References

Is education causal? Yes

From MARCUS RICHARDS1* and AMANDA SACKER2

1MRC Unit for Lifelong Health and Ageing, London, UK and 2Institute for Social and Economic Research, Essex, UK

*Corresponding author. MRC Unit for Lifelong Health and Ageing, 33 Bedford Place, London WC1B 5JU, UK.

E-mail: m.richards@nshd.mrc.ac.uk

In a provocative analysis piece Deary and Johnson critique the lack of theory behind the use of education as an explanatory variable in epidemiology. There are of course several well-defined sociological theories of education in relation to health, although it is a fair comment that these are not always sufficiently
heed in population-based analyses. More specifically, they also challenge the extent to which education has a causal effect on subsequent intelligence (and by implication other health-related outcomes). One of several studies singled out for criticism in this context is that of our path model linking circumstances of origin, childhood intelligence assessed at 8 years, educational attainment by 26 years, occupational attainment (social class) by 43 years and cognitive function a decade later in the British 1946 cohort. Briefly, we found strong direct paths between childhood and midlife cognition, and between the former and education; but also a modest independent path from education to midlife cognition, with only a small additional contribution from adult occupation. We conclude that education has a causal influence on subsequent cognition, which is not fully explained by social class of origin, or childhood intelligence, or subsequent occupational attainment.

Deary and Johnson direct three criticisms at this conclusion, which we list in ascending order of challenge. First, they concentrate on only one of our outcomes, the NART, which they suggest is, as a measure of knowledge accumulation, particularly sensitive to education; in other words, our argument is circular. This need not detain us since we observed a similar pattern of results for memory, even with the NART controlled. In any case, the NART is so highly predictive of full-scale IQ that it can be thus prorated. If Deary and Johnson are making a subtle claim that verbal knowledge represented by this measure (or cousins such as the Vocabulary and Information components of the Wechsler Adult Intelligence Scale) should be downgraded as aspects of intelligence, then they should be explicit about this. Indeed, Johnson herself has shown that a general cognitive ability factor can be derived from numerous diverse cognitive ingredients.

The second caveat is that intelligence may not be fully developed by age eight, so that some of its full variance is being picked up by educational attainment. In fact, Hatch et al. showed, in the same cohort on which our path model is based, that adult education and training were associated with higher NART scores even after controlling not only for formal education but also for verbal ability at 26 years, by which time intelligence is presumed to be fully formed. As we shall see, it is just as much a category error for Deary and Johnson to suggest that education is a ‘proxy’ for intelligence as it is for others to presume that it represents early circumstances or adult socioeconomic position, even though all four of these variables are highly intercorrelated.

The third and perhaps most complex criticism is based on evidence that intelligence and educational attainment share a genetic common cause, which is implied to be the real cause of the putative ‘environmental’ effect of education. More carefully stated, they suggest a shared genetic influence on intelligence and other factors that drive educational attainment, most probably personality traits or related ‘non-cognitive skills’ such as self-regulation. We do not disagree with this, only note that it does not negate the possibility of an environmental influence of education, but merely qualifies this in reference to a partial selection effect. In one study quoted, for example, 34% of the genetic influence on educational attainment was shared by intelligence, which means of course that 66% was not. And even if we agree that educational attainment per se is substantially genetically determined, it is hard to appreciate exactly how this genetic potential is reified outside the learning environment; even if the ‘soft’ skills that children bring to the classroom have no more reached their genetic potential than have their intellectual skills, it is a straining credibility to suggest that this environment has little to do with their structuring and refinement.

At this point, it is worth raising an issue that is under-emphasized by Deary and Johnson; in a relatively large number of studies of health in relation to prior cognition, education attenuates the effect of cognition towards the null but not vice versa, particularly when the outcomes in question are phenotypes related to chronic physical disease. And for those still sceptical, it is probably worth noting a study comparing our path model between the British 1946 and 1958 cohorts, using the more functional outcome of everyday literacy and numeracy problems. If genetic influence is the key driver of an educational effect on this outcome, then there should be little difference between these models separated by a mere 12 years; in fact, there are systematic differences, not least in the path between cognition and education, but most strikingly a significantly stronger path between the latter and the outcome in the younger cohort following a raising of the school leaving age in 1972, and thus a large increase in the number of children leaving school with educational qualifications.

In sum, there are strong grounds for maintaining our position that there are specific causal effects of education on later health-related outcomes, including cognition.

References
Author’s Response
From IAN J DEARY* and WENDY JOHNSON

Centre for Cognitive Ageing and Cognitive Epidemiology, Department of Psychology, University of Edinburgh, UK

*Corresponding author. Centre for Cognitive Ageing and Cognitive Epidemiology, Department of Psychology, University of Edinburgh, 7 George Square, Edinburgh EH8 9JZ, UK. E-mail: i.deary@ed.ac.uk

We should like to make six brief points—to make sure readers understand what our essay on education and intelligence1 set out to achieve, and to encourage more discussion about this important matter.

(1) We are pleased that Marcus and Amanda have engaged with us in addressing some of the issues we raised.2 We had hoped to begin just such a full and frank discussion about intelligence and education, and their causal associations.

(2) We repeat that our essay demonstrated, among researchers, a wide range of underlying assumptions about: the origins of variation in education (some more exogenous, some more endogenous); the reasons for education’s association with intelligence; and the interpretation of epidemiological findings involving education. Our goal was to urge researchers to make these often tacit assumptions explicit so that they can be examined and tested, and so that all tenable causal possibilities are considered and not just those favoured by the investigators.

(3) We had intended not to be partisan about the origins and causal associations between intelligence and education. It was our intention to urge researchers in epidemiology and differential psychology to consider interpretations that their disciplinary traditions tended to downplay or omit.

(4) Even though we are regular users of the method, we do not think that staring more closely at structural equation model-based path analyses will answer the causal questions we raised. However, as we and Marcus and Amanda and others have shown, such thinking can raise an appropriately wide range of possible interpretations of the same dataset.

(5) We urged, and still urge, researchers in this important area to consider appropriate research designs that can address causal matters. We gave twin studies as one example. Where they are possible, intervention studies will help too.

(6) We do not think these matters are impossible to sort out, but we wish to emphasize that few studies, especially cohort studies (including our own), afford straightforward conclusions. New research designs and an open mind to causal interpretations will help in making progress.

References


* The Author 2010; all rights reserved.