mdn_{\text{Power}} = 93.10\%$; $OR\ Mdn_{\text{Power}} = 99.90\%$). See Supplementary Figs. 1 and 2 for additional details.

Specification curve analysis

Two interactive, web-based dashboards (available at https://pietschniglab.univie.ac.at/?page_id=11) allow readers to examine our specification curve and multiverse analyses in great detail (we recommend viewing the application on a large, high-resolution screen). In all, specification curve analyses showed small and predominantly positive summary effect sizes in *HR*s, with 54 out of 58 specifications reaching statistical significance, indicating a remarkable generality of the intelligence and health link. In other words, it neither mattered which subsets of studies were combined meta-analytically nor which meta-analytical estimators were used – the effect size remained

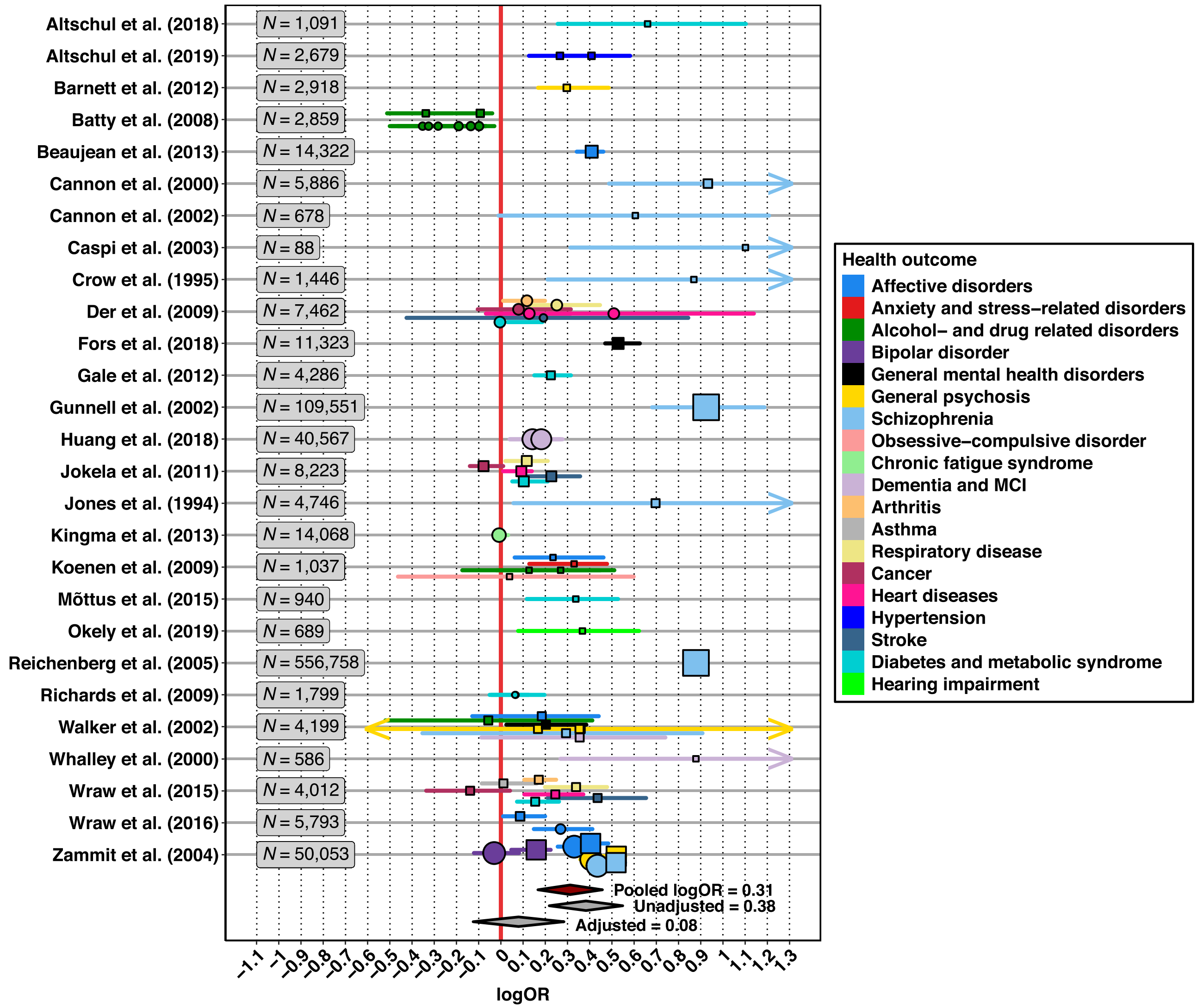

Fig. 3 | Forest plot for the meta-analysis of ORs. Squares represent log*OR*s from unadjusted models, circles represent log*OR*s from adjusted models. Larger square and circle sizes indicate higher study precision. The colors of symbols and confidence intervals indicate disease type. log*OR* = 0.00 is provided as reference line. Diamonds indicate summary (i.e., pooled) effects and confidence intervals from multilevel models for the overall model as well as for unadjusted and adjusted effect syntheses. The arrows on some confidence intervals indicate that the interval has been shortened to decrease the plotting area.

Table 1 | Multilevel meta-analyses

Model	*k*	Summary effect (95% CI)	*p*	I^2
*HR*s				
Overall	79	0.20*** [0.13; 0.26]	<0.001	99.56
Unadjusted	36	0.24*** [0.16; 0.32]	<0.001	99.67
Adjusted	43	0.17** [0.09; 0.24]	0.002	99.26
*OR*s				
Overall	72	0.31*** [0.17; 0.46]	<0.001	97.35
Unadjusted	49	0.38*** [0.22; 0.55]	<0.001	96.41
Adjusted	23	0.08 [−0.13; 0.28]	0.364	96.22

k number of studies, *HR*s hazard ratios, *OR*s odds ratios, I^2 proportion of variance explained by between-study heterogeneity.
***$p < 0.001$.

meaningful and positive. The median effect size was log*HR* = 0.20, supporting the meaningful, positive association between early-life intelligence and later health outcomes. The smallest effects were found in studies using register data and adjusting for education, whereas the largest ones were observed in analyses without any adjustments (see Fig. 4 and Supplementary Fig. 3). In *OR*s, the results were less consistent, with 52 out of 76 effect sizes reaching statistical significance. The median effect size (log*OR* = 0.20) was virtually identical compared to *HR*s, although the variability was larger. The smallest effect sizes in *OR*s were observed in UK studies with self-reported health data and the largest in studies without covariate adjustments (Fig. 5 and Supplementary Fig. 4).

Combinatorial meta-analysis

In combinatorial meta-analyses, effect sizes from a large number of randomly combined study subsets were examined, indicating that the meta-analytic effect estimates in *HR*s and *OR*s were not substantially affected by leverage points and outliers. Moreover, no distinct patterns of effect subgroups emerged, thus corroborating the generality of the intelligence and health link (see GOSH plots in Supplementary Figs. 5 and 6).

Discussion

In this meta-analysis of almost three million individuals, we demonstrate that lower scores on standardized intelligence tests in early life are associated with a higher risk of mental and physical illnesses in later life. The effect proved robust and generalizable across various health conditions.

Table 2 | Subgroup multilevel meta-analyses for individual health conditions

Model	k	Summary effect (95% CI)	p	I^2
*HR*s				
Affective disorders	5	0.30 [−0.98; 1.59]	0.206	99.70
Alcohol- and drug-related disorders	9	0.40** [0.37; 0.43]	0.004	99.79
Schizophrenia	5	0.35 [−0.05; 0.75]	0.065	97.19
Dementia and MCI	7	0.17 [−0.12; 0.47]	0.125	95.72
Cancer	5	−0.01 [−0.21; 0.20]	0.685	68.79
Cardiovascular disease	4	0.05 [−0.43; 0.52]	0.437	63.74
Heart diseases	20	0.20*** [0.16; 0.24]	<0.001	94.50
Stroke	13	0.11* [0.04; 0.17]	0.010	91.43
Diabetes and metabolic syndrome	5	0.24** [0.20; 0.27]	0.007	67.06
*OR*s				
Affective disorders	7	0.29** [0.15; 0.44]	0.005	80.75
Alcohol- and drug-related disorders	11	−0.04 [−0.65; 0.58]	0.820	92.08
General mental health disorders	2	0.39 [−1.79; 2.56]	0.265	91.49
General psychosis	6	0.40* [0.15; 0.65]	0.020	58.01
Schizophrenia	10	0.70*** [0.53; 0.88]	<0.001	93.63
Dementia and MCI	4	0.29 [−1.75; 2.32]	0.326	82.01
Respiratory disease	3	0.21 [−1.03; 1.46]	0.275	77.57
Cancer	3	−0.06 [−0.20; 0.08]	0.118	0.05
Heart diseases	4	0.13 [−0.73; 1.00]	0.297	58.29
Stroke	3	0.30 [−0.77; 1.37]	0.176	36.39
Diabetes and metabolic syndrome	8	0.16* [0.01; 0.31]	0.041	83.04

k number of studies, *HR*s hazard ratios, *OR*s odds ratios, I^2 proportion of variance explained by between-study heterogeneity.
$^*p < 0.05$; $^{**}p < 0.01$; $^{***}p < 0.001$.

Specifically, a 15-point lower IQ was linked to a 22% (hazard ratios) and 36% (odds ratios) higher risk of illness, respectively. Our findings have considerable societal implications, identifying individuals with lower early-life intelligence as an at-risk group for various health problems. In terms of strength, the effects we found were modest. However, on a societal scale, minor associations can lead to major impacts, as evidenced by how a small increase in mental health issues during the COVID-19 pandemic drastically strained health services globally[109,110].

Our analyses reveal that the association of intelligence with later health diminishes when education is statistically held constant. This finding is important for developing strategies to lower health risks in individuals with lower intelligence, with improved education and health literacy as promising mitigating approaches. Nevertheless, despite attenuating the intelligence and health link, socioeconomic and educational factors do not fully account for the observed variance. Corroborating what many studies have previously reported[51,88,94,95,111], this indicates that education is not the sole driver of the relationship.

The varying effect strength across health conditions suggests distinct mechanisms linking intelligence and health. Mental health disorders proved to be the most affected, with schizophrenia showing the largest effect size among all analyses (logOR = 0.70). This is consistent with a previous meta-analysis of intelligence and schizophrenia that yielded a similar effect[39] (logOR = 0.78). Lower intelligence in individuals who later develop mental health problems may be an early manifestation of the conditions[112]. Alternatively, higher intelligence in early life may protect against later mental illness by improving coping strategies[113] or mental health literacy[50], enabling individuals to intervene early on. Notably, recent evidence suggests a causal genetic connection between intelligence and mental health[114–116]. Here, we demonstrate a link between intelligence and health that cannot be fully explained by socioeconomic factors. A combination of genotype and education may explain the link between intelligence and mental health. However, as our meta-analytic evidence does not afford inferences of causality, this must be addressed using specific research designs, such as twin studies or genome-wide association studies.

We observed that effect sizes varied across countries. Countries with better healthcare access and quality (i.e., HAQ index) showed smaller associations and vice versa. Notably, all contributing countries were predominantly Western, educated, industrialized, rich, and democratic (WEIRD[117]), with the least equitable healthcare system in our data (i.e., the USA in 2019) still ranking high globally. This suggests that the effects might be even more pronounced if countries with less equitable healthcare systems were to be included. Enhancing healthcare access and quality might thus reduce the detrimental link between low intelligence and morbidity.

In all, analyses of *HR*s and *OR*s showed similar results, although *HR*-based summary effect estimates were more precise, while *OR*s showed higher variability. This was especially evident in our subgroup analyses of adjusted models, where the summary effect estimate remained statistically significant in *HR*s. In contrast, the analysis yielded an imprecise, statistically insignificant summary effect in *OR*s. This difference likely stems from the heterogeneous methodologies in *OR* studies. All studies reporting *HR*s were cohort studies using patient register data or physical examinations for health outcomes. *OR* studies additionally included self-reported outcomes. Consequently, the larger confidence intervals in *OR*s suggest more inherent variability in these analyses due to noisier data.

Notably, alcohol- and drug-related conditions showed substantial effect sizes in *HR*s but trivial ones in *OR*s. However, *OR* studies mainly used self-report questionnaires[17], while *HR* studies relied on more objective data like psychiatric registers[31]. Our specification-curve analyses suggest that the assessment methods impacted the findings, with self-reports reflecting subclinical behaviors and registers indicating severe cases requiring treatment. The difficulty of assessing problematic alcohol consumption is a known issue in cognitive epidemiology. It has been proposed that individuals with higher intelligence tend to reflect more on their consumption habits, leading to higher scores on self-report scales, while individuals with less intelligence tend to underestimate their own consumption[17].

There was evidence for some effect inflation due to publication bias in the *OR* subset. Studies reporting positive effects (i.e., higher early-life intelligence associations with lower risk) were somewhat overrepresented among studies with smaller samples. Nevertheless, because the overall study power was high, this is unlikely to have skewed the summary effect estimates.

Limitations

Some limitations of this meta-analysis should be acknowledged. Many included reports were based on male-only conscript data[5,7,108,118], with only a few datasets (e.g., Israeli[106]) including women. Many cohort studies involving IQ use intelligence test scores obtained during conscription, typically at ages 18–20. However, because women were exempt from military service in most countries during the 20th century, these datasets predominantly include men. Consequently, men outnumbered women 10:1 in our analyses. While we found no moderating effect of the male percentage in our analyses, several studies provided analytic models adjusted for sex[2,104], potentially obscuring sex differences. Future studies should, therefore, aim to improve female representation.

Our meta-analytic sample comprised only WEIRD countries. Even though these countries exhibited relatively limited variability in prosperity, variations at the country level emerged. Consequently, it is reasonable to assume that even more substantial differences might be observed when investigating a broader range of countries.

We found that adjusting for education reduced the association between intelligence and health, although the effect sizes remained

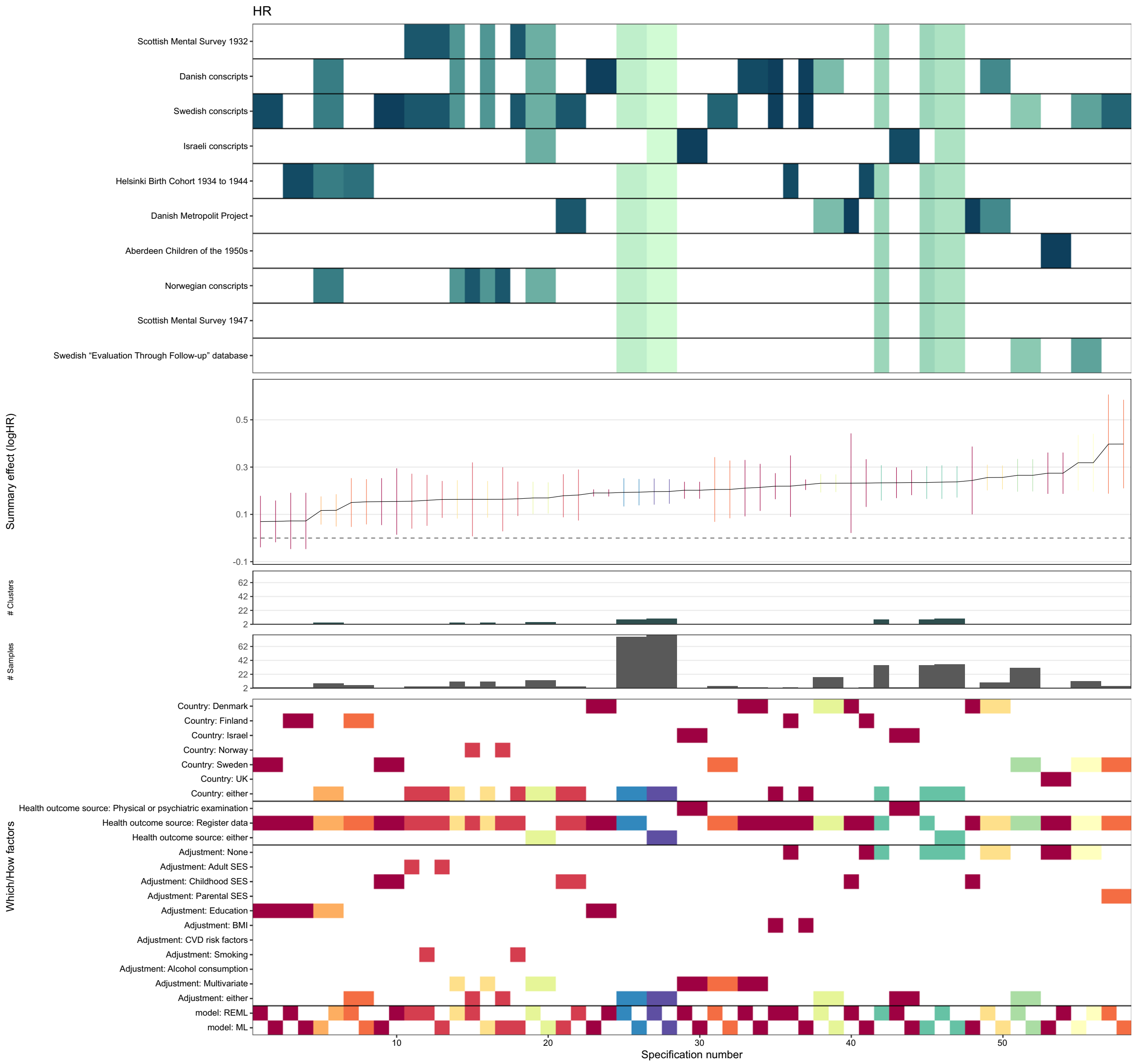

Fig. 4 | Descriptive specification plot for HRs. Descriptive meta-analytic specification plot of summary effects from all reasonable specifications for *HR*s. The first panel at the top displays the participant cohorts included in each analysis. The second panel presents the summary effect estimates for each subset along with their 95% confidence intervals; the line of null effect is indicated by a dashed horizontal line. The third panel shows the count of participant cohorts (clusters) included in the respective subsets. The fourth panel details the number of samples in each subset. The final (bottom) panel explains the factors considered ("which" and "how") in each analysis, where spectral colors ranging from warmer to cooler signify the precision of the estimates (from lower to higher) for the summary effects shown in the second panel, along with their respective 95% confidence intervals. Thus, the x-axis represents various combinations of study characteristics (specifications) along with the corresponding samples, ordered by effect size. Here, no specifications yielded negative effect sizes; therefore, the effect sizes are largest at the right edge of the plot. The figure is best read by drawing an imaginary vertical line through the panels and reading the information along this line. For example, the leftmost specification was composed of Swedish conscripts for whom register health data was available, the analytic model was adjusted for education, and a restricted maximum-likelihood estimation was applied; this combination of study characteristics exhibited the smallest effect sizes among *HR*s. This figure can be viewed in more detail at https://pietschniglab.univie.ac.at/?page_id=11.

meaningful. However, adjusting for variables closely linked to intelligence also decreases the variance attributable to intelligence[58,119]. This does not necessarily imply that improving education or socioeconomic position reduces the association between early-life intelligence and later health outcomes, as these variables are in bidirectional causal relationships[119]. However, some evidence from cohort studies suggests that education and socioeconomic status mediate the relationship between intelligence and health[120]. This could mean that a proportion of the intelligence-health association is due to higher education in more intelligent individuals and vice versa, although causal inferences cannot be drawn from these results. Importantly, education can be enhanced through dedicated intervention programs[121–123]. Some studies indicate small effects of longer education[124] and more appropriate early developmental environments[125,126] on population-level intelligence. Therefore, education and socioeconomic conditions appear as potential levers to mitigate the adverse effects of the intelligence-health association. Future work should explore how improving these variables can weaken the link between intelligence and alleviate the health inequalities that may arise from it.

In our analyses, cancer showed a negligible link with intelligence. However, intelligence-related risk behaviors differentially affect cancer types

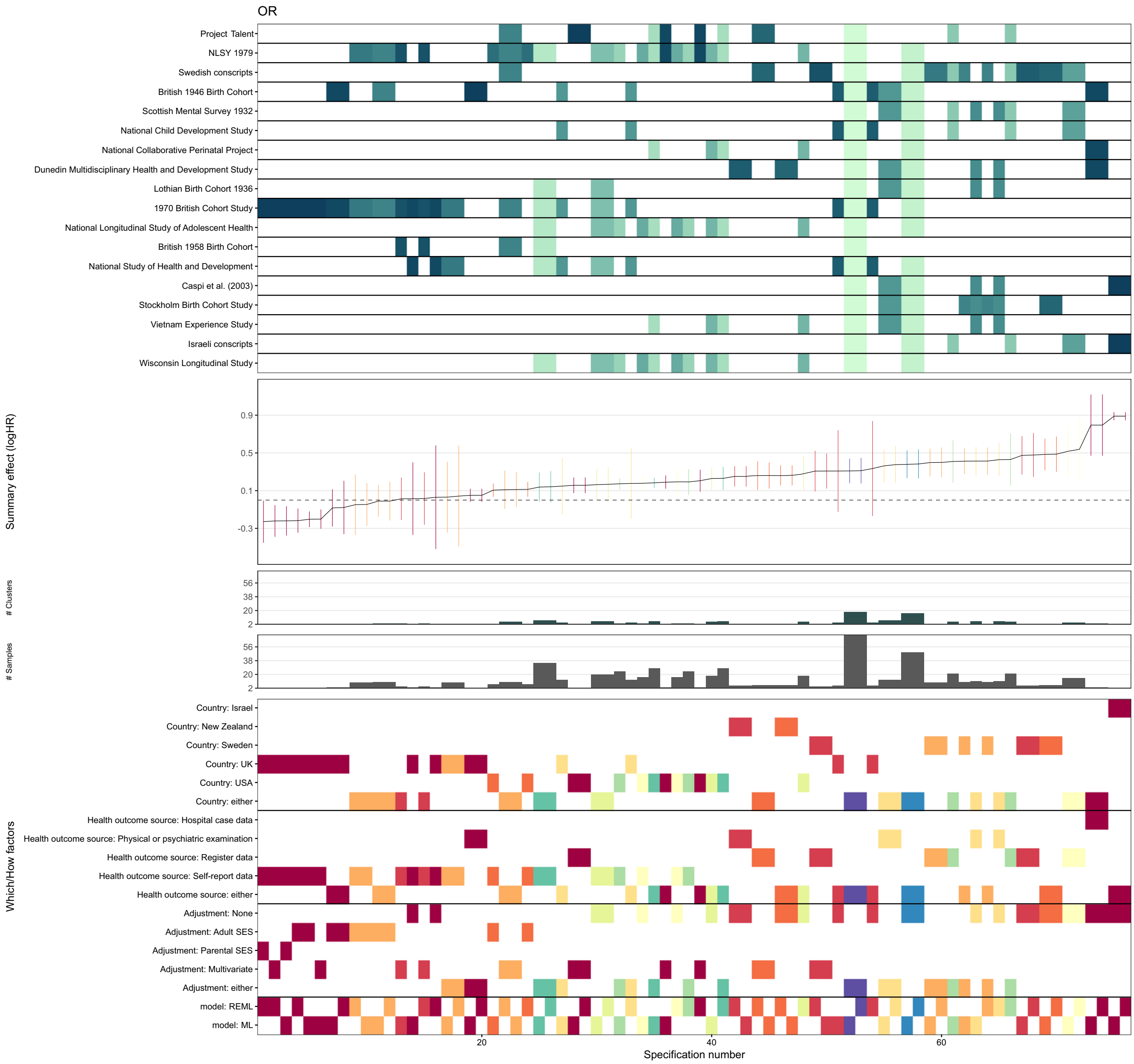

Fig. 5 | Descriptive specification plot for ORs. Descriptive meta-analytic specification plot of summary effects from all reasonable specifications for *ORs*. The first panel at the top displays the participant cohorts included in each analysis. The second panel presents the summary effect estimates for each subset along with their 95% confidence intervals. The third panel shows the count of participant cohorts (clusters) included in the respective subsets. The fourth panel details the number of samples in each subset. The final (bottom) panel explains the factors considered ("which" and "how") in each analysis, where spectral colors ranging from warmer to cooler signify the precision of the estimates (from lower to higher) for the summary effects shown in the second panel, along with their respective 95% confidence intervals. Here, few specifications yielded negative effect sizes; therefore, the effect sizes are largest at the right edge of the plot. The figure is best read by drawing an imaginary vertical line through the panels and reading the information along this line. For example, the leftmost specification was composed of Participants in the 1970 British Cohort study who provided self-reports of their health outcomes, the analytic model was adjusted for parental socioeconomic status, and a restricted maximum-likelihood estimation was applied; this combination of study characteristics exhibited the smallest effect sizes among *ORs*. This figure can be viewed in more detail at https://pietschniglab.univie.ac.at/?page_id=11.

(e.g., lung cancer, which is related to smoking behavior, vs. bone cancer, which is not[94]). Therefore, the present lack of a relationship may be due to insufficient data to examine specific cancer conditions separately. If more data become available in the future, this limitation should be addressed in a targeted quantitative synthesis of the relationship between intelligence and different types of cancer.

Conclusions

In this formal systematic review and meta-analysis of the intelligence and health association, we show that individuals with lower intelligence test scores in early life face an increased risk of both physical and mental illnesses in later life. This pattern emerged across various health conditions, with the strongest associations observed for mental health. Enhancing the quality of education and healthcare may not eliminate but could mitigate the adverse health impacts associated with lower intelligence. Public health strategies could, therefore, aim to improve general and health-specific education as well as access to high-quality healthcare to potentially address health concerns linked to lower intelligence.

Data availability

The data used in this meta-analysis is available at https://doi.org/10.17605/OSF.IO/ASTHV. The repository contains an R data file (.RDS; can be imported via the R code) as well as a spreadsheet version of the data (.xlsx; can be opened via Microsoft Excel or any other spreadsheet software).

Code availability

The analysis code used in this meta-analysis is available at https://doi.org/10.17605/OSF.IO/ASTHV. The repository contains a code file (.R; can be opened with a text editor and executed via R).

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References

1. Deary, I. J., Hill, W. D. & Gale, C. R. Intelligence, health and death. *Nat. Hum. Behav.* **5**, 416–430 (2021).
2. Wraw, C., Deary, I. J., Gale, C. R. & Der, G. Intelligence in youth and health at age 50. *Intelligence* **53**, 23–32 (2015).
3. Schmidt, M. et al. Cognitive test scores in young men and subsequent risk of type 2 diabetes, cardiovascular morbidity, and death. *Epidemiology* **24**, https://doi.org/10.1097/EDE.0b013e31829e0ea2 (2013).
4. Lawlor, D. A., David Batty, G., Clark, H., McIntyre, S. & Leon, D. A. Association of childhood intelligence with risk of coronary heart disease and stroke: Findings from the Aberdeen Children of the 1950s cohort study. *Eur. J. Epidemiol.* **23**, 695–706 (2008).
5. Osler, M., Lawlor, D. A. & Nordentoft, M. Cognitive function in childhood and early adulthood and hospital admission for schizophrenia and bipolar disorders in Danish men born in 1953. *Schizophr. Res.* **92**, 132–141 (2007).
6. Tiihonen, J. et al. Premorbid intellectual functioning in bipolar disorder and schizophrenia: Results from a cohort study of male conscripts. *Am. J. Psychiatry* **162**, 1904–1910 (2005).
7. Christensen, G. T., Rozing, M. P., Mortensen, E. L., Christensen, K. & Osler, M. L. Young adult cognitive ability and subsequent major depression in a cohort of 666,804 Danish men. *J. Affect. Disord*. **235**, https://doi.org/10.1016/j.jad.2018.04.035 (2018).
8. Gottfredson, L. S. & Deary, I. J. Intelligence predicts health and longevity, but why? *Curr. Dir. Psychol. Sci.* **13**, 1–4 (2004).
9. Schmidt, F. L. & Hunter, J. General mental ability in the world of work: Occupational attainment and job performance. *J. Personal. Soc. Psychol.* **86**, 162–173 (2004).
10. Berkman, N. D., Sheridan, S. L., Donahue, K. E., Halpern, D. J. & Crotty, K. Low health literacy and health outcomes: An updated systematic review. *Ann. Intern. Med.* **155**, 97–107 (2011).
11. Deary, I. J. et al. Intelligence and persisting with medication for two years: Analysis in a randomised controlled trial. *Intelligence* **37**, 607–612 (2009).
12. Reeve, C. L. & Basalik, D. Is health literacy an example of construct proliferation? A conceptual and empirical valuation of its redundancy with general cognitive ability. *Intelligence* **44**, https://doi.org/10.1016/j.intell.2014.03.004 (2014).
13. Gottfredson, L. S. Mainstream science on intelligence: An editorial with 52 signatories, history, and bibliography. *Intelligence* **24**, 13–23 (1997).
14. Strenze, T. Intelligence and socio-economic success: A meta-analytic review of longitudinal research. *Intelligence* **35**, 401–426 (2007).
15. Colom, R. & Flores-Mendoza, C. E. Intelligence predicts scholastic achievement irrespective of SES factors: Evidence from Brazil. *Intelligence* **35**, 243–251 (2007).
16. Prokosch, M. D., Coss, R. G., Scheib, J. E. & Blozis, S. A. Intelligence and mate choice: Intelligent men are always appealing. *Evol. Hum. Behav.* **30**, 11–20 (2009).
17. Batty, G. D. et al. Childhood mental ability and adult alcohol intake and alcohol problems: The 1970 British Cohort Study. *Am. J. Public Health* **98**, 2237–2243 (2008).
18. Schwartz, J. A. et al. Intelligence and criminal behavior in a total birth cohort: An examination of functional form, dimensions of intelligence, and the nature of offending. *Intelligence* **51**, 109–118 (2015).
19. Gottfredson, L. S. Intelligence: Is it the epidemiologists' elusive "fundamental cause" of social class inequalities in health? *J. Pers. Soc. Psychol*. **86** https://doi.org/10.1037/0022-3514.86.1.174 (2004).
20. Lubinski, D. & Humphreys, L. G. Incorporating general intelligence into epidemiology and the social sciences. *Intelligence* **24**, 159–201 (1997).
21. Calvin, C. M. et al. Childhood intelligence in relation to major causes of death in 68 year follow-up: Prospective population study. *BMJ* **357**, j2708 (2017).
22. Deary, I. J., Weiss, A. & Batty, G. D. Intelligence and personality as predictors of illness and death: how researchers in differential psychology and chronic disease epidemiology are collaborating to understand and address health inequalities. *Psychol. Sci. Public Interest* **11** https://doi.org/10.1177/1529100610387081 (2010).
23. Hart, C. L. et al. Childhood IQ, social class, deprivation, and their relationships with mortality and morbidity risk in later life: Prospective observational study linking the Scottish Mental Survey 1932 and the Midspan Studies. *Psychosom. Med*. **65**, 877–883 (2003).
24. Twig, G. Cognitive function in adolescence and the risk for premature diabetes and cardiovascular mortality in adulthood. *Cardiovasc. Diabetol*. **17** https://doi.org/10.1186/s12933-018-0798-5 (2018).
25. Whalley, L. J. & Deary, I. J. Longitudinal cohort study of childhood IQ and survival up to age 76. *BMJ* **322**, https://doi.org/10.1136/bmj.322.7290.819 (2001).
26. Deary, I. J. & Batty, G. D. Cognitive epidemiology: A glossary. *J. Epidemiol. Community Health* **61**, https://doi.org/10.1136/jech.2005.039206 (2007).
27. Karpinski, R. I., Kinase Kolb, A. M., Tetreault, N. A. & Borowski, T. B. High intelligence: A risk factor for psychological and physiological overexcitabilities. *Intelligence* **66**, 8–23 (2018).
28. Fries, J., Baudson, T. G., Kovacs, K. & Pietschnig, J. Bright, but allergic and neurotic? A critical investigation of the "overexcitable genius" hypothesis. *Front. Psychol*. **13** https://doi.org/10.3389/fpsyg.2022.1051910 (2022).
29. Guénolé, F. et al. Behavioral profiles of clinically referred children with intellectual giftedness. *BioMed. Res. Int.* **2013**, 540153 (2013).
30. Jokela, M., Batty, G. D., Deary, I. J., Silventoinen, K. & Kivimäki, M. Sibling analysis of adolescent intelligence and chronic diseases in older adulthood. *Ann. Epidemiol.* **21**, 489–496 (2011).
31. Gale, C. R., Batty, G. D., Tynelius, P., Deary, I. J. & Rasmussen, F. Intelligence in early adulthood and subsequent hospitalization for mental disorders. *Epidemiology* **21**, 70–77 (2010).
32. Fries, J. & Pietschnig, J. An intelligent mind in a healthy body? Predicting health by cognitive ability in a large European sample. *Intelligence* **93**, 101666 (2022).
33. Williams, C. M. et al. High intelligence is not associated with a greater propensity for mental health disorders. *Eur. Psychiatry* **66**, e3 (2023).
34. Duplenne, L., Bourdin, B., Fernandez, D. N., Blondelle, G. & Aubry, A. Anxiety and depression in gifted individuals: A systematic and meta-analytic review. *Gifted Child Q.* **68**, 65–83 (2023).
35. Caspi, A. Childhood forecasting of a small segment of the population with large economic burden. *Nat. Hum. Behav.* **1** https://doi.org/10.1038/s41562-016-0005 (2016).
36. Calvin, C. M. et al. Intelligence in youth and all-cause mortality: Systematic review with meta-analysis. *Int. J. Epidemiol*. **40** https://doi.org/10.1093/ije/dyq190 (2011).
37. Dobson, K. G., Chow, C. H. T., Morrison, K. M. & Van Lieshout, R. J. Associations between childhood cognition and cardiovascular

events in adulthood: A systematic review and meta-analysis. *Can. J. Cardiol.* **33**, 232–242 (2017).
38. McHutchison, C. A., Backhouse, E. V., Cvoro, V., Shenkin, S. D. & Wardlaw, J. M. Education, socioeconomic status, and intelligence in childhood and stroke risk in later life: A meta-analysis. *Epidemiology* **28**, 608–618 (2017).
39. Khandaker, G. M., Barnett, J. H., White, I. R. & Jones, P. B. A quantitative meta-analysis of population-based studies of premorbid intelligence and schizophrenia. *Schizophr. Res.* **132**, 220–227 (2011).
40. Page, M. J. et al. The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. *BMJ* **372**, n71 (2021).
41. University of Vienna. *University of Vienna Ethics Committee: Tasks of the Ethics Committee*, https://satzung.univie.ac.at/en/more-parts-of-the-statutes/ethics-committee/ (2017).
42. Kanazawa, S. Mind the gap…in intelligence: Re-examining the relationship between inequality and health. *Br. J. Health Psychol.* **11**, 623–642 (2006).
43. Wicherts, J. M., Borsboom, D. & Dolan, C. V. Why national IQs do not support evolutionary theories of intelligence. *Pers. Individ. Dif.* **48**, 91–96 (2010).
44. Alemayehu, D. & Sineshaw, T. A commentary on Satoshi Kanazawa's study of intelligence and health. *Br. J. Health Psychol.* **12**, 185–190 (2007).
45. GBD 2019 Healthcare Access and Quality Collaborators. Assessing performance of the Healthcare Access and Quality Index, overall and by select age groups, for 204 countries and territories, 1990-2019: A systematic analysis from the Global Burden of Disease Study 2019. *Lancet Glob. Health* **10**, e1715–e1743 (2022).
46. GBD 2016 Healthcare Access and Quality Collaborators. Measuring performance on the Healthcare Access and Quality Index for 195 countries and territories and selected subnational locations: A systematic analysis from the Global Burden of Disease Study 2016. *Lancet* **391**, 2236–2271 (2018).
47. O'Quigley, J. *Proportional hazards regression* (Springer, 2008).
48. Higgins, J. P. T., Li, T. & Deeks, J. *Cochrane handbook for systematic reviews of interventions* (Wiley, Hoboken, USA, 2019).
49. Borenstein, M., Hedges, L. V., Higgins, J. P. T. & Rothstein, H. R. *Introduction to meta-analysis*. 2nd edn, (Wiley, 2021).
50. Wraw, C., Deary, I. J., Der, G. & Gale, C. R. Intelligence in youth and mental health at age 50. *Intelligence* **58**, 69–79 (2016).
51. Altschul, D. M., Wraw, C., Der, G., Gale, C. R. & Deary, I. J. Hypertension development by midlife and the roles of premorbid cognitive function, sex, and their interaction. *Hypertension* **73**, 812–819 (2019).
52. Der, G., Batty, G. D. & Deary, I. J. The association between IQ in adolescence and a range of health outcomes at 40 in the 1979 US National Longitudinal Study of Youth. *Intelligence* **37**, 573–580 (2009).
53. Welz, T., Viechtbauer, W. & Pauly, M. Cluster-robust estimators for multivariate mixed-effects meta-regression. *Computational Stat. Data Anal.* **179**, 107631 (2023).
54. Viechtbauer, W. I2 for multilevel and multivariate models, https://www.metafor-project.org/doku.php/tips:i2_multilevel_multivariate (2022).
55. Cohen, J. *Statistical power analysis for the behavioral sciences*. 2nd ed. (L. Erlbaum Associates, 1988).
56. Funder, D. C. & Ozer, D. J. Evaluating effect size in psychological research: Sense and nonsense. *Adv. Methods Pract. Psychological Sci.* **2**, 156–168 (2019).
57. Pustejovsky, J. E. & Tipton, E. Meta-analysis with robust variance estimation: Expanding the range of working models. *Prev. Sci.* **23**, 425–438 (2022).
58. Hegelund, E. R. et al. The influence of educational attainment on intelligence. *Intelligence* **78**, 101419 (2020).
59. Gale, C. R., Boot, T., Starr, J. M. & Deary, I. J. Intelligence and socioeconomic position in childhood in relation to frailty and cumulative allostatic load in later life: the Lothian Birth Cohort 1936. *J. Epidemiol. Community Health* **70** https://doi.org/10.1136/jech-2015-205789 (2016).
60. Gelman, A. & Loken, E. The statistical crisis in science. *Am. Sci.* **102**, 460–465 (2014).
61. Voracek, M., Kossmeier, M. & Tran, U. S. Which data to meta-analyze, and how? *Z. Psychol.* **227**, 64–82 (2019).
62. Vesely, D. *Multiverse tools*v. 1.0 (Vienna, Austria, 2023).
63. Petkari, E., Nikolaou, E., Oberleiter, S., Priebe, S. & Pietschnig, J. Which psychological interventions improve quality of life in patients with schizophrenia-spectrum disorders? A meta-analysis of randomized controlled trials. *Psychol. Med*. **54**, 221–244 (2024).
64. Sterne, J. A. C., Becker, B. J. & Egger, M. in *Publication bias in meta-analysis* 73–98 (Wiley, Hoboken, USA, 2005).
65. Rodgers, M. A. & Pustejovsky, J. E. Evaluating meta-analytic methods to detect selective reporting in the presence of dependent effect sizes. *Psychol. Methods* **26**, 141–160 (2021).
66. R Core Team. *R: A language and environment for statistical computing* v. 4.2.2 (2022).
67. Viechtbauer, W. Conducting meta-analyses in R with the metafor Package. *J. Stat. Softw.* **36**, 1–48 (2010).
68. Wickham, H. *ggplot2: Elegant graphics for data analysis*. (Springer, 2016).
69. Kossmeier, M., Tran, U. S. & Voracek, M. *metaviz: Forest plots, funnel plots, and visual funnel plot inference for meta-analysis* v. 0.3.1 (2020).
70. Thériault, R. *rempsyc: Convenience functions for psychology* v. 0.1.0 (2022).
71. Gunnell, D., Harrison, G., Rasmussen, F., Fouskakis, D. & Tynelius, P. Associations between premorbid intellectual performance, early-life exposures and early-onset schizophrenia: Cohort study. *Br. J. Psychiatry* **181**, 298–305 (2002).
72. Hemmingsson, T., Essen, J. V., Melin, B., Allebeck, P. & Lundberg, I. The association between cognitive ability measured at ages 18–20 and coronary heart disease in middle age among men: A prospective study using the Swedish 1969 conscription cohort. *Soc. Sci. Med.* **65**, 1410–1419 (2007).
73. Huang, A. R., Strombotne, K. L., Horner, E. M. & Lapham, S. J. Adolescent cognitive aptitudes and later-in-life Alzheimer disease and related disorders. *JAMA Netw. Open* **1**, e181726–e181726 (2018).
74. Jones, P., Murray, R., Jones, P., Rodgers, B. & Marmot, M. Child developmental risk factors for adult schizophrenia in the British 1946 birth cohort. *Lancet* **344**, 1398–1402 (1994).
75. Kajantie, E. et al. Stroke Is predicted by low visuospatial in relation to other intellectual abilities and coronary heart disease by low general intelligence. *PLoS One* **7**, e46841 (2012).
76. Kingma, E. et al. The prospective association between childhood cognitive ability and somatic symptoms and syndromes in adulthood: The 1958 British birth cohort. *J. Epidemiol. Community Health* **67**, 1047 (2013).
77. Koenen, K. C. et al. Childhood IQ and adult mental disorders: A test of the cognitive reserve hypothesis. *Am. J. Psychiatry* **166**, 50–57 (2009).
78. Latvala, A., Kuja-Halkola, R., D'Onofrio, B. M., Larsson, H. & Lichtenstein, P. Cognitive ability and risk for substance misuse in men: Genetic and environmental correlations in a longitudinal nation-wide family study. *Addiction* **111**, 1814–1822 (2016).
79. Mõttus, R., Luciano, M., Starr, J. M., McCarthy, M. I. & Deary, I. J. Childhood cognitive ability moderates later-life manifestation of type 2 diabetes genetic risk. *Health Psychol*. **34** https://doi.org/10.1037/hea0000184 (2015).
80. Okely, J. A., Akeroyd, M. A., Allerhand, M., Starr, J. M. & Deary, I. J. Longitudinal associations between hearing loss and general

cognitive ability: The Lothian Birth Cohort 1936. *Psychol. Aging* **34**, 766–779 (2019).

81. Rantalainen, V. et al. Cognitive ability in young adulthood predicts risk of early-onset dementia in Finnish men. *Neurology* **91**, e171 (2018).
82. Richards, M. et al. IQ in childhood and the metabolic syndrome in middle age: Extended follow-up of the 1946 British Birth Cohort Study. *Intelligence* **37**, 567–572 (2009).
83. Sjölund, S., Hemmingsson, T., Gustafsson, J. E. & Allebeck, P. IQ and alcohol-related morbidity and mortality among Swedish men and women: The importance of socioeconomic position. *J. Epidemiol. Community Health* **69**, https://doi.org/10.1136/jech-2014-204761 (2015).
84. Sjölund, S., Allebeck, P. & Hemmingsson, T. Intelligence quotient (IQ) in adolescence and later risk of alcohol-related hospital admissions and deaths-37-year follow-up of Swedish conscripts. *Addiction* **107**, https://doi.org/10.1111/j.1360-0443.2011.03544.x (2012).
85. Toulopoulou, T., Picchioni, M., Mortensen, P. B. & Petersen, L. IQ, the urban environment, and their impact on future schizophrenia risk in men. *Schizophr. Bull.* **43**, 1056–1063 (2017).
86. Twig, G. Cognitive function and the risk for diabetes among young men. *Diabetes Care* **37**, https://doi.org/10.2337/dc14-0715 (2014).
87. Walker, N. P., McConville, P. M., Hunter, D., Deary, I. J. & Whalley, L. J. Childhood mental ability and lifetime psychiatric contact: A 66-year follow-up study of the 1932 Scottish Mental Ability Survey. *Intelligence* **30**, 233–245 (2002).
88. Wennerstad, K. M., Silventoinen, K., Tynelius, P., Bergman, L. & Rasmussen, F. Association between intelligence and type-specific stroke: A population-based cohort study of early fatal and non-fatal stroke in one million Swedish men. *J. Epidemiol. Community Health* **64**, 908 (2010).
89. Whalley, L. J. et al. Childhood mental ability and dementia. *Neurology* **55**, 1455–1459 (2000).
90. Altschul, D. M., Starr, J. M. & Deary, I. J. Cognitive function in early and later life is associated with blood glucose in older individuals: analysis of the Lothian Birth Cohort of 1936. *Diabetologia* **61**, https://doi.org/10.1007/s00125-018-4645-8 (2018).
91. Ariansen, I. et al. The educational gradient in coronary heart disease: The association with cognition in a cohort of 57,279 male conscripts. *J. Epidemiol. Community Health* **69**, 322–329 (2015).
92. Barnett, J. H. et al. Childhood cognitive function and adult psychopathology: Associations with psychotic and non-psychotic symptoms in the general population. *Br. J. Psychiatry* **201**, 124–130 (2012).
93. Batty, G. D., Mortensen, E. L., Nybo Andersen, A.-M. & Osler, M. Childhood intelligence in relation to adult coronary heart disease and stroke risk: Evidence from a Danish birth cohort study. *Paediatr. Perinat. Epidemiol.* **19**, 452–459 (2005).
94. Batty, G. D. et al. IQ in early adulthood and later cancer risk: Cohort study of one million Swedish men. *Ann. Oncol.* **18**, 21–28 (2007).
95. Beaujean, A. A., Parker, S. & Qiu, X. The relationship between cognitive ability and depression: A longitudinal data analysis. *Soc. Psychiatry Psychiatr. Epidemiol.* **48**, 1983–1992 (2013).
96. Cannon, M. et al. Evidence for early-childhood, pan-developmental impairment specific to schizophreniform disorder: Results from a longitudinal birth cohort. *Arch. Gen. Psychiatry* **59**, 449–456 (2002).
97. Cannon, T. D. et al. Childhood cognitive functioning in schizophrenia patients and their unaffected siblings: A prospective cohort study. *Schizophr. Bull.* **26**, 379–393 (2000).
98. Caspi, A. et al. Cognitive performance in schizophrenia patients assessed before and following the first psychotic episode. *Schizophr. Res.* **65**, 87–94 (2003).
99. Crow, T. J., Done, D. J. & Sacker, A. Childhood precursors of psychosis as clues to its evolutionary orgins. *Eur. Arch. Psychiatry Clin. Neurosci.* **245**, 61–69 (1995).
100. Deary, I. J., Whiteman, M. C., Starr, J. M., Whalley, L. J. & Fox, H. C. The impact of childhood intelligence on later life: Following up the Scottish mental surveys of 1932 and 1947. *J. Pers. Soc. Psychol.* **86**, 130–147 (2004).
101. Fardell, C., Torén, K., Schiöler, L., Nissbrandt, H. & Åberg, M. High IQ in early adulthood is associated with Parkinson's disease. *J. Parkinsons Dis.* **10**, 1649–1656 (2020).
102. Fors, S., Torssander, J. & Almquist, Y. B. Is childhood intelligence associated with coexisting disadvantages in adulthood? Evidence from a Swedish cohort study. *Adv. Life Course Res.* **38**, 12–21 (2018).
103. Gale, C. R., Deary, I. J., Fowkes, F. G. & Batty, G. D. Intelligence in early adulthood and subclinical atherosclerosis in middle-aged men: The Vietnam Experience Study. *J. Epidemiol. Community Health* **66**, e13 (2012).
104. Hart, C. L. et al. Childhood IQ and cardiovascular disease in adulthood: Prospective observational study linking the Scottish Mental Survey 1932 and the Midspan studies. *Soc. Sci. Med.* **59**, 2131–2138 (2004).
105. Osler, M. et al. Influence of early life characteristics on psychiatric admissions and impact of psychiatric disease on inflammatory biomarkers and survival: a Danish cohort study. *World Psychiatry* **14**, 364–365 (2015).
106. Reichenberg, A. et al. Elaboration on premorbid intellectual performance in schizophrenia: Premorbid intellectual decline and risk for schizophrenia. *Arch. Gen. Psychiatry* **62**, 1297–1304 (2005).
107. Russ, T. C. et al. Childhood cognitive ability and incident dementia: The 1932 Scottish Mental Survey cohort into their 10th decade. *Epidemiology* **28**, https://doi.org/10.1097/EDE.0000000000000626 (2017).
108. Zammit, S. et al. A longitudinal study of premorbid IQ score and risk of developing schizophrenia, bipolar disorder, severe depression, and other nonaffective psychoses. *Arch. Gen. Psychiatry* **61**, 354–360 (2004).
109. Carey, E. G., Ridler, I., Ford, T. J. & Stringaris, A. Editorial Perspective: When is a 'small effect' actually large and impactful? *J. Child Psychol. Psychiatry* **64**, 1643–1647 (2023).
110. Mansfield, R. et al. The impact of the COVID-19 pandemic on adolescent mental health: A natural experiment. *R. Soc. Open Sci.* **9**, 211114 (2022).
111. Anderson, E. L. Education, intelligence and Alzheimer's disease: evidence from a multivariable two-sample Mendelian randomization study. *Int. J. Epidemiol*. **49** https://doi.org/10.1093/ije/dyz280 (2020).
112. Ambelas, A. Preschizophrenics: Adding to the evidence, sharpening the focus. *Br. J. Psychiatry* **160**, 401–404 (1992).
113. Cederblad, M., Dahlin, L., Hagnell, O. & Hansson, K. Intelligence and temperament as protective factors for mental health. A cross-sectional and prospective epidemiological study. *Eur. Arch. Psychiatry Clin. Neurosci.* **245**, 11–19 (1995).
114. Hill, W. D., Harris, S. E. & Deary, I. J. What genome-wide association studies reveal about the association between intelligence and mental health. *Curr. Opin. Psychol.* **27**, 25–30 (2019).
115. Arden, R. The association between intelligence and lifespan is mostly genetic. *Int. J. Epidemiol.* **45**, 178–185 (2015).
116. Deary, I. J., Harris, S. E. & Hill, W. D. What genome-wide association studies reveal about the association between intelligence and physical health, illness, and mortality. *Curr. Opin. Psychol.* **27**, 6–12 (2019).
117. Henrich, J., Heine, S. J. & Norenzayan, A. The weirdest people in the world. *Behav. Brain Sci.* **33**, 61–83 (2010).
118. Osler, M., Christensen, G. T., Garde, E., Mortensen, E. L. & Christensen, K. Cognitive ability in young adulthood and risk of dementia in a cohort of Danish men, brothers, and twins. *Alzheimer's Dementia* **13**, https://doi.org/10.1016/j.jalz.2017.04.003 (2017).

119. Lee, J. J. et al. Gene discovery and polygenic prediction from a genome-wide association study of educational attainment in 1.1 million individuals. *Nat. Genet.* **50**, 1112–1121 (2018).
120. Wrulich, M. et al. Childhood intelligence and adult health: The mediating roles of education and socioeconomic status. *Intelligence* **41**, 490–500 (2013).
121. Cheng, L., Ritzhaupt, A. D. & Antonenko, P. Effects of the flipped classroom instructional strategy on students' learning outcomes: a meta-analysis. *Educ. Technol. Res. Dev.* **67**, 793–824 (2019).
122. Corcoran, R. P., Cheung, A. C. K., Kim, E. & Xie, C. Effective universal school-based social and emotional learning programs for improving academic achievement: A systematic review and meta-analysis of 50 years of research. *Educ. Res. Rev.* **25**, 56–72 (2018).
123. Higgins, S. *Improving learning: Meta-analysis of intervention research in education* (Cambridge University Press, 2019).
124. Ritchie, S. J. & Tucker-Drob, E. M. How much does education improve intelligence? A meta-analysis. *Psychol. Sci*. **29** https://doi.org/10.1177/0956797618774253 (2018).
125. Pietschnig, J. & Voracek, M. One Century of Global IQ Gains: A Formal Meta-Analysis of the Flynn Effect (1909–2013). *Perspect. Psychol. Sci.* **10**, 282–306 (2015).
126. Bates, T. C., Lewis, G. J. & Weiss, A. Childhood Socioeconomic Status Amplifies Genetic Effects on Adult Intelligence. *Psychol. Sci.* **24**, 2111–2116 (2013).

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