


Commentary: Intelligence in youth and all-cause mortality: some problems in a recent meta-analysis

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I would like to congratulate Cathrine Calvin and co-authors for their meta-analysis, recently published by *IJE*. Research addressing the relation between social circumstances, cognitive ability and health has grown rapidly. The association between early IQ and all-cause mortality is an excellent place to start. Their review will benefit all working in the field. I will highlight some of its problems in order to help us to move forward, both in understanding whether cognitive ability has any causal effect on health and in addressing the public health consequences of this knowledge.

Early intelligence or pre-morbid intelligence?

There is some inconsistency in the way the authors think and define the causal factor in focus: is it intelligence in youth or pre-morbid intelligence? Both terms are used and in fact treated as equivalent. However, they can hardly be equivalent if one at the same time sees IQ as a measure of ‘bodily insult’, as some of the authors do. Bodily insult is a somewhat vague concept, but I assume that it refers to external influences on the body which influence a person’s development negatively. Malnutrition in utero (resulting in low birth-weight), infection in infancy or physical abuse as a child, may influence physiological, emotional and cognitive development, thus constituting ‘bodily insult’. IQ in youth would already have been affected (reduced) by such early events, and should therefore not be described as pre-morbid intelligence. And as far as I understand there is no information about morbidity, or exclusion of persons reporting morbidity in childhood, adolescence or early adulthood, in any of the studies included in the meta-analysis.

The concept of pre-morbid intelligence may be borrowed from studies of cognitive decline, where ‘pre-morbid intelligence’ often refers to intelligence before the ageing process affects the mind severely. It can simply mean intelligence measured in a person with no disease diagnosis, in which case it clearly differs from early intelligence.

Cognitive ability and health probably develop together from Day 1 (conception) and influence each other mutually during the fetal, infant, childhood and adult periods. At the end of life they decline together. A mutual, reciprocal, influence between health and intelligence need not look the same early and late in life; the predominant causal direction could change...
with ageing and at the end of life, disease may more commonly determine cognitive ability than the other way round.

Therefore, the correct question to ask would seem to be: is early IQ a causal determinant of long-term health and survival, independent of early health status and independent of any preceding morbidity? It seems that the answer to this question is yes. However, the authors are quite generous in defining early intelligence; any study where the mean age of respondents at IQ testing is 25 years or less qualifies for inclusion. Such generosity in defining early intelligence may bias estimates of its effect on mortality upwards. There is, in fact, an indication in their meta-analysis that IQ has a smaller effect on mortality the earlier it is measured.

Heterogeneity of effect and effect modification

An increase of 1 SD of IQ is estimated to give a 24% lower all-cause mortality. ‘Evidence of a low degree of heterogeneity’ leads the authors to conclude that early IQ has a robust effect of this magnitude on mortality, independent of context: gender, birth cohort, age at IQ measurement and type of study. Yet a quick look at Figure 2 immediately reveals that risk estimates in the 16 selected studies vary from 0.61 to 0.90.

The question of heterogeneity can also be formulated as a question of whether IQ interacts with other determinants in causing death. Intuitively one would assume that this was the case. Gender is a case in point. The authors conclude that the effect of IQ on mortality is the same for men and women; obviously, a very important conclusion. But the composition of causes-of-death in men and in women differs substantially. Therefore, if the effect of IQ on all-cause mortality is identical for men and women it could hardly be identical for specific causes-of-death, unless IQ had exactly the same effect on every cause-of-death, which it clearly has not.

Of the seven studies that include both men and women, three report a formal test of interaction between gender and IQ. Two found no such interaction and one did. Of the remaining four, one reported no difference in IQ effect on mortality and three reported small effects (without testing for interaction). One paper which was excluded from the review, reference 46, also reported a gender difference in the effect of IQ. All this looks to me like a very unclear picture.

Apart from gender, there are many other determinants which could modify the effect but are not considered here. For instance, if a boy or a girl has a below average IQ, could the corresponding mortality risk be modified by school meals or one year extra in school? Should we not look for determinants of mortality that interact with early intelligence?

Dynamics of social circumstances, education, IQ and health

Childhood circumstances and education are important because both of them may either confound or mediate the association between early intelligence and mortality.

An excellent aspect of this study is that the authors try to systematically take into account the role of childhood socio-economic status (SES), education and adult SES, to ‘discover their magnitude of influence as potential confounders or mediators of the intelligence–mortality association’. Childhood SES is estimated to reduce the association somewhat (4%).

Childhood socio-economic status is of course only one aspect of childhood circumstances. If one thinks about circumstances that will influence a child’s early cognitive development, their variation within a social class is considerable. Attachment between child and parents, the extent to which parents talk and read to their child as well as violence and trauma are factors that, independently of social class, are likely to influence both IQ and long-term mortality. In other words, we cannot exclude confounding from childhood social circumstances on the grounds that controlling for childhood SES only reduced hazard ratios marginally. Premature birth for instance, more frequent in low SES but occurring in all classes, has a large effect on IQ and is linked to long-term mortality from stroke.

Education and adult SES were found to be mediators in the association between IQ and mortality. Education attenuates the association by 50% and adult SES by some 30%. They represent a considerable influence, one of the most important findings in this review. However, social circumstances, education and IQ mutually influence each other in a dynamic way, across the life course. IQ is measured at at the age of 18 years in the largest studies and at the age of 24 years in some smaller ones. At this point in life, educational experience will certainly have influenced cognitive test results, implying that IQ may be a mediator between education and mortality, rather than (or as well as) education being the mediator.

The role of education in the association between IQ and mortality must be one of the most important to clarify. The authors write about the possibility that ‘education and adult SES… are partial surrogates for intelligence’, and speculate about ‘the extent to which intelligence shares genetic and environmental causes with health, education and social class’. The idea that education and adult SES are mere
epiphenomena, surrogates or just imprecise measures of the more fundamental characteristic of intelligence, is far-fetched and challenges most modern social science. The term ‘surrogate’ or even ‘partial surrogate’ sounds inconsistent with the idea of a reciprocal influence between intelligence and education, and reveals perhaps a belief that intelligence is unchangeable across most of the life span.

Public health opportunity
IQ measured early in life is fundamentally influenced by experience, schooling and abstract reasoning, in line with modern ideas about the plasticity of the brain. Blair et al.\(^8\) suggested that ‘the experience associated with the utilisation and repeated practice of pre-frontally based fluid cognitive skills that begin relatively early in life are likely to lead to relatively enduring changes in performance of measures of fluid intelligence’. Their ‘neurodevelopmental-schooling hypothesis’ is used to explain the fact that IQ increased substantially across the last century (the so called Flynn effect). Incidentally, mortality was falling across much of the same period, giving support to the hypothesis that IQ and health may be causally connected, and that they share closely related environmental determinants.

Improvement of early family and social circumstances, as well as schooling, represents opportunity for public health interventions. One of the ways through which such interventions will work appears to be through their effect on cognitive development and intelligence.

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References