Intelligence and life expectancy in late adulthood: A meta-analysis

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ABSTRACT

In an aging society, it is crucial to understand why some people live long and others do not. There has been a proliferation of studies in recent years that highlight the importance of psycho-behavioural factors in the ways of aging, one of those psychological components is intelligence. In this meta-analysis, the association between intelligence and life expectancy in late adulthood is analysed through the Hazard Ratio (HR). Our objectives are: (i) to update Calvin's meta-analysis, especially the estimate of the association between survival and intelligence; and (ii) to evaluate the role of some moderators, especially the age of the participants, to explore intelligence–mortality throughout adulthood and old age. The results show a positive relationship between intelligence and survival (HR$_\bullet$: 0.79; 95% CI: 0.81–0.76). This association is significantly moderated by the years of follow-up, the effect size being smaller the more years elapse between the intelligence assessment and the recording of the outcome. Intelligence is a protective factor to reach middle-high age, but from then on survival depends less and less on intelligence and more on other factors.

1. Introduction

Population aging is a global phenomenon. Increasing survival is increasing population; furthermore, demographic studies highlight that we can find more older old cohorts in our aging society.

Traditionally, two major lines of research have been applied to understand why some people live long and others do not; studies on genetic or intrinsic components and environmental or extrinsic factors (e.g., Fernández-Ballesteros, 2017). As pointed out by Fernández-Ballesteros and Sánchez-Izquierdo (2019), "environmental or extrinsic factors, include a broad heterogeneity of conditions (physical, economic, social, and cultural aspects as well as some behavioural ones such as lifestyle) but do not include personal conditions, such as psycho-behavioural characteristics".

There has been a proliferation of studies in the last years highlighting the importance of psycho-behavioural factors intervening in the ways of aging, specifically personality traits, positive emotion and control, psychosocial, self-stereotypes, intelligence and cognitive functioning, physical conditions, and lifestyles, all of which are highly associated with active aging, health, longevity, and survival (see e.g., Calvin et al., 2011; Chida & Steptoe, 2008; Deary, Batty, Pattie, & Gale, 2008; Deary, Hill, & Gale, 2021; Gottfredson, 2004; Kern, Friedman, Martin, Reynolds, & Luong, 2009; Lindahl-Jacobsen & Christensen, 2019; Wilson, Mendes, Bienias, & Da., 2004; Wilson et al., 2005; Deary, 2009).

One of these psychological components is intelligence. Individual differences in intelligence (cognitive ability, mental ability) test scores, as measured by standardized IQ-type tests in childhood, predict later achievements in schooling, educational attainments, social adjustment, and job success or job satisfaction (Kaplan, Pamuk, Lynch, Cohen, & Balfour, 1996; Schmidt & Hunter, 1998) and show an inverse association with risk of death from all causes throughout adulthood (i.e., Deary et al., 2008, 2021; Jokela, Batty, Deary, Gale, & Kivimäki, 2009).

The association between intelligence in youth and all-cause-mortality was the subject of a meta-analysis (Calvin et al., 2011), in which all 16 studies that met the inclusion criteria demonstrated an inverse relationship between intelligence (quantifying a 1-standard deviation (SD)) and a 24% lower risk of dying during a 17- to 69-year follow-up, with no differences between men and women, and which was not explained by socio-economic differences in early life, as indicated by parental occupation or income.

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There were insufficient studies to comprehensively address a number of pertinent questions from this research domain, one issue being whether or not the association between intelligence and mortality is the same for younger adults as for older adults. There was no meta-analysis including data from older adults, analysing whether or not the association between intelligence and mortality remains the same across adulthood and old age.

A second issue to re-evaluate was the influence of Socio-economic status (SES) as a predictor of mortality. Although Batty, Deary, and Gottfredson (2007) found some studies that suggested intelligence had independent effects on risk of mortality from those of early socio-economic influences, it was Calvin et al. (2011) that suggested that SES was not a confounder of the intelligence–mortality association. Along this line, Gottfredson (2004) argued that underlying IQ differences explained social inequalities in health and that these were not necessarily mediated via socioeconomic status.

Following Kilgour, Starr, and Whalley (2010) a number of methodological considerations in intelligence–mortality studies must be addressed: taking into account ascertainment bias, age and sex. In this article, we addressed the influence of these factors as possible moderators.

In summary, this report aimed to (i) update Calvin's meta-analysis and confirm the quantification of the association with intelligence––and (ii) conduct subgroup analyses on studies according to the age of participants, to discover the intelligence–mortality association across adulthood and old age.

2. Materials and methods

This report follows the guidelines of the APA task force recommendations regarding reporting standards for quantitative research in Psychology and, especially, meta-analysis articles (Appelbaum et al., 2018, table 9).

2.1. Search strategy

A systematic search was performed on three websites that provide access to multiple databases related to academic articles. The websites reviewed were PsycINFO, MEDLINE, and EMBASE, from February 1st, 2010, to November 15th, 2021. That period is a successive period from Calvin et al. (2011) and allowed us to update Calvin's original meta-analysis. The search strategy was guided by a specific question: Is intelligence related to mortality? The selected keywords were based on Calvin's publishing. The keywords for cognitive ability were: "Aptitude or Cognition" or "Cognitive function" or "Cognitive ability" or "Cognitive characteristics" or "Cognitive style" or "intellectual ability" or "Intelligence measures" or "Intelligence quotient" or "Intelligence test" or "Intelligence" or "IQ or Language test" or "Memory" or "Mental ability" or "Mental capacity" or "problem-solving" or "Problem solving" or "Psychological performance" or "Psychometrics". And for mortality terms were: "Cause of Death" or "Cause of Death trends" or "Death" or "death rate" or "Incidence" or "Morbidity" or "Morbidity trends" or "Mortality Rate" or "Mortality risk" or "Mortality" or "Mortality trends". The words selected were introduced as free terms and were searched in the title, abstract, and keywords boxes. The search strategy is available in Supplementary material (Table S1) Two authors (M.S-I. and E.L.) independently scanned each title and abstract, retrieving articles based on their relevance to intelligence and mortality.

2.2. Inclusion and exclusion criteria

The inclusion criteria were as follows: (1) the study assessed the association between intelligence and mortality; (2) the design of the study was a prospective, longitudinal study; (3) the study reported statistical results on the association between intelligence and mortality. The exclusion criteria were as follows: (1) the study did not specifically address mortality as an outcome variable; (2) the study did not assess intelligence in youth; (3) intelligence was not considered a predictor; (4) the design of the study was not longitudinal. Additionally, if two studies were based on the same dataset, even partially, the most recent study was selected. When a study did not report the statistics reflecting the association assessed and their confidence interval, an exact p-value was required to estimate the confidence interval. If the confidence interval or exact p-value were not reported, the study was excluded. The flow diagram of study identification and selection is shown in Fig. 1.

2.3. Reviewing procedure and data extraction

Database searches were conducted on November 15th, 2021. Potentially eligible studies were selected in two steps; the first step was based on the title and abstract screening. Irrelevant references were removed. The second step was based on the full-text reading of potentially relevant studies. The pre-specified eligibility criteria were checked in both phases. For each reference, the following variables were systematically extracted and entered into a summary table: (1) Author, year; (2) number of follow-up years; (3) sample size at baseline; (4) percentage of female participants; (5) average years of birth; (6) mean age at baseline; (7) overall death rate at the end of the study; (8) Dataset or project name; (9) outcome measured at the original study; (10) intelligence assessment (name)/Cohort type; (11) intelligence test description; (12) control variables; (13) Index type (Hazard ratio, odds ratio, relative risk); (14) the magnitude of the effect size (1-SD or percentiles); (15) Confidence interval of the effect size; (16) Reference group. The collected data is available via the author's mail account (see Table S2).

2.4. Intelligence measures

A previous meta-analysis (Calvin et al., 2011) analysed the effect size for mortality per 1-SD of IQ score. However, we identified that some previous and new studies reported effect sizes based on two different operationalisations: (1) categorization of the IQ score based on being 1-SD above the mean or not; and (2) categorization based on percentiles, such as quintiles, quartiles, or tertiles. The first case implied the comparison between the group with an IQ score with a 1-SD advantage and the reference group (subjects with any other IQ score). The latter scenario implied the comparison of the high-percentile group with the reference group comprising participants in the low percentile. For example, a group with individuals in the third tertile, i.e., above the 67th percentile, and the other with individuals below the 33rd percentile. We extracted both effect sizes, when reported in the study, and it allowed us to perform a second analysis. When studies reported separate estimates by gender, these were taken as two different samples in the same study.

Additionally, the effect sizes after analysing a model with control variables were also extracted and analysed. Those control variables were socio-economic factors, such as childhood SES, adult SES, and education. In this article, we addressed the influences of childhood SES using subgroup analyses. The other factors, such as adult SES and education, were not separately analysed in the selected new studies.

Finally, 22 studies assessed intelligence, 16 studies were selected from Calvin's meta-analysis and 6 were new studies. Twenty-one studies
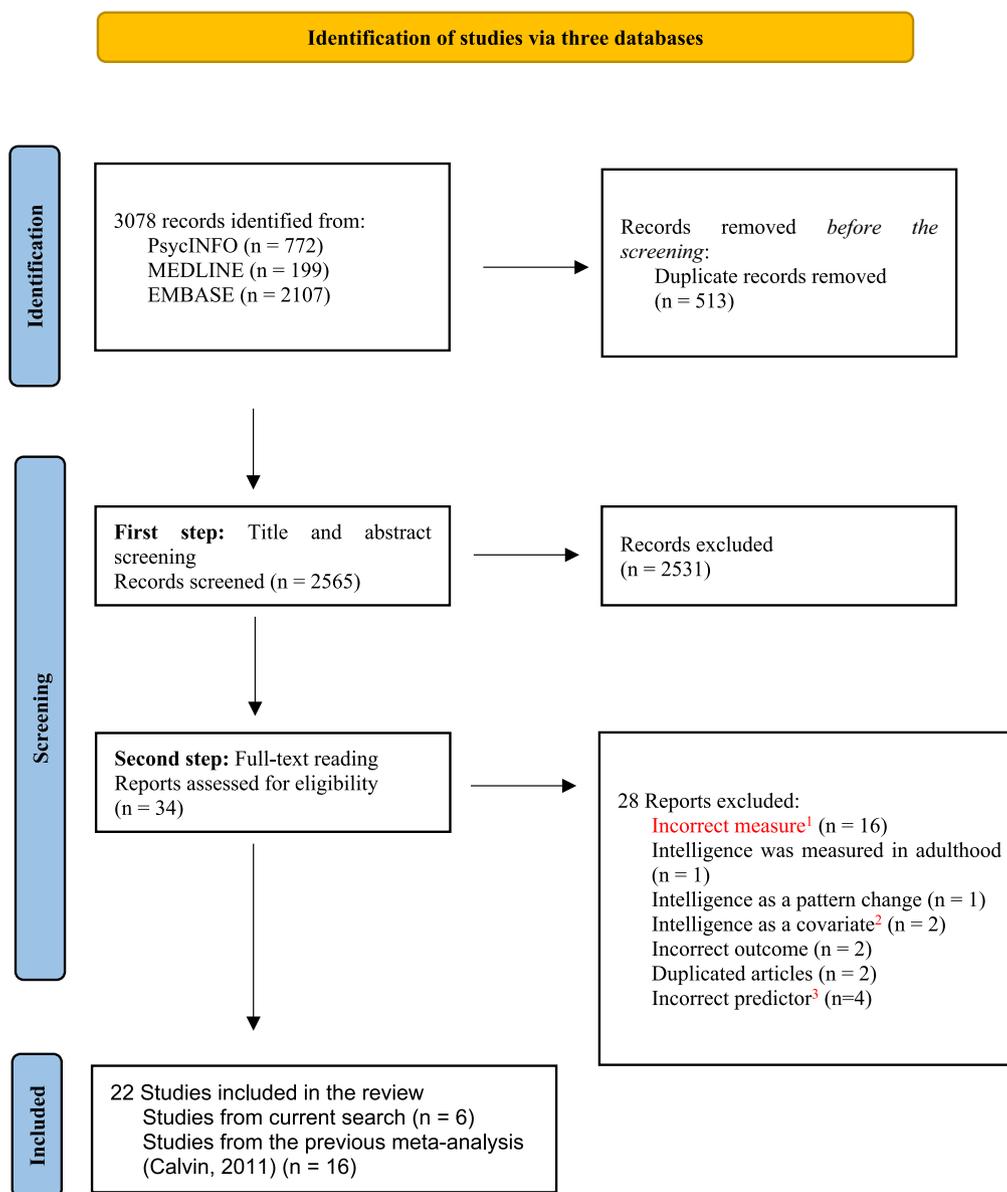
Identification of studies via three databases

3078 records identified from:
  PsycINFO (n = 772)
  MEDLINE (n = 199)
  EMBASE (n = 2107)

Records removed *before the screening*:
  Duplicate records removed (n = 513)

First step: Title and abstract screening
Records screened (n = 2565)

Records excluded (n = 2531)

Second step: Full-text reading
Reports assessed for eligibility (n = 34)

28 Reports excluded:
  Incorrect measure[1] (n = 16)
  Intelligence was measured in adulthood (n = 1)
  Intelligence as a pattern change (n = 1)
  Intelligence as a covariate[2] (n = 2)
  Incorrect outcome (n = 2)
  Duplicated articles (n = 2)
  Incorrect predictor[3] (n=4)

22 Studies included in the review
  Studies from current search (n = 6)
  Studies from the previous meta-analysis (Calvin, 2011) (n = 16)

Identification / **Screening** / **Included**

Fig. 1. Flow diagram of study identification and selection.
From: Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ 2021;372:n71. doi: https://doi.org/10.1136/bmj.n71
[1] Intelligence was measured by tests with an unacceptable degree of validity.
[2] In these studies, intelligence was not a relevant variable and, therefore, its estimation was not reported in the results table.
[3] The measure of intelligence was not considered an independent variable, the predictor of mortality being another construct.

informed a total of 25 effect sizes based on 1-SD categorization, four from the studies that reported separate estimates by gender. Note that these 25 estimates represent the basic, raw models tested in the original studies, that is, the effect size before controlling for the variables included in the model.[1] The total number of participants amounted to 1,197,946 (8.6% more than in the Calvin MA), while the total number of events (deaths) amounted to 52,667 (1.3-fold more than in the Calvin MA).

From the second categorization of effect sizes based on percentiles, nine studies were analysed, and three studies informed separate estimates by gender. In the end, 12 estimates were available from the second scenario analysis. Of the total of six new studies, three reported an additional effect size after controlling for childhood SES. These studies were added to the previous nine studies analysed by Calvin and colleagues.

The complete database is included in Supplemental material (see Table S3).

2.5. Description of the moderators assessed

Five moderators were considered in the analysis: (1) participant's mean age at the study's baseline (MAB); (2) follow-up years of the study (FUY); (3) percentage of women in the study (SEX); (4) year of publication (PUBY); (5) mean age at the study's endpoint (MAE).

The first four moderators were extracted directly from the selected articles. The fifth moderator, MAE, was calculated to answer a second question derived from the main goal: "Does the relationship between intelligence and mortality change in the older adults?" In that sense, MAE is the sum of the mean age of the subjects during the first assessment and the total number of years of the study. The fifth moderator was coded as 1 when studies obtained an MAE value equal to or over 65

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[1] We are aware that the values obtained with the raw, basic model are not strictly comparable with those provided by estimates after controlling for some basic moderators, such as sociodemographic ones. To preserve all the studies within the meta-analysis but at the same time prevent distorted results, we carried out a sensitivity analysis. Specifically, we compared the ES of studies without moderators with those of studies that included the basic moderators. As can be seen in the supplementary material (Table S4), the difference is small, and not statistically significant, so we have continued the analyses without taking this distinction into account.

years, the other studies were coded as 0, i.e., MAE value lower than 65 years.

2.6. Effect size and statistical model

The effect size index for this meta-analysis was the (Okun, Yeung, & Brown, 2013) (HR) for mortality. The numerator value always refers to the condition assessed, and the denominator is the category for control. Thus, values <1 reflect lower mortality (greater survival), whereas values larger than 1 reflect higher mortality (lower survival). The results of the primary studies were generally presented directly as HR values but sometimes as proportions, relative risks (RR), or odds ratios (OR). The formulas of Zhang and Kai (1998); (see Okun et al., 2013) were used to convert the OR and/or RR values to HR values. Variances were obtained from the reported confidence intervals or the exact *p*-values of the significance tests. The values were previously transformed to their logarithms for statistical analysis in order to have a more symmetric distribution. The results reported below have already been back-transformed to the original metric, appearing as HR values throughout the paper.

Variability between studies was evaluated using the Q statistic, as a test of heterogeneity, and the I^2 statistic (Huedo-Medina, Sánchez-Meca, Marín-Martínez, & Botella, 2006). For the pooled estimate, the values were weighted by the inverse of their variances. Random effects models were assumed instead of the fixed-effect model. Random effect models are generally preferred because they are more conservative and allow generalising conclusions beyond the specific set of studies analysed (Borenstein, Hedges, Higgins, & Rothstein, 2010). The specific variance was estimated via the restricted maximum likelihood method.

Meta-regression moderator analyses were performed to assess five potential sources of heterogeneity: participants' mean age at baseline, length of follow-up, gender (percentage of women), year of publication, and mean age at the endpoint of the study.

As reflected in the asymmetry of the funnel plot, the risk of publication bias was assessed by visual inspection of the figure and some statistical tests, such as Egger's test, the rank correlation test, and the Trim and Fill method. We also calculated the fail-safe numbers (Rosenthal, 1979). The results of these analyses are summarized in Table 3. The analyses and figures were performed using the R package "metafor" (Viechtbauer, 2010). We did not apply other methods, such as p-uniform, because the number of significant studies was generally too small to obtain reliable results (Blázquez, Botella, & Suero, 2017).

3. Results

3.1. Study characteristics

The database search initially produced 3078 records and, after removing 513 duplicated articles, it afforded 2565 unique articles. Of these, 2531 were excluded in the first step, based on the title and abstract (Fig. 1). Of the remaining 34 articles, 28 were excluded in the second step, based on full texts. This process resulted in a total of six new articles in the current research. Additionally, 16 studies were already included in the previous meta-analysis (Calvin et al., 2011). In short, 22 studies were included in the meta-analysis (Table S2 provides an overview), totalling 1,197,946 participants, of whom 52,667 died (4.4%).

The average sample size was 47,917.84, with a median sample size of 4316, ranging from 610 to 994,262. The average age at baseline was 12.5 years (7–20 years). The 25 independent estimates (1-SD increase of IQ categorization) were part of studies conducted in Europe (32%, n = 8), the United States (16%, n = 4), the United Kingdom (48%, n = 12), and Australia (4%, n = 1). The average follow-up time was 49.7 years, varying between 17 and 69 years (See Table 1).

Table 1

Characteristics of the five moderators studied, mean values (range) and frequency.

Moderators studied		Categorization 1: IQ score by 1-SD standard deviation (n = 25)	Categorization 2: IQ score by percentiles (n = 12)
Average mean age at baseline of the study (MAB) [range]		12.5 [7–20]	11 [7–18]
Average % of women [range]		34.7 [0–100]	39.6 [0–100]
Average follow-up years (FUY) [range]		48.9 [17–69]	49.8 [17–69]
Average publication year (PUBY) [range]		2009 [1988–2020]	2007 [1988–2015]
Mean age at the endpoint of the study (MAE) [% of studies]	MAE = 0	<65 [48%, n = 12]	–
	MAE = 1	≥65 [52%, *n* = 13]	–

MAB = Mean of participant's age at the baseline of the study; FUY = follow-up years of the study; SEX = percentage of women in the study (0 = studies with male participants only); PUBY = year of publication; MAE = mean age at the endpoint of the study.

3.2. Association of intelligence with mortality risk

Fig. 2 shows the forest plot with the 25 separate estimates when the factor is defined as being (versus not being) above the mean by at least 1-SD. These estimates correspond to the basic, raw model analysed in the original studies. The combined estimate revealed a significant association of high intelligence with the risk of mortality [HR• = 0.784; 95%CI: 0.757–0.811], supporting the hypothesis that intelligence is an important factor associated with a significantly lower rate of mortality. Having an intelligence of at least 1-SD above the mean reduces the mortality rate by about 21.6%. The 12 independent estimates that controlled for childhood SES revealed a similar pooled effect size [HR• = 0.788; 95% CI: 0.759–0.817], according to which high intelligence seems to reduce mortality by about 21.2%. Childhood SES does not moderate the potential of intelligence for predicting mortality. In both cases, the heterogeneity of the estimates was significant [Q(24) = 182.96 and Q(11) = 23.40, respectively; *p* < .001 in both cases], although the I^2 value was much higher when the estimate did not control for childhood SES (91.5%) than when it did (60.6%).

As expected, when the intelligence groups are defined as high versus low IQ (excluding individuals with intermediate values), the index is more extreme [HR• = 0.68; 95%CI: 0.62–0.75], but the interpretation does not change: the rate of mortality is smaller for high intelligence (about 31.6%). The heterogeneity is again significant [Q(11) = 13.45, p < .001], but the I^2 value is much smaller (20.8%).

3.3. Moderator analysis

Five variables (MAB, FUY, SEX, PUBY, and MAE) were analysed to evaluate the role of potential moderators. As the power of the tests greatly reduces when the number of studies is small, we performed these analyses with the operationalization that provided a larger number of estimates, i.e., with the 25 estimates that defined the focus group as having an IQ score of at least 1-SD above the mean, and the reference group as any other IQ score. As no role for childhood SES was observed, we did not follow it with any additional analysis.

Table 2 shows the results of the meta-regression models for the five factors. The non-significant slope of the sex factor suggested that sex did not moderate the association between intelligence and mortality. Essentially, that association was the same for men and women. The other three slopes were significant (MAB, FUY, and PUBY), revealing that they play a relevant role in the association between intelligence and mortality. In order to interpret the results, it must be borne in mind that
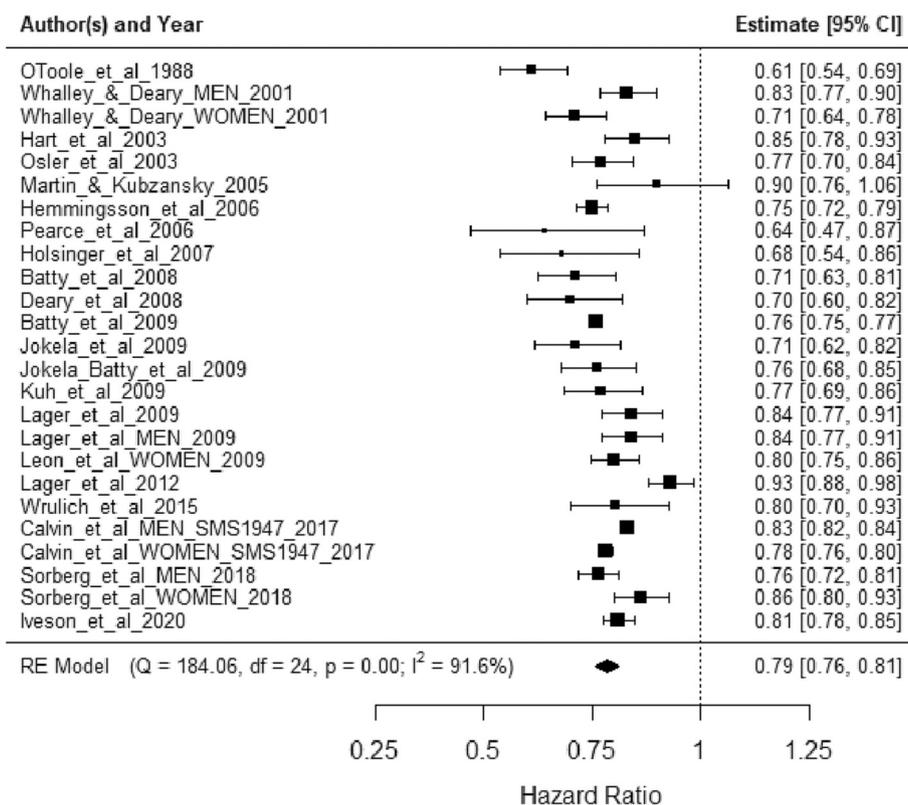
Fig. 2. Forest Plot with the 25 separate estimates of intelligence[1].

[1]Intelligence is defined as being (versus not being) above the mean in at least one standard deviation (See description in Method).

Table 2
Results of meta-regression models on intelligence[1].

Moderator	k	Slope	95%CI	Q_R
MAB	24	−0.011 **	−0.018 - -0.003	7.73 **
FUY	25	0.004 ***	0.002–0.005	18.11 ***
SEX	24	0.0001 n.s.	0.001–0.001	0.02 n.s.
PUBY	25	0.006 *	0.001–0.010	6.19 *
MAE	25	0.083 **	0.023–0.142	7.47 **

"*" $p < .05$, "**" $p < .01$, "***" $p < .001$.

[1] High intelligence is defined as at least 1-SD above the mean. MAB = Mean of participant's age at the baseline of the study; FUY = follow-up years of the study; SEX = percentage of women in the study (0 = studies with male participants only); PUBY = year of publication; MAE = mean age at the endpoint of the study (MAE = 0, when participants' mean age at the end of the study is lower than 65 year, and MAE = 1 when the mean age is equal to or over 65 years).

a lower HR index indicates a higher association, as values are <1, the no-effect value. Specifically, the negative slope for MAB means that the association was stronger as mean age increased. In many studies, intelligence was assessed in childhood or adolescence. The studies with very young baseline samples had less predictive potential than those in which intelligence was assessed at adult ages. The significant (although small) positive slope for FUY reflected that the association was slightly smaller as more years elapsed between time1 (intelligence assessment) and time2 (check for survival). The significant and positive slope for PUBY must be interpreted as recent studies tending to show a weaker association between intelligence and mortality than older studies. Of course, it is logical that PUBY and MAE show convergent results, since they are constructs that overlap to some degree. In theory the value of MAE has to be small if PUBY is very large. In fact, the 24 studies that

provided this value showed a significant negative correlation ($r = −0.508$; $p = .011$).

The fifth potential moderator assessed was the MAE. As explained above, we were interested in whether the predictive power of intelligence for survival changed when survival was checked in old people. For that purpose, we categorized the studies into two groups (see Method, "*Description of the moderators assessed*"). The model fitting this categorical classification revealed a significant role (Slope = 0.08, $p < .01$, 95% IC [0.02–0.14]). The latter result allows us to answer the second question: "Does the relationship between intelligence and mortality change in older adults?" Yes, that relationship changes when the most long-lived studies were compared with the youngest studies. In other words, the positive slope of MAE (mean age at the endpoint of the study) reflected a weaker association the older the participants were at the endpoint of the studies. The studies with older samples at the endpoint had less predictive potential than those in which the participants were under the age of 65 at their endpoint. This conclusion had already been reached in some primary studies (e.g., Hart et al., 2003).

More specifically, the studies with older samples at the endpoint had less predictive potential ($HR_\bullet = 0.815$) than those in which the participants were under the age of 65 at their endpoint ($HR_\bullet = 0.750$).

3.4. Publication bias

As publication bias can give rise to overestimates of the effect size, we evaluated the degree to which this anomaly could be a potential threat to the results of this meta-analysis. Egger's test (Test = −20.73; $p = .04$) revealed a significant asymmetry that was clearly visible in the funnel plot (Fig. 3), whereas the rank correlation test did not reach significance (Test = −0.15; $p = .32$). When applying the Trim and Fill
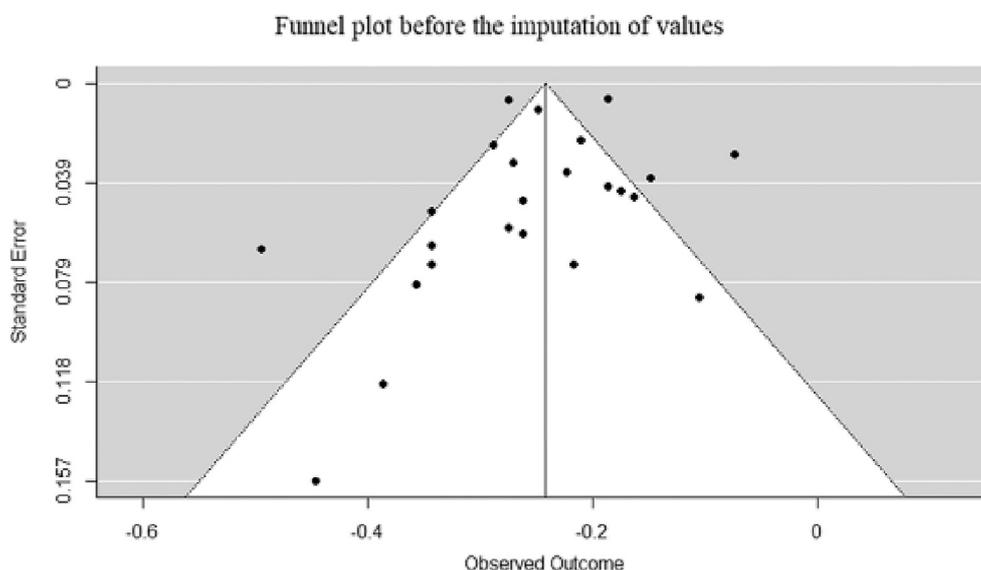
Funnel plot before the imputation of values

Fig. 3. Publication bias analysis based on the funnel plot.

method, four missing estimates were imputed. Of course, the estimated association after the imputation was smaller [HR. = 0.79; 95%CI: 0.77–0.83], but close to the uncorrected estimation. Furthermore, the fail-safe number was very high (Nfs = 15,778) and well above Rosenthal's criterion (see also Table 3). In summary, we believe that the observed effect is not the product of massive publication bias and that the possible effect of overestimating the pooled ES is small and does not change the conclusions of the meta-analysis.

4. Discussion

>20 longitudinal studies from several countries (Australia, Denmark, Luxembourg, Sweden, United Kingdom, and USA) have demonstrated the link between higher intelligence and longer life. This gave rise to the field of cognitive epidemiology, which focuses on understanding the relationship between cognitive functioning and health.

This study aimed to update Calvin's meta-analysis, confirm the quantification of this association and also analyse whether the intelligence–mortality association varies across adulthood and old age. We found evidence that having intelligence of at least 1-SD above the mean seems to reduce the mortality rate, although our rate was a little lower (21.6%) than that of Calvin's meta-analysis (24%).

Another objective of the study was to analyse the influence of several factors as possible moderators, specifically bias, age, and sex. Our results showed that recent studies tend to find a weaker association between intelligence and mortality than older studies. Along this line, Calvin and colleagues have shown a trend for larger cohorts accumulating in more recent years (Calvin et al., 2011).

Table 3
Analysis of publication bias associated with significant estimates.

Factors	N_{fs}	RC	Rank[T]	Egger[T]	Trim&Fill	
					Imputed values	Corrected estimation
IQ score by 1-standard deviation	15,778	155	0.32	0.04*	4	HR. = 0.79 (0.77–0.83)
IQ score by percentiles	214	85	0.95	0.62	3	HR. = 0.791 (0.67–0.94)

"*" $p < .05$. Egger's Asymmetry Test and the rank correlation test p-value $< .05$ are in bold. RC = Rosenthal's criterion. [T] = p-value for Egger's Asymmetry Test and the rank correlation test. IQ = Intellectual coefficient.

The average life expectancy of women exceeds that of men, however, sex does not moderate the association between intelligence and mortality, being the same for men and women, as previously reported in twin longitudinal studies (Arden et al., 2016) and Calvin et al. (2011).

The question "What causes the relationship between intelligence and longevity/ mortality?" remains unsolved and crucial. Factors such as childhood environment, family income, schooling, and healthy/unhealthy lifestyle habits (diet, exercise, tobacco use, alcohol, illnesses), have been studied (Deary, Weiss, & Batty, 2010; Whalley & Deary, 2001). As Deary et al. (2021) suggested, there seems to be a reciprocal dynamic association between intelligence and health throughout life, and although there are several constructs associated with health/ illness and death (e.g., parental social class, intelligence in youth, more education, higher health literacy, healthy behaviors, and more affluent social class) shared genetic differences are likely to account for only a small proportion of these associations.

In this study we wanted to re-evaluate the influence of Socioeconomic status as a predictor of mortality; our results showed that childhood SES did not moderate the potential of intelligence for predicting mortality. Although several studies (Batty et al., 2007; Hemmingsson, Melin, Allebeck, & Lundberg, 2009) suggested that intelligence had effects on the risk of mortality independent from those of early socio-economic influences, other studies suggested that SES was not a confounder of the intelligence–mortality association (Calvin et al., 2011, 2017). Furthermore, a study of over 900 Scottish participants (Hart et al., 2003) found that statistically controlling for economic class and a measure of "deprivation" reflecting unemployment, overcrowding, and other adverse living conditions accounted for only about 30% of the IQ-mortality correlation.

Along this line, Gottfredson (Gottfredson, 2004) argued that underlying IQ differences explained social inequalities in health and that these were not necessarily mediated via adult/person's-own SES. This idea was tested by Batty, Der, Macintyre, and Deary (2006) who found that IQ does not completely explain socioeconomic inequalities in health, however, it might contribute to them through a variety of processes.

Another line of research suggested that genes may contribute to the link between IQ and mortality. Arden and colleagues (Arden et al., 2016) analysed three twin studies (from the U.S., Denmark, and Sweden) and found a small positive phenotypic correlation between intelligence and lifespan, furthermore, in the combined sample, the genetic contribution to covariance was 95%; in the US study, 84%; in the Swedish study, 86%, and in the Danish study, 85%. As the authors

highlighted, any genetic factors that contribute to intelligence and mortality may operate indirectly via good health choices or higher income which leads to better healthcare. Deary, Harris, and Hill (2019); Deary et al. (2021)) reviewed the genetics through genome-wide association studies (GWASs), Genome-wide complex trait analysis (GREML), and LD regression studies, which allowed them to estimate genetic correlations between phenotypes (intelligence and health).

The second question of this study: "Does the relationship between intelligence and mortality change in the older adults?" Yes, that relationship changes when the most long-lived studies were compared with the youngest studies. As several studies have suggested (Arden et al., 2016; Hart et al., 2005), the causes of the association between intelligence and lifespan may vary between ages. Childhood IQ has been related to mortality in Scottish populations: Hart et al., (2005) showed that childhood IQ was significantly related to deaths occurring up to age 65, but not to deaths occurring after age 65, whereas the Aberdeen study found that people with a lower IQ were less likely to be alive at age 76 (Whalley & Deary, 2001).

Analysing whether the relationship between intelligence and mortality changes in the older adults, our results showed a small but significant positive slope for FUY, which reflected that the association was slightly smaller as more years elapsed between time 1 (intelligence assessment) and time 2 (check for survival). This means that the relationship between intelligence and survival is dampened. Our findings confirm results from the Midspan studies (Hart et al., 2005). As suggested by several studies, one possible reason might be that higher IQ might be associated with better healthcare and engaging in healthier behaviors (Deary et al., 2019; Hart et al., 2005; Wraw, Der, Gale, & Deary, 2018), which is associated to a lower mortality risk (Gottfredson, 2004; Gottfredson & Deary, 2004). IQ might also predispose to conditions of adult life (Marmot & Kivimäki, 2009), quitting smoking in later life (Batty et al., 2007; Daly & Egan, 2017), and entry into safer environments (Whalley & Deary, 2001), which promote staying healthier and living longer.

One way of discovering why intelligence and mortality are related and why this association seems to be smaller at higher ages might be to review the specific causes of death to which intelligence relates from childhood and adulthood. Along this line, several studies have shown its association with most of the major causes of death. The main literature has reported inverse patterns of the association between childhood intelligence and respiratory disease (Batty, Deary, & Zaninotto, 2016; Calvin et al., 2017), coronary heart disease (e.g., Calvin et al., 2017; Christensen, Mortensen, Christensen, & Osler, 2016; Hart et al., 2004; Lawlor, Ronalds, Clark, Davey Smith, & Leon, 2005;), stroke (Calvin et al., 2017), total cardiovascular disease (Batty et al., 2016; Calvin et al., 2017; Christensen et al., 2016; Hart et al., 2003, 2004; Hemmingsson, Melin, Allebeck, & Lundberg, 2006; Leon, Lawlor, Clark, Batty, & Macintyre, 2009), digestive disease (Calvin et al., 2017), cancer (Batty et al., 2009, Batty et al., 2016; Leon et al., 2009), specifically with smoking-related diseases (Calvin et al., 2017), dementia (Calvin et al., 2017; Russ et al., 2013), and suicide (Hemmingsson et al., 2006,

Deary et al. (2021) presented consistent results showing intelligence associated with several causes of death (cardiovascular disease, coronary heart disease, stroke, respiratory disease, diabetes, digestive disease, dementia, non-smoking-related cancers, accidents and suicide), illnesses (hypertension, metabolic syndrome, diabetes, schizophrenia, and major depression), health biomarkers (e.g. systolic and diastolic blood pressure, heart rate, triglycerides and cholesterol, body mass index), and health behaviors (smoking and physical inactivity). As the authors highlight, intelligence's long-term association with health is mediated via adult social factors and health behaviors.

5. Limitations

The present meta-analysis includes large published studies representing in total >47,000 average sample size. However, it includes 22

studies: 16 studies were already included in the previous meta-analysis (Calvin et al., 2011) and six new articles.

Although all studies were adjusted for multiple potential moderators, there are likely to remain different factors, such as SES in adulthood, cause of death in the intelligence–mortality association, etc., that could substantially affect the results.

For those reasons, the combined models are not strictly comparable, since other moderators are frequently added to childhood SES and it is not possible to disentangle their effects in the meta-analysis. Although we are aware of this potential weakness, we have preferred to perform and report this analysis. If we had found an effect, it would have been difficult to interpret, but we did not any effect of this moderator. As might be expected, the inclusion of moderators leads to a significant increase in heterogeneity, which we may interpret to mean that some of the moderators increase the effect and others reduce it, but we cannot identify the role each of them plays at the meta-analytic level.

Also, future research should explore mediating effects on a pathway from premorbid intelligence to the risk of mortality, taking into account common genetic effects (e.g. with GWAS) and the role of socioeconomic status, health literacy, and adult environments and behaviors.

It should also be important to include other countries and cultures in the studies.

6. Conclusions

(1) There is a positive relationship between intelligence and survival.
(2) The most robust moderator is years of follow-up, in the sense that the relationship between intelligence and survival is dampened. This makes sense, as health other situational factors and causes of death at different ages become more important over the years and the role of intelligence necessarily becomes less important.
(3) Intelligence is a protective factor for reaching upper-middle age, but thereafter survival depends less and less on intelligence and more on other factors.

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Institutional review board statement

Not applicable.

Declaration of Competing Interest

The authors declare no conflict of interest.

Data availability

Data will be made available on request.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.intell.2023.101738.

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