and injuries in later life: the Metropolit 1953 male birth cohort. *Int J Epidemiol* 2007;**36:**212–19.

[21] Lleras-Muney A. The relationship between education and adult mortality in the United States. *Rev of Eco Stud* 2005; **72:**189–221.

[22] Auld MC, Sidhu N. Schooling, cognitive ability and health. *Health Eco* 2005;**14:**1019–34.

[23] Herrnstein RJ, Murray C. *The Bell Curve*. New York, NY: Free Press, 1994.

[24] McNamee R. Confounding and confounders. *Occ Environ Med* 2003;**60:**227–34.

[25] Batty GD, Deary IJ, Macintyre S. Childhood IQ in relation to risk factors for premature mortality in middle-aged persons: the Aberdeen Children of the 1950s study. *J Epidemiol Comm Health* 2007;**61:**241–47.

[26] Bouchard TJ, McGue M. Familial studies of intelligence: a review. *Science* 1981;**212:**1055–1059.

[27] Deary IJ, Spinath F, Bates TC. Genetics of intelligence. *Eur J Hum Gen* 2006;**14:**690–700.

[28] Plomin R, Spinath F. Intelligence: genetics, genes, and genomics. *J Person Soc Psychol* 2004;**86:**112–29.

[29] Bartels M, Rietveld MJH, Van Baal GCM, Boomsma DI. Heritability of educational achievement in 12-year-olds and the overlap with cognitive ability. *Twin Res* 2002;**5:** 544–53.

[30] Johnson W, McGue M, Iacono WG. Genetic and environmental influences on academic achievement trajectories during adolescence. *Dev Psychology* 2006;**42:**514–32.

[31] Johnson W, Deary IJ, Silventoinen K, Tynelius P, Rasmussen F. Educational attainment: family background buys it in Minnesota but not in Sweden. *Psychol Science* (in press).

[32] Johnson W. Genetic and environmental influences on behavior: capturing all the interplay. *Psychol Rev* 2007; **114:**423–40.

[33] Johnson W, Deary IJ, Iacono W. Genetic and environmental transactions underlying educational attainment: something for everyone but everything for no one. *Intelligence* 2009;**37:**466–78.

[34] Carmelli D, Swan GE, Cardon LR. Genetic mediation of the relationship of education to cognitive function in older people. *Psychol Aging* 1995;**10:**48–53.

[35] Hernán MA, Hernández-Díaz S, Werler MM, Mitchell AA. Causal knowledge as a prerequisite for confounding evaluation: an application to birth defects epidemiology. *Am J Epidemiol* 2002;**155:**176–84.

Published by Oxford University Press on behalf of the International Epidemiological Association *International Journal of Epidemiology* 2010;**39**:1369–1371

doi:10.1093/ije/dyq123

Commentary: Is it time to redefine cognitive epidemiology?

Archana Singh-Manoux[1,2,3]

[1]INSERM, U1018, Centre for Research in Epidemiology and Population Health Hôpital Paul Brousse, 94807 Villejuif Cedex, France, [2]Department of Epidemiology and Public Health, University College London, London, UK and [3]Centre de Gérontologie, Hôpital Ste Périne, AP-HP, Paris, France

Correspondence to. INSERM, U1018, Centre for Research in Epidemiology and Population Health, Hôpital Paul Brousse, Bât 15/16, 16 Avenue Paul Vaillant Couturier, 94807 Villejuif Cedex, France. E-mail: archana.singh-manoux@inserm.fr

Accepted 14 June 2010

In this issue of *IJE*,[1] Deary and Johnson discuss the association between education and intelligence and argue that their use in epidemiological research as interchangeable entities is incorrect. They highlight at least three areas where this ambiguity is evident: choice of education rather than intelligence as a confounder of the association between an exposure and an outcome, the direction of the causal association between education and intelligence and, finally, their main effects on health outcomes.

Confounder/common cause

The authors argue that education is more widely available in epidemiological data sets, leading to its greater use as a 'control variable' when in fact the confounder or the common cause in the analyses ought to be intelligence. Epidemiologists generally agree that the association between an exposure and a health outcome contains two elements; the true causal effect of the exposure on health and the

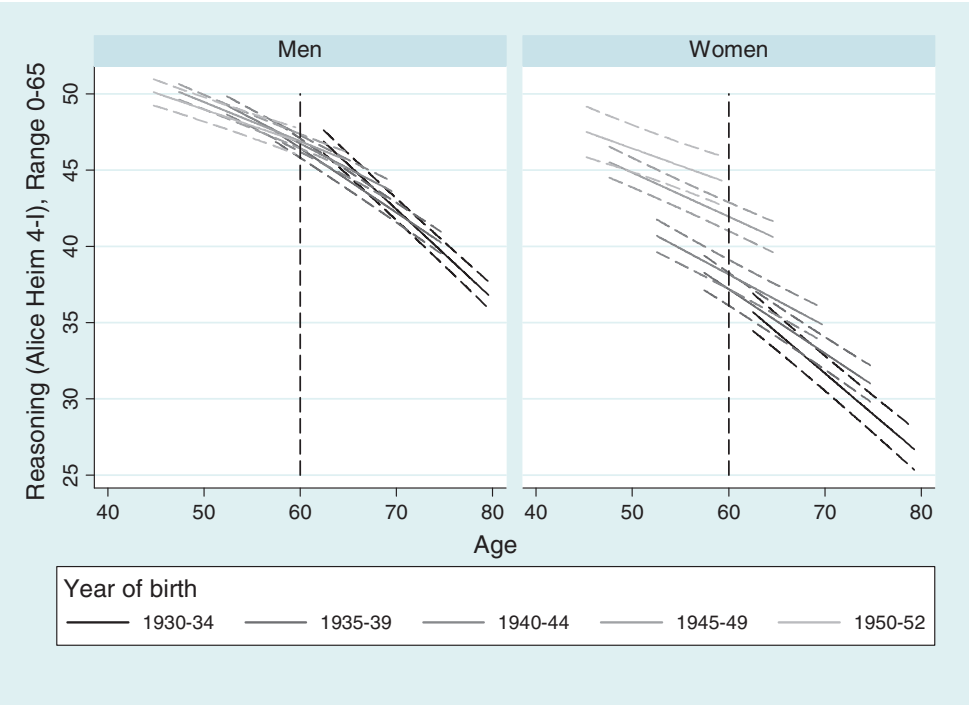

Figure 1 Decline [and 95% confidence interval (CI)] in reasoning (Alice-Heim 4-I) estimated from three repeat measures over 10 years as a function of year of birth in 5247 men and 2233 women in the Whitehall II study

effect due to a shared common cause, the confounder.[2,3] Analyses are adjusted for the confounder (common cause) in order to remove its spurious effect on the association between an exposure and the health outcome in question. If intelligence is the real confounder of these associations then, in the absence of a direct measure of intelligence, is it correct to adjust for education? The answer is yes. Hernan and colleagues[3] use directed acyclic graphs to show that when the hypothesized causal confounder (in this case intelligence) is not measured, adjusting for the measured variable (education) is correct as it acts as a surrogate confounder and adjustment for it removes most of the effect of the common cause (intelligence) from the association between the exposure and the outcome.

Two-way relationship between education and intelligence

It is possible that there is a two-way association between education and intelligence; although I am not sure that the two criteria, temporality and heritability, discussed by Deary and Johnson[4] are sufficient to enable us to determine the causal direction of the association. Temporality alone is not a sufficient criterion,[4] and the heritability estimate of most human traits at ~50% does not help us resolve issues of causality. The authors suggest that 'the temporal cascade between intelligence and education will be clearer when repeated measures of each are available' in longitudinal studies. However, at present, statistical methods that allow causality to be established in the presence of reciprocal effects do not exist.

Natural experiments tell us something about causality. A large body of research supports the existence of the Flynn effect, the trend for mean IQ scores to increase between generations, leading to much discussion of the explanations (improved nutrition, better care of children, better education, increasing familiarity with tests, heterosis, etc.) for the malleability of intelligence scores.[5] The increase in test scores can be dramatic as evident in Figure 1, which shows three repeat assessments on a test of reasoning (AH4-I),[6] also a test of fluid intelligence with a large loading on the general intelligence factor, in men and women of the Whitehall II study, a cohort of British civil servants.[7] Gender differences in child care, nutrition, familiarity with multi-choice questions or heterosis appear unlikely. However, we know that education levels in these women have improved; 22.6% of the oldest women (birth years 1930–34) completed high school education compared with 66.5% of the youngest women (birth years 1950–52) in this cohort. Does this prove that education causes intelligence? No, but how important is it for epidemiology to establish this causal direction? Epidemiology is a practical discipline concerned with identifying modifiable determinants to improve health at the population level. While intelligence lies beyond the reach of policy makers, education is a much easier

target. Many middle- and low-income countries set improving education as a major policy goal with a view to improving both the economic and health prospects of the population. Policies directed at education span the range from minimum school leaving age to increasing percentage of the population with a university degree.

Defining cognitive epidemiology

The final point made by Deary and Johnson is that the direction of the association between education and intelligence is important for the 'newly emerging field of cognitive epidemiology', defined previously as 'the use of cognitive ability test scores as risk factors for human health and disease outcomes, including mortality'.[8] Given the association between intelligence and education, extensively discussed by Deary and Johnson, this definition of cognitive epidemiology puts it squarely in the domain of social epidemiology, a discipline concerned with the social distribution of determinants of health. Location in this broader church, rather than the micro-discipline of cognitive epidemiology, will avoid a narrow focus on intelligence that ignores its associations with markers of social position such as education, income and occupation.

My concern is also with the 'cognitive' in this definition of cognitive epidemiology. Cognitive function and intelligence are not interchangeable concepts. For one, cognitive function is not a unitary concept like intelligence; it is composed of multiple aspects involved in the input, storage, processing and output of information.[9] Not all cognitive functions decline with age at the same rate. Some, like vocabulary, show little decline. Others, such as measures of executive function, are more likely to be affected by cardiovascular risk factors. Decline in memory, besides its importance for Alzheimer's disease, might have a stronger association with mortality.[10] Furthermore, most epidemiologists study cognitive function as an outcome rather than an exposure, making the definition proposed by Deary and colleagues rather restrictive.

Continuing increases in life expectancy imply fundamental changes to the population structure. Impaired cognitive status is one of the biggest challenges of the future because of its impact on both the individual and society. We now know that decline in multiple domains of cognitive function starts in mid-life, which is evident in those <50 years in the Whitehall II study (Figure 1). We also know that the inter-individual differences steadily increase with age,[11] even though the determinants of this increasing heterogeneity are not entirely clear. For these reasons, more than the impact of cognition on health, cognitive epidemiology should principally be the study of the pathophysiology of cognitive decline, involving etiological models to identify the biological, behavioural, psychological and social risk factors. Although age itself is not modifiable, given its heterogeneous impact on cognitive decline it is time to deconstruct this effect in order to meet the challenge of ageing populations. Epidemiology's contribution to medicine is the study of the causes, distribution and control of disease in populations; cognitive epidemiology need not be any different.

Funding

I acknowledge support from the European Science Foundation via a 'European Young Investigator Award' and the National Institute on Aging, National Institutes of Health (R01AG013196; R01AG034454).

Acknowledgements

I would like to thank Aline Dugravot, Jane Ferrie, Herman Nabi, Séverine Sabia and Silvia Stringhini for comments on a previous draft of this article.

Conflict of interest: None declared.

References

[1] Deary IJ, Johnson W. Intelligence and education: causal perceptions drive analytic processes and therefore conclusions. *Int J Epidemiol* 2010;**39:**1362–69.

[2] Weinberg CR. Toward a clearer definition of confounding. *Am J Epidemiol* 1993;**137:**1–8.

[3] Hernan MA, Hernandez-Diaz S, Werler MM, Mitchell AA. Causal knowledge as a prerequisite for confounding evaluation: an application to birth defects epidemiology. *Am J Epidemiol* 2002;**155:**176–84.

[4] Rothman KJ, Greenland S. Causation and causal inference in epidemiology. *Am J Public Health* 2005;**95 (Suppl 1):**S144–50.

[5] Dickens WT, Flynn JR. Heritability estimates versus large environmental effects: the IQ paradox resolved. *Psychol Rev* 2001;**108:**346–69.

[6] Heim AW. *AH 4 Group Test of General Intelligence*. Windsor, UK: NFER-Nelson, 1970.

[7] Marmot M, Brunner E. Cohort profile: the Whitehall II study. *Int J Epidemiol* 2005;**34:**251–56.

[8] Deary IJ, Batty GD. Cognitive epidemiology. *J Epidemiol Community Health* 2007;**61:**378–84.

[9] Lezak MD. *Neuropsychological Assessment*. New York: Oxford University Press, 1995.

[10] Sabia S, Gueguen A, Marmot MG, Shipley MJ, Ankri J, Singh-Manoux A. Does cognition predict mortality in midlife? Results from the Whitehall II cohort study. *Neurobiol Aging* 2010;**31:**688–95.

[11] Brayne C. The elephant in the room—healthy brains in later life, epidemiology and public health. *Nat Rev Neurosci* 2007;**8:**233–39.