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Intelligence and Education: Causal Perceptions Drive Analytic Processes and Therefore
Conclusions

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Summary

Background

Educational attainment is associated with many life outcomes, including income, occupation, and many health and lifestyle variables. Many researchers use it a control variable in epidemiological and other social scientific studies, often without specifying exactly what environmental effects or set of personal characteristics is being controlled. Other researchers assume that genetically-influenced intelligence drives educational attainment, and think that intelligence is the appropriate control variable. Researchers' different and often unstated causal assumptions can lead to very different analytical approaches and thus to very different results and interpretations.

Methods, results, and conclusions

We document several examples of this important variation in the treatment of education and intelligence and their association. We recommend: greater clarity in stating underlying assumptions and developing analytical approaches; and greater objectivity in interpreting results. We discuss implications for study designs.

Brighter people tend to get more schooling, and the longer-schooled tend to be brighter. These simple facts elicit surprisingly different interpretations among the many epidemiologists and social scientists who measure education and intelligence for research use. Their different interpretations contribute to differences in methodological and analytical treatments that can have profound impacts on study design, methodological choice, results, and interpretation of results. Implicit interpretation of the association between these two variables is common throughout epidemiological and other social science research. With regard to health and other outcomes, this observationally ambiguous association involves the statistical issues of mediation, moderation, confounding, and direct and indirect effects. These issues are always troublesome because their treatment depends not only on timing of available measurements but understanding of causal pathways. The issues involved in the association between these particular two variables, however, are especially important to the newly emerging field of cognitive epidemiology. One or the other—especially education, due to its greater availability in datasets—is very commonly used as a control variable; intelligence and education are closely inter-related, and they may be measured with varying degrees of precision. Moreover, there is probably some form of longitudinal cascade between them, quite possibly with reciprocal causal and selection effects (1); yet, the optimal longitudinal data sequence to understand the processes involved in these reciprocal and selection effects is often unavailable. At the same time, because they are not perfectly correlated, neither education nor intelligence is a perfect proxy for the other. It is thus often important to understand objectively which (if either) exerts a causative effect on an outcome.

Intelligence and education: clearly correlated, but what is the direction of causation?

Intelligence and education have been studied together since the earliest empirical research on these topics. Spearman (2) found teachers' estimates of intelligence to be correlated with school exam results. Binet (3) developed what we now know as IQ tests to identify those children who would not benefit from normal education. When intelligence and educational outcomes—often assessed as years of full time education or as highest achieved qualification, but also by school grades or educational achievement test scores—are measured at about the same time, a typical correlation is about 0.5 (4). Like any other correlation, a cross-sectional correlation between intelligence and education demands an open mind with regard to causal interpretation. Perhaps more intelligent people gain access to more and higher-level education. Perhaps exposure to more education causes higher intelligence test scores. The problem is one that is basic to epidemiology: what is person and what is situation, what is genetic and what is environmental, and what is cause and what is effect? Influences may flow in both directions, and longitudinal studies can help to quantify their relative magnitudes.

Does higher intelligence beget better educational outcomes? In longitudinal studies that measure psychometric intelligence first and educational attainments later (thus assessing that causal chain), there is a moderate to strong correlation between the two, as assessed by years spent in full-time education, the highest qualification obtained by a person, or the scores obtained on educational assessments (5). For example, in a study of about 70,000 children in the United Kingdom, the general factor from the Cognitive Abilities Test (CAT) battery taken at age 11 correlated about 0.8 with the general factor of grades on the General Certificate of Secondary Education (GCSE) examinations taken at age 16 (6). The general factor of the CAT test had very similar loadings from the three domains of verbal, non-verbal (abstract), and quantitative

reasoning. Older studies have reported correlations ranging from 0.60 to 0.96 (e.g., 7, 8, 9). The conclusion from such studies might be that intelligence has stronger causal effects on educational results than vice versa.

Does more education beget higher intelligence? Most studies of the influence of education on intelligence have not been longitudinal, but they have carefully examined the relation between length of schooling and intelligence, thus attempting to assess the reverse causal chain. Findings generally support the observation that more time in school does lead to greater intelligence. For example, Baltes and Reinert (10) compared the intelligence scores of three cross-sections of German 8- to 10-year-olds who were separated in age by 4-month intervals. The intelligence tests used were assessments of induction, verbal comprehension, numerical facility and processing speed from the German Begabungstestsysteem, which was based on Thurstone's theory and classification of Primary Mental Abilities. Since the German school system at the time required the entering children to be 6 years old by April 1, it was possible to compare the scores of children whose birthdays fell either just before or just after that dividing point, so that the children were effectively the same chronological ages but had a 1-year difference in schooling. Baltes and Reinert found that 8-year-olds who had received an extra year of schooling performed more like the least schooled 10-year-olds than the least schooled 8-year-olds. They noted, also, that the test most affected in this way—the Grundrechnen test of numerical facility—“is heavily loaded with material that is covered in the grade levels used”. Tests of more fluid skills were less affected, for example, the Buchstabenzaehlen test of letter counting which assessed processing speed, and which contained material much less based on taught materials. Schmidt (11) reported analogous results from a South African community of East Indian immigrants who had varying exposure to school that was not dependent on ability.

There, the correlations between schooling and two measures of nonverbal intelligence and one measure of verbal intelligence ranged from 0.49 to 0.68. The conclusion from such studies might be that education influences the development of intelligence. However, this requires the caveat that the so-called ‘intelligence tests’ should be scrutinised to examine the extent to which they contain materials that appear in the taught curriculum.

So, it is possible that intelligence causes differences in educational outcomes, or that education causes intelligence differences, or a bit of both. Indeed, it is probably more complex than this. Readers can find further detailed consideration of possible non-linear effects of schooling on mental test scores, and the parts played by measurement error in intelligence and education measurement in a rather technical paper by Hansen et al. (12). In this paper, too, is the interesting idea of using a latent trait of ‘ability’ that might underlie both schooling and scores on achievement tests that are often used as indicators of intelligence.

Within epidemiology, educational attainment or performance in young adulthood is often implicitly or explicitly assumed to be an outgrowth of social position in childhood, reflecting causal familial environmental effects. For example, in a study modeling the effects of education and childhood and adult socioeconomic position (SEP) on midlife cognitive function, Singh-Manoux et al. (13) concluded (p. 577) that,

“A major part of the effect of education on cognition... is also indirect. In these data, the influence of education on cognition is mostly through its influence on adult SEP.... The total impact of socioeconomic circumstances [both childhood and adult] on cognitive abilities... is substantial.... This merits the appropriate modeling of the impact of socioeconomic circumstances.”

Ironically, despite their note regarding appropriate modeling, in their analysis they assumed rather than tested the directions of the ‘causal’ arrows linking education and cognitive function. In their study, cognition was a latent trait comprising the following manifest variables: verbal

memory, Alice Heim 4 Test Part 1 (verbal and numerical reasoning), Mill Hill vocabulary, phonemic fluency, and semantic fluency. Education was assessed by allocating subjects to five levels of attainment, from no formal education to higher university degree.

Different views about education and intelligence and their association in epidemiology

Intelligence and education are commonly used as possible causes and mediators of other outcomes. Epidemiologists, sociologists, psychologists, economists, social geographers and demographers include intelligence and education as possible influences upon a variety of human factors, including health and illnesses, late-life cognitive function, social mobility, and subsequent status attainment. Among such researchers there are striking differences in how the association between intelligence and education is viewed and treated analytically.

Examples assuming education is causal. Richards and Sacker (14) studied life-course contributions to scores on the National Adult Reading Test (NART) at age 53, a measure which they took to be an index of cognitive reserve, or peak cognitive ability in adulthood. The NART asks the subject to pronounce 50 words that do not follow the normal rules of grapheme-phoneme association or stress. Therefore, the subject has to have met the words previously to know how to pronounce them. Among the contributors to differences in NART scores at age 53 were cognitive ability at age 8 (the sum of four tests) and educational attainment by age 26 (a variable with five levels). A path analysis showed standardized path weights of 0.45 between cognition at 8 years and education by 26 years, and 0.24 between education by age 26 years and NART at age 53. Here is some verbatim discussion of these results by the authors,

The second path to midlife cognition, that via educational attainment, is easier to conceptualize, since there is clear evidence that schooling per se can lead to cognitive gains, even in late adolescence... Indeed, data from the 1946 birth cohort show that academic performance of the primary school (i.e., its record in sending pupils to selective

secondary schools) was predictive of increased cognitive performance... Furthermore, it has been shown in the British 1958 birth cohort that the academic performance of the school is one of the major contributors to social class differences in childhood cognitive function... (p. 621).

The study results suggest at least two possibilities that are ignored in this interpretation.

First, there was a strong association between cognition at age 8 and education by age 26. As childhood cognition or IQ is subject to genetic influences, there may be genetically-influenced individual differences that contribute directly to educational attainment. Second, though this study measured both direct and indirect (via education) paths between cognition at age 8 and NART at age 53, it is likely that the tests given at age 8 did not fully capture all the cognitive variance that was present in the NART at age 53. This is because there are individual differences in the rates at which intelligence develops in children (15, 16), and there is some evidence that the intelligences of brighter children may develop more slowly relative to their mature levels than those of the less bright (17) relative to their mature levels. It is possible that education by age 26 acted as a surrogate marker for cognitive variance that was not picked up by the age 8 tests. That is, some of the differences in what is actually cognitive development from age 8 to age 53 might be indexed by education as a proxy measure. Moreover, the NART is a measure of intelligence that reflects accumulated knowledge to a particularly high degree. As knowledge is one of the clear benefits of education, performance on the NART may be especially subject to this effect.

Thus, in two ways, the association between education and NART in the Richards and Sacker (14) study might be caused at least in part by inherent cognitive ability per se. Richards's and Sacker's discussion of education ignored these possibilities and considered only the possible environmental effects of the educational setting.

Epidemiologists also routinely use education as an indicator of socioeconomic position in studies of health, or adjust for education as a possible confounding (causally prior) factor in studies of the determinants of health outcomes. For example, Singh-Manoux and Marmot (18) followed this practice, concluding that,

The effect of blood pressure on cognition was stronger among women, and was stronger for some measures of cognitive ability than others... Confounding factors of age, educational level, occupational position, smoking, alcohol consumption, use of antihypertensive medication, diagnoses of diabetes, and cardiovascular disease were controlled in the analyses.... (p. 1312)

Cognition and education were assessed as described above (13). It is rare to see full discussion or even statement of the assumptions underlying these adjustments for educational level and occupational position. Sometimes, the assumption is that education represents childhood social position. For example, in an important study that compared education and adult occupational social class, Davey Smith et al. (19) commented that, “The stronger association of education with death from cardiovascular causes than with other causes of death may reflect the function of education as an index of socioeconomic circumstances in early life, which appear to have a particular influence on the risk of cardiovascular disease” (p. 153). They noted, however, that this way of assessing early life socioeconomic circumstances was far from universally used. “In the UK, studies of socioeconomic differentials in mortality have generally relied upon occupational social class as the index of socioeconomic position, while in the US, measures based upon education have been widely used” (p. 153). Other times, it is at least recognised that education might lie between mental ability and health outcomes on a causal path. For example, Osler et al. (20) stated, “The attenuation towards the null of this association [between prior IQ and later injuries] with adjustment for educational attainment suggests that an overall increase in educational level may result in a reduction in adult injury risk” (p. 7). Here, education is cast as

the causal factor, though it might be acting as a surrogate for other aspects of the earlier-measured cognitive ability.

Some economists, too, have examined education as a variable related to health, without considering the role of intelligence in the creation of educational variance. For example, the large study of the US censuses of 1960, 1970 and 1980 found that education was related to mortality. People with less education had greater mortality rates (21). The conclusion was that, “education has a causal impact on mortality” (p. 189), and that “we need to consider education policies more seriously as a means to increase health” (p. 215). Some possible mediating variables were mentioned, including stress, depression, and hostility, but the place of intelligence as a possible influence on educational outcomes was not mentioned. On the other hand, other economists have been nuanced in looking at the contributions of intelligence and education to health. In an analysis of the National Longitudinal Survey of Youth 1979, there was an interaction between them: “the causal effect of schooling on health is greatest for individuals with low cognitive ability” (22).

Examples assuming education is an outcome of intelligence. A contrast to the treatment of education as causal is that by Herrnstein and Murray (23) in *The Bell Curve*. They argued that education should not be statistically controlled at all in examining the association between adolescent cognitive ability and later life outcomes, because intelligence is a determinant of education. Their argument was that there is movement of people into higher levels of education based upon prior intelligence differences which are in part caused by genetic variation. This is actually consistent with current teachings of statistical practice in epidemiology (24), but both interpretations and statistical approaches rely on causal models of the processes involved that should be tested rather than assumed.

Batty et al. (25) noted the potential implications of adjusting for education in evaluating the impact of childhood IQ on premature mortality in middle age. Rather than conceptualise education as an indicator of childhood social position—a confounding factor—they explained how it also might be a mediating factor or a proxy indicator of intelligence.

Higher IQ test scores may lead to educational success, and entry into well remunerated, high-status employment with a concomitantly high salary. An alternative, but often ignored, explanation is that educational attainment may represent a proxy for IQ, rather than the converse. That is, people with higher IQs stay longer within education, gaining more and higher qualifications. In this study, IQ at age 11 was moderately strongly correlated with subsequent educational attainment ($r = 0.61$; $p = 0.001$)... including education in our statistical models may be regarded as overadjustment (p. 243-244).

Such a treatment, though perhaps also arbitrary, at least makes the alternative causal accounts explicit. The IQ tests in this study were two of the Moray House Test series, which mainly contain items requiring verbal reasoning, but not material that is explicitly taught in school. Education was assessed using qualifications, which were classified into six categories, from none to postgraduate qualifications.

We have now shown that intelligence and education are correlated, and given illustrations of how education is sometimes assumed to be causal in epidemiology without considering that it might be in part an outcome of intelligence, and might even share genetic as well as environmental influences with it. Next, we examine the extent to which this is found.

Intelligence and education: do they share genetic and environmental influences?

One way to resolve some of the confusion over the causes of the association between intelligence and education is to examine the transactions among the genetic and environmental influences contributing to them. As we have already noted, the presence of genetic influences on intelligence is well established. These influences increase from less than 50% of variance in

childhood to around 70% in adulthood (26, 27, 28). The variance accounted for by shared environmental influences on intelligence declines from early childhood to a near-to-zero contribution in adulthood. Non-shared environment contributes a sizeable minority of the influence through most of life, though this term also contains error of measurement.

Multivariate variance decompositions can take this exploration further. They can estimate the environmental and genetic contributions to the correlation between two measured variables such as intelligence and education, and the extent to which the two variables share common genetic and environmental influences. For example, the national test of educational achievement used in the Netherlands at age 12 (the CITO test; 29) correlated between 0.41 and 0.63 with intelligence test scores gathered at ages 5, 7, 10 (using the Revised Amsterdamse Kinder Intelligentie Test) and 12 (using the Wechsler Intelligence Test for Children). The additive genetic contributions to variance in the CITO were about 60%, and genetic influences were the principal reason for the correlations between the intelligence test measures and the CITO. Similar results were obtained by Johnson, McGue, and Iacono (4, 30) in an adolescent sample, where a latent variable representation of school grades formed the measure of achievement and intelligence was measured using abbreviated Wechsler Scales (the children's scales for the under-16s, and the adult scales for those aged 16 and older). Almost 70% of the educational variables's variance could be attributed to genetic influence, and over 56% was common to genetic influences on intelligence. Even after other predictors of school grades including engagement, family risk, and disruptive behaviors were included in addition to intelligence, 34% of the genetic influences on school grades were shared with intelligence. In a Swedish twin-based study, intelligence was assessed at military conscription—using tests of reasoning, synonym detection, visuospatial perception, and mathematics/physics—and education was based

on seven categories from less than nine years of education to doctoral studies. The genetic correlation between intelligence and education was greater than 0.5, and varied little (from .53 to .56) across the range of intelligence, and the shared environmental correlation between the two variables was 1.0 (31). This evidence of shared sources of influence is useful for epidemiologists to know and recognise in discussing results.

In fact, the causes of the association between intelligence and education might be more complex (32). Analyses of educational attainment at age 24 in the USA, based on data from the the Minnesota Twin Family Study, showed that the genetic and environmental contributions to educational outcomes can differ at different levels of intelligence (33). The genetic variation in educational attainment increased four-fold from low intelligence (people two standard deviations below the mean intelligence level) to high intelligence (two standard deviations above the mean). By contrast, the shared environmental variation increased more than ten-fold across the same range of intelligence. In simpler terms, this means that, in this particular geographical and temporal setting, one's rearing environment (including family resources, broadly conceived) was a much more important source of variance in educational outcomes at lower than at higher levels of intelligence, where genetic sources were much more important. A similar set of analyses was conducted in Sweden, with importantly similar and different results. At higher levels of intelligence, as was found in the Minnesota twin sample (33), genetic variance in educational outcomes were greater than at low levels of intelligence. For shared environment variance, however, the two countries had opposing results: in Sweden, there was more shared environmental variance at higher than lower levels of intelligence (31). One should not forget, however, the genetic and shared environmental correlations between intelligence and educational attainments, which were strong in both locations.

Genetic influences common to educational attainment and cognitive ability are also found among older people. In a sample aged between 50 and 70 years, a common genetic factor accounted for 40% or more of the variance in two measures of cognitive ability (Mini-Mental State Examination and Iowa Screening Battery for Mental Decline) and 21% of the variance in educational attainment (34). The authors of this study concluded with the following comment,

At present, clinicians are taught to discern cognitive loss when a diagnosis of dementia is considered, and final diagnostic criteria specify that a decline in ability must have occurred before a definite diagnosis of dementia is made. Because in most situations no data on premorbid level of function are available, the general practice is to use education and occupational attainment as substitute measures of premorbid levels of function. In this regard, education adjustment seems useful and necessary, and the present finding of a common genetic factor supports this practice (p. 52).

This provides a marked contrast to the quotation from Richards and Sacker (14), above. Whereas Richards and Sacker viewed education as an environmental contributor to peak cognitive ability, these researchers (34) viewed education as a proxy for peak prior cognitive ability precisely because it captured at least some of the genetic influences on intelligence. Importantly, although the statistical approach these researchers (34) recommended is the opposite of that recommended by Herrnstein and Murray (23), their conceptions of the role of education in cognitive function are the same. This emphasizes that the appropriateness of statistical approaches are dependent not only on the accuracy of the causal conceptualizations underlying their use, but also on the specific timing of measurement of the variables involved.

Conclusions and recommendations

These examples illustrate the diversity of assumptions that underlie approaches to study design involving education and intelligence among epidemiologists and other health and social scientists. At the same time, they highlight the impact that such assumptions can have on study

design, results, and interpretation of results. Because these assumptions are often unstated and unacknowledged, these examples also demonstrate that part of the difficulty in disentangling the possible causal associations linking these two variables can be traced to less-than-objective examination of all of the causal possibilities during study design and interpretation. Some of these difficulties can be remedied by greater attention to, awareness and statement of, underlying assumptions, and the consideration of reasonable alternatives by all researchers making use of education and intelligence and other closely related variables. This is important if we are to understand how cognitive function is involved in the development, maintenance, improvement, and deterioration of physical health.

We are far from being the first to state that one must be suspicious about inferences after statistical tests to assess confounding, or mediation. We concentrated narrowly on this matter with respect to how education and intelligence are treated in epidemiology because these closely related variables are critical to understanding the role of cognitive function in epidemiology. And we tried to argue that knowledge about causal background enhances analytical decisions and interpretations. This point is made well, in the context of birth defects epidemiology, and more generally, by Hernán et al. (35).

In order to make the points above, we have not always gone into detail on how educational assessments can differ. What are referred to as 'educational outcomes' can refer to quite distinct empirical phenomena: e.g., years of schooling completed, highest credential obtained, subjective assessments of academic performance (e.g. class rank), and standardized tests of academic achievement in some content domain. These have different correlations with intelligence test scores, because all result from somewhat different personal traits and circumstances, and they are measured with varying degrees of accuracy. Making such

distinctions will be crucial for forming meaningful causal hypotheses about education and intelligence and how they combine to influence people's lives.

It is clear that not everyone derives the same benefit from any given educational opportunity and that the same educational opportunities are not available to everyone. Distinguishing between the processes involved in education and intelligence is difficult because it requires measurement that can simultaneously establish causal attributions through precise timing and identify both genetic and environmental influences and their relations to the timing of measurement. The data necessary to do this with respect to education and intelligence are not often available. There are clear implications of the above points for study design.

First, the temporal cascade between intelligence and education will be clearer when repeated measures of each are available. This would allow longitudinal models to examine the direction and strengths of the mutual causal influences. Second, genetically informative designs—such as twin studies—can help to uncover the environmental and genetic aetiologies of the correlations between intelligence and education, and the other life outcomes with which both are associated. It will be especially interesting when specific genetic variants are found that are associated with intelligence differences, as these can also be examined to discover whether they are associated with educational differences. Third, it should be kept in mind that, even though intelligence and education are correlated, one can still act as a moderator of the other with respect to life outcomes, such as health (22). Therefore, study designs powerful enough to include interactions between the two are desirable. Fourth, where it is possible to do so, multiple assessments of intelligence and educational outcomes at a single time point will alleviate the problems of measurement error through the construction of latent variables (6,12). Fifth, a mind that is kept open to the various plausible interpretations of analyses which involve education and

intelligence helps, even when the above design strengths are not available. Sixth, we should not be blinkered by considering only intelligence and education. It should be kept in mind that there might be other variables that contribute to the association between intelligence and education.

Possible candidates could be personality traits and their influences on coping styles and motivations. Therefore researchers should consider measuring such constructs.

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Key messages

We illustrate that the use of education and intelligence measurements comes with different views about their environmental and genetic origins, and the reasons for their being correlated

We show how this influences approaches to analyses and the interpretations of results

We provide some information about the reasons for intelligence and education being correlated

We provide some suggestions for study designs and argue for researchers to consider all likely interpretations of results involving education and intelligence