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Childhood socioeconomic status does not explain the IQ-mortality gradient

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ABSTRACT

Background: Cognitive ability correlates 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 large difference in mortality risk is evident across Norwegian males: We find that the mortality risk of the lowest ability bracket, relative to that of the median bracket, is 2.31 (Confidence Interval (CI): 2.12, 2.52, $p < 0.0005$), declining to 0.64 (CI: 0.56, 0.73, $p < 0.0005$) for the highest ability bracket. Estimated differences 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.

Conclusions: The IQ-mortality gradient is not due to confounding from family background or childhood SES. Higher IQ-scores are associated with substantially reduced mortality risk within a modern welfare-state setting, and the relationship was largely stable across a 30-year period.

1. 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" (Deary, 2009), 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 (Calvin et al., 2011).

The underlying mechanisms relating 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 (Batty, Deary, et al. 2008; Hemmingsson, Melin, Allebeck, & Lundberg, 2006; Meincke et al., 2014; Osler, Andersen, Laursen, & Lawlor, 2007), as well as natural causes (Meincke et al., 2014), specifically cardiovascular mortality (Batty, Shipley, et al. 2008; Hart et al., 2003; Hemmingsson et al., 2006; Silventoinen, Modig-Wennerstad, Tynelius, & Rasmussen, 2007; Wallin et al., 2015; Wraw, Deary, Gale, & Der, 2015), alcohol-related mortality (Hemmingsson et al., 2006; Sjölund, Hemmingsson, Gustafsson, & Allebeck, 2015), and (less conclusively) cancer (Batty et al., 2007; Batty et al., 2009; Deary, Whiteman, Starr, Whalley, & Fox, 2004; Hart et al., 2003; Hemmingsson et al., 2006; Wraw et al., 2015). The broad nature of the relationship is consistent with research on IQ and health, which reports associations between early IQ and a broad number of later health-related outcomes (Batty, Mortensen, & Osler, 2005; Der, David Batty, & Deary, 2009; Schmidt et al., 2013; Sörberg et al., 2013; Wraw et al., 2015; Wrulich et al., 2014) as well as with measures of biological aging (Schaefer et al., 2015).

Several mechanisms may account for the overall association (Deary et al., 2004; Whalley & Deary, 2001): 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, reflecting a "correlation between neurodevelopment and that occurring

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in other organs" (Deary et al., 2004), 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 (Mackenbach et al., 2003), socio-economic status (SES) remains the most plausible confounder for the IQ-mortality gradient. The meta-analysis of Calvin et al. (2011) lists three studies that simultaneously adjusted for both adult SES and own education. In all cases, these adjustments entirely attenuated the gradient (Jokela, Batty, et al. 2009; Jokela, Elovainio, et al. 2009; Kuh et al., 2009). 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 over-adjustment (Schisterman, Cole, & Platt, 2009). 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 (Calvin et al., 2011). Similar results were found in the recent study by Lager, Seblova, Falkstedt, and Lövdén (2016). Concern remains, however, that such proxies are inadequate controls for childhood SES. Social class and family-fixed factors statistically explain a substantial share of variation in childhood intelligence (Lawlor et al., 2005), social class is correlated with educational attainment which itself may have an effect on later IQ scores (Brinch & Galloway, 2012; Carlsson, Dahl, Öckert, & Rooth, 2014; Cascio & Lewis, 2006; Ceci, 1991), and it has been argued that important parental and family factors vary substantially within crude measures of social class (Vågerö, 2011). In addition, variation in verbal IQ measures may themselves reflect social class rather than true verbal ability (Chapman, Fiscella, Duberstein, Kawachi, & Muennig, 2014). On the other hand, a recent study of three twin samples indicates that the intelligence-mortality association was largely driven by genetic influences (Arden et al., 2015), 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. 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.

2. Methods

2.1. 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» (Sundet, Eriksen, Borren, & Tambs, 2010).

The data on intellectual ability have been extensively used and described in past research (Sundet, Barlaug, & Torjussen, 2004; Sundet, Borren, & Tambs, 2008; Sundet et al., 2010). They include a General Ability score (henceforth referred to as 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 ability 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 of the offspring, 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.

2.2. 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 ability 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