



Premorbid (early life) IQ and Later Mortality Risk: Systematic Review

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PURPOSE: Studies of middle-aged and particularly older-aged adults found that those with higher scores on tests of IQ (cognitive function) had lower rates of later mortality. Interpretation of such findings potentially is hampered by the problem of reverse causality: such somatic diseases as diabetes or hypertension, common in older adults, can decrease cognitive function. Studies that provide extended follow-up of the health experience of individuals who had their (premorbid) IQ assessed in childhood and/or early adulthood minimize this concern. The purpose of the present report is to systematically locate, evaluate, and interpret the findings of all such studies.

METHODS: We systematically identified individual-level studies linking premorbid IQ with later mortality by using four approaches: search of electronic databases (MEDLINE, EMBASE, and PSYCHINFO); scrutiny of the reference sections of identified reports; search of our own files; and contact with researchers in the field. Study quality was assessed by using predefined criteria.

RESULTS: Nine cohort studies met the inclusion criteria. Overall, study quality was moderate. All reports showed an inverse IQ–mortality relation; i.e., higher IQ scores were associated with decreased mortality risk. The nature of this relation (i.e., dose–response or threshold) and whether it differs by sex was unclear. The IQ–mortality association did not appear to be explained by reverse causality or selection bias. Confounding by other early-life factors also did not seem to explain the association, although some studies were not well characterized in this regard. Adult socioeconomic position appeared to mediate the IQ–mortality association in some studies, but this was not a universal finding.

CONCLUSIONS: In all studies, higher IQ in the first two decades of life was related to lower rates of total mortality in middle to late adulthood. Some plausible mechanistic pathways exist, but further examination is required. The precise nature of the IQ–mortality relation (particularly in ethnic minorities and women) and the link between IQ and disease-specific outcomes also warrants further research.

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INTRODUCTION

Developed a century ago, psychometric tests of intelligence (denoted by IQ) have been used most commonly in educational and workplace settings. During the last decade, investigators began to examine the predictive significance of IQ for health outcomes (1). Several studies of middle-aged (2, 3) and, particularly, older-aged (4, 5) adults found that those with higher scores on tests of mental ability had lower rates of later all-cause mortality. These associations may occur

across the full range of test scores (6, 7), rather than just in persons with severe mental impairment, although this is not a universal observation (4). Mechanisms advanced to explain these findings include the suggestion that individuals with higher cognitive function are more likely than their lower-scoring counterparts to recognize early symptoms of disease, adhere to medication use, and engage in behaviors conducive to health (8, 9).

Although these cognition–mortality gradients do not appear to be explained by confounding, their interpretation is hampered by the issue of reverse causality: it is well established that such somatic diseases as diabetes or hypertension, common in middle-aged and, particularly, older-aged adults, can lead to a decrease in cognitive function (10, 11). Therefore, it may be this morbid load, rather than low cognitive function itself, that generates the elevated risk for mortality. Studies that provide extended follow-up of the mortality experience of individuals who had their IQ assessed in childhood or early adulthood, when scores are likely to be premorbid, minimize this concern.

Several such studies have appeared, particularly in recent years (12–14). Although brief nonsystematic overviews of these studies exist (15–18), to our knowledge, there has

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Selected Abbreviations and Acronyms

SMS = Scottish Mental Survey
CI = confidence interval

been no systematic review. Therefore, the purpose of the present report is to systematically locate all studies relating early-life IQ to adult mortality, evaluate their method quality, assess the consistency of their results, and interpret their findings.

METHODS

Study Identification

We searched for relevant studies by using four approaches. First, an electronic search was conducted of MEDLINE (from its inception in January 1966 until March 2006), PSYCHINFO (1872 until March 2006), and EMBASE (1980 until March 2006). In MEDLINE, publications were identified by using a combined text word and recognized medical subject headings (indicated here by italics) approach: intelligence (*intelligence, intelligence tests, cognition, aptitude, mental ability, cognitive function, IQ, memory, attention, language tests, language ability, vocabulary, and intelligence quotient*) and non-psychiatric health outcomes (*mortality and death [including sudden, sudden cardiac]*). We also included somatic disease-specific outcomes, reasoning that the IQ–total mortality association also might be reported. These terms included *cause of death, cardiovascular diseases, coronary heart disease, coronary disease, stroke, cerebrovascular accident, cancer, neoplasms, respiratory tract diseases, lung diseases [including obstructive], respiratory diseases, suicide [including assisted, attempted], and accidents [including occupational, home, traffic]*. Second, reference sections of identified and related reports were scrutinized for additional publications. Third, we searched our own files. Finally, some researchers in the field were contacted for relevant reports.

Inclusion Criteria

Publications were included in the present review if the study was conducted at the level of the individual, rather than group; IQ was assessed in persons younger than 24 years, consistent with the World Health Organization definition of the period spanning childhood, adolescence, and youth (19); and a statistical estimate of the relation between IQ and all-cause mortality was provided.

Quality Assessment

Assessment of the method quality of each study was based on an approach used by Miller and Thoresen (20). This involved two authors (G.D.B. and I.J.D.) independently rating each study on a scale of A to C (A = recognized test of IQ,

high study power, minimal loss to follow-up, and inclusion of confounding and mediating variables; B = one of the mentioned method weaknesses and methods incompletely reported; and C = two or more of the mentioned method weaknesses, methods incompletely reported, and not peer reviewed).

RESULTS

Identified Studies

Nine studies met our inclusion criteria (12–14, 21–26). Their characteristics and results are listed in Table 1. Some reports that initially appeared to be relevant were excluded because results in some (22, 25) were partially replicated in others (27–29), the IQ–mortality relation was not quantified (30), the report was available in only abstract form (31), and the study sample was not population based, but drawn from a group of intellectually gifted former school children (the “Termites”) (32). However, two reports of mortality surveillance in the same cohort, the 1932 Scottish Mental Survey (SMS) (33), were included because they were based on nonoverlapping subgroups drawn from the northeast (24) and west of Scotland (25).

All nine studies used a cohort design; three studies were prospective (12, 14, 21) and the remainder were retrospective. Despite the preponderance of studies using the latter design, most were well characterized for covariate data, a common problem in such investigations (see Discussion section). Drawing on individuals from Australia (22), Denmark (13), England and Wales (12), Scotland (24–26), Sweden (14, 21), and the United States (23), most, but not all (22, 23), study samples were representative of their general populations. All study populations appeared to be Caucasian. Cohort size ranged between 180(23) and 49,262 persons (14), and number of deaths varied from 58(23) to 2022 (24). Five studies were males and females (12, 21, 24–26), three studies were males only (13, 14, 22), and one study included solely females (23).

Measurement of IQ

If reported, the content of each IQ test is described briefly in Table 1. IQ was assessed between 1932 (24) and 1971 (13, 22), with age at measurement ranging from 8 (12) to 22 years (23). With one exception (23), for which cognitive ability was inferred from written autobiographies, study populations were administered a standard IQ test of the period. These were either group administered (usually by teachers who paced children through the test) (13, 24) or individually administered (respondents were self-paced) (22). In some studies, tests were administered in a research context (13, 24), whereas in others, they were extracted from

routinely collected records that had been established for the purposes of military selection (14, 22).

The validity of the IQ tests used in these studies was examined by comparison with existing well-regarded tests (concurrent validity) or school performance and later indicators of socioeconomic position (predictive validity). Three publications (24–26) based on data from follow-up of the 1932 or 1947 SMSs used a version of the Moray House Test (no. 12). The high correlation between this test and the Stanford-Binet, regarded as a gold standard among IQ tests, in both boys ($r = 0.81$) and girls ($r = 0.78$) suggests a high degree of concurrent validity (33). In the Australian Army Conscripts study, participants were administered three tests. The Army General Classification Test correlates highly with both the Otis ($r = 0.90$) and Raven Progressive Matrices ($r = 0.78$) (34). Two other tests used in this study, the Speed and Accuracy Test and the Mechanical Comprehension Test, showed good agreement with the Army General Classification Test ($r = 0.66$ to 0.76) (22). We were unable to identify studies that specifically examined the concurrent validity of tests in the three remaining studies included in this review: the Swedish Conscripts Study (unnamed test) (14), the 1946 Birth Cohort Study (The National Foundation for Educational Research tests) (12), and the Malmo Study (the Hallgren test) (21). However, scores from these tests showed the well-established associations with perinatal (35) and postnatal early growth in the 1946 Birth Cohort Study (36), current educational performance and later occupational social class in the Malmo Study (37), and early life socioeconomic position (indexed by attained height) in the Swedish Conscripts Study (38). This evidence suggests they have a reasonable degree of validity. Finally, using the National Foundation for Educational Research tests, 108 school children aged 8 years were administered the battery and retested a maximum of 32 days later (39). Correlations across the four subtests suggest reliability was high (range, 0.86 to 0.96).

Ascertainment of Mortality

In three studies, the means of ascertaining mortality were not described (21–23). The remaining studies used linkage with national death registers.

Study Quality

Overall, study quality was moderate, with a grade of “A” allocated to three studies (14, 22, 25); “B”, to four studies (12, 13, 24, 26); and “C”, to two studies (21, 23). Studies not given the top grade were hampered by marked loss to follow-up (13) (raising concerns about selection bias), absent or insufficient covariate data (21, 24, 26) (raising concerns

about confounding), incomplete reporting of results (21), low number of deaths in some analyses (12) (offering suboptimal statistical power), and nonstandard assessment of cognitive ability (raising concerns about test validity) (23).

Results of the Systematic Review

The nine studies included quantified the relation between IQ and mortality in different ways. Two studies calculated the mean IQ difference between decedents and survivors (21, 22). The others compared mortality rates across various groupings of IQ; specifically, quartiles (13, 25), dichotomies (12), and IQ score continua (23, 26). One study (21) showed the mean difference in IQ scores between decedents and survivors only graphically, but reported an accompanying probability value. Because of both the different IQ tests used across studies and variable categorization of scores from them, it was not possible to statistically aggregate results in the form of a meta-analysis.

An inverse relation between IQ and total mortality was apparent in all nine studies, such that risk for death increased in the lower IQ scoring groups. The aforementioned problems of study comparison notwithstanding, in reports that computed effect estimates by comparing mortality rates in the lowest IQ group with those in the highest IQ group, an increase in risk of approximately 50% to 100% was found (Table 1). Three of nine studies (13, 24, 25) explicitly examined dose–response effects by computing mortality rates across three or more IQ groups. Two studies (13, 24) found a stepwise increase in mortality risk in successively lower quartiles of IQ, whereas in a third study, the 1932 SMS–Midspan linkage study (25), there was a suggestion of a threshold effect.

Only two (12, 24) of five studies that contained both men and women reported sex-specific analyses. In the SMS 1932 (24), odds ratios for *survival* in the highest IQ quartile relative to the lowest quartile increased in both men (1.81; 95% confidence interval [CI], 1.29–2.52) and women (2.88; 95% CI, 1.98–4.19). Although an (unadjusted) IQ–mortality effect was seen among males in the 1946 British Birth Cohort Study (hazards ratio bottom quartile versus remainder, 2.1; 95% CI, 1.4–3.2), it was essentially null in women (1.1; 95% CI, 0.66–1.8) (12). However, the number of deaths in the latter analysis was low ($N = 96$). In the women-only Nun Study, both markers of cognitive ability, idea density and grammatic complexity, showed inverse associations with mortality, although only the former (1.49; 95% CI, 1.17–1.89) and not the latter index (1.37; 95% CI, 0.97–1.92) was statistically significant at conventional levels. This study was the smallest and, as indicated, had the least valid measure of IQ.

TABLE 1. Studies relating early-life IQ to later all-cause mortality

Study (reference) (quality rating)	Study description (including period from IQ testing to completion of follow-up)	Intelligence measure (reference)	Outcome (source)	IQ-mortality association, effect estimate (95% confidence interval)		
				Unadjusted (unless otherwise stated)	Adjustment for socioeconomic position	Adjustment for other covariates
Malmö Study (21) (C)	Swedish prospective cohort study from 1938–1979	Hallgren group test at 10 years (items concerning antonyms, sentence completion, identical figures, and disarranged sentences) (82)	61 deaths in 831 men (NR)	Test results at age 10 years lower in decedents compared with survivors for deaths occurring at 10–29 and 30– 39 years (both $p < 0.05$), but not 40–51 years	—	—
Australian Veterans Health Study (22) (A)	Australian retrospective cohort study from 1965/ 1971–1982	Three tests at 18 years: AGC test (assesses verbal and nonverbal IQ; 100 items concerning analogies, problem solving, series completion, digit symbol); ASA test (160 items concerning number and word checking); MC test (45 items concerning mechanical ideas presented as diagrams and drawings)	523 deaths in 1786 men (NR)	Relative deaths rates in $>$ vs. $<$ median IQ score: AGC, 0.65 ($p = 0.001$) ASA, 0.75 ($p = 0.01$) MC, 0.74 ($p = 0.001$)	—	Hazard ratio/1-unit increase in AGC ^a : 0.96 (0.93–0.99) ASA and MC no longer predictive in multivariable models (results not reported)
Nun Study (23) (C)	American retrospective cohort study from 1931/1943–1998 (death surveillance covers 1991–1998)	Two linguistic measures at 22 years: idea density and grammatical complexity ascertained from autobiographies	58 deaths in 180 women (NR)	Relative risk/1-unit decrease in score ^b : Idea density: 1.49 (1.17–1.89) Grammatical complexity: 1.37 (0.97–1.92)	Relative risk/1-unit decrease in score ^c : Idea density: 1.56 (1.21–2.02) Grammatical complexity: 1.27 (0.91–1.78)	—
Scottish Mental Survey (1932) (24) (B)	Scottish retrospective cohort study from 1932–1997	Version of the Moray House Test No. 12 at 11 years (principally assesses verbal reasoning; 71 items concerning general, spatial and numerical reasoning) (33)	633 deaths in 1167 men; 438 deaths in 1050 women (national death registers)	Hazards ratios for <i>survival</i> (men)/1-SD decrease in IQ: 0.79 (0.75–0.84) Odds ratios (men [77]) Quartile 1: 1.0 (referent) Quartile 2: 1.52 (1.08–2.14) Quartile 3: 1.59 (1.13–2.23) Quartile 4: 1.81 (1.29–2.52)	No change in risk ^d : (data NR) (24)	—
Danish Metropolit Study (13) (B)	Danish retrospective cohort study from 1965–2002	Härnqvist test at 12 years (40 items concerning spatial, inductive, and verbal) (83, 84)	522 deaths in 7319 men (national death registers)	Hazard ratios: Quartile 1: 1.71 (1.34–2.19) Quartile 2: 1.30 (1.00–1.69) Quartile 3: 1.12 (0.83–1.47) Quartile 4 (highest): 1.0 (referent)	Hazard ratios ^e : Quartile 1: 1.55 (1.21–2.00) Quartile 2: 1.22 (0.94–1.59) Quartile 3: 1.08 (0.82–1.43) Quartile 4 (highest): 1.0 (referent)	Hazard ratios ^f : Quartile 1: 1.53 (1.19–1.97) Quartile 2: 1.20 (0.92–1.41) Quartile 3: 1.07 (0.81–1.41) Quartile 4: 1.0 (referent)

Scottish Mental Survey 1932-Midspan Studies Linkage (25) (A)	Scottish retrospective cohort study from 1932–1995 (death surveillance covers 1970–2001)	Version of the Moray House Test No. 12 at 11 years (principally assesses verbal reasoning; 71 items concerning general, spatial, and numerical reasoning) (33)	422 deaths in 922 men and women (national death registers)	Relative rates ^g : Quartile 1: 1.47 (1.12–1.92) Quartile 2: 1.14 (0.87–1.51) Quartile 3: 1.13 (0.86–1.49) Quartile 4 (highest): 1.00 (referent) <i>p</i> (trend) = 0.001	Relative rates ^h : Quartile 1: 1.26 (0.94–1.70) Quartile 2: 1.01 (0.76–1.36) Quartile 3: 1.03 (0.77–1.37) Quartile 4 (highest): 1.00 (referent) <i>p</i> (trend) = 0.038	
Scottish Mental Survey (1947) – “Six Day Sample” (26) (B)	Scottish retrospective cohort study from 1947–2003 (death surveillance covers 1968–2001)	Version of the Moray House Test No. 12 at 11 years (principally assesses verbal reasoning; 71 items concerning general, spatial, and numerical reasoning) (33)	125 deaths in 908 men and women (national death registers)	Hazard ratio for <i>survival</i> /1-unit decrease in score ^g : 0.977 (0.964–0.986)		
National Survey of Health and Development (12) (B)	UK prospective cohort study from 1955–2001	Mean of 4 tests at 8 years (items concerning reading comprehension, pronunciation, vocabulary, & non-verbal reasoning) (39)	133 deaths in 2192 men; 96 deaths in 2057 women (national death registers)	Hazards ratio for bottom quartile vs. remainder: Men: 2.1 (1.4–3.2) Women: 1.1 (0.66–1.8)	Hazards ratio for bottom quartile vs. remainder: Men: 1.8 ⁱ (1.1–2.7); 1.5 (0.88–2.7) ^j Women: 0.9 ⁱ (0.52–1.6); 0.89 (0.46–1.7) ⁱ	Hazards ratio for bottom quartile vs. remainder: Men: 1.29 ^k (0.67–2.49) Women ^k : no relationship (hazards ratio NR)
Swedish Conscription Survey (14) (A)	Swedish retrospective cohort study from 1969–2000	Aggregation of results from four IQ tests into a 9-point scale administered at 18–20 years (items concerning logical/general intelligence; synonym detection; visuospatial/geometric perception; technical/mechanical skills) (62)	2022 deaths in 49262 men (national death registers)	Hazards ratio/1-point decrease in IQ score: 1.15 (1.13–1.18)	Hazards ratio 1-point decrease in IQ score: 1.15 (1.12–1.17) ^l	1-Point decrease in IQ score: 1.12 (1.09–1.5) ^m

Reports ordered by ascending publication date.

NR = not reported, AGC = Army General Classification, ASA = Army Speed and Accuracy, MC = Mechanical Comprehension, *p* (trend) = probability value for linear trend across IQ groups.

^aAbsence without leave, motor vehicle offence, alcohol offence, postschool academic course, number of jobs, and duration of hospital stay.

^bAdjusted for age and birth year.

^cAdjusted for education at time of biography completion and birth year.

^dAdjusted for index of overcrowding.

^eAdjusted for paternal occupational social class.

^fAdjusted for paternal social class and birth weight.

^gAdjusted for sex.

^hAdjusted for sex, adult occupational social class, and area-based deprivation.

ⁱAdjusted for childhood socioeconomic position (paternal occupational social class and care of the home and child).

^jAdjusted for adult socioeconomic position (occupational social class and home ownership).

^kAdjusted for childhood serious illness, childhood socioeconomic position, educational qualifications by age 26 years, adult socioeconomic position, and smoking.

^lAdjusted for parental occupational social class.

^mAdjusted for subject's own occupational social class (available for a subgroup).

DISCUSSION

The purpose of the present report is to systematically locate all studies relating early-life IQ to adult mortality, evaluate their method quality, assess the consistency of their results, and interpret their findings. The present review found some evidence for an inverse IQ–mortality relation in the nine studies that met inclusion criteria. The association seemed to be present irrespective of study population, IQ test used, and era of data collection (i.e., prewar and postwar).

To varying degrees, all IQ tests in the reviewed studies measure general cognitive ability (often referred to as *g*) (40). The narrower the focus of a mental test (e.g., the Australian Veteran study's Speed and Mechanical tests are narrower than its IQ-like Army General Classification [22]), or the less standardized the assessment (the Nun Study used ratings of "idea density" and "grammatic complexity" as an index of cognition [23]), the less effectively it will capture general mental ability. If the construct measured by IQ tests truly is related to total mortality, one would expect to find the same general pattern of results across all studies, as we did. One also would anticipate that studies with superior IQ measures would yield steeper IQ–mortality gradients. However, the different approaches to data presentation across identified studies, which negated a meta-analytical approach, also precluded examination of this important issue.

At least three alternative explanations exist for the apparent protective effect of high IQ scores against premature mortality. These are the role of reverse causality, bias, and confounding. In studies relating mortality to IQ in middle or later life, there is a strong possibility that preexisting disease, clinically evident and/or occult, could have an IQ-lowering effect in these age groups. As described earlier, reverse causality is much less likely to be a problem in the studies reported here because they assessed IQ in early life when chronic disease is much rarer than in middle and older age. This supposition was tested empirically by Kuh et al. (12) in the 1946 British Birth Cohort Study, in which childhood illness (defined as hospitalization for ≥ 28 days between birth and 9 years of age) had little impact on unadjusted effect estimates for IQ in relation to mortality ($RR_{\text{bottom quartile versus remainder}}$ 1.9; 95% CI, 1.2–2.9) after it was entered into the multivariable model (2.1; 95% CI, 1.4–3.2) for male study participants (no IQ–mortality association was evident in women in any analyses). Selection bias would occur in these follow-up studies if the intelligence–mortality gradient differed markedly between persons included in analyses and those not included; that is, if it were flat or positive among nonparticipants. Given that this group typically represents a small proportion in samples reviewed here, such effects are highly unlikely.

Of the three potential alternative explanations described, confounding is the principal concern in the present review. The majority of studies included used a retrospective cohort design (six of nine studies). A problem with such studies is that they often lack important covariate data. However, with few exceptions (24, 26), these cohorts were reasonably well characterized in this regard. Even in military cohorts, data collected at induction to the army often were extensive. The two cohorts with a paucity of such data are the follow-ups of the SMS of 1932 (24) and 1947 (26), in which the only information available to investigators at the individual level was IQ and mortality experience (group-level information on socioeconomic position [overcrowding] subsequently was obtained from linkage to the 1931 UK census).

Given its relation with both childhood IQ (40) and adult mortality risk (41, 42), socioeconomic disadvantage (usually indexed by education, occupational social class, or income) most frequently is posited as an important confounder in the IQ–mortality link (15). Three (12–14) of nine studies we identified evaluated the effect of early-life social circumstances on the IQ–mortality gradient. In two (12, 13) studies, there was modest attenuation of risk; in all, statistical significance was retained. All three studies featured postwar birth cohorts in which living conditions were markedly more favorable than those born before the war. It would be of value to assess the impact of controlling for early-life socioeconomic position when the prevailing conditions were more varied and, presumably, any confounding effect would be stronger.

Birth weight also represents a potential confounding variable in the IQ–mortality relation because this indicator of growth in utero repeatedly was shown to be related to both IQ (43) and mortality, particularly that attributable to cardiovascular disease (44). We are aware of only one study that controlled for birth weight (13); adjustment for birth weight did not appear to have an appreciable impact on the magnitude of the IQ–mortality relation.

Several mechanisms may explain how higher intelligence in early life (measured by IQ) might exert an apparent protective effect against premature mortality (15–17, 24). A path diagram illustrates five such pathways (Fig. 1): through adult socioeconomic advantage, improved disease/injury prevention, better disease/injury management, reduced psychiatric disease, and "body system integrity."

Whereas socioeconomic position in childhood may be a potential confounding variable, assessed in later life, this index is more likely to lie on the pathway linking IQ to mortality and therefore should be considered as a mediator. It often is suggested, for example, that high cognitive ability in childhood leads to educational success, high social status, and a well-remunerated job, and these factors, not cognitive function itself, reduce mortality risk by, for example,

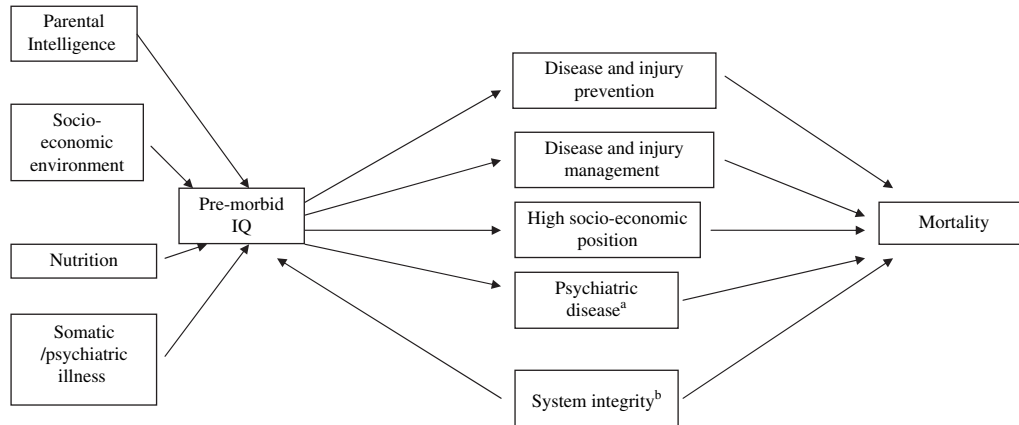


FIGURE 1. Simplified model of influences on pre-morbid IQ and potential pathways linking pre-morbid IQ with later mortality.

^a Although psychiatric disease is shown as a mediating variable between IQ and mortality, it might also be an antecedent variable if, for example, suboptimal neurodevelopment were the prior cause of both psychiatric disease and early mortality. Both of these possibilities are captured in the text.

^b Note that system integrity is shown as antecedent to both IQ and mortality. In this pathway, lower IQ is not a cause of mortality, but both IQ and mortality are influenced by this more fundamental physiological integrity.

increasing access to health care or reducing environmental risks (45–48). Five studies reviewed here (12, 14, 22, 23, 25) evaluated whether adult socioeconomic position could account for the IQ–mortality gradient. In three of these, the Australian Veterans Study (22) (“post–high school academic course”), the Nun Study (23) (educational qualifications at mean age of 22 years), and the 1946 British Birth Cohort Study (12) (educational qualifications at age 26 years), the indicator of social circumstances was education. The pattern of effects after adjustment for the different markers of education is not consistent across studies, with both strengthening (23) and attenuation (12, 22) of the IQ–mortality relation reported. Given that education and IQ correlate highly, adjusting for the former is highly debatable (18).

Higher cognitive ability also may reduce mortality by promoting more healthful behaviors. Individuals may be able to better prevent illness and injury when they know more about the behavioral risk factors for them (9). For example, analyses of data from some longitudinal studies featured in the present review showed decreased likelihood among children who scored highly on tests of IQ to smoke regularly by their early 30s (30) and middle-age (49) and an increased chance in two Scottish cohorts of quitting in midlife, having earlier taken up the habit (49, 50). In addition, individuals reporting alcohol-induced hangovers (a proxy for binge drinking) in middle age had lower childhood IQ scores than their more moderate consuming counterparts (51). Although associations with IQ are yet to be tested for other behaviors (e.g., physical activity and dietary characteristics), some of their physiological consequences, such as obesity and height (an indicator of early-life nutrition),

show associations with childhood IQ. Specifically, people with a high IQ may be less likely to experience later obesity/overweight (49, 52, 53) and suboptimal height in adulthood (29, 49), although no relation was evident for adult blood lipid levels (29). Higher childhood IQ also is related to greater pulmonary function (54). The only study to explore whether later life behaviors or their physiological consequences mediate the IQ–mortality relation found that adjusting for smoking levels in early adulthood did not alter the strength of the IQ–mortality effect (12).

Although chronic disease is the major cause of death during the last half of life in developed nations, external causes (mostly unintentional injury) make an important contribution in the first four decades. In studies that followed up participants into early or late middle age only (12–14, 22), accident prevention behaviors therefore may at least partially explain the IQ–mortality relation. Such precautions may include seatbelt wearing and motorbike and cycle helmet use, all more prevalent among individuals with higher educational attainment (55). It was suggested that learning and reasoning are crucial in promoting health-protective behavior (9), and education is likely to be a marker for these cognitive resources (56).

The daily self-management of chronic conditions that may be classified as illnesses in their own right, but which also are powerful risk factors for mortality (e.g., diabetes and hypertension) is cognitively demanding because it requires ongoing learning and independent decision making (56). Persons with low levels of education or health literacy, both strongly related to IQ (9, 40), are less likely to appreciate when their conditions require medical attention, be aware of the appropriate actions to take when they do

(57), and correctly comprehend instructions for self-medication (58). They also might fail to seek prompt medical advice after an illness episode (e.g., myocardial infarction) and treatment at a facility most appropriate to their clinical requirements, even in Nordic countries that purport to offer complete equity of access to health care (59). Such nonadherence to medical regimens is likely to lead to increased mortality rates (9).

Higher childhood intelligence is associated with reduced rates of major psychiatric disease, such as psychosis and depression (60–62). If real, at least two plausible explanations exist for this relation. First, it may be that IQ tests capture suboptimal neurodevelopment or even the early signs of these illnesses (60). Second, IQ may influence interpretation of stressful life events and the selection and use of strategies to cope with them. These explanations aside, persons carrying psychiatric morbid loads also are more likely to die prematurely than those without these loads (63–65). Psychiatric morbidity therefore may represent another pathway by which early-life IQ influences later mortality, but one that has not yet been empirically examined.

Finally, in a potentially related point, the fifth link between childhood intelligence and mortality shown in Figure 1 is used to signify the possibility that IQ could be an indicator of “body system integrity” (24). That is, scoring higher on an IQ test might reflect a superior constitution, and this favorable “wiring” of complex physiologic systems leads to resistance to environmental insults throughout life (3). In support of this suggestion, one study found that after adjusting for reaction time, there was no longer a significant association between intelligence and mortality (3). Thus, the general integrity of the body (perhaps indexed by information-processing efficiency in the form of reaction time) in part might underlie both intelligence and mortality. A possible indicator of system integrity is developmental instability, indexed in some studies by fluctuating asymmetry. Measured as deviation from symmetry of an individual’s bilateral characteristics, e.g., left–right differences in foot breadth, wrist breadth, or ear length (66), fluctuating asymmetry was associated with both IQ (67) and reaction time (68).

The general learning and reasoning ability captured by IQ tests may be important in the successful management of a person’s health and their health behaviors. It may be that individual cognition levels should be considered when preparing health promotion campaigns and in the health professional–client interaction (51). Thus, audits could be made of tasks critical to health self-care (e.g., obtaining preventative care and managing a chronic illness) with the objective of reducing unnecessary complexity when possible (e.g., simpler documents, fewer medications, or simpler dosing schedule). When possible, supplementary cognitive assistance also could be provided (e.g., more

detailed feedback) to patients unable to cope with the inherent complexities of treatment and self care.

There is no evidence to suggest that adult IQ can be raised, but both head trauma (69) and chronic disease (10, 11) are associated with lower levels. Normal aging also is related to reduced aptness in learning and reasoning, especially in old age. There is evidence that IQ can be increased in childhood, but the full extent of this modification, if present, currently is unclear (70). Thus, two recent systematic reviews of early learning and school readiness interventions (71, 72), one of which focused on randomized trials only (71), concluded that these programs led to important improvements on tests of reading, arithmetic ability, and general intelligence that extended to secondary high school ages. However, with a modest duration of follow-up in these trials, crucially, the extent to which these improvements are maintained across the life course is not clear. If the early IQ–death associations reported in the present review genuinely are causal, these interventions may have an impact on premature mortality many decades later. Although problematic given the logistical considerations, long-term follow-up of participants in such intervention studies would provide valuable insights about whether this was the case.

Given the infancy of this research area, a number of outstanding questions remain. First, information regarding the shape of the IQ–mortality relation (incremental or threshold) and tests of the existence (or not) of an association in women and ethnic minority groups are required. That a relation between markers of socioeconomic position (including education) and mortality is seen among women (73, 74) and ethnic minorities (75) suggests that similar gradients for IQ and mortality would be expected. Second, although all nine studies reported an IQ–all-cause mortality relation, other research suggested that IQ does not relate in the same way to all specific types of morbidity and mortality. This is to be anticipated given that different diseases have different causes. For example, although high IQ scores appear to be cardioprotective (25, 76), the IQ–stroke relation may be null (25, 76). Moreover, there is a suggestion that low scores are associated with elevated risk for some cancers (e.g., lung and stomach), but lower risk for others (e.g., colorectal and breast) in women (77). The recent suggestion that Swedish persons with lower IQ scores in early adulthood experience elevated rates of alcohol-related causes of death (14) is consistent with the observation that binge-drinking adults (indexed by means of self-reported hangovers) had lower IQ scores in childhood than their more moderate-drinking counterparts (51). Further examination of disease-specific links with IQ, currently a literature very modest in size, will provide insights into cause, as would the use of data sets that hold information on IQ, intermediary risk factors, and health outcomes.

Third, further insights into mechanisms underlying the association between IQ and mortality would be provided by genetic covariance analysis (78). In this type of analysis, it is not the environmental and genetic contributions to a single phenotype that are measured, but the environmental and genetic contributions to the association between two phenotypes. Because both IQ and mortality are partly heritable (79, 80), some fraction of their phenotypic correlation may represent a genetic correlation (common genetic roots). Moreover, there is evidence from a study of adoptees that part of the association between parental socioeconomic position and mortality might be heritable (81). Determining the degree to which the (phenotypic) IQ–mortality association is mediated by genetic and environmental factors will require genetically informative data sets, e.g., monozygotic and dizygotic twins for whom there are IQ scores and data for subsequent health and mortality.

In conclusion, in all nine studies identified, higher IQ in the first two decades of life was related to lower rates of total mortality in mid to late adulthood. This association does not appear to be explained by confounding, reverse causality, or selection bias, but additional studies are needed. Some plausible pathways exist, but much more mechanistically orientated work is required. The precise nature of the IQ–mortality relation (particularly in ethnic minorities and women) and the link between IQ and disease-specific outcomes also warrant additional attention.

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