Childhood socioeconomic status does not explain the IQ-mortality gradient

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The IQ-mortality association and family background[1]

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Abstract

Background: Cognitive ability correlates strongly with mortality risk, but confounding from childhood social class has been a persistent concern. While studies controlling for indicators of childhood social status report limited attenuation of coefficients, important parental and family factors are likely to vary substantially within social class.

Methods: Norwegian administrative register data with high-quality intelligence scores measured at age 18-19 for the large majority of males in the 1962-1990 birth cohorts (n=720 261) were used to assess the IQ-mortality gradient using progressively stronger controls for childhood social class in Cox proportional hazard and linear probability models. A family-fixed effects specification avoids confounding from any family or childhood characteristics fixed over time within families (e.g., childhood socio-economic status, parenting style, and neighborhood environment).

Results: A strong IQ-mortality gradient is present and robust to controls for childhood background and a family-fixed effects specification. To illustrate: In the linear probability model of mortality at age 40, the excess mortality risk of the lowest score (relative to the median score) declined from

[1] Acknowledgments: This work was supported by the Norwegian Research Council (project grants 227117, 227103). Data on ability scores have been obtained by consent from the Norwegian Armed Forces, who are not responsible for any of the findings and conclusions reported in the paper. Data made available by Statistics Norway have been essential for this research.
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0.026 (Confidence Interval (CI): 0.023, 0.029, p<0.0005) without controls, to 0.025 (CI: 0.021,0.029, p<0.0005) with controls for family background, to 0.019 (CI: 0.008, 0.030, p=0.001) with family-fixed effects added to the model. Other stanine score groups saw changes of comparable or smaller magnitude. Results from the Cox and linear probability models were substantively equivalent.

Conclusions: The IQ-mortality gradient is robust to controls for childhood family social status, including models with family-fixed effects. Childhood environment does not substantially confound the IQ-mortality gradient.

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Keywords:

Intelligence; cognitive ability; mortality; childhood environment; socioeconomic status; cognitive epidemiology

Key messages:

- Higher cognitive ability predicts longer lifespan. We examined confounding from childhood environment using large-scale Norwegian administrative register data.
- The IQ-mortality gradient was estimated with family-fixed effects, largely avoiding confounding from socio-economic status, parenting style and neighborhood.
- The IQ-mortality gradient was largely unaffected by controls for childhood environment, and social class should not be seen as a substantial confounder.

Introduction

Cognitive epidemiology is the study of how intelligence is correlated and causally linked with morbidity and mortality. The field has seen growing interest, earning a special issue in "Intelligence" (1), with research consistently reporting strong and robust associations between all-cause mortality and IQ measured in childhood or early adulthood: a meta-analysis of 16 studies found a one standard deviation advantage in IQ associated with a 24% reduction in the risk of death (2).

The underlying mechanisms linking IQ to mortality remain unclear, although evidence is accumulating linking IQ to a broad class of mortality causes. This includes unnatural causes such as suicides, accidents and homicides (3–6), as well as natural causes (3), specifically cardiovascular mortality (6–11), alcohol-related mortality (6,12), and (less conclusively) cancer (6,10,11,13–15). The broad nature of the relationship is also confirmed by research on IQ and health, which reports associations between early IQ and a broad number of later health-related outcomes (10,16–20) as well as with measures of biological aging (21).

Several mechanisms may account for the overall association (15,22): A) IQ may be a measure of bodily insults, such as low birth weight, poor childhood nutrition and early disease, that affect both later health and later intelligence, B) IQ and robust health may both be measures of bodily system integrity, signaling the ability to grow a trait in normal form despite mutations and environmental challenges (i.e., developmental stability), C) IQ may be a predictor of healthy behaviors, and D) IQ may be a predictor of selection into safe environments (e.g., workplaces).

In light of well-documented and persistent social gradients in health (23), socio-economic status (SES) remains arguably the most plausible potential confounder for the IQ-mortality association. In the three studies simultaneously adjusting for both adult SES and own education, the adjustment entirely attenuated the IQ-mortality association (2). Since there will be IQ-based sorting into educational attainment and adult SES, however, this attenuation of the coefficient on IQ is not necessarily evidence against the IQ-health hypothesis. If education and adult SES reduce mortality risk, selection

on IQ into education and SES would make these part of the causal chain whereby IQ affects health (i.e., mechanisms in the C and D category of the last paragraph), in which case the attenuation would reflect statistical overadjustment (24). Education and adult SES will also *reflect* IQ when there is IQ-based selection, causing them to serve as alternative proxies for intelligence. Including these variables in the regression would then attenuate the IQ-score coefficient even if there were no causal effects on mortality of either education or adult SES.

Unlike adult SES, childhood SES is unlikely to be affected causally by own IQ. In nine studies that included proxies for childhood SES, such as father's occupation or income, attenuation of the IQ-mortality relationship was insubstantial (2). Similar results were found in a recent study not included in the meta-review (25). Concern remains, however, that this may understate the importance of childhood SES. Social class and family-fixed factors statistically explain a substantial share of variation in childhood intelligence (26), social class is correlated with educational attainment which itself has a causal effect on later IQ scores (27–30), and it has been argued that important parental and family factors vary substantially within crude measures of social class (31). In addition, variation in verbal IQ measures may themselves reflect social class rather than true verbal ability (32). On the other hand, a recent study of three twin samples indicates that the intelligence-mortality association was largely driven by genetic influences (33), with substantially stronger associations observed in di-zygotic twin pairs than in mono-zygotic twin pairs that differ in phenotypic intelligence only.

In the present study, we examine the relationship between IQ and mortality risk in a longitudinal data set covering the majority of Norwegian males in birth cohorts spanning 1962-1990 (n=720 261), with mortality outcomes observed up to and including 2015. The data set contains stanine scores measuring IQ from military conscription tests taken at age 18-19. To examine confounding from childhood SES and family-fixed factors we compare coefficients for the risks of death by age 40 in models with and without controls for family background, and with and without family-fixed effects. The fixed effects model is of particular interest, in that it estimates the IQ-mortality association using

only within-family variation in IQ scores, relying on variation in mortality outcomes for differentially scored brothers in families with two or more scored male siblings. This avoids confounding from any family or childhood characteristics fixed over time within families, minimizing confounding from factors such as childhood socio-economic status, parenting style, and neighborhood environment.

Methods

Data

We use intelligence test data from the Norwegian National Conscript Service, and limit the analysis populations to males born between 1962 and 1990 to two Norwegian-born parents. Military service was compulsory for all able-bodied men in the data period, with most males meeting before a conscription board and given a test of intellectual ability prior to service. «Around 90% of the men liable for service attend, and most of them (around 95%) meet between their 18th and 21st birthday» (34).

The data on intellectual ability have been extensively used and described in past research (34–36). They include a General Ability score, expressed in stanine units and calculated from the scores on speeded tests of arithmetic (30 items), word similarities (54 items) and figures (36 items). The test and its scoring remained unchanged throughout the analysis cohorts, apart from the arithmetic test that changed to a multiple-choice format in the beginning of the 1990s, affecting those born after 1973 in our data.

The intelligence data contain a person identifier allowing the data to be linked to other administrative data sets. We combine the GA score data with records from the Central Population Register containing dates of birth and death, and with our extract covering all registered deaths through 2015. The data also contain identifiers for biological mother and father, allowing us to identify brothers. We exclude birth cohorts prior to 1962 because they were subject to a different

testing norm, and restrict the main body of analyses to cohorts born before 1976 because we wish to study mortality through age 40.

For each male, we compute a number of characteristics measuring socio-economic status during childhood. These include parental earnings, calculated as the average annual earnings of mother and father combined over the age interval 6 to 15, with annual earnings inflated to year 2000 currency using the index of the national pension system ("G"). The earnings data stem from the register of the welfare administration and are available from 1967. We also include parental education, measured by the higher educational attainment of mother and father when the son is 15 years of age. Educational attainment data are extracted from the national education data base ("NUDB"). Finally, we account for whether or not the mother was in her teens at the time of child birth, drawing on the birth date information contained in the population register.

Statistical methods

Mortality rates across stanine groups are shown using shares deceased at various ages for pooled cohorts.

Using individual level data, we first assess the relationship between GA scores and mortality using Cox proportional hazard model regressions on different samples: A) The full set of male birth cohorts 1962-1975, B) a subset of the 1962-1975 cohorts containing scored males with scored brothers (defining families as children with the same biological mother and father), and C) the full set of male birth cohorts 1976-1990.

Using the full 1962-1975 set, we estimate the IQ-mortality association with and without controls for birth year and observable indicators of family background. The brother sample is used to assess whether this subsample – used for a later analysis with family-fixed effects – differs substantively from the full cohort. The later set of cohorts, whose mortality can only be assessed up to ages from 25 (the 1990 cohort) to 39 (the 1976 cohort), is used to assess whether the IQ-mortality relationship

has remained stable over time or whether the relationship appears substantially different in the later period compared to the first.

To implement a model with family-fixed effects we turn to a linear probability model of survival at age 40, and compare coefficients from models a) without other covariates, b) with controls for family SES characteristics such as parental education and earnings, and c) with family-fixed effects (using the subsample of brothers).

A major benefit of the Cox model is that it uses mortality at each age in the estimation, taking full advantage of the longitudinal data structure to improve the statistical precision of estimates. Although it only considers mortality evaluated at a certain age, the advantage of the linear probability model is that it allows for implementation of the fixed-effects estimator. All analyses were conducted using the Stata statistical software.

Results

Sample descriptive statistics

Figure 1, panel A, shows the sizes of the 1962-1990 male birth cohorts under study and the number of scored males in each cohort. In total the 39 birth cohorts cover 817 946 births, with the scored individuals accounting for 88.1 percent of births over the period leaving us with 720 261 observations with a valid general ability (GA) score. The frequency distribution of stanine scores in the data follows a bell-shaped density (Figure 1, panel B).

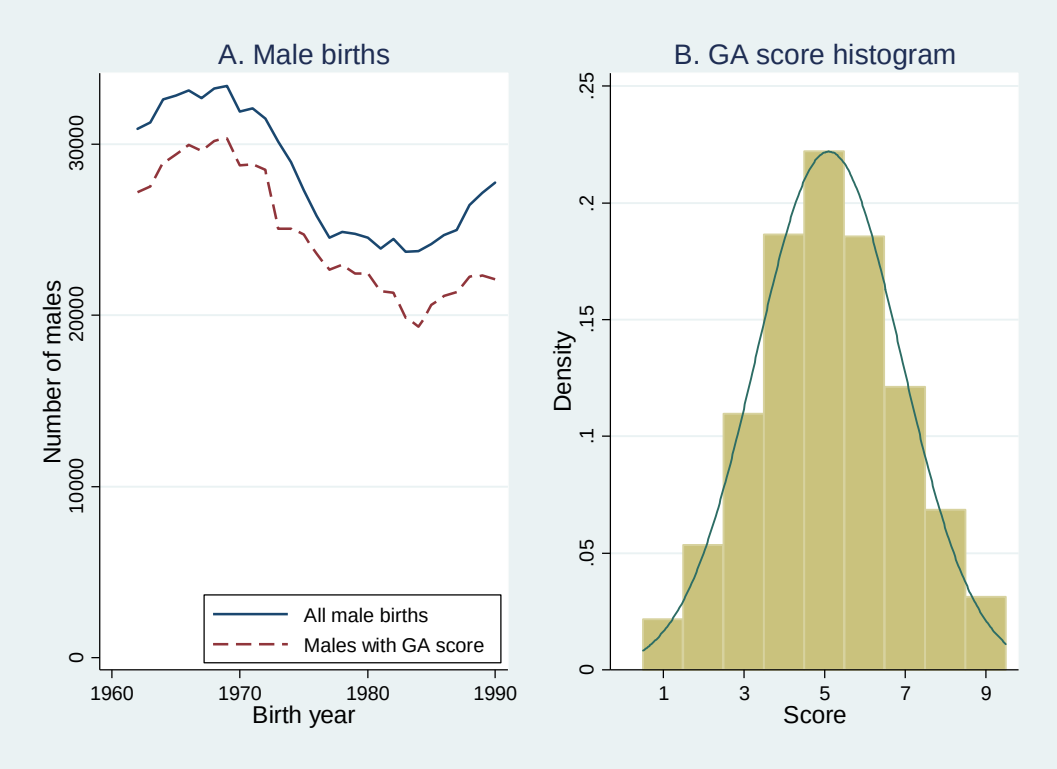

Fig 1: Number of births, coverage, and distribution of general ability score. Note: Population restricted to males born in Norway 1962-1990 with two Norwegian-born parents (N= 817 946).

The crude association between mortality and GA scores is evident at early ages, becoming more pronounced as the cohorts age (Figure 2). The clearly discernible gradient at age 30 reflects mortality differences over roughly a decade of early adulthood, since the sample is conditional on participants having attended military conscription testing (age 18-19). Larger differences are apparent at higher ages, with proportionally bigger changes observed in the lower-scoring groups. *Relative* mortality rates by GA score are stable, supporting the proportional hazard assumption of the Cox model.

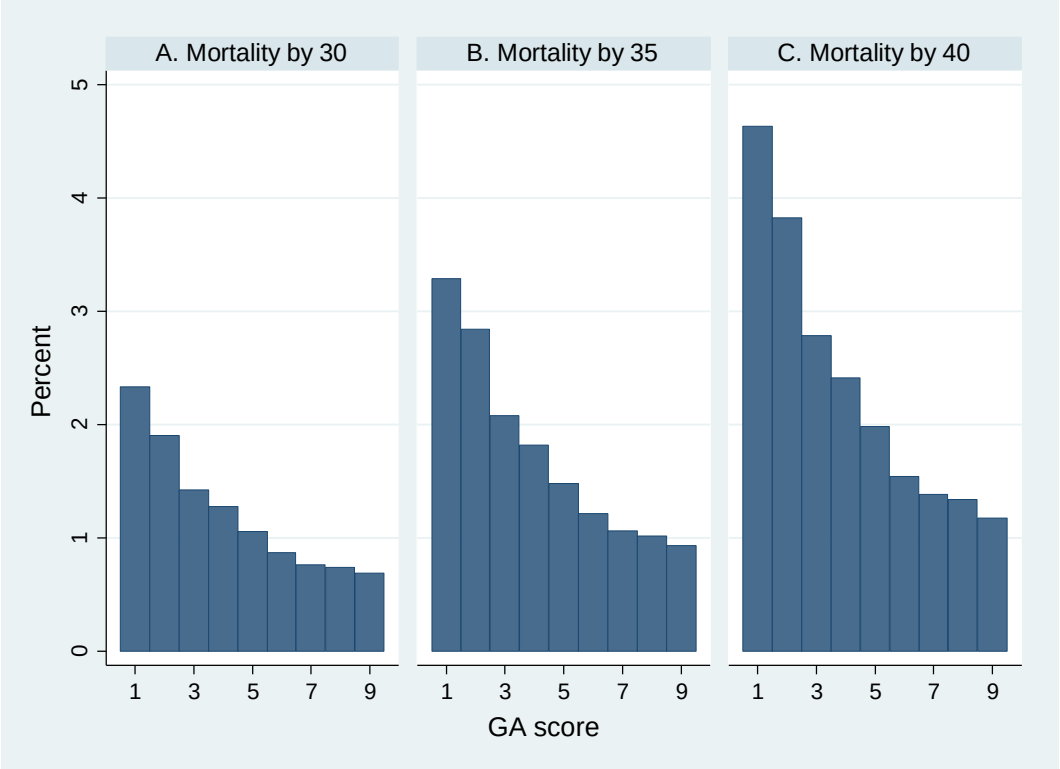

Fig 2: Mortality by age and general ability. Note: Population restricted to males born in Norway 1962-1975 with two Norwegian-born parents and with valid GA score (N=394 291).

The data cover almost 30 birth cohorts in full. Comparing the early cohorts (1962-1975) to the later cohorts (1976-1990), we find that the later cohorts tend to have older parents with substantially higher real earnings and educational attainment (see Table 1). The brother sample, however, is largely similar to the earlier cohorts from which it is drawn. Teenage mothers are less common in the brother sample, but this is to be expected since children born to teenage mothers will now only be included only if they have a younger brother born to the mother at a higher age.

Table 1: Descriptive statistics, analysis samples

	All males born 1962-1975 (1)	Brothers born 1962-1975 (2)	All males born 1976-1990 (3)
Mortality by 2015 (%)	3.08	2.95	1.10
Mortality by age 40 (%)	2.08	2.00	
GA score	5.10	5.06	5.12
Parental earnings	370 508	366 985	469 795
Parental education:			
Compulsory (%)	20.59	20.82	10.42
Secondary (%)	54.86	54.95	47.73
Higher than secondary (%)	24.55	24.23	41.86
Teenage mother (%)	6.95	5.71	4.85
Birth year	1968	1968	1983
Observations	390 140	171 699	323 482
Families	297 167	78 726	260 050

Note: Samples are restricted to males born to two Norwegian-born parents with a valid personal identifier for both mother and father, as well as non-missing data on GA score, parental earnings, and parental education. Parental earnings are computed as average annual earnings from work for both parents over the age interval 6 to 15, and are inflated to year 2000 NOK using the national pension system index ("G"). Parental education is the higher attainment of mother and father at offspring age 15.

Statistical analyses

Cox regressions

Results from Cox regressions are shown in Table 2. Columns 1 and 2 display the coefficients from estimations using the full set of early cohorts, with column 2 including controls for birth year and childhood socio-economic status. This has no substantive effect on coefficient estimates or confidence intervals. In the specification controlling for childhood background (column 2), males in the lowest GA score bracket have a mortality risk that is 2.31 (CI: 2.12, 2.52, $p<0.0005$) times that of the median bracket. Relative mortality risk declines monotonically with general ability, reaching 64 percent (CI: 0.56, 0.73, $p<0.0005$) for the highest scoring group.

Results are similar for the full sample and the brother sample (column 3), indicating that the sample conditioned on family type does not introduce compositional change.

Finally, column 4 shows estimates of mortality differences using the younger birth cohorts. The results remain largely unchanged. The relative mortality risk in the lower score brackets declines slightly relative to the reference category, but the same is true for the higher score brackets. As a result, the relative risk difference between the next lowest and next highest GA score groups remains constant over time (GA score 2 is associated with 2.6 times the mortality risk of GA score 8 in both the old and the young samples, cfr columns 2 and 4).

Table 2: GA score and mortality, Cox proportional hazard models

	(1)	(2)	(3)	(4)
GA score:				
1	2.42 (2.22, 2.64) P<0.0005	2.31 (2.12, 2.52) P<0.0005	2.38 (2.09, 2.72) P<0.0005	2.15 (1.78, 2.60) P<0.0005
2	1.92 (1.80, 2.06) P<0.0005	1.86 (1.73, 1.99) P<0.0005	1.92 (1.73, 2.14) P<0.0005	1.67 (1.46, 1.92) P<0.0005
3	1.49 (1.40, 1.58) P<0.0005	1.45 (1.36, 1.54) P<0.0005	1.47 (1.34, 1.62) P<0.0005	1.53 (1.37, 1.70) P<0.0005
4	1.24 (1.18, 1.32) P<0.0005	1.23 (1.16, 1.30) P<0.0005	1.27 (1.17, 1.39) P<0.0005	1.20 (1.08, 1.32) P<0.0005
5 (reference)				
6	0.81 (0.76, 0.87) P<0.0005	0.82 (0.77, 0.88) P<0.0005	0.83 (0.75, 0.92) P<0.0005	0.84 (0.75, 0.94) P=0.002
7	0.73 (0.67, 0.78) P<0.0005	0.74 (0.69, 0.80) P<0.0005	0.79 (0.70, 0.88) P<0.0005	0.66 (0.58, 0.76) P<0.0005
8	0.69 (0.63, 0.76) P<0.0005	0.71 (0.65, 0.78) P<0.0005	0.71 (0.61, 0.83) P<0.0005	0.64 (0.53, 0.76) P<0.0005
9	0.62 (0.54, 0.71) P<0.0005	0.64 (0.56, 0.73) P<0.0005	0.66 (0.53, 0.82) P<0.0005	0.43 (0.31, 0.59) P<0.0005
Controls	None	Birth year, parental SES	Birth year, parental SES	Birth year, parental SES
Sample	All 1962-1975		Brothers 1962-1975	All 1976-1990
Observations	390 140		171 699	323 482
Families	297 167		78 726	260 050

Note: 95% confidence intervals, based on standard errors clustered within families, are reported in parentheses. Parental SES characteristics include average annual earnings of both parents in age interval 6-15, the higher parental education (7 indicators for attainment) at age 15, and an indicator variable for teenage mother. Only observations with valid parental SES data are included. Family identifier consists of unique combination of identifier for mother and father.

Linear probability models

The results from the estimated linear probability models are shown in Table 3.

The models in columns 1 to 3 correspond to models 1-3 in the Cox regression table, except that the outcome is now an indicator variable for mortality by age 40. As before, we estimate a strong IQ-mortality gradient (model 1) that is unaffected by the inclusion of birth year and parental SES controls (column 2), and we find similar results in both the full early sample and the brother subsample (columns 2 and 3). The mortality rate of GA score 1 is 2.5 percentage points higher than that in the reference bracket (i.e., 2 percent; see the constant term in column 1). The mortality risk declines monotonically with higher GA score, with the top score bracket having a 0.7 percentage point advantage compared to the reference category.

Coefficients estimated in the model with family-fixed effects use only the variation in mortality and GA score within families, i.e., by comparing the mortality of males to their own brothers (column 4). This avoids the confounding of unobserved factors constant within families, but at the cost of broader confidence intervals as the estimator ignores between-family variation. Another drawback is that fixed-effects models are "notoriously susceptible to attenuation bias from measurement error" (37). Specifically, since sibling GA scores are correlated, a larger share of the GA score variation used by the fixed-effects estimator will reflect measurement error, biasing coefficients towards zero. In light of this, the point estimates remain surprisingly stable.

Table 3: GA score and mortality by age 40, linear probability models

	(1)	(2)	(3)	(4)
GA score:				
1	0.026 (0.023, 0.029) P<0.0005	0.025 (0.021, 0.029) P<0.0005	0.024 (0.018, 0.031) P<0.0005	0.019 (0.008, 0.030) P=0.001
2	0.018 (0.016, 0.020) P<0.0005	0.018 (0.015, 0.020) P<0.0005	0.017 (0.013, 0.021) P<0.0005	0.014 (0.007, 0.021) P<0.0005
3	0.008 (0.006, 0.010) P<0.0005	0.007 (0.005, 0.009) P<0.0005	0.007 (0.004, 0.010) P<0.0005	0.005 (0.000, 0.010) P=0.073
4	0.004 (0.003, 0.006) P<0.0005	0.004 (0.003, 0.006) P<0.0005	0.004 (0.002, 0.007) P<0.0005	0.003 (-0.001, 0.007) P=0.108
5 (ref)				
6	-0.004 (-0.006, -0.003) P<0.0005	-0.004 (-0.005, -0.003) P<0.0005	-0.004 (-0.006, -0.003) P<0.0005	-0.005 (-0.009, -0.001) P=0.010
7	-0.006 (-0.007, -0.004) P<0.0005	-0.005 (-0.007, -0.004) P<0.0005	-0.005 (-0.007, -0.003) P<0.0005	-0.005 (-0.009, -0.001) P=0.027
8	-0.006 (-0.008, -0.004) P<0.0005	-0.006 (-0.007, -0.004) P<0.0005	-0.005 (-0.008, -0.003) P<0.0005	-0.005 (-0.010, -0.000) P=0.039
9	-0.008 (-0.011, -0.006) P<0.0005	-0.007 (-0.010, -0.005) P<0.0005	-0.007 (-0.010, -0.004) P<0.0005	-0.006 (-0.013, 0.001) P=0.073
Constant	0.020 (0.019, 0.021) P<0.0005			
Controls	None	Birth year, parental SES	Birth year, parental SES	Birth year, parental SES, family fixed effects
Sample	All 1962-1975		Brothers 1962-1975	
Observations	390 140		171 699	
Families	297 167		78 726	

Note: 95% confidence intervals, based on standard errors clustered within families, are reported in parentheses. Parental SES characteristics include average annual earnings of both parents in age interval 6-15, the higher parental education (7 indicators for attainment) at age 15, and an indicator variable for teenage mother. Only observations with valid parental SES data are included. Family identifier consists of unique combination of identifier for mother and father.

Comparing the results from the Cox and the linear probability models

The linear probability model uses outcomes measured at a single age, while the Cox model uses information on the timing of all deaths observed up to the end of 2015. The linear probability model, on the other hand, allows us to control for all factors that remain fixed within families over time, largely avoiding confounding from family socioeconomic background, parenting styles, neighborhoods, etc.

We compare the coefficient estimates across models by computing the relative risk of mortality by age 40 (from Table 3), evaluated at the observed mortality rate of the reference category. The

estimates are strikingly consistent across the estimation methods (Figure 3), though the precision of the family-fixed effects model are lower, as reflected in the wider confidence intervals of the plot.

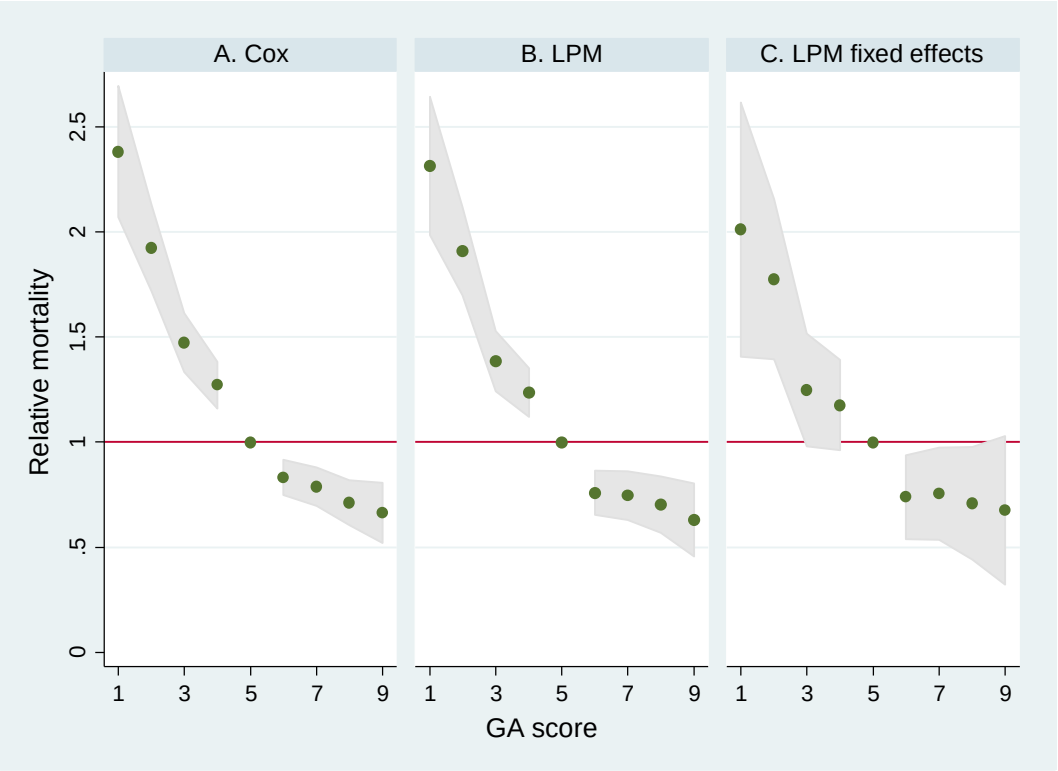

Fig 3: Comparing predicted mortality across estimation methods. Note: Shaded areas indicate 95% confidence interval around point estimate. Regressions control for parental SES and birth year, panel C adds family-fixed effects. Sample restricted to those in the brother data and with valid observation of parental SES characteristics (N=171 699); see also notes to Tables 2 and 3.

Discussion

Principal findings

We document a large difference in mortality risk across Norwegian males with different scores on a high-quality intelligence test performed around age 18. For the 1962-1975 cohorts we find that the

estimates are similar in linear probability models with and without controls for birth year and parental SES, in Cox models with birth year and parental SES controls, and in a linear probability model with family-fixed effects. This provides strong evidence that the IQ-mortality association is largely unrelated to family background and childhood SES, and that IQ-differences are associated with substantial differences in mortality risk within a modern welfare-state setting. We also find that similar differences in mortality risk persist for the later cohorts born 1976-1990, indicating that the relationship was largely stable across a 30-year period.

The results indicate a non-linear association of IQ and mortality risk. Following convention in defining the standard deviation (SD) of the GA scale as 2 and starting with the lowest score group, men with a GA score of 3 have a 37% lower mortality risk than those scoring 1. A score of 5 is associated with 31% lower mortality than score 3. Continuing in one SD increments, higher GA scores are associated with further risk reductions of 26% and 14% respectively (see Table 2, column 2). Translated into *absolute* risk differences, this non-linear pattern appears even starker: using the same one-SD increments in GA score, our results imply that the *absolute* reductions in mortality rates measured in percentage points is 1.8, 0.7, 0.5 and 0.2 respectively (see Table 3, column 2).

Strengths and limitations

The primary strength of the present study is the quality, breadth and coverage of the underlying data: A population sample of 719 198 individuals, covering 88 percent of the male birth cohorts analyzed, with outcomes observed in high quality administrative registers.

The data allow us to examine statistical associations in a representative population sample of 29 birth cohorts, controlling for birth year and parental SES. Finally, the data volume allows us to estimate a data-demanding model with family-fixed effects, which provides the strongest control against confounding from family background and other factors fixed over time within families.

A limitation in the study is that we lack information on cause of death, thus preventing us from exploring the causal mechanisms at work. A second limitation is that GA scores may reflect more than the intellectual ability that they measure, as intellectual ability may correlate with personality traits and other genetic factors. One study reported strong attenuation of the IQ-mortality relationship in a sample of monozygotic twins with differing IQ-scores (33), while controlling for emotional control led to some attenuation of the IQ-mortality relationship in a large sample of Swedish males whose IQ and emotional control were both assessed at military conscription (25).

Conclusions

Analyses based on Norwegian administrative data reproduce established associations between IQ and all-cause mortality, and indicate that the IQ-mortality gradient has been stable throughout the data period. Controls for birth year and childhood SES have no substantive impact on estimates, and coefficient point estimates are attenuated only modestly, and then only for the lowest GA scores, when using family-fixed effects. This is strong evidence that the gradient is not due to confounding from childhood environment, socio-economic status or neighborhoods.

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