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Archana Singh-Manoux

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Commentary: is it time to redefine cognitive epidemiology?

Archana Singh-Manoux [1 2 3 *]

[1] *CESP, Centre de recherche en épidémiologie et santé des populations INSERM : U1018 , Université Paris Sud - Paris XI , Université de Versailles-Saint Quentin en Yvelines , INED , FR*

[2] *Department of Epidemiology and Public Health University College of London (UCL) , 1-19 Torrington Place London WC1E 6BT,GB*

[3] *Centre de Gérontologie Assistance publique - Hôpitaux de Paris (AP-HP) , Hôpital Sainte Périne , FR*

* Correspondence should be adressed to: Archana Singh-Manoux <archana.singh-manoux@inserm.fr >

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In this issue of the 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 datasets, 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 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 use directed acyclic graphs to show that when the hypothesised 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 [3].

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 are sufficient to enable us to determine the causal direction of the association [4]. Temporality alone is not a sufficient criterion, [4] and the heritability estimate of most human traits at around 50% does not help us resolve issues of causality. The author's 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 childcare, 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 1930–1934) completed high school education compared to 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 % 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 due to its impact on both the individual and society. We now know that decline in multiple domains of cognitive function starts in midlife, evident in those under 50 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 etiologic 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.

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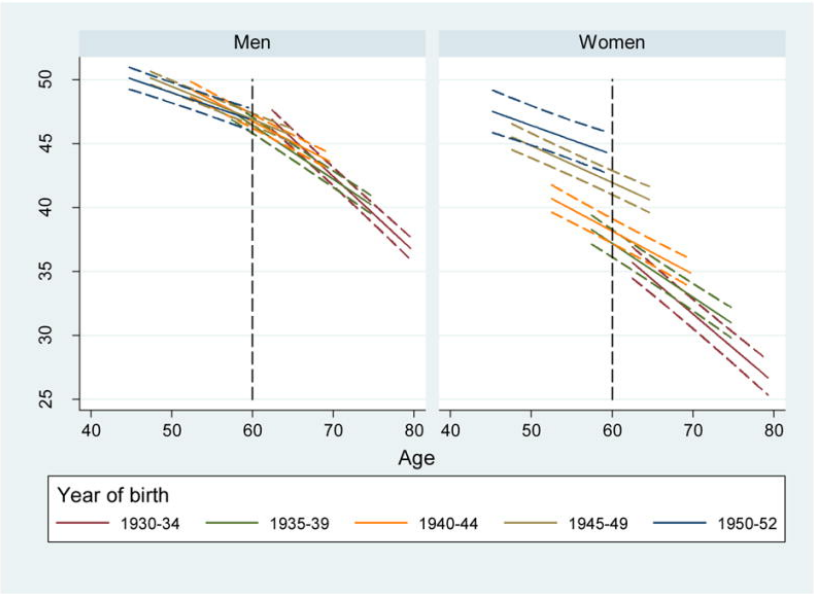
Figure 1

Decline (and 95% CI) in reasoning (Alice-Heim 4-I) estimated from 3 repeat measures over 10 years as a function of year of birth in 5247 men and 2233 women in the Whitehall II study.