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Cognitive epidemiology: its rise, its current issues, and its challenges

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ABSTRACT

Cognitive epidemiology is the study of intelligence—as measured using psychometric tests—as an associate of mortality, illness, and health. This essay has three parts. In Part 1, the rise of cognitive epidemiology—mostly in the last decade—is surveyed. Some reasons for its becoming convincing and well-established are enumerated and discussed. In Part 2, many of the current issues of interest within cognitive epidemiology are listed and exemplified. These address specific causes of death and specific illnesses and health behaviours with which intelligence is associated. The studies which attempt to address mechanisms of association are highlighted and discussed. In Part 3, a series of challenges facing cognitive epidemiology are examined. These include the better integration with epidemiology, the better uncovering of mechanisms, and the application of cognitive epidemiology's findings to health policy and practices.

It was an honour to be invited to give a talk at the festschrift for Tom Bouchard. One of his visions was that intelligence should work at an epidemiological level (Bouchard and Loehlin, 2001). Here, I describe and discuss how, in the field of health, that is now happening.

1. The rise of cognitive epidemiology

1.1. From 'neo-Nazi' to medical sociology

Our research team had reported—in the British Medical Journal (Batty, Der, Macintyre, & Deary, 2006)—findings that offered some partial confirmation for Gottfredson's (2004) hypothesis that psychometric intelligence might account for some of the variance shared between socioeconomic status and health. After the article had appeared one of our authors—a medical sociologist—was written to by a senior UK academic clinician,

I am particularly interested to see that the Unit's programme includes exploration of possible links between cognitive ability and health. Sometime around 1980, I... presented a paper suggesting that since human intelligence probably evolved through a beneficial effect on longevity and reproductive fitness, it might well have an influence on health in modern societies. This idea was received with severe disapproval and a friendly mole at the Department of Health confided to me that "they" had raised questions about what affiliations I might have to certain neo-Nazi political groups!

Things have changed. This year, Professor Sir Michael Marmot (2009)—Chairman, since 2006, of the World Health Organisation's Commission on Inequalities in Health—wrote an editorial in response to one of our recent cognitive epidemiology articles (Batty, Shipley, Dundas, Macintyre, Der et al., 2009). The title of his piece was, "Social inequalities in mortality: a problem of cognitive function?". The answer was not an unequivocal 'no'. In it, he wrote

Some observers have put forward the proposal that the only way to understand health inequalities is to examine the material conditions in which people live. This study on cognitive function makes clear that what happens in the mind, whether the influences come from the material world or the social, has to be taken into account if we are to understand how the socioeconomic circumstances in which people live influence health and wellbeing.

This section now examines some possible factors about cognitive epidemiology that led to this change in attitude, and the acceptance within epidemiology and medical sociology that individual differences in intelligence might play a role in explaining health differences.

1.2. Systematic review

It clearly helps if the data, taken as a whole, support the idea. A systematic review of the association between early life (childhood to young adulthood) intelligence and mortality discovered nine relevant studies, published between 1984 and 2006 (Batty, Deary, & Gottfredson, 2007). All had found that people with higher intelligence tended to live longer. The studies were conducted in three continents (Australia, America and Europe), on deaths from middle to old age, and from mental ability tests taken before adolescence. Some of the follow-ups spanned almost 70 years, and all of them involved a number of decades. Studies since then have replicated and extended the association between intelligence and all-cause mortality (e.g., Batty, Shipley, Mortensen et al., 2008a; Batty, Wennerstad, Davey Smith et al., 2009; Jokela, Batty, Deary, Gale, & Kivimaki, in press; Jokela, Elovainio, Singh-Manoux, & Kivimaki, 2009; Leon, Lawlor, Clark, Batty, & Macintyre, 2009; Sabia, Guegen, Marmot, Shipley, Ankri, & Singh-Manoux, in press), but none has refuted it.

1.3. Getting the message out

In part, the rise in cognitive epidemiology can be put down to the fact that a broad range of scientific and even more general lay readers have found the topic naturally interesting, and it has been given a forum as overviews and editorials in general journals. For example, the general journals *British Medical Journal* (Batty & Deary, 2004), *Current Directions in Psychological Science* (Gottfredson & Deary, 2004) and the British Psychological Society's *The Psychologist* (Deary, 2005) ran favourable pieces, as did *Nature* (Deary, 2008). In addition to such accessible overviews, there has been an attempt to nudge scientists to think about intelligence as a relevant variable in their own work. Our team has several times had letters published in response to journal articles in which intelligence could have been, but was not, considered as a possibly relevant variable to the health outcomes in their studies: especially when the predictors were social class or education. An example there was Sapolsky's (2005) essay in *Science* which overviewed data and speculated mechanistically on the association between human social hierarchies and health. His original piece contained no mention of intelligence—it did not appear to play a part in the thinking about how hierarchies might come about. However, when it was suggested that intelligence might have a part to play (Deary, Batty, & Gottfredson, 2005), he responded favourably to the suggestion. Therefore, intelligence as a variable might simply be off people's radar of mechanistic variables—of course, some will be more antagonistic towards it—and spreading the message that it is useful to consider will be helpful.

1.4. Reaching out

The best scientific work on cognitive epidemiology and the best way of making sure that intelligence does not become ignored will be for the research to involve a collaboration of at least individual differences psychologists and epidemiologists. One benefit of the collaboration is that the former have a tendency to look more to genetic factors and the latter

more to environmental variables. Whatever the biases, both sets of knowledge and analytical skills are needed for success in cognitive epidemiology. Moreover, intelligence will have its best influence if epidemiologists and their journals and policy influences take up the measurement and application of psychometric intelligence as relevant to health outcomes, especially alongside the more favoured variables of education and social class. The two sets of researchers tend to have complementary sets of statistical tools. These can usefully be brought together to illuminate findings in cognitive epidemiology. For example, we have used both Cox proportional hazards regression and structural equation modelling to study the mediating effect of social class on the association between intelligence and mortality: first, by using the two techniques in parallel (Hart, Taylor, Davey Smith, Whalley, Starr et al., 2003); and then by combining them in an omnibus analysis (Weiss, Gale, Batty, & Deary, 2009).

1.5. Slapping a name on it

The idea that cognitive ability may be related to health has been furthered by labelling it as field rather than burying cognitive ability within general epidemiology as just another variable that happens to be associated with health. The first use our group made of the name was in an article on how reaction time accounted for some substantial proportion of the association between intelligence and death (Deary & Der, 2005). That usage was in response to the recognition that others had urged that intelligence should be included in epidemiological studies and, indeed, that researchers in intelligence should raise their sights and start working at the population-representative level rather than merely using convenience samples. Bouchard and Loehlin (2001) and Lubinski and Humphreys (1997) had urged correction of the neglect of intelligence within epidemiology. And Krueger, Caspi and Moffitt (2000) were keen to see individual differences variables applied to population-based sampling frames. There is an accumulation of other ways in which the name appears too: a

glossary of ‘cognitive epidemiology’ appeared (Deary & Batty, 2007), a special issue of the journal *Intelligence* is currently in press on the topic of ‘cognitive epidemiology’, and our own Centre’s title—the Centre for Cognitive Ageing and Cognitive Epidemiology—is half devoted to the topic. Having a name is important: it is the consequence of there being enough data and ideas concerning intelligence and health and death; and the cause of a more focussed and lively interest in the topic by a wide range of researchers.

1.6. Strength in numbers #1: researchers

It does appear that scientists are responding to cognitive epidemiology as if it were a going concern. Take the example of the special issue of *Intelligence* on ‘cognitive epidemiology’ (see Deary [2009] for an overview of the papers and a summary of the samples used and their key findings). There are 15 empirical papers from different research groups. The sample sizes used for their analyses were mostly in the 1000s. The contributing scientists include differential psychologists, and some cognitive and neuro- psychologists, but there are also behaviour geneticists, epidemiologists (many), neurologists, other medical specialists, medical sociologists, other sociologists, and statisticians. It’s a good sign that the study of the application of intelligence differences can attract some of the best work from such a range of researchers.

1.7. Strength in numbers #2: the cohorts

Although left to the last topic in this section, perhaps the most helpful contribution to the rise of cognitive epidemiology has been the sheer weight of numbers and quality of the data involved in its cohorts. Among those—this is not a complete list—cohorts contributing to cognitive epidemiology are the following, with an indication in parentheses of the sorts of

numbers of subjects they have brought to bear in empirical studies: ¹*Aberdeen Children of the 1950s (11,000); Aspirin for Asymptomatic Atherosclerosis (2,000); *British Cohort Study 1970 (6,000); Danish Twin Studies (34,000); Generation Scotland (6,000); Minnesota Twin Family Study (1,000); *National Child Development Study (UK 1958 birth cohort; 6,000); *National Longitudinal Study of Youth 1979 (7,000); *National Survey of Health and Development (UK 1946 Birth cohort; 3,000); **Scottish Mental Surveys of 1932 and 1947 (1000s); *Swedish Conscripts Study (1,000,000); UK Health and Lifestyle Survey (9,000); West of Scotland Twenty-07 Study (900); Whitehall II Study (5,000); and *Vietnam Experience Study (4,000). Many of these cohorts have had their subjects' details and main contributions to cognitive epidemiology to date summarised in Deary and Batty (in press).

2. Current issues in cognitive epidemiology

2.1. IQ: health-associated lightweight or heavyweight?

Cognitive epidemiology would deserve to attract only a little attention if the effect sizes with mortality and other health outcomes were trivial in absolute size; or very small with respect to the effect sizes of other well-established risk factors for illness and death, against which they might be insignificant. There are many large studies that may be used to evaluate absolute effect sizes—but two will do. In the first study that examined the influence of childhood intelligence and death up to old age (76 years), a two-standard deviation disadvantage in intelligence at age 11 was associated with about twice the risk of being dead by age 76 (Whalley & Deary, 2001). Thus, the absolute effect is far from trivial (and the relative effect size will be discussed below). The largest study to date is the Swedish Conscripts Study (Batty, Wennerstad, Davey Smith et al., 2009). It examined 994,270 men

¹ Those studies that are marked with an asterisk have intelligence tested from either childhood or young adulthood, and have been followed up into adulthood or even old age.

born between 1950 and 1976. They were tested for intelligence at military conscription at a mean age of 19.5 years. There were four tests—logical, spatial, verbal, and technical—and they were used to construct a general mental ability (*g*) factor. They were followed up for 19.9 years. There were 14,498 deaths in the follow-up period. A one standard deviation advantage in intelligence at conscription was associated with 24% reduced risk of dying in the 20-year follow-up period. The 95% confidence interval for the estimate was 23% to 25%, so the effect is robust as well as sizeable; however, it may be seen from the sample size and the number of deaths that the chances of dying between 20 and 40 are low. The effect appears more dramatic when one looks at extremes of intelligence groupings. In the Swedish Conscripts Study data, the intelligence scores are grouped into nine categories of intelligence—stanines—to make a normal-type distribution. Compared with the lowest-scoring group, the most intelligent group had 70% reduced risk of mortality in the follow-up period, a very sizeable effect. When this association was adjusted for age at test, birth year, blood pressure, parental socioeconomic status, height, weight, and physical and psychiatric illness at conscription, the highest IQ group still had a 60% reduced risk compared with the lowest. Therefore, the advantage that the highest IQ group had for survival was not explained very much at all by these several death-relevant factors. Individual causes of death—and the association with intelligence—in this large sample are discussed more below.

The second issue raised above is whether intelligence is a relatively powerful associate of intelligence when compared with other risk factors. Evidence for this comes, for example, from the Vietnam Experience Study (Batty, Shipley, Mortensen, Gale, & Deary, 2008). This was based on 18,313 male USA Vietnam veterans. They took the Army General Technical Test at recruitment at 22.5 years. There was a telephone interview to gather data at 38 years for 15,288, a comprehensive medical examination at age 39 for 6,443 in 1986, and then

mortality surveillance until 2000. The analyses used produce hazard ratios for all predictor variables on the same scale. For all-cause mortality in the Vietnam Experience Study the hazard ratios were as follows for the available risk factors: smoking = 4.00; IQ at conscription = 3.32; fasting blood glucose = 1.73; systolic blood pressure = 1.63; high density lipoprotein = 1.59; diastolic blood pressure = 1.59; total cholesterol = 1.07; body mass index – 0.96. In this sample, and in others (Batty, Deary, Benzeval, & Der, in press), intelligence ranked highly as an associate of mortality.

In both absolute and relative strength, therefore, intelligence is a sizeable predictor of death. These absolute and relative strengths of intelligence apply to all-cause and cardiovascular disease mortality. The latter is mentioned as it tends to be the largest cause of death in samples in middle age and beyond, and its risk factors are much studied.

2.2. People die of something

With an intelligence-mortality association firmly established, it is natural that researchers and policy makers wish to know the causes of the association. One way to make progress is to study individual causes of death. It seems unlikely that the same set of causes will account for the association between intelligence and all types of mortality, so exploring whether and why intelligence is associated with specific causes of death can make a start in the discovery of the different sets of mechanisms involved. The first peer-reviewed dataset that was used to examine individually-tested intelligence and mortality was the Australian Vietnam Veterans Study, and it noted that there were associations between intelligence and external causes of death, for example motor vehicle accidents (O'Toole & Stankov, 1992). There are also specific associations between intelligence test scores from early life and deaths from, for example, cardiovascular disease (e.g., Batty, Deary, Benzeval, & Der, in press; Batty,

Mortensen, Nybo, Andersen, & Osler, 2005; Batty, Shipley, Mortensen, Gale, & Deary, 2008; Lawlor, Batty, Clark, Macintyre, & Leon, 2008; Silventoinen, Modig-Wennerstad, Tynelius, & Rasmussen, 2007), vascular dementia (McGurn, Deary, & Starr, 2008), accidents (Batty, Gale, Tynelius, Deary, & Rasmussen, 2009), suicide (Gunnell, Magnusson, & Rasmussen, 2005), and homicide (Batty, Deary, Tengstrom, & Rasmussen, 2008; Batty, Mortensen, Gale, & Deary, 2008). Whether intelligence is related to death from cancers of different types is equivocal: some of the smaller studies found links with lung and or stomach cancer deaths (Batty, Mortensen, Gale, Shipley, Roberts, & Deary, 2009; Hart, Taylor, Davey Smith, Whalley, Starr et al., 2003). By far the largest study found significant but slight evidence of low intelligence as a risk factor for stomach (where any protective effect of intelligence was seen only in the highest intelligence tertile), but not lung or other cancer death; it also found that high intelligence was a risk factor for skin cancer (Batty, Wennerstad, Davey Smith, Gunnell, Deary, Tynelius, & Rasmussen, 2007).

One of the largest hazard ratios found—in the Swedish Conscripts Study—was that a standard deviation disadvantage in intelligence at conscription was associated with 5.82 times the risk of death by accidental poisoning (Batty, Gale, Tynelius, Deary, & Rasmussen, 2009). The link with homicide was similar, almost five-fold (Batty, Deary, Tengstrom, & Rasmussen, 2008). We shall discuss below some ideas about mechanisms of association for specific causes of death such as those from cardiovascular disease, but those suggested for homicide were rather different. Ideas put forward were that people with lower intelligence might live in more dangerous neighbourhoods; that there might be a social milieu effect such that people tend to live among people with similar levels of intelligence and that people with lower intelligence are more likely to commit homicide; people with lower intelligence might

have poorer risk perception; and people with lower intelligence might be less adept in using verbal ability to effect conflict resolution.

2.3. Illness precedes death

People who become ill are more likely to die. So, it is of interest to see if part of the intelligence-mortality association can be accounted for by low intelligence predisposing to illnesses. It is also part of cognitive epidemiology to inquire, for its own sake, whether intelligence is associated with illness states. Several studies have examined intelligence-illness associations. Lower intelligence is associated with, for example, a greater risk of developing cardiovascular disease (e.g. Hart, Taylor, Davey Smith, Whalley, Starr et al., 2004). A study of the National Longitudinal Survey of Youth 1979 provides an unusually large range of illness states (Der, Batty, & Deary, 2009). The Armed Forces Qualification Test was administered to subjects at ages 16-22, and subjects were followed up to about age 40. People with lower intelligence at baseline were more likely to have received physician's diagnoses of chronic lung disease, heart problems, hypertension, diabetes, and arthritis/rheumatism. They were also more likely to have self reports of eye trouble (not glasses), ulcers, severe tooth or gum problems, epilepsy or fits, stomach or intestinal ulcers, lameness or paralysis or polio, frequent problems sleeping, frequent headaches or dizzy spells or fainting, chest pain or palpitations, anaemia, leg pain or bursitis, foot and leg problems, asthma, depression or anxiety, and kidney or bladder problems. Only a few self-reported illness states were reported more frequently in people with higher intelligence: chronic colds or sinus problems, high cholesterol, thyroid trouble or goitre, and tumour or growth or cyst. It is possible that some of these latter associations could be due to people with higher intelligence being more likely to understand the terms (e.g. thyroid), be aware of the states (e.g. thyroid, cholesterol), and be monitoring them (e.g. cholesterol, tumours).

Separately, the Swedish Conscripts Survey, the Vietnam Experience Study, and the Dunedin Study between them examined intelligence in early life and found significant and strong prospective associations with the following psychiatric states: schizophrenia, other non-affective psychosis, mood disorders, neurotic and somatoform disorders (including generalised anxiety disorder), adjustment disorders, post-traumatic stress disorder, personality disorders, alcohol-related disorders, other substance use disorders (Gale, Batty, Tynelius, Deary, & Rasmussen, in press; Gale, Deary, Boyle, Barefoot, Mortensen, & Batty, 2008; Gale, Hatch, Batty, & Deary, 2009; Koenen, Moffitt, Roberts, Martin, Kubzansky et al., 2009).

2.4. Your life in your hands

One originally-suggested (Whalley & Deary, 2001) cause for the link between intelligence and illness and death was that people with higher intelligence might have better health behaviours. There is growing evidence that this is the case, although the studies linking early life intelligence and health behaviours are often based on subjects too young yet to have accrued many deaths. Therefore, intelligence-health behaviour associations are found, but it is largely not yet known if these mediate associations with later chronic illness and death. People with higher early life intelligence are more likely, decades later to avoid smoking; eat more fresh fruit and vegetables, wholemeal bread, white meat, and fish; cook with vegetable oil; take more exercise; and comply in the long run with prescribed medications. People with lower intelligence in early life are more likely, decades later, to eat chips, cakes and biscuits, and white bread; cook with animal fats; binge drink and have hangovers; and be obese or overweight. The original reports of these findings may be found in the following journal articles: Batty, Deary, & Macintyre (2006); Batty, Deary, Schoon, & Gale (2007a,b);

Chandola, Deary, Blane, & Batty, 2006); Gale, Deary, Schoon, & Batty, 2007; Hemmingsson, Kroebel, Melin, Allebeck, & Lundberg, 2008; Taylor, Hart, Davey Smith, Starrm Hole et al., 2003). However, some types of alcohol problems were more common in people with higher intelligence in a UK cohort born in 1970 (Batty, Deary, Schoon, Emslie, Hunt, & Gale, 2008).

Some of these factors, and the association between childhood intelligence and hypertension in adulthood, led to the idea that the metabolic syndrome might mediate the association between intelligence and mortality. The metabolic syndrome is a complex of high blood pressure, overweight/obesity, high blood lipids, and poor glucose metabolism. In the Vietnam Experience Study one standard deviation advantage in intelligence at enlistment was associated with 14% reduced risk of the metabolic syndrome in midlife (Batty, Gale, Mortensen, Langenberg, Shipley, & Deary, 2008). Moreover, this association attenuated the intelligence-cardiovascular disease mortality association by about a third in the same sample. This is a rare example of an intermediate illness state that appeared to have some mediating power between intelligence and death from a specific cause. The intelligence-metabolic syndrome association was replicated in the UK's National Survey of Health and Development (the 1946 British Birth Cohort) (Richards, Black, Mishra, Gale, Deary, & Batty, 2009).

Parents also have their offspring's lives in their hands. In two British birth cohorts intelligence in childhood was a significant predictor of which mothers would smoke during pregnancy (Gale, Johnson, Deary, Schoon, & Batty, 2009).

2.5. *Well assembled?*

Another possible explanation for the association between intelligence and death was the idea that intelligence might be a marker for system integrity (Whalley & Deary, 2001). That is, intelligence could be an indicator of a body that has generally been put together well, and which can respond well to stressful challenges from the environment. This has not been easy to test. Tests of this idea have tended to use other variables as possible indicators of system integrity and to see if they can account for the intelligence-mortality association. One such variable was reaction time. It was found that reaction time could account for the majority of the association between intelligence and mortality in a sample in which intelligence and mortality had been assessed at age 55 years and mortality surveillance took place up to age 70 years (Deary & Der, 2005). The association between reaction time and mortality is now replicated (Shipley, Der, Taylor, & Deary, 2006, 2007, 2008).

Another study used physical co-ordination of the upper and lower limbs as a possible indicator of system integrity. This study used the data from the 1958 and 1970 British Birth Cohorts (Gale, Batty, Cooper, & Deary, 2009). They had taken intelligence tests in childhood at age 11 and 10, respectively. They also took tests of co-ordination at these ages. Intelligence was significantly associated with co-ordination in both groups. Also, in both groups, intelligence and co-ordination from childhood were associated with illness outcomes at about age 30: psychological distress, poor self-rated health, and obesity. However, neither attenuated the effect of the other's association with the health outcomes, which fails ultimately to support the system integrity hypothesis. Intelligence and physical co-ordination appear to be largely independent predictors of these health states, not indicators of the same latent trait.

2.6. *"It's all just SES... or education... or income..."*

One idea that was mooted early on in cognitive epidemiology was that the association between intelligence and mortality and other illness outcomes could be explained by indicators of socioeconomic status, either from childhood or adulthood (Whalley & Deary, 2001). There is little attenuation of the intelligence-mortality association after adjusting for childhood socioeconomic status (e.g. Batty, Wennerstad, Davey Smith, Gunnell, Deary et al., 2009). There is greater attenuation after adjusting for the person's own socioeconomic status in adulthood (e.g. Weiss, Gale, Batty, & Deary, 2009). However, it should be asked what these latter findings mean. Indeed, there might be mediation of the effects of intelligence via more education, and/or the obtaining of more professional work with more income and entry to safer environments, as suggested by Whalley & Deary (2001). But it should also be asked—as Gottfredson (2004) did—if intelligence is the fundamental variable here: causal to a greater or lesser extent to both health and adult socioeconomic status, with the latter having less causal role than the statistical adjustments might indicate.

The statistical adjustments can also be done the other way around. In the West of Scotland Twenty-07 study intelligence measured at age 55 had a strong-to-complete ability to account for the associations between five different indicators of socioeconomic status and six health outcomes (Batty, Der, Macintyre, & Deary, 2006). Intelligence could account for all of the associations between the two most objective health outcomes—total mortality and coronary heart disease—and the two most used indicators of adult socioeconomic status—a person's occupation and education. Also, in the Vietnam Experience Study, intelligence was better than a basket of traditional risk factors (hypertension, obesity/overweight, smoking, cholesterol, blood sugar, lung function, and resting heart rate) in accounting for the associations between death from cardiovascular disease and army income, occupational prestige, mid-life income, and education (Batty, Shipley, Dundas, Macintyre, Der et al.,

2009). Again, though, it should be emphasised that socioeconomic factors can have a powerful attenuating effect on intelligence-mortality/illness associations. For example, education and income entirely mediated the association between intelligence and mortality in the Vietnam Experience Study (Weiss, Gale, Batty, & Deary, 2009). Future studies should examine whether indicators of socioeconomic status are mediators of the influence of intelligence, or if they are largely surrogates for intelligence. Of course this is not an either-or situation. It could be that intelligence launches the person into the better environment, but it is the better environment that causes the health benefits.

2.7. Look behind you! #1

Sometimes, cognitive epidemiology findings can appear when an association in the other direction has been looked for; that is, when examining for effects of health-related variables on cognition. For example, the Lothian Birth Cohort 1936 sample of the Scottish Mental Survey of 1947 was used to study the influence of the blood inflammatory marker C-reactive protein on cognitive ability at age 70 (Luciano, Marioni, Gow, Starr, & Deary, 2009). There was a replication of the association, similar in effect size to that which had been found by others. However, in the same sample, the association between mental ability at age 11 and C-reactive protein at age 70 was just as strong as the contemporaneous correlation between cognitive ability and C-reactive protein at age 70. Moreover, adjusting for cognitive ability at age 11 reduced the latter correlation almost totally, and to non-significant levels. Thus an exploration of a possible contribution to cognitive ageing became largely a new discovery in cognitive epidemiology.

Similar types of findings are that lung function (forced expiratory volume in 1 second) and blood pressure in later life are associated with childhood intelligence (Deary, Whalley, Batty

& Starr, 2006; Richards, Strachan, Hardy, Kuh, & Wadsworth, 2005; Starr, Taylor, Hart, Davey Smith, Whalley et al., 2003). Both of these factors are variables which have been explored as possible contributors to cognitive ageing, so it is useful additionally to realise that there is some possible reverse causation, with prior intelligence being associated with variation in them in later life.

2.8. ...and what about personality?

Intelligence is not the only individual differences measure that is associated with mortality. There is growing evidence that the personality traits of conscientiousness and neuroticism are too. In a representative sample of the Scottish Mental Survey of 1947, a personality variable called dependability (similar to conscientiousness) and intelligence—both estimated in childhood—were associated with mortality, and had additive but not interactive effects (Deary, Batty, Pattie, & Gale, 2008). Children who fell in the bottom halves both of ratings for dependability and scores for intelligence in childhood were about two-and-a-half times as likely to have died up to age 66 than those in the upper half of both distributions. In the Vietnam Experience Study neuroticism and intelligence were both predictors of death to middle age (Weiss, Gale, Batty, & Deary, 2009). They interacted: intelligence had a greater influence on mortality at higher levels of neuroticism, and neuroticism had greater effects on mortality at lower levels of intelligence.

3. Challenges for cognitive epidemiology

3.1. Keeping intelligence in people's minds

It is important not to become complacent and think that intelligence will naturally be included when scientists from different disciplines consider the causal nexus of social factors on health. For example, recent papers considering social mobility (Bartley & Plewis, 2007) and

education (Lleras-Muney, 2005) as key influences on health outcomes failed to consider the role that intelligence plays in both of these.

3.2. Challenge the ‘this variable is environmental’ assumption

Many studies make implicit assumptions about the aetiology of individual differences in a variables. Especially, it is common in epidemiology to see the assumption that certain variables are caused by environmental factors. For example, height is frequently used as an indicator of childhood social deprivation (Davey Smith, Hart, Upton, Hole, Gillis, Watt, & Hawthorne, 2000), despite its heritability being very high (Silventoinen, Sammalisto, Perola, Boomsma, Cornes et al., 2003). Data about the sources of variance that contribute to a variable should be used to support the assumptions. For example, data from the Generation Scotland: Scottish Family Health Study found that education, income, smoking, and fruit and vegetable consumption all had substantial heritability, genetic correlations with general intelligence, and bivariate heritability (Luciano et al., in submission). The situation might be even more complex than this; in a Danish twin sample variation in self-rated health was moderated by education level, with additive genetic variation being much lower at higher levels of education (Johnson, Kyvik, Mortensen, Skytthe, Batty, & Deary, in press).

3.3 Look behind you! #2

Developing the above idea, there are some variables that have been suggested as factors from early life that might be partly causal to childhood intelligence differences and to later ill health, and perhaps mortality. These include maternal smoking, breast feeding and birth weight. However, using data from the National Longitudinal Survey of Youth 1979, it was found that the associations between all three of these variables and childhood intelligence were almost entirely accounted for by maternal intelligence (Batty, Der & Deary, 2006;

Deary, Der, & Shenkin, 2005; Der, Batty, & Deary, 2006). This raises the possibility that these variables might, to some considerable extent, be picking up the genetic association between mother and child.

3.4 Behavioural and molecular genetic studies

Developing the above, it will be important to conduct twin and adoption studies in samples that have intelligence tested and are then followed up for mortality. This will help to reveal the degree to which there is shared genetic and environmental aetiology in intelligence and mortality and illness. Molecular genetic studies will also be useful in finding specific genetic variants that tie intelligence and mortality influences together.

3.5. Statistical versus mechanistic mediation

In attempts to explain the association between intelligence and mortality and illness care needs to be taken not to draw over-strong conclusions from statistical adjustments. For example, consider the following statement from Marmot and Kivimaki (2009) in the Editorial that was referred to at the beginning of this piece,

The fact that IQ drops out of the predictive model when education and income are included... suggests we are not looking primarily at a direct IQ effect. Social inequalities seem not to be a problem of genetic predisposition and deficient brain information processing at an individual level

This might be correct, but it will take more than statistical adjustment to establish that this is the case. It is important to engage with lifecourse epidemiologists, so that assumptions and even biases from different viewpoints can be balanced and criticised, and extended. Again referring to Marmot and Kivimaki's (2009) piece for an epidemiologists's view, they

sketched three possibilities for intelligence-health associations that a differential psychologist might wish to extend,

intelligent people knowing how to look after themselves

IQ is a determinant of social and economic success in life

early conditions, ranging from foetal programming to parental interest in child's education, influence both IQ and subsequent risk of disease

Again, any or all might be correct and contribute to the mechanistic story, but the clear leaning is toward environmental explanations; possible shared genetic contributions to intelligence and health are not mentioned, for example. This can be broadened and, perhaps most easily, by intelligence experts working alongside epidemiologists.

3.6 Engage with lifecourse epidemiologists, evolutionary biologists, and policy makers

For cognitive epidemiology to progress in explanatory power and relevance, practitioners must collaborate with those who can offer explanatory mechanisms, and those who can translate findings into practice. There is growing interest from theoretical and experimental biologists with regard to lifecourse epidemiology, and their work is relevant to intelligence differences and its links with mortality, illness and health status. Factors and processes such as inflammation, oxidative stress, and immunity are obvious candidates as mediators between intelligence and health outcomes. Such well-recognised biological mechanisms should be part of the picture in exploring the causes of intelligence-health associations. Medical sociologists and epidemiologists are already engaging with policy ideas. Alongside exploring mechanisms, intelligence researchers can make a contribution to answering how health might be made better for all, especially those whose health outlook is poor. The application of

cognitive epidemiology will be in finding out what brighter people do that is beneficial to their health and making that information available for translation from research to practice.

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