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Intelligence and personality as predictors of illness and death:
How researchers in differential psychology and chronic disease epidemiology are collaborating to understand and address health inequalities

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Abstract

We describe the research findings that link intelligence and personality traits with health outcomes: health behaviors, morbidity, and mortality. The former field is called cognitive epidemiology, and the latter is known as personological epidemiology. However, intelligence and personality traits are the principal research topics studied by differential psychologists, and so the combined field might be termed differential epidemiology. The importance of bringing this field to wider attention lies in the facts that: the findings overviewed here are relatively new, often known neither to researchers or practitioners; the effect sizes are on a par with better-known, traditional risk factors for illness and death, so they should be broadcast as important; mechanisms of the associations are largely unknown, so they must be explored further; and the findings have yet to be applied, so we write this to encourage diverse interested parties to consider how this might be done.

To make the work accessible to as many relevant researchers, practitioners, policy makers and laypersons as possible, we first provide an overview of the basic discoveries regarding intelligence and personality. In both of these areas we describe the nature and structure of the measured phenotypes. Both are well established even though we recognize that this is not always appreciated beyond the cognoscenti. Human intelligence differences are well described by a hierarchy that includes general intelligence (*g*) at the pinnacle, strongly correlated broad domains of cognitive functioning at a lower level, and specific abilities at the foot. The major human differences in personality are described by five personality factor that attract wide consensus with respect to their number and nature: neuroticism, extraversion, openness, agreeableness and conscientiousness. As a foundation for the health-related findings, we provide a summary of the research which shows that intelligence and personality differences are: measured reliably and

validly; stable traits across many years, even decades; substantially heritable; related to important life outcomes. Cognitive and personality traits are fundamental aspects of the person that have relevance to life chances and outcomes; and here we discuss health outcomes.

There is an overview of the major and mostly recent research that has studied associations between intelligence and personality traits and health outcomes. These outcomes include mortality from all causes, specific causes of death, specific illnesses, and other health outcomes including health-related behaviors. Intelligence and personality traits are significantly and substantially (by comparison with traditional risk factors) related to all of these. The studies we describe are unusual in psychology: mostly they are larger in sample sizes (typically thousands of subjects, and sometimes around one million), the samples are more representative of the background population, the follow-up times are long (sometimes many decades, almost the whole human lifespan), and the outcomes are objective health measures (including death) not just self-reports. In addition to the associations, possible mechanisms for the associations are described and discussed, and some attempts to test these are illustrated. It is relatively early in this research field, and so much remains to be done here.

Finally, some preliminary remarks are made about possible applications. These are made in the knowledge that the psychological predictors addressed are somewhat stable aspects of the person, with substantial genetic causes. Nevertheless, the view taken is that this does not preclude useful interventions that can make wider appreciation of differential epidemiology a useful component of interventions to improve individual and public health. Intelligence and personality differences are the loci of later health inequalities; to the extent that it is possible, the eventual aim of cognitive and personological epidemiology is to reduce or eliminate these inequalities and

provide information that helps people toward their own optimal health through the life course. We offer up these findings to a wider audience so that: more associations will be explored; a better understanding of the mechanisms of health inequalities will be produced; and inventive applications will ensue based on what we hope will become to be seen practically useful knowledge.

1. Intelligence and personality traits

Humans differ from each other. Not just in physical characteristics, like sex, height, weight, hair and eye color, facial attractiveness, and so on. People also differ in their psychological make-up. This monograph addresses a research area in the fields of health psychology and psychosomatic medicine, namely how prominent human individual differences in the psychological traits of intelligence and personality are associated with death, illness, and other aspects of health such as health behaviors (e.g., smoking and diet, including alcohol intake). Before that, for the readers who are not psychologists working in these fields, we describe and explain the nature of these traits. Similarly, for readers who are not epidemiologists, we also introduce some key concepts in that field. Both intelligence and personality are topics within psychology which, from the outside, could seem to be mired in controversy and disagreements about even the most basic facts. This is far from the truth of the matter. In both intelligence and personality research there are core discoveries and knowledge about them that is buttressed by large bodies of data. In the account presented here we have tried to limit what we claim only to those findings which are empirically well established.

1.1 Structure and nomological network of intelligence

People differ with respect to the efficiency with which their brains operate, and this is the domain of psychologists interested in intelligence differences. Given that intelligence differences are to be an important part of this piece, it is important to understand how they are structured and how they affect other aspects of people's lives. For those wishing a more extended but accessible to guide to intelligence we recommend a short introduction to this topic by Deary (2001) and the consensus document provided by Neisser et al. (1996).

1.1.1 The structure of intelligence differences. The key question here is how many types of intelligence one needs to consider in studying people's differences in intelligence, and their contribution to health differences. In the past, psychologists differed with respect to whether just one 'general intelligence' existed—people were just generally smart or not so smart—or whether there were many different types of intelligences, and that some people were good at some types of mental task and some people were good at others. Everyday experience offers some support for both options. By observation, there are people who seem mentally to excel at many things. On the other hand, some people seem to have obvious cognitive strengths, with some of their abilities seeming stronger than others. Consider, for example, the mental task of trying to multiply two numbers using mental arithmetic. Why are some people better than others at this type of task? Is it because some people are more intelligent than others, and that this applies to all mental work? Is it because that some people are better than others at all types of numerical ability, but not necessarily better at, say, verbal reasoning or spatial ability? Is it because some people are better than others at the specific task of multiplication, but not necessarily better at other number tasks or mental work more generally? The answer is that all three are correct to some extent, which we now explain.

When a diverse range of mental tests is performed by a large group of people, the associations among the test scores form a very well-replicated pattern. The correlations among the test scores are universally positive. That is, no matter what type of mental work the tests involve, the general rule is that people who do well on one type of mental task tend to do well on all of the others. This is the phenomenon known as general intelligence—or general mental ability, or general cognitive ability—and it is usually shortened to just a lowercase italicized g: *g*. It was discovered by Charles Spearman in 1904, has been replicated in every database—several hundreds of them

(Carroll, 1993)—since then, and accounts for about half of the differences among people in their mental capability. There is also a clear finding that some types of test tend to have higher correlations among themselves than they do with others. For example, verbal test scores generally correlate more highly among themselves than they do with spatial ability tests or mental speed tests, each of which also generally have higher associations within its own type of test than with different types of test. This is the phenomenon that has led to the idea of multiple intelligences. This was first suggested—as a challenge to Spearman's idea of general intelligence—by Thurstone (1938), and more recently in the popular Multiple Intelligences theory of Howard Gardner (1983). The problem with these theories is that they never accorded with data from real people: the supposedly separate intelligences typically had positive correlations among themselves and people who did well on them also tended to do well on the others, thus re-stating Spearman's *g* (Johnson & Bouchard, 2005; Visser, Ashton, & Vernon, 2006). The fact is that there are separable domains of cognitive ability—such as reasoning, spatial ability, memory, processing speed, and vocabulary—but they are highly correlated (Deary, Penke, & Johnson, 2010). People who do well in one area also tend to do well in the others, a phenomenon which is explained by *g*. However, apart from *g* some of the differences in people's mental capabilities can be accounted for by differences in these domains; but not very much. Indeed, apart from *g*, the main types of mental capabilities in which people differ are those which are specific to each mental task. This results in what is known as the hierarchical model of intelligence differences. This model fits every data set that has been gathered pretty well and explains that people differ in three types of capability: general intelligence, broad domains of mental capability, and specific mental abilities (which includes error and occasion-specific variance), with the first and last explaining most of the differences. The three-level hierarchy was suggested in the first half of the 20th century, but was consolidated mostly clearly by Carroll (1993), and has been replicated—

with some revisions to the domain-level intelligences—in large data sets since then (Johnson & Bouchard, 2005). Importantly, it has also been shown clearly that the *g* factor that results from different test batteries ranks people in almost identical ways (Johnson, te Nijenhuis, & Bouchard, 2008).

1.1.2 Intelligence's nomological network. The three-level hierarchical model of intelligence differences has been useful both for finding out how intelligence is associated with important aspects of people's lives, and the causes of differences in intelligence. Indeed, for most of these types of study, the prime source of interest has been *g*. As will be seen below, with respect to its effects on health, it is *g* that seems to be the important factor, and not the more specific cognitive abilities. And, when individual tests are used in cognitive epidemiology, they appear to be associated with health as a result of their tapping *g*. Some tests seem to be especially good at calling on general intelligence for their performance; this includes nonverbal reasoning tests like Raven's Progressive Matrices, and broad IQ-type tests like the Moray House Test series and the Alice Heim test series (see Deary & Batty, 2007). Ideally, in health research, one would hope to see people being given a diverse battery of mental tests from which a *g* factor score would be calculated for each person from, for example, the Wechsler Adult Intelligence Scale-III (Wechsler, 1997), the Kaufman Adolescent and Adult Intelligence Test (Kaufman & Kaufman, 1993), or the Stanford Binet Intelligence Scale[1] (Thorndike, Hagen, & Sattler, 1986). Sometimes this is done but, just as frequently people have been given a single test which has a substantial *g* loading.

[1] An early version of the Stanford-Binet Scale was used to validate the Moray House Test which was used in the national intelligence surveys that formed the basis for some Scottish-based cognitive epidemiology studies (Deary, Whalley, & Starr, 2009, chapter 1).

Intelligence differences—the rank order of individuals—do not come and go. In healthy individuals they show considerable stability of individual differences across the life course. For example, from age 11 years to almost age 80 years, the correlation is such that around half of the variance is stable (Deary, Whalley, Lemmon, Crawford, & Starr, 2000). Stability across shorter periods of time is, of course, even higher. Intelligence differences have a major impact in people's lives. Health is a newcomer to what is called the predictive validity of intelligence. However, it has been known for many years that intelligence—especially general intelligence—strongly predicts people's success at work, in education, and in their social lives; and in everyday practical decision making (Gottfredson, 1997). A large meta-analysis showed that scores on a general intelligence test were the best predictors of hiring success and in job performance (Schmidt and Hunter, 1998). In datasets with tens of thousands of people, *g* scores at age 11 very strongly predict success in national school exams five years later (Deary, Strand, Smith, & Fernandes, 2007). Intelligence in childhood and early adulthood is also an important predictor of success in obtaining social mobility, adult social status, and income (Strenze, 2007).

In addition to the impressive predictive validity of intelligence differences for life chances, it is also important to understand the origins of intelligence and quite a bit is known (Deary, Penke, & Johnson, 2010). Genetic factors account for a substantial proportion of the individual differences in intelligence (Deary, Johnson, & Houlihan, 2009). This applies to individuals within groups, and not to the origins of any between-group differences (Neisser et al., 1996). The principal genetic contribution is to differences in the *g* factor. The proportion of intelligence differences explained by genetic differences rises from low levels (20% to 30%) in early childhood, to levels as high as 70% to 80% in young and middle adulthood, with possibly some slight decline in old age. There is some evidence that genetic influences on intelligence, at least in childhood, are

stronger in more affluent by comparison with more deprived socioeconomic groups (e.g. Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003). As yet, no variants of individual genes have been discovered that underlie this high heritability, apart from a small contribution from genetic variation in the gene for apolipoprotein E (*APOE*) which explains about 1% of the variation in some mental ability in old age (Wisdom, Callahan, & Hawkins, in press), and possibly even smaller contributions from *COMT* and *BDNF* genes (Deary, Penke, & Johnson, 2010). There is a well-established modest correlation between intelligence and brain size—based on structural brain imaging in healthy people—but its cause is not known (McDaniel, 2005). Various types of functional brain scanning studies strongly suggest that more intelligent brains are also more efficient in how they process information (Neubauer & Fink, 2009).

General intelligence declines with age, and there are probably some additional age-related declines in the cognitive domains of memory and processing speed (Salthouse, 2004; Hedden & Gabrieli, 2004; Schaie, 2005). However, aging raises an important distinction between two types of intelligence: fluid and crystallized (Horn, 1989). The types of cognitive ability that show a mean age-related decline are usually called aspects of fluid intelligence. They are assessed using tests that require active engagement with information, especially that which is novel and abstract, and completed under time pressure. Fluid intelligence involves working things out mentally on the spot. On the other hand, crystallized intelligence shows little age-related decline, and some tests of these capabilities even survive in the early stages of dementia (McGurn et al., 2004). Crystallized intelligence tests typically assess things like vocabulary and general knowledge, which involve the retrieval of well-established knowledge. Indeed, this type of knowledge is so stable that some tests are used in old age as highly accurate estimates of peak prior intelligence: a way at getting back to a person's high-water mark of intelligence before the aging process

started. These tests include the National Adult Reading Test (in the UK), and the Wechsler Test of Adult Reading (more widely). The decline in intelligence with age brings with it decreased everyday capability and independence (Kirkwood, Bond, May, McKeith, & Teh, 2008) and—especially in the context of aging societies—has meant that there is an economic mandate to find out why some people decline in intelligence more than others (Hendrie et al., 2006). Causes have been found in genetic variation (e.g. *APOE*), illness, biomarkers, physical fitness, brain structure and function, and demographic and social factors, including socioeconomic adversity (Deary et al., 2009). This means, of course, that there are additional causes of intelligence differences in old age when compared with youth.

The topic of intelligence differences is perennially controversial. We submit the above brief summary as mainstream opinion within the differential psychology research community, including the reservations posed by Gardner (1983) and Turkheimer et al. (2003). However, it should be stated that there are additional influential contrary views, and some findings that challenge aspects of the account. For example, the Flynn (1987; Dickens & Flynn, 2001) effect—whereby it is well attested that scores on standard intelligence tests rose throughout a substantial proportion of the 20th century, with those born in the later cohorts scoring better—suggests that IQ-type test scores are not immutable to environmental influences. And Nisbett (2009) has queried aspects of the twin and family designs used to derive heritability estimates and emphasized the possibility that cultural differences might generate differences in intelligence. However, these data and ideas should be understood with respect to their implications. For example, the Flynn effect, as recognized by the author himself, does cast doubt on the reliability and validity of intelligence differences found within a cohort. And when, for example, Nisbett suggests that parenting practices might be the origin of 'environmentally'-caused intelligence

differences, it behooves him to examine whether such practices could be caused, at least in part, by differences in parental genotype (Hunt, 2009). It is our opinion that the summary of major facts about intelligence given above does not alter as a result of these writers contributions. Again, because the topic of intelligence can be controversial, it is important to have access to unbiased accounts. Once more, we recommend the American Psychological Association's consensus overview for an even-handed summary of many important topics in intelligence differences (Neisser et al., 1996).

Of special importance for this piece is the fact that there is sometimes reverse causation between intelligence and its purported causes. That is, when a correlation is found between some risk factor and intelligence in old age, the usual assumption is that the researcher has discovered a contribution to cognitive aging. However, with the right database, we can check the reverse, i.e., that long-standing differences in intelligence might, instead, have given rise to differences in the risk factor. That is not cognitive aging, it is cognitive epidemiology. We shall see an example of this with intelligence and C-reactive protein in old age (Luciano, Marioni, Gow, Starr, & Deary, 2009). A third possibility is that there is some prior factor or set of factors that has caused differences in both the risk factor and intelligence, and that any correlation between them is spurious, and just a reflection of the fact that they both have an association with something more fundamental. Epidemiologists refer to this as confounding, and it is a perennial problem: it is discussed further in section 5.3.

1.2 Structure and nomological network of personality traits

In addition to intelligence, or cognitive abilities, people differ with respect to personality, which encompasses several stable traits related to behavior, affect, interpersonal interactions, and

cognitive dispositions. When you are asked, “what’s he like?” something physical might be intended. But, more often, the request is for a psychological description. Is the person typically generous or mean, irritable or placid, shy or outgoing? These descriptions and guesses about people’s general reactions and feelings are the phenomena that inspire personality trait theories. There are no given categories for classifying people into psychological types, and there is no *a priori* basis on which to allocate a given number of major traits. The major dimensions of personality along which people differ have emerged clearly only in the last few decades, after much large-scale psychometric research. For those wishing a more extended but accessible guide to personality traits, we recommend the short book by Nettle (2001). A more advanced account of personality trait research is provided by Matthews, Deary, and Whiteman (2009).

1.2.1 The five personality factors and their measurement instruments. By about 1990, psychologists were converging on a consensus that there might be only five principal personality traits (Matthews, Deary, & Whiteman, 2009). Personality psychologists often refer to these traits as the Big Five, or the Five-Factor Model. The arrival and broad acceptance of the Five-Factor Model of personality is a major scientific advance in the understanding of human psychology. For many decades of the 20th century, two prominent theorists in the personality trait world were Hans Eysenck and Raymond Cattell. Eysenck’s (1916-1997) theory was that there were three main personality traits, called neuroticism, extraversion, and psychoticism. To measure these, he devised and revised the Eysenck Personality Questionnaire (Eysenck & Eysenck, 1975). Cattell’s (1905-1998) theory was that there were 16 main personality traits, narrower in psychological content than Eysenck’s. He devised and revised a questionnaire called the 16PF (Cattell, Eber, & Tatsuoka, 1970). There were many more systems, each offering different numbers of personality traits with different names. For anyone wanting the true story of human personality it was not to

be had. However, apparently different trait theories had more in common than had been superficially obvious. For example, the overlaps in coverage of Cattell's, Eysenck's, and the Five-Factor Model's traits are substantial (e.g., Aluja, Garcia, & Garcia, 2002). The history of, and convergence around, the currently-dominant Five-Factor Model of personality traits has been described by Digman (1990, 1996).

A brief sketch of each of the five traits in the Five Factor Model is as follows. We shall rely on the most common names of each factor, though others have been used elsewhere.

Neuroticism: a tendency to feel anxiety and other negative emotions versus a tendency to be calm and emotionally stable.

Extraversion: a tendency to be outgoing and to take the lead in social situations versus a tendency to stay in the background socially and to be timid.

Conscientiousness: a tendency to be organized and to follow rules versus a tendency to be somewhat careless, disorganized and not to plan ahead.

Agreeableness: a tendency to be trusting and deferential versus a tendency to be distrustful and independent.

Openness to Experience: a tendency to be open to new ideas and feelings and to like reflection versus shallowness and narrow in outlook.

Such brief sketches do not cover the richness of personality traits. The Five-Factor Model's personality traits are broader. They describe general tendencies in people's behaviors, feelings, attitudes and thinking that are not well-suited for a single phrase or sentence. Table 1 is taken from the summary sheet from the most widely-used brief questionnaire for the Five-Factor Model: The NEO-Five Factor Inventory (Costa & McCrae, 1992). It is used to indicate to the person being tested roughly what their score was and what it means in practical terms. Recall that

each of the traits has a normal distribution in the population, and a long range of scores from very high to very low, and that what is being described here is only each extreme and the middle. In the measurement scheme devised by Costa and McCrae (1992), in their full Revised NEO Personality Inventory, each of the five factors has six facets. Facets are psychologically narrower aspects of the broad traits (see Table 2). They are strongly correlated with each other within a trait. Much of the application of personality traits to health outcomes is done using the broad factors (sometimes called domains, dimensions, or traits, so do not be confused by variation in the terminology), but some is done using the facets. In each case, the way to think of personality traits is like measuring rulers. They are scales that measure aspects of human personality. Most people will have a middling score with fewer and fewer people as the scores become more extreme; just like height and weight, for example.

Thus, there is a general consensus, though there are detractors (e.g., Eysenck, 1992; Lee, Ogunfowora, & Ashton, 2005), that five broad dimensions or factors underlie and describe individual differences in non-cognitive traits (Digman, 1990). While there was early skepticism about the reality or validity of personality traits in a general sense (Mischel, 1968), there have since been many findings supporting their status as real and important psychological variables. In what follows we briefly recount some of the major issues that personality psychologists address with regard to the validity of personality traits.

1.2.2 The nomological network of personality traits. There is ongoing research which addresses whether the five factors are too few. For example, some argue that honesty-humility is a sixth trait, important to humans and separate from the five factors (Ashton & Lee, 2005; Lee & Ashton, 2006). Others suggest that there are yet more traits that could be important. There is also

a parallel tendency to look for higher-order factors which supersede the Five-Factor Model. Noting some correlations among the five factors, Digman (1997) and later DeYoung (2006) emphasized two broad higher-order traits of stability and plasticity, which were thought to be important biological factors. Similarly, some have examined the correlations among the five personality traits and argued for a single, general personality factor (Musek, 2007). However, there is considerable evidence that the general personality factor is a methodological artifact (see, e.g., Bäckström, Björklund, & Larsson, 2009). Thus, it is our evaluation that the five personality factors should be considered separately with respect to health—not least because some appear to predict health outcomes whereas other do not—and that, unlike general intelligence, there is not such a compelling case to address general personality. For the most part, the suggested revisions to the Five-Factor Model are not large. The Five-Factor Model (or, at least, four of its factors, with openness as a partial exception) does account for variation in abnormal as well as normal personality variation (Markon, Krueger, & Watson, 2005).

Some or all of the five factors of personality are found in different language groups and cultures, making them universally applicable to health outcomes. The Revised NEO Personality Inventory has been translated into many different languages. In 26 cultures, many non-Western, McCrae (2001) found very similar personality structures for translations of the NEO-Personality Inventory. McCrae, Terracciano, and 78 other researchers (2005) asked 12,000 students in 50 cultures to rate another person's traits and found concordance with the American self-report structure. De Raad et al. (2009) examined 14 trait taxonomies in 12 languages and found especially strong replication for the five factor traits of extraversion, agreeableness, and conscientiousness, though less so for emotional stability (the reverse of neuroticism) and intellect/imagination (similar to openness to experience). There is especially good agreement

across some languages. For example, English and German have very similar five factor structures in their lexicons (Saucier & Ostendorf, 1999).

Health outcomes research is predicated on personality trait ratings being relatively stable aspects of the person and not transient states, such as mood (e.g., anxiety and depression). Stability has two aspects: the stability of mean levels, and the stability of individual differences. The five factors are mostly stable throughout adulthood, showing only slight mean declines in neuroticism, extraversion, and openness to experience, and slight increases in agreeableness and conscientiousness (McCrae & Costa, 2003; Roberts & DelVecchio, 2000; Roberts, Walton, & Viechtbauer, 2006). A review of over 152 longitudinal studies with over 3000 correlation coefficients found that trait stability of individual differences increased from childhood to adulthood, rising from about 0.3 to over 0.7 (Roberts & DelVecchio, 2000). This supported earlier research with traits from the Five-Factor Model (Costa & McCrae, 1994) and Eysenck's factors (Sanderman & Ranchor, 1994), which had found stability coefficients of well above 0.6, rising to above 0.8, for periods of between 6 and 30 years. The stability of individual differences among children can be high, given an appropriate measurement instrument (Measelle et al., 2005).

Most studies—including health studies—use self-ratings of traits. Therefore, it is important to establish that these ratings are indicators of objective differences, not some accident of self-misperception. This is done using consensual validation studies, in which self-ratings are compared with ratings made by people who know the subject well. McCrae et al. (2004) reviewed 19 studies of cross-observer agreement in different cultures. They concluded that people, "include trait information in their self-reports and observer ratings". Self- versus spouse-

ratings were the highest of those reported with median consensual validity coefficients of .44, .57, .51, .50, and .42 for neuroticism, extraversion, openness, agreeableness, and conscientiousness, respectively. Personality traits are also related to outcomes such as behavior (Funder & Sneed, 1993), values (De Raad & Van Oudenhoven, 2008), music preferences (Rentfrow & Gosling, 2003, 2006), the characteristics of one's work or living environments (Gosling, Ko, Mannarelli, & Morris, 2002), subjective well-being (DeNeve & Cooper, 1998; Steel, Schmidt, & Shultz, 2008), and mood as well as its disorders (Ivkovic et al., 2006; Stewart, Ebmeier, & Deary, 2005).

Understanding of personality associations is better informed when the origins of personality variation are known. There is good evidence for the biological bases of personality dimensions. Personality traits, including the five factors, are substantially heritable (Bouchard & Loehlin, 2001). Additive genetic factors account for about one third to a half of the personality trait variation among adults. This is true for all of the five factors. There are some differences between studies and some studies indicate some substantial non-additive genetic variance. Of course, even greater understanding would be possible if the contributions of individual genes to personality variation were known. However, molecular genetic studies still have found no solid associations between genetic variations and personality traits (Ebstein, 2006). Finally, as stated previously, the five dimensions of personality appear to be a human universal, being present in at least 50 Western and non-Western cultures (McCrae et al., 2005). There is even evidence that other species have analogues of some of these dimensions (Gosling, 2001) and that chimpanzees, our closest living nonhuman relative, have six dimensions, including the five found in humans (King & Figueredo, 1997).

Besides intelligence and personality traits, few psychological constructs could muster such rich information on psychometric structure and nomological networks. Findings in the field of cognitive and personological epidemiology, therefore, can be addressed with the knowledge of these background strengths.

2. Intelligence and health

Whereas there are early reports of a link between early life intelligence and total mortality (mortality from all causes of death)—some nearly eight decades old (Maller, 1933)—research attention was not maintained and, instead, the focus shifted to the role of cognition in the etiology of mental health. This may simply have reflected the prevailing understanding that cognitive function, perhaps as a measure of sub-optimal neurodevelopment, would be more likely to influence psychological rather than physical well-being. It is also the case that the incidence of several of these mental health outcomes (e.g., depression, psychosis) peak, or at least first emerge, in early adulthood, many years before major physical disease such as cancer and cardiovascular disease become common enough to facilitate study. Accordingly, investigators working on longitudinal (cohort) studies could most robustly assess the links between intelligence and mental illness simply owing to the number of events. This section will first consider the role of intelligence in the etiology of mental outcomes, including the related outcomes of intentional injury (particularly completed and attempted suicide). We shall then review links with total mortality and some of its major constituent elements (cardiovascular disease, cancer).

2.1 Mental Health

Understanding the determinants of mental health problems is important because such problems are likely to recur across the life course and lead to reduced life expectancy, perhaps because people affected by mental illness have poorer health behaviors. Whereas it is perhaps to be expected that the presence of mental illness, such as depression, elevates the risk of suicide (Miles, 1977), there is also a suggestion that sufferers experience higher rates of cardiovascular disease (Phillips et al., 2009). There is evidence from both the 1958 and 1970 British Birth Cohort studies (Gale, Hatch, Batty, & Deary, 2009) that the prevalence of self-reported psychological distress—formerly referred to as common mental disorder—in early adulthood is lower in study members who had higher intelligence test results in childhood relative to their lower performing counterparts. However, requesting an individual who is experiencing significant bouts of anxiety or depression accurately to rate their mood raises concerns over validity. One solution is to utilize a more objective measure of mental health such as data on hospital admissions/discharge or interviews with a trained mental health professional.

Well-characterized cohort studies typically reveal an association between low intelligence test scores and the risk of hospital admission for any psychological disorder by middle age. There is some support that this may point to a general susceptibility in studies which have the capacity to examine the association between measured intelligence and a range of specific, important mental health problems. In one of the most sizeable studies, conducted in a cohort of one million Scandinavian men, mental health outcomes were based on conditions serious enough to warrant in-patient care (Gale, Batty, Tynelius, Deary, & Rasmussen, 2010). Lower intelligence at about age 20 years was associated with a greater risk of eight psychiatric disorders by midlife (Gale et al., 2010): for a one SD disadvantage in intelligence—assessed using a general score derived from four diverse mental tests—there was a 60% greater risk in the hazard of being admitted for

schizophrenia, a 50% greater risk for mood disorders, and a 75% greater risk of alcohol-related disorders. In the Vietnam Experience Study (VES) cohort, very unusually, study members had an interview with a psychologist in middle age from which it was possible to ascertain both serious conditions, but also mental health problems of a more moderate nature (Gale et al., 2008). Intelligence at enlistment at a mean age of about 22 years—based on a combination of verbal and numerical tests—was inversely related to the risk of alcohol disorders, depression, generalized anxiety disorder, and post-traumatic stress disorder (Gale et al., 2008). Moreover, there was evidence that those with comorbid psychiatric problems had especially low intelligence. Elsewhere, and again using Swedish data, in a cohort of school children followed for over three decades, there was a suggestion that low cognitive ability was related to a raised risk of personality disorder, an effect that was seen across the full range of intelligence (Moran, Klinteberg, Batty, & Vagero, 2009). This graded association is a common observation in studies exploring links between intelligence and mental health, and suggests that the raised risk of disease is not merely confined to men and women with below average intelligence test scores. Notably, the associations described above typically hold after adjusting for a range of markers of socioeconomic status which included parental occupational social class and income.

2.1.1 Intentional injury. Mental illness is frequently implicated as a cause of intentional injury (Miles, 1977). With the relationships described above between intelligence and a range of mental health problems, there is therefore a degree of circumstantial evidence that intelligence may have a role in intentional injury, chiefly suicide and homicide. Intentional injury or death can be self-inflicted, for example attempted or completed suicide, or it can be the result of others' actions, including physical attack and homicide. There are inherent problems in exploring the causes of these outcomes. For suicide, for instance, attempted and completed (death) are thought to have

different etiologies; that is, the circumstances and mental processes that lead an individual to self-harm versus the taking of their own life may be very different. For example, completed suicide is more common in men, whereas non-fatal suicidal-type behaviors are more common in women and in younger individuals (Nock et al., 2008). Additionally, as a result of the low numbers of suicide and homicide cases in most cohorts relative, for instance, to chronic disease (e.g., cancer) and unintentional injury (e.g., road traffic accidents), very few studies are sufficiently well powered to evaluate their associations with premorbid intelligence.

A cross-sectional ecological study of census data from almost one hundred European and Asian countries reported a *positive* association between the estimated mean standardized intelligence score (an IQ-type estimate) of each country and incidence of suicide among older adults (Voracek, 2004). Whereas such studies are regarded in epidemiology as being of some value because they lead to hypothesis generation, they offer very little insight into disease processes. There are also several examples in chronic disease epidemiology of the ecological fallacy; that is, results from such group-based studies do not replicate findings seen at the level of the individual. Published in the same year, investigators using the Swedish Conscripts Study reported a robust reverse gradient; that is, lower premorbid intelligence test scores were associated with an increased risk of death by suicide up to midlife (Gunnell, Magnusson, & Rasmussen, 2005) (see Figure 1).

Within the same Swedish cohort, Batty and his colleagues related the Swedish conscripts' intelligence test scores to homicide mortality after twenty years of follow-up. A one SD advantage in premorbid intelligence was associated with a 51% reduced risk of death by homicide, and the effect was incremental across the intelligence range (Batty, Mortensen, Gale,

& Deary, 2008; Batty, Deary, Tengstrom, & Rasmussen, 2008). This association was only marginally attenuated by controlling for a range of covariates. This finding prompted the same group of investigators to explore the link between intelligence and hospitalization for assault via various means (Whitley et al., 2010a). These results supported those for homicide: men with higher intelligence were less likely to experience an assault of any description, and a similar pattern of association was apparent for stabbings, attack using a blunt instrument, or injury caused by a fight/brawl (see Figure 2). Figure 2 shows that, in the age-adjusted model, the hazard of being involved a fight/brawl is over eight times as great for the lowest versus the highest IQ group. The raw numbers given by Whitley et al. (2010a, Table 3) show that, given that this is just one cause of injury/illness, the effect is not trivial. Combining the three highest IQ groups, only 0.5% had had a hospital admission over an average of 24 years of follow-up. Combining the lowest two IQ groups, the figure was 2.5%. In both the homicide and the assault reports these authors have considered a number of possible explanations for the associations, including: neighborhood effects, risk perception differences, differences in verbal skills for conflict resolution, perpetrator-victim correlation of traits such as intelligence, and alcohol intoxication.

Figure 2 also illustrates a persistent issue in the field of cognitive epidemiology and epidemiology in general: possible confounding by various indicators that are often used to indicate socioeconomic position, in this case educational attainment. As a research group, where the data are available, we have always presented intelligence-medical outcome associations with and without adjustment for education and other available factors. Typically, adding education to a multivariable model leads to very marked attenuation (see Figure 2) and, in some cases, nullification, of the intelligence-health outcome gradient. However, this may simply be a reflection of multicollinearity, because education and intelligence are strongly correlated. Indeed,

the more detailed the educational outcome variable, the stronger the relation with intelligence, such that the coefficient of association nears 1.0 (Deary, Strand, Smith, & Fernandes, 2007). This being the case, controlling for education in this scenario raises concerns of over-adjustment: educational outcomes could be acting to some extent as proxies for cognitive ability. We also recognize that there is evidence that education might increase scores in intelligence-type tests (Ceci, 1991), and we have contributed an examination of the education-intelligence association as it applies in epidemiology for those who wish to consider this important topic at greater length (Deary & Johnson, in press).

2.1.2 Dementia. The studies described above typically assess mental health no later than middle age. They therefore do not have the capacity to explore the link between cognition and cognitive decline such as dementia and its sub-types (e.g., Alzheimer's Disease) which typically occur in older age. With a demographic shift towards a rapidly aging population, allied to the absence of successful treatments, understanding the causes of dementia is crucial in efforts to prevent the disorder. One of the few studies that have several decades of follow-up between intelligence assessment and ascertainment of dementia was a sample from the Scottish Mental Survey that took place in 1932. This Survey tested the intelligence of almost all children born in 1921 and attending school in Scotland on one day in June 1932 (Deary, Whalley, & Starr, 2009). The intelligence test used was one of the Moray House series of tests. These are group-administered mental tests with a range of items, but especially verbal reasoning. Test scores correlate very highly (~.8) with the individually administered Binet scales (Deary, Whalley, & Starr, 2009). The study found an association between low childhood intelligence and the risk of late-onset, but not early-onset, dementia (Whalley et al., 2000). A larger follow-up sample of the Scottish Mental Survey of 1932 enabled late-onset dementia cases to be separated into vascular dementia and

Alzheimer's type dementia. The investigators reported that lower childhood intelligence was a risk factor for late-onset vascular dementia, but not Alzheimer's-type dementia, suggesting that vascular processes rather than cognitive reserve are likely mediators in the pathway between early life intelligence and later cognitive decline (McGurn, Deary, & Starr, 2008). This is consistent with an inverse association between intelligence and later cardiovascular disease, in particular coronary heart disease and, most relevantly, cerebrovasular accident (stroke), both of which have vascular origins (see later discussion).

2.1.3 Unintentional injury. A small cluster of studies have examined links between intelligence and unintentional injuries, drawing on data from the Aberdeen children of the 1950s study (Batty et al., 2004), the Danish Metropolit study (Osler et al., 2004), and the Swedish conscripts study (Batty et al., 2007e). Whereas the two former studies found graded associations—unintentional injuries were more common in people with lower prior intelligence—they were somewhat underpowered to examine links with specific injury outcomes. Again, the Swedish conscripts study, because it is up to three orders of magnitude larger in scale, has the power to explore these links. What is immediately evident is that the effects estimates seen in these analyses are markedly larger than those apparent for somatic disease and mental health outcomes. In the Swedish studies, on comparing the lower end of the intelligence spectrum with the higher end, there is typically a doubling of risk. However, when different types of unintentional injury are the outcome of interest, up to a six-fold elevated risk is seen. We have also examined links between intelligence and hospital admissions for unintentional injury in this cohort (Whitley et al., 2010b), and results accord with those described for mortality.

2.1.4 Possible mechanisms. The intelligence-health gradients described above do not appear to have artifactual explanations: confounding, sample bias, reverse causality, chance. This has led to speculation about the underlying causal mechanisms. There are likely to be a series of shared or overlapping processes linking intelligence with the above-described mental health outcomes. When psychological illness is the outcome of interest, one possibility is that intelligence might capture sub-optimal neurodevelopment or, perhaps, the early subclinical stages of mental illness itself (Batty, Mortensen, & Osler, 2005). It is possible that the link is related to sociodemographic variables, such that stress and thereafter mental illness arise from being less adept at school and work. There are some strong advocates of such an explanation (Marmot, 2004; Sapolsky, 2005) though evidential links in the causal chain are missing (Deary, Batty, & Gottfredson, 2005). As indicated, the link between low intelligence and increased suicide risk may be mediated via mental illness, such as depression and psychosis. An alternative explanation is that having reduced cognitive function limits an individual's capacity to resolve problems or personal crises, such that suicide/self-harm occurs more prominently as a solution (Gunnell et al., 2005). For unintentional injury, low cognitive ability may signal either a sub-optimal perception of risk (Batty, Deary, Schoon, & Gale, 2007b) and/or longer reaction times as intelligence and reaction time are inversely related (Deary, Der, & Ford, 2001). Both of these processes may elevate the risk of occupational and domestic injury such as the operation of machinery, and negotiating a hazardous environment more generally.

2.2 Somatic Health

2.2.1 Total mortality. A systematic review identified nine independent longitudinal cohort studies, each of which found an association between lower premorbid intelligence test scores and greater risk of all-cause mortality in adulthood (Batty, Deary, & Gottfredson, 2007a). There was

a suggestion that the intelligence-mortality association was stepwise and there was, at best, a very modest influence of confounding by early life socioeconomic circumstances. Subsequently, there has been an increase in the publication frequency of intelligence versus all-cause (total) mortality studies and we are currently in the process of updating this review within the context of a meta-analysis. As an outcome, total mortality comprises a range of causes of death, both external and internal, not all of which are, *a priori*, likely to demonstrate associations with intelligence. It is therefore more informative—especially with an eye to making the research relevant to public health—to explore disease-specific effects. In brief, we do so now for cardiovascular disease and site-specific cancers.

2.2.2 Cardiovascular disease. In middle- to older-age Western populations, the most common cause of death and disability is cardiovascular disease. Accordingly, this disorder has most frequently been examined in relation to intelligence. Cardiovascular disease can be broadly subdivided into coronary heart disease and stroke. Coronary heart disease is the leading cause of death in the United States and occurs when the coronary arteries which supply blood to the heart are blocked by fatty deposits (atherosclerosis). When this occurs, heart muscles die and an individual is said to have a heart attack. This subdividing is necessary because the epidemiology of these conditions differs. For instance, raised blood cholesterol is risk factor for coronary heart disease but not stroke. The first examination of the intelligence-coronary heart disease link was conducted in Scotland. In this study, 938 participants from the Midspan prospective cohort studies, initiated in the 1970s, were, based on their birth date, linked to their intelligence test scores at age 11, as captured using the Scottish Mental Survey 1932 (Hart et al., 2004). After approximately three decades of mortality and morbidity surveillance, a 1 SD disadvantage in intelligence at age 11 was related to 11% increased risk of hospital admission or death due to

cardiovascular disease. This observation has been replicated in other cohorts drawn from Scotland (Deary, Whiteman, Starr, Whalley, & Fox, 2004), and Sweden (Hemmingsson, Melin, Allebeck, & Lundberg, 2006).

In studies of cardiovascular disease sub-types, the Midspan study (Hart et al., 2004) found a 16% increased risk of coronary heart disease (hospital admission or death) per SD disadvantage in childhood intelligence. Again, these results accord with those from cohorts drawn from Denmark (Batty, Mortensen, Nybo Andersen, & Osler, 2005), Sweden (Batty et al., 2009), and the United States (Batty, Shipley, Mortensen, Gale, & Deary, 2008b)—all of which sampled men—and in a rare mixed-gender sample from Scotland where there was no strong evidence of a differential effect by gender (Lawlor, Batty, Clark, MacIntyre, & Leon, 2008). Adjusting for childhood and early adult covariates had little impact on these gradients.

Studies of the association between premorbid intelligence and stroke have revealed less clear findings. This may result from the low numbers of stroke events in many studies, so leading to sub-optimal statistical power. However, in a sufficiently large study—the Aberdeen Children of the 1950s cohort—a one SD advantage in intelligence at age 11 years was associated with a 32% reduced risk of incident stroke by middle age (Lawlor et al., 2008). The effect that was stronger in women than men. Furthermore, the Swedish Conscripts cohort was large enough to estimate the effects of premorbid intelligence on risk of stroke subtype: ischemic and hemorrhagic (Modig, Silventoinen, Tynelius, Bergman, & Rasmussen, 2009). Again, these associations were robust to the adjustment of collateral data.

2.2.3 Cancer. Cancers share some common modifiable risk factors with cardiovascular disease, including obesity and tobacco smoking. This has led to speculation that premorbid intelligence and selected cancers are also related. Despite some reasonably well-designed studies, the evidence to date suggests that the association is weak. For instance, data from two studies essentially found no relation between intelligence and cancer from all sites combined (Batty et al., 2007e; Hemmingsson et al., 2006). However, as a total cancer endpoint comprises dozens of different cancer sub-types, many of which have no unifying etiology, exploring the relationship, if any, between intelligence and the more common malignancies such as lung cancer would be more informative.

Perhaps owing to the relationship between intelligence and later smoking habits—initiation *and* cessation—an elevated risk of lung cancer has been reported in adult Scottish men and women who had lower intelligence test scores in childhood (Batty, Deary, & MacIntyre, 2007b; Taylor et al., 2003). Similar results have been reported for stomach cancer which, like carcinoma of the lung, is strongly related to cigarette smoking (Hart et al., 2003). Again, analyzing the much larger Swedish conscripts study, Batty and colleagues (Batty et al., 2007e) found little evidence of an association between intelligence and 19 different malignancies. The only exception was skin cancer which was positively related to intelligence. This may be ascribed to the much replicated relation between higher intelligence and job income (Neisser et al., 1996), and the resulting increased frequency of holidays taken in sunny climates, although the association was only slightly attenuated after controlling for socioeconomic status.

2.2.4 Possible mechanisms. The mechanisms that might explain the relations between intelligence and cardiovascular disease—we focus on this outcome owing to the dearth of

convincing evidence, to date, to link intelligence and cancer—are likely to differ from those mechanisms advanced above for the link between intelligence, mental illness, and injury. In a figure that also depicts some of the early life determinants of pre-adult cognition, these possible mechanistic pathways have been set out previously (see Figure 3). Having alluded to several of the mechanisms elsewhere in this piece, here we focus on disease prevention, adult socioeconomic position, and so-called system integrity.

Tobacco smoking (Taylor et al., 2003; Batty et al., 2007b; Batty, Deary, Schoon, & Gale, 2007a), excessive alcohol consumption/alcohol abuse (Batty, Deary, & MacIntyre, 2006; Batty et al., 2007c; Gale et al., 2008), physical inactivity (Batty, Deary, Schoon, & Gale, 2007c), and poor diet (Batty et al., 2007c)—all of which may elevate the risk of cardiovascular disease and selected cancers—appear to be more common in men and women who have lower scores on intelligence tests in childhood and early adulthood. Similarly, some of the physiological consequences of these behaviors, such as obesity (Chandola, Deary, Blane, & Batty, 2006) and raised blood pressure (Starr et al., 2004), are also related to lower childhood intelligence test scores. Perhaps unsurprisingly, given the generally low correlation between behavior and physiology (a diet rich in cholesterol does not necessarily lead to high blood cholesterol), the magnitude of the relationship between intelligence and physiological characteristics appears to be lower than that seen for intelligence and health behaviors. Some of the afore mentioned components (obesity, blood pressure) comprise the metabolic syndrome, and there is also a suggestion that lower intelligence test scores are associated with an increased risk of this disorder (Batty et al., 2008a; Richards et al., 2010). In the study by Batty et al. (2008a), the influence of intelligence on the metabolic syndrome was independent of education, and adjusting for the

metabolic syndrome removed about one third of the now reasonably well-established association between intelligence and cardiovascular disease mortality.

Plausibly, then, these risk factors may partly mediate the relationship between intelligence and cardiovascular disease. To examine this issue requires a dataset with information on intelligence, later measurement of these risk factors, and then subsequent ascertainment of cardiovascular disease. Two such studies—the Vietnam Experience Study (Batty et al., 2008b) and the Midspan-Scottish Mental Survey 1932 linkage (Hart et al., 2004)—have found that, whereas behavioral and physiological do not fully explain the relationship, controlling for later socioeconomic status appears to have a large impact. This potentially points to chains of events: high intelligence test scores lead to educational success, placement into a high social status profession and increased income. Higher adult social status confers protection against cardiovascular disease. However, it is possible that the often-impressive attenuation of the intelligence-health associations found after adjusting for education and/or socioeconomic status could occur because variation in these factors, to a large extent, reflect variation in earlier intelligence (Deary, Strand, Smith, & Fernandes, 2007; Strenze, 2007). Causally informative studies are required to pick apart such possibilities.

Finally, the system integrity hypothesis (Whalley & Deary, 2001; Deary, 2008) posits that individual differences in the integrity of an underlying general physiological make-up may explain the association between premorbid intelligence and health outcomes. This, often rather vaguely articulated, idea is that intelligence tests reflect not just brain efficiency; rather, they are detecting the brain aspect of a well-put-together body more generally; one that is well placed to respond to environmental challenges, and to be able to return to equilibrium after allostatic load.

Therefore, testing this hypothesis demands a search for other possible markers of system integrity; other measurable indicators of bodily and brain efficiency. Reaction time tasks, which measure information processing efficiency, have been significantly associated with all-cause-mortality, in that faster reaction times are associated with reduced risk (Deary & Der, 2005). In this Scottish adult cohort of 898 study members, reaction time also very substantially attenuated the association between prior intelligence and all-cause mortality after 14 years of follow up. This finding lends support to the system integrity theory of intelligence's associations with health outcomes, if processing speed is an effective indicator of neurological integrity which reflects overall physiological integrity. However, without full understanding of why intelligence and reaction time correlate significantly, the interpretation of mechanisms remains problematic. Moreover, the construct of system integrity remains to be explicated more fully. A further attempt to test the system integrity hypothesis used psychomotor coordination and intelligence test scores from childhood in the 1958 and 1970 British birth cohorts (Gale, Batty, Cooper, & Deary, 2009). The health outcomes were obesity, self-rated health and psychological distress assessed when people were in their early 30s. In accordance with the system integrity idea, both intelligence and psychomotor coordination were significantly correlated; and both were significantly associated with all of the health outcomes thirty-plus years later. However, the association between intelligence and the health outcomes was not attenuated after adjusting for psychomotor coordination; and the association between psychomotor coordination and the health outcomes was not attenuated after adjusting for intelligence. Childhood intelligence and psychomotor coordination were, thus, independently associated with health in the 30s. This did not support the idea that intelligence and psychomotor coordination were both markers of some more general body integrity that is relevant to long-term health.

3. Personality and your health

Interest in "epidemiological personology" (Krueger, Caspi, & Moffitt, 2000, p. 967) is not new. The Roman physician and philosopher Galen promoted the long held belief that health was a condition in which there was balance among four bodily fluids, called humors (blood, phlegm, yellow bile, and black bile) and that imbalance would adversely influence a patient's health and personality. Long since Galen's time, considerable research has shown that personality traits and health are interrelated. One can roughly divide this research into areas focusing on four types of health outcomes. The first examines the relationship between personality and physical health outcomes such as disease and death. The second examines the relationship between personality and precursors of disease such as inflammatory markers, dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, and the metabolic syndrome. The third avenue of this research examines the relationships between personality dimensions and either behaviors or demographic risk factors which directly or indirectly impact health. The fourth, and largely unexplored, avenue of this research examines the possibility that personality traits are not causally related to disease but are, instead, biomarkers for risk. Because it is a massive area of research on its own and because they are better-known findings, owing to space constraints, we shall not include the associations between personality and mental disorders here.

3.1 Personality and coronary heart disease: Type A and Hostility

One large area in the study of personality and health outcomes has focused on coronary heart disease and mortality. Whereas it was not the earliest paper examining personality predictors of coronary heart disease (see, e.g., Storment, 1951), a seminal paper by Friedman and Rosenman (1959) noted that, compared to healthy matched controls, the behavior of men who had coronary heart disease was characterized by an "intense, sustained drive for achievement and as being

continually involved in competition and deadlines, both at work and in their avocations" (p. 1286). This seminal study between coronary heart disease and what came to be known as the Type A personality spawned a wave of studies on the relationship between personality and coronary heart disease which lasted for decades. In a review of this literature, Booth-Kewley and Friedman (1987) revealed modest relationships between Type A personality and coronary heart disease. They also found that these relationships were stronger in cross-sectional studies than in prospective studies—suggesting the possibility of some reverse causality—and when a structured interview was used to assess Type A personality as opposed to self-reports. Finally, this same review found evidence that other personality traits were risk factors for coronary heart disease, namely those indicative of depression, angry hostility or aggression, and anxiety. A subsequent meta-analysis (Matthews, 1988) questioned Booth-Kewley and Friedman's conclusions regarding Type A personality, arguing that it may, instead, be related to other risk factors for coronary heart disease in the general as opposed to the at-risk population (see H. S. Friedman & Booth-Kewley, 1988 for a rebuttal). To try and better understand the apparent relationship between Type A personality and CHD, researchers sought to identify whether specific subcomponents of Type A personality were responsible for the relationship. The toxic subcomponents of the Type A personality—namely aspects of Type A personality significantly associated with coronary heart disease—were those which described antagonistic hostility as opposed to components such as speech style or verbal competition (Dembroski, MacDougall, Costa, & Grandits, 1989). In a study which sought to base antagonistic hostility in the context of the five personality factors, Dembroski and Costa (1987) showed that it was most strongly related to lower agreeableness, and it was also moderately related to higher neuroticism.

3.2 Personality and CHD: Other personality risk factors

In addition to the findings with respect to Type A personality and antagonistic hostility as predictors of coronary heart disease, researchers have examined other traits identified by Booth-Kewley and Friedman. A development in this area has been the identification of the distressed type or Type D personality (Denollet, 2005; Denollet, Sys, & Brutsaert, 1995; Kupper & Denollet, 2007). Individuals exhibiting a Type D personality are both high in negative affect (unhappy, irritated, and worrying) and social inhibition (shy, inhibited in social interactions, and closed).[2] Cardiac patients who exhibit a Type D personality are at substantially greater risk for poorer outcomes, including death (Pedersen & Denollet, 2006). Finally, cardiac patients higher in four facets of openness to experience—including openness to aesthetics, feelings, actions, and ideas—were at reduced risk for cardiac mortality (Jonassaint et al., 2007). Openness has a modest positive correlation with intelligence, which could explain some of this finding.

3.3 Personality and your life: The Terman Life-Cycle Study

The other major area of research in epidemiological personology concerns whether certain personality dimensions are related to a longer or shorter lifespan. Initial studies focused on hostility and neuroticism as predictors of mortality from all causes (e.g., Almada et al., 1991). However, since that time, conscientiousness has been identified as the key personality trait predictor of longevity. This association was first uncovered in a follow-up study of over 1,178 participants in Terman's Life-Cycle Study (Friedman, Tucker, Tomlinsonkeasey, Schwartz, Wingard, & Criqui, 1993). The participants, sometimes referred to as the Termites, were a representative sample of bright school children whose Stanford-Binet IQs were at least 135 (Terman, 1925). In 1922 when the children were approximately 12 years old, they were rated on

[2] The description of this construct factor as a 'type' is a misnomer. Similar combinations using Neuroticism and Extraversion have been described as a gloomy pessimist style of well-being (Costa & Piedmont, 2003).

25 traits by one or both of their parents and their teachers. In addition to using these ratings to create scales related to neuroticism (“Permanency of Mood”), extraversion (“High Energy and Sociability”), and agreeableness (“Cheerfulness”), Friedman and his colleagues constructed a scale related to conscientiousness using ratings on “prudence,” “conscientiousness,” and “truthfulness”. Survival analysis revealed that students who had been higher in conscientiousness in childhood were more likely to be alive when mortality was assessed 64 years later. In addition to neuroticism, and contrary to expectations, cheerfulness was related to greater mortality risk (Friedman et al., 1993).

3.4 Personality and your life: Beyond the Termites

Whereas Friedman’s study could be criticized for the homogeneity of the sample on cognitive and social grounds, a review of studies on 20 independent samples (Kern & Friedman, 2008), many of which differed dramatically from the Termites, showed that conscientiousness was a clear predictor of mortality across samples and held even when controlling for traditional risk factors.

Other studies of personality and longevity have examined either all, or subsets of, personality trait measures related to the Five-Factor Model. A review of this literature (Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007) found that, overall, lower conscientiousness, lower extraversion/positive emotions, higher neuroticism, and lower agreeableness conferred greater mortality risk. Moreover, they noted that the magnitude of risk posed by these personality predictors was equal to (or greater than) that posed by low socioeconomic status and even lower intelligence. It is worth noting that, whereas the effects of conscientiousness were consistent

across studies, there was some variability in the direction of the effect for other personality factors (e.g., higher neuroticism was related to greater longevity in some studies).

3.5 Personality and other health outcomes

3.5.1 Other diseases. Compared to the research on personality and either coronary heart disease or longevity, there is considerably less research on personality predictors of other diseases. However, progress has been made on this front. In a study of the MIDUS national representative sample, Goodwin and Friedman (2006) found that, of the five personality dimensions, conscientiousness and neuroticism were consistently related to the presence of several self-reported diseases. Of this sample, participants reporting diabetes, high blood pressure, hernia, or bone and joint problems were lower in conscientiousness but did not differ in neuroticism. Participants reporting ulcers, asthma or bronchitis, and other lung problems were higher in neuroticism but did not differ in conscientiousness; and participants reporting persistent skin problems, sciatica/lumbago, urinary/bladder problems, stroke, or tuberculosis were both lower in conscientiousness and higher in neuroticism. Similarly, Chapman, Lyness, and Duberstein (2007) found that the same pattern of results held for the aggregate medical illness burden as assessed by patient records.

Personality dimensions have also been identified as risk factors for physician-diagnosed conditions. Of note is a study which showed that, among a sample of nearly 1,000 older members of religious orders, those with high as opposed to low conscientiousness were at reduced risk for Alzheimer disease and mild cognitive impairment (Wilson, Schneider, Arnold, Bienias, & Bennett, 2007).

3.5.2 Disease Progression. Personality dimensions may also influence the course of diseases. One notable example is the case of cancer. A review of the literature suggested that, whereas traits related to negative affect and depression are not related to the development of cancer, they adversely influence the course of the disease and lead to a greater likelihood of mortality (Denollet, 1999). A second notable example is the case of HIV disease progression; higher conscientiousness, extraversion, and openness were related to slower disease progression as indicated by reductions in viral load and increases in CD4 counts over time (Ironson, O'Cleirigh, Weiss, Schneiderman, & Costa, 2008; O'Cleirigh, Ironson, Weiss, & Costa, 2007).

3.5.3 Precursors: Inflammatory Markers. Alongside health outcomes such as mortality, disease incidence, and disease progression, researchers have explored the possibility that personality could impact precursors to diseases. A study by Sutin et al. (2009) found that high neuroticism and low conscientiousness were associated with higher levels of interleukin-6 and C-reactive protein, markers related to chronic inflammation, morbidity, and mortality. They also found that participants in the top and bottom 10% of neuroticism and conscientiousness, respectively, were at significantly increased risk of exceeding clinically-relevant levels of interleukin-6.

3.5.4 Precursors: HPA-axis dysregulation. Similarly, studies have examined whether personality is a risk factor for HPA-axis dysregulation. The HPA-axis is activated in times of stress and readies the body for 'fight or flight' responses. However, if there is chronic activation of this system, it contributes to allostatic load or wear and tear on the body and organs (McEwen, 2000). At least three studies have shown a relationship between higher neuroticism and traits related to neuroticism and dysregulation of the HPA-axis as measured by cortisol responses to chemical challenges (Mangold & Wand, 2006; Tyrka et al., 2006; Tyrka et al., 2008). These findings

suggest that the HPA-axes of individuals higher in these traits are either more vulnerable to the stressors which they experience, experience more stressors, or simply have higher levels of activation throughout the day.

3.5.5 Precursors: Metabolic syndrome. Finally, researchers have examined whether neuroticism is a risk factor for the metabolic syndrome and its components. The metabolic syndrome, as discussed previously, describes a confluence of conditions that are major risk factors for diabetes and cardiovascular disease. Phillips et al. (in press) found that neuroticism was a risk factor for metabolic syndrome and three of its components: obesity, high triglycerides, hypertension, and high blood glucose levels. Most of these associations were no longer significant after controlling for other risk factors and intelligence, though neuroticism remained a risk factor for obesity and hypertension.

4. Mechanisms

Given these many associations, by what means could personality influence health? Personality traits are related to many potentially important factors impacting health, including coping style, social support, and depression, and it would be beyond the scope of this article thoroughly to review the literature. Instead, we will focus on two predominant classes of possibilities: health behaviors and socioeconomic status.

4.1 Health Behaviors

One possibility is that personality traits are related to health-harming or health-promoting behaviors which directly effect health. This mechanism is highly plausible: a review of 194 studies by Bogg and Roberts (2004) showed that high conscientiousness was consistently related

to more health promoting (e.g., exercise and healthy diet) and fewer health-harming behaviors (e.g., alcohol abuse and fast driving). In a study of the five personality dimensions and smoking, Terracciano and Costa (2004) showed that, in addition to low conscientiousness, high neuroticism and low agreeableness were related to smoking. Moreover, participants who had high neuroticism and low conscientiousness scores—i.e., those whose style of impulse control was classified as undercontrolled—were particularly at risk. Personality's influence on health behaviors may also impact how well patients manage diseases. This was confirmed in a study of patients with end-stage renal disease, a chronic condition requiring kidney dialysis and a complex treatment regimen. Of the five dimensions, conscientiousness predicted better adherence to medication (Christensen & Smith, 1995), something that is also found with intelligence (Deary et al., 2009).

4.2 Socioeconomic status

Another important route by which personality may impact health is via socioeconomic status, which we earlier identified as a well-known predictor of health outcomes, and a possible mediator of the association between intelligence and health outcomes. Lower neuroticism and higher extraversion, openness, agreeableness, and conscientiousness are related to several indicators of higher socioeconomic status (Jonassaint, Siegler, Barefoot, Edwards, & Williams, in press). Whereas the relationship is likely to be reciprocal, it is not hard to envisage how this configuration of traits could lead to higher educational achievement, income, and social status, which, subsequently, could impact health.

4.3 Mediation studies: Health Behaviors

Surprisingly, formal tests of whether these potential mediators actually mediate have revealed that, at best, mediators only partly account for the personality-mortality relationship. A follow-up study of the Termites (Martin, Friedman, & Schwartz, 2007) showed that the effects of childhood conscientiousness were not reduced after controlling for later alcohol use, smoking, and educational achievement. This study also found that the relationship between adult conscientiousness and mortality was only partly reduced, though it was no longer significant. Similarly, Terracciano, Löckenhoff, Zonderman, Ferrucci, and Costa (2008) found that smoking and obesity did not mediate the relationship between low neuroticism and longevity, and were only very slightly involved in the relationship between conscientiousness and longevity. Likewise, Nabi and his colleagues (2008) showed only a modest mediation of the relationship between neurotic hostility and mortality by the combination of smoking, drinking, and body mass index. Also, Chapman, Fiscella, Kawachi, and Duberstein (2010) showed that smoking and physical inactivity partly mediated the effects of neuroticism on mortality. On the other hand, Weiss, Gale, Batty, and Deary (2009a) found no evidence that the relationship between neuroticism and mortality was directly mediated via health behaviors.

The study of inflammatory markers by Sutin et al. (2009) also investigated the possible mediating effects of health behaviors including smoking, body mass index, and the use of aspirin. They found that the impulsivity facet of neuroticism led to higher interleukin-6 in part via its effect on smoking and body mass index. They also found that smoking partly mediated the relationship between lower levels of four conscientiousness facets (competence, deliberation, achievement striving, and deliberation) and higher levels of interleukin-6. Finally, higher body weight partly mediated the relationship between lower scores on the order facet of conscientiousness and higher levels of interleukin-6.

4.4 Mediation studies: Socioeconomic status

Most studies on the relationship between personality and health include measures of socioeconomic status, such as income and educational achievement. Oddly enough, despite this—and by contrast with the situation we described in intelligence-health research—few studies have formally tested whether personality-mortality relationships are partly or wholly mediated by these variables. One recent exception is the previously described study of neuroticism, cognitive ability, and mortality in Vietnam-era veterans by Weiss et al. (2009) who found no evidence that the risk posed by higher neuroticism was mediated by education or family income. A second exception was the study by Chapman et al. (2010). Their study showed that the effects of socioeconomic status on mortality were, in part, explained by the five major personality dimensions and that, conversely, the effects of personality were very slightly mediated by their effects on socioeconomic status.

4.5 Mediation studies: The patient-health care practitioner relationship

One potentially important way by which individual differences in cognitive abilities and personality may impact health is via their effects on how individuals interact and communicate with health-care practitioners. In the case of cognitive abilities, one possibility is that more intelligent patients are likely to have larger vocabularies and may have investigated their condition before seeing a health care practitioner. As such, they may be better able to communicate their symptoms. Similarly, the greater vocabulary of more intelligent patients may be better able to understand any advice they are given on how to conceptualize, treat and/or manage a health condition.

The effects of personality on this relationship may be particularly important, especially given that several of the domains and facets of personality may directly or indirectly influence how an individual interact with others. This may be especially true in the case of agreeableness. Patients who are higher in agreeableness may be more compliant, more willing to place their trust in health care practitioners, and more honest and frank in discussing their condition. Conversely, individuals who are cynical and distrustful of medical practitioners may be more likely to turn to unorthodox and untested treatments, which could be ineffective and even dangerous. It is also possible that the effects of intelligence and personality on the relationship are more subtle and that intelligent and congenial patients may elicit more empathy from their health care providers.

With respect to the effects of personality, there are findings that support these possibilities. For example, of the participants of the Western Electric Study, those who scored higher in a measure of cynicism were at greater risk of coronary and all-cause mortality even after controlling for several behavioral and physiological risk factors (Almada et al., 1991). Similarly, a large sample of 65 to 100 year old Medicare patients also supports this possibility; when examining the facets of the five personality factors they found that, even after controlling for several behavioral, psychological, and physiological risk factors, that the protective effect of agreeableness was underpinned by its straightforwardness facet.

4.6 Moderators

Another mechanism by which a personality dimension could impact health is by modifying or moderating other risk factors whether they are demographic factors, socioeconomic status, health

behaviors, or cognitive abilities and other personality dimensions.[3] Few studies have examined this realm of possibilities, which is surprising particularly as it might help explain some inconsistencies within the literature such as why neuroticism is a mortality risk factor in some studies and a protective factor in others (see Weiss & Costa, 2005; or Friedman, 2008 for a brief review).

Most studies that have examined moderators have looked at interactions among traits, i.e., whether certain combinations lead to greater risks than the traits alone could account for. One research program which investigated this possibility is that of Type D personality and coronary heart disease. In short, a particular combination of traits or personality style marked by high neuroticism and low extraversion is a particularly potent risk factor for poorer prognosis in patients with coronary heart disease (Denollet, 2005; Denollet et al., 1995; Kupper & Denollet, 2007). Similarly, in studying mortality risk, Chapman et al. (2010) found that high conscientiousness was only a protective factor at high levels of agreeableness. Finally, Weiss et al. (2009) found an interaction between neuroticism and intelligence in their study of the Vietnam Experience Study cohort. This finding could be interpreted as showing that the protective effects of high intelligence was reduced among individuals who were high in neuroticism or that the risk posed by high neuroticism was reduced among subjects who were more intelligent (see Figure 4).

5. How Should Future Studies be Designed and Analyzed?

Our inability to identify consistent and strong mediators of the intelligence-health (though we note both the attenuating effects and the possible problems of interpretation with adult

[3] It should be noted that it is also possible to examine whether the effects of personality are dependent on other factors such as age. For example, Lee, Wadsworth, and Hotopf (2006) showed that anxiety was related to greater accident risk among older subjects but reduced accident risk among younger subjects.

socioeconomic status) and personality-health associations hampers our ability to understand how intelligence and personality affect health. Moreover, we are only beginning to understand how intelligence and personality may interact with other health-related predictors. We suggest that future studies should focus on identifying and ruling out potential mediators and moderators, particularly because they may be modifiable risk factors. Unfortunately, while identifying and ruling out possible mediators should be a seemingly simple task, the research to date suggests that such variables are elusive at best. As we described earlier, combined, most mediators have only partly explained the relationship between personality and health. We should be clear about what such a suggestion entails. Many of the cohort studies we described are already heroic—in terms of their numbers of subjects, the representativeness to the background population, the follow-up period, or the quality of the variables gathered, or some combination of these—and we are suggesting that, in addition to high-quality studies that have both predictors (personality and/or intelligence) and outcomes (health, broadly conceived), they also include likely mediators and moderators. Some studies will have such characteristics—we note the richness of data in the Vietnam Experience Study and the British 1958 birth cohort study, for example—there will always be limitations, not least because new biomarkers cannot be included in studies until they have been identified and can be measured.

5.1 Where are the mediators?: Measurement and socioeconomic status

Socioeconomic status only accounts for a modest amount of the relationship between personality and health, whereas it has a larger attenuating effect with intelligence. One reason that may account for this is that socioeconomic status variables such as family income, educational achievement, and occupational prestige are poor proxies of a host of specific values, goals, desires, and other factors that impact health; and, to an extent, for intelligence. As such, by

relying on socioeconomic measures we are only accounting for a very small portion of the true link between personality and health. And, with respect to intelligence, the direction of causation is unclear, because each attenuates the other's association with mortality and mental and physical ill health (Batty, Der, Macintyre, & Deary, 2006).

5.2 Where are the mediators?: Complex causality

Another possibility is that the pathways leading from intelligence and personality to health outcomes are not as straightforward as assumed by our models (see Friedman, 2008 for a discussion). We must therefore ask ourselves whether our theories are adequate or whether we require new theories and new models to test them. Before doing this, it is important to review the predominant theory and the models used to test that theory. Most present models assume that intelligence or some personality trait leads to one or more mediators which subsequently have an impact on health outcomes (see Figure 3 and 5a). Whereas there are multiple ways of testing these models, the most common way is via a regression-based approach described by Baron and Kenney (1986). This approach involves first regressing a factor conceptualized as a mediator. For example we may wish to regress education onto intelligence or smoking onto a personality trait such as neuroticism. The second step is to regress the health outcome (e.g., mortality) onto intelligence or the personality trait. The final step then involves regressing the health outcome onto the predictor variable and the mediator. If, in these cases, education mediates the relationship between intelligence and mortality, or smoking mediates the relationship between personality and mortality, three things should be demonstrated in the results: 1) intelligence must be a significant predictor of education, or neuroticism must be a significant predictor of smoking in the first regression; 2) intelligence must be a significant predictor of mortality, or neuroticism must be a significant predictor of mortality in the second regression; 3) education must predict

mortality, or smoking must predict mortality in the second regression, and the effect size of intelligence or neuroticism, respectively should be partly or completely reduced (Baron & Kenny, 1986, p. 1177).

If the relationship between intelligence or personality and health followed this simple mediation model, researchers should then be able to understand the mechanisms by which intelligence or personality influences health simply by examining plausible (and hopefully well-measured) mediators. Unfortunately, this simple model does not appear to be good at capturing the nature of the personality-disease relationship (Friedman, 2008) and may also be poor at capturing the nature of the intelligence-disease relationship. As such, we hope that researchers turn to other models. For example, another way in which personality could impact health is via a chain model or what we refer to as a cascade model (see Figure 5b). Here the impact of personality sets off a series of events which determine poorer health outcomes. For example, low intelligence could lead to poorer diet choices and uninformed health habits which lead to diabetes or atherosclerosis and, ultimately, earlier death. This possibility can be examined with an extension of the methods described by Baron and Kenny (1986), though other regression-based methods such as sequential canonical analysis (Figueredo & Gorsuch, 2007) can also be used to test these models.

A third model is similar to the classic mediation model shown in Figure 5a. However, this model does not assume that the relationship between intelligence or personality and the mediators is necessarily causal. Instead, intelligence or personality and mediators in this model are believed to influence one another (see Figure 5c). This model quite possibly better reflects reality in the event that there is no theory specifying causal direction or the theory indicates that causality may flow both ways. For example, in the former case, a specific health risk behavior such as smoking

likely arises after intelligence or personality differences have developed. In such cases, it would make more sense to look at a model similar to that proposed in Figure 5a. However, the case is not so simple with variables such as socioeconomic status. As Chapman and his colleagues (2010) pointed out, there is sufficient evidence to believe that socioeconomic and personality dimensions influence one another. Chapman and his colleagues (2010; Table 2) also demonstrated that this can be handled by two sets of the regression approaches described by Baron and Kenny (1986). In the first set traits (personality dimensions in their case, though it also would apply to intelligence test scores) are treated as predictors and socioeconomic status is treated as the mediator. In the second set, this is reversed with socioeconomic status being treated as the predictor and the traits as the mediators. With regard to intelligence, such reversal—intelligence being considered a mediator of socioeconomic influences in health outcomes—has been attempted, and found to indicate that, indeed, intelligence—at least, statistically—can substantially appear as a mediator between socioeconomic status and morbidity and mortality (Batty, Der, Macintyre, & Deary, 2006).

The fourth possible model is also similar to the classic mediation model. However, in this model the regressions of mediators onto intelligence or personality traits, and of health onto the mediators are random and not fixed effects (see Figure 5d). Thus, whereas each regression coefficient (*b*) has a specific average or mean, there is also between-subject variance in the size of the coefficients as denoted by the subscript i. This, therefore, permits the possibility that, among some individuals, the relationships between certain mediators and traits as well as the relationships between mediators and health outcomes may be stronger or weaker. These differences may be related to other characteristics of the participants (e.g., age, sex, or other psychological or physical traits) or be unexplained residual variation around the mean effect.

Unlike the other models described thus far, testing this model requires the use of statistical analyses such as multilevel modeling (see Singer & Willett, 2003 for an exceptionally clear treatment) or, in the case of survival data, frailty analysis (Hosmer & Lemeshow, 1999).

The fifth model differs from the previous models in that there are no direct or indirect relationships between the variables (see Figure 5e). Instead, this model postulates that traits as well as the mediators are biomarkers of health. One possible means by which this state of affairs may come about is via genetic pleiotropy (Falconer & Mackay, 1996) where a single gene influences multiple phenotypes (traits, mediators, and health outcomes in our example). Another possibility is that genes for intelligence and personality traits, mediators, and health outcomes are close enough on the chromosome so that they are inherited together. Both of these possibilities cannot be tested in the same way as the first three models. Moreover, to estimate whether there are genetic correlations among variables, one requires a different design than the other models, namely one which incorporates data on genetic relatedness such as a twin or family study (Neale & Cardon, 1992). Personality may also be a health biomarker for other reasons. For example, it may be the case that high levels of circulating hormones may lead to health risks as well as trait differences among individuals.

5.3 Where are the mediators?: Confounding

Finally, as with any non-experimental study examining the relationship between two variables, one possibility is that the relationship between personality or intelligence and health are confounded by some unmeasured or ‘third’ variable (Cook & Campbell, 1979). In other words, some or all of the relationship between the individual differences variables on the one hand and health on the other reflects some common cause.

In introductory statistics and research classes this is commonly illustrated by discussing the correlation between ice cream consumption and drowning. Of course, eating ice cream does not cause drowning, but both the amount of ice cream eaten and people entering bodies of water (a pre-requisite for drowning) are higher during the summer. With respect to relationship between individual differences variables and health, there are several possible mechanisms. For example, it may be that the relationship between lower intelligence and poorer health may be explained by social deprivation effects which impact both of these variables. In the case of the relationship between higher neuroticism and poorer health outcomes, both may be caused by persistent or early life stressors.

There are two means by which researchers can rule out the possibility of confounders. The most common approach is a regression-based approach in which the researcher runs two separate models. In the first model the health outcome is predicted by some individual differences variable or variables. In the second model the effect of the individual differences variable or variables are tested after statistically controlling for the possible third variable (e.g., socioeconomic status or stressful life events). If the effects of any predictor variable decreases or is no longer statistically significant, the relationship between this variable or variables and health is said to be confounded by the third variable. The second approach, and one which is gaining in popularity, is to use covariance structure modeling. This is illustrated in Figure 6 in which a base model which specifies that some third variable (III) predicts both the trait of interest and health outcomes via paths $b_{T.III}$ and $b_{H.III}$, respectively, and that health is also predicted by the trait via path $b_{H.T}$. To test whether the effects of the third variable confound the relationship between traits and health, a model in which $b_{H.T}$ is free to vary should be compared to a model in which it is fixed to 0. If the

models are significantly different, i.e., the model with the pathway between traits and health is better, then the effects of the trait on health are not confounded by a third variable. If, on the other hand, there is no difference between these models it suggests that the relationship between the trait and health, when controlling for the third variable, is not different from 0. That is to say, it is confounded by the third variable. An accessible account of this and related issues in the context of social inequalities in health is provided by Singh-Manoux (2005).

5.3 Everything in moderation

Given the large volume of data that exists on intelligence and personality traits and health outcomes, it is surprising that not much more work has been done on identifying whether traits moderate or are moderated by other traits and risk factors. This is a relatively simple enterprise, requiring little additional work than including interaction terms which test specific hypotheses concerning these possibilities. Moreover, with the growing number of studies which include measures of intelligence (or specific cognitive domains) and/or the five major personality dimensions, one can also examine the impact of personality styles, combinations of high or low scores on two of the dimensions (Costa & Piedmont, 2003). Using styles may reveal that, for example, whereas individuals high in neuroticism are generally more at risk for poor health outcomes, this effect may not be true among individuals who, for example, are high in intelligence, or in conscientiousness. This last possibility has strength, especially in light of evidence that intelligence and personality styles are related to mortality (Chapman et al., 2010), cigarette smoking (Terracciano & Costa, 2004), and health risk factors such as depression (Weiss et al., 2009b). As such, we would encourage researchers to look beyond the main effects of the traits and risk factors they examine in their studies.

5.4 New studies

We realize that the possibilities outlined above are daunting, especially if one considers that they are not exhaustive. Our point was not to suggest that all possibilities need to be examined, but merely to try to explain the relatively modest amount of mediation discovers to date in cognitive and personological epidemiology. We feel that it is important to remember the role of theory, parsimony, and what is clinically significant. Theory and prior research should be able to rule out several possibilities for the relationships between intelligence, personality traits and health. Also, whereas complex models may do a better job at explaining relationships, do they explain more additional variance than is justified by the loss in elegance or ability to communicate the findings? In particular, is the additional information gained likely to be of clinical significance or useful to practitioners?

Given that the study of possible mediators and moderators of trait effects on health is in its infancy, we recommend first making the search for mediators, either causal or reciprocal, a key priority for differential (intelligence and personality) epidemiology. Many existing data sets can be used to these ends and re-analyses could yield many important insights. In addition, just as genetically informative data sets have been influential in understanding the comorbidity between personality traits and major and minor psychiatric disorders (e.g., Kendler, Gatz, Gardner, & Pedersen, 2006; Ivkovic et al., 2007), these data sets, where possible, should be used to rule out or rule in the possibility that intelligence and personality traits are biomarkers for health.

Alongside using existing data, we emphasize to researchers and funding bodies the need to incorporate personality and cognitive ability measures in future health studies and especially randomized control trials of health interventions. These measures are well-understood, reliable,

partly tractable, and highly cost effective in that they can be had at low prices or for free. Moreover, they are relevant to health outcomes. Such new studies can either explore the possible impact of traits on their intervention, e.g., determine whether providing printed health information is more likely to be useful for patients who are higher in intelligence. However, the relationship between traits and interventions can also be the focus of the study, e.g., is a specific health intervention, say a change in diet, useful in reducing the cardiovascular risk posed by low intelligence, or low agreeableness.

We also advocate experimental studies using animal models, especially as traits such as personality (Gosling, 2001) and intelligence (Banerjee et al., 2009) can be reliably measured in nonhuman species and that the ability to control diet, environmental risk, and other factors could help better understand how these traits impact health either directly, indirectly, or in combination with other factors. Research in this area has already found that rhesus macaques higher in a trait named ‘sociable’ show a greater reduction in viral copies of the simian immunodeficiency virus over time (Capitanio, Mendoza, & Baroncelli, 1999), a finding which presaged by nearly a decade findings on extraversion and disease progression among humans with HIV (Ironson et al., 2008).

5.5 New analyses

To explore plausible mechanisms and analyses, data from new study designs requires appropriate analytical techniques. At present, there seemingly are two families of techniques. One family is more familiar to epidemiologists and is based on statistics related to regression such as multiple regression, general linear models, logistic regression, survival analysis, and multilevel modeling. The second family is more familiar to differential psychologists/psychometricians and those

studying areas such as program evaluation and behavior genetics. It subsumes regression based approaches and other analyses and is commonly referred to as latent variable modeling, covariance structure modeling, structural equations modeling, or path analysis (Loehlin, 1998). In this approach, relationships among a series of variables are modeled and then fit to the actual data set. The goal is to find a set of paths which describes relationships that best fit the data. As such, this approach can allow researchers to formally test whether the effects of traits are mediated by one or more other variables and whether different mediators are interrelated. Deary (2010) has urged a closer integration of these types of analyses, and for more differential psychologists and epidemiologists to work more closely together to solve the problems of why intelligence and personality are so consistently and strongly associated with morbidity and mortality.

Because the number of possible paths that can be used to relate traits and health variables to each other is exceptionally large in many large datasets, we recommend combining these two approaches in two steps (see Hart et al., 2003; Chandola et al., 2006; Weiss et al., 2009a for examples). First, regression-based approaches are used to identify plausible mediators of a given trait or traits and to rule others out. Second, using this information, the relationships among the traits, mediators, and outcomes such as mortality are formally modeled and tested. In the latter example, we used *Mplus* (Muthén & Muthén, 1998-2007), particularly as it allows outcome variables to be continuous, categorical, or censored variables, and thus can be readily used with much health data.

6. Putting Research into Practice: why should medical practitioners be interested?

Intelligence and personality traits comprise several characteristics which should make them of interest to health researchers and medical practitioners. First, intelligence has many real-world

impacts; and the fact that there is agreement between self- and rater-reports of personality traits suggests that patients, their family, or, if they are familiar with the patient, the primary care providers, could easily assess the personalities of patients using any one of the readily available personality measures. Given that these dimensions are human universals, in an increasingly diverse society, measures should apply equally to patients from a wide range of backgrounds. Moreover, as intelligence and personality are relatively stable in adulthood, a single assessment in adulthood would usually be informative over long periods of time.

Whereas knowledge of how traits predict health outcomes is, at present, nascent, we do not believe it is too early to speculate about how what we do not and what we might discover could be used by health practitioners and policy makers to improve public health. To illustrate we propose two thought experiments. First, what could health practitioners and policy makers do with information on a patient's intelligence and personality? Health practitioners who encounter a range of patients regularly are most likely aware of differences in their intelligence and personalities. However, what could they do with this information? We offer four possibilities, though, undoubtedly, many more exist. The first is targeted surveillance: a patient lower in intelligence or agreeableness, or who displays a distressed type of personality, could have his or her cardiovascular health monitored more regularly. This would be helpful in managing costs as regular and costly monitoring would be targeted at those most at risk whereas those at less risk could undergo less frequent, albeit still regular, monitoring. Moreover, the increased surveillance in those at risk, although costing more in the short run, could lead to large savings to health care organizations and societies gained from a reduced likelihood of myocardial infarction.

A second possible use of intelligence and personality data on patients would be to tailor and develop more effective intervention strategies for particular patients. For example, when faced with patients high in conscientiousness, a physician or nurse's advice to change one's diet or give up smoking would be likely to be met by a high self-directed effort on the part of the patient. However, for patients low in conscientiousness, this advice may need to be accompanied with short-term incentives and regular monitoring and reminders, or behavior modification either by the health care providers or other experts. Similarly, whereas individuals who are high in intelligence and conscientiousness could adhere to a complex treatment regimen such as highly active antiretroviral therapy, those who are low in both could have difficulties. The contrasting long-term survival likelihood of those who are, in childhood, high intelligence-high conscientiousness versus those who are low intelligence-low conscientiousness is marked (Deary, Batty, Pattie, & Gale, 2008). In these cases, patients in the latter group could be supplied with mental prostheses which remind them of when they need to take a particular medication or be the recipients of newer, less complex treatments. Again, the additional costs borne by these prostheses or newer treatments are likely to be outweighed by a reduction in serious future complications and the evolution of resistance. Finally, future findings in pharmacogenetics may be able to better match drugs to patients on the bases of their personality and reduce the number of side effects and other complications.

A third possible way that personality can improve the patient experience is in helping the physician choose drugs which the patient can tolerate. All medications come with potential side effects. However, where a range of treatment options exist, physicians could choose the option which would least bother or upset a patient. For example, patients high in conscientiousness may have more mental resources to tolerate treatments that effect their concentration whereas those

who are high in extraversion may be upset if a treatment interferes with their activity levels or causes drowsiness. As such, information about personality could not only improve health and patient compliance, but also improve patient satisfaction and well-being.

A fourth possible use of these data is to improve relationships between healthcare practitioners and patients. These relationships are likely key to better health outcomes in patients and may be influenced by personality. For example, patients who are low in agreeableness may need more time before they trust nurses or physicians and so this aspect of the relationship could be worked on so as to insure better compliance, more disclosure of health problems and complications as they arise, and other matters. Similarly, patients high in openness to experience may appreciate being provided with more information and a host of treatment options whereas those who are closed might prefer unambiguous instructions from their healthcare providers.

Our second thought experiment is to ask how one could tailor personality information for health care practitioners. With a large number of patients and other information on their charts, the addition of more information would be most beneficial if it was clear, concise, and relevant. We suggest simple reports like those of the Revised NEO Personality Inventory which we reproduced as Table 1 (Costa & McCrae, 1992) that are often provided to subjects in research studies or to possible employers. The report could briefly describe what characteristics are expected by the individual based on whether they are low, average, or high in that personality domain. In particular, they could be described in ways relevant to health practitioners, i.e., their disposition, risk factors for any diseases, and ability to comply with medication regimens. In addition, certain personality styles such as those who are high in neuroticism and low in extraversion could be flagged as being at much higher risk for specific problems. Such reports could be developed

together with physicians and epidemiologists. Moreover, after developing these questionnaires, randomized control trials could determine whether physicians provided with this information provide better healthcare and have better outcomes than those who are not.

Specifically with regard to intelligence, we do not think it is practicable to emphasize a route that goes toward raising intelligence throughout the life course to improve health. Of course, it is possible that optimal health and bodily care though life will lead to better intelligence (mental capital; Kirkwood, Bond, May, McKeith, & Teh, 2008) in any case. What seems more likely to be effective is to encourage phenocopies of high intelligence with respect to health. To the extend that we can discover what smart people do to look after their bodies and health and manage their illnesses, these strategies can be made widely known and available as valid and useful health care rules. The same would apply to the behavioral choices of people with high conscientiousness.

In concluding, we emphasize that it is early in the development of this field for suggested interventions. We stressed earlier that there are many new types of studies and analyses that require to be done in cognitive and personological epidemiology. However, it is important at this early stage that the clear and new findings that link very well established individual differences to health outcomes are much more widely known. This foundational knowledge will be important in urging researchers and practitioners to include cognitive and personality variables in their work. For example, when epidemiologists are planning large scale observational studies and interventions they will be encouraged to include intelligence and traits. To date, too many of the studies in cognitive and personological epidemiology have been undertaken simply because, by luck, there happen to have been personality or intelligence measures assessed in what turned out to be a sample that could be linked to health, morbidity, and mortality. It is our aim that, having

introduced the strong findings and many unanswered questions in the field, differential epidemiology can start to be the subject of studies that take place through design rather than luck.

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Table 1

Descriptions of the Five Factor Model personality traits from the middle and both extremes.

	High scorer	Average	Low scorer
Neuroticism	Sensitive, emotional, and prone to experience feelings that are upsetting	Generally calm and able to deal with stress, but you sometimes experience feelings of guilt, anger, or sadness	Secure, hardy, and generally relaxed even under stressful conditions
Extraversion	Extraverted, outgoing, and high-spirited. You prefer to be around people most of the time.	Moderate in activity and enthusiasm. You enjoy the company of others but you also value privacy.	Introverted, reserved, and serious. You prefer to be alone or with a few close friends.
Conscientiousness	Conscientious and well-organised. You have high standards and always strive to achieve your goals.	Dependable and moderately well-organised. You generally have clear goals but are able to set your work aside.	Easygoing, and sometimes careless. You prefer not to make plans.
Agreeableness	Compassionate, good-natured, and	Generally warm, trusting, and	Hardheaded, sceptical, proud,

	eager to cooperate and avoid conflict.	agreeable, but you can sometimes be stubborn and competitive.	and competitive. You tend to express your anger directly.
Openness to Experience	Open to new experiences. You have broad interests and are very imaginative.	Practical but willing to consider new ways of doing things. You seek a balance between the old and the new.	Down-to-earth, practical, traditional, and pretty much set in your ways.

Table 2

Facets of the Five Factor Model personality traits.

Trait	Facets
Neuroticism	anxiety, angry hostility, depression, self-consciousness, impulsiveness, vulnerability
Extraversion	warmth, gregariousness, assertiveness, activity, excitement-seeking, positive emotions
Openness	fantasy, aesthetics, feelings, actions, ideas, values
Agreeableness	trust, straightforwardness, altruism, compliance, modesty, tender-mindedness
Conscientiousness	competence, order, dutifulness, achievement striving, self-discipline, deliberation

Figure captions

Figure 1

From Gunnell et al. (2005): Fully adjusted suicide hazard ratios by categories of logic intelligence test score with category 5 as the referent. The same results were apparent for a global IQ score.

Figure 2

From Whitley et al. (2010): Hazard ratio (95% CI) for fight or brawl by IQ adjusted for (a) age (■) and (b) all factors including education (♦).

Figure 3

From Batty et al. (2007a): A simplified model of influences on pre-morbid IQ and potential pathways linking pre-morbid IQ with later mortality. [a]Although psychiatric disease is shown as a possible mediating variable between IQ and mortality, it might also be an antecedent variable if, for example, suboptimal neurodevelopment were the prior cause of both psychiatric disease and early mortality. [b]Note that system integrity is shown as antecedent to both IQ and mortality. In this pathway, lower IQ is not a cause of mortality, but both IQ and mortality are influenced by this more fundamental physiological integrity.

Figure 4

From Weiss et al. (2009): Hazard ratios for combinations of high (+1 SD), average (0 SD), and low (-1 SD) levels of Neuroticism (N) and general intelligence (*g*).

Figure 5

Five models showing how the influence of a trait (T) on health (H) may be mediated by other variables (M_n). Note that, while traits may directly impact health, this is not assumed in these models. The models include a) the classic model commonly used in differential epidemiology. The first set of arrows represents the regressions of the three mediators onto the trait. The second set of arrows represents regressions of health onto the three mediators; b) the chain or cascading model in which the first mediator is regressed onto the trait, the second mediator is regressed onto the first mediator, and health is regressed onto the second mediator; c) the reciprocal model in which one trait has a reciprocal relationship with two possible mediators which influence health outcomes; d) the random effects model where the relationship between each individual's (i) traits and possible mediator variables and those mediator variables and health outcomes may differ; e) the biomarker model in which traits, potential mediators, and health outcomes are all manifest variables of underlying biological health.

Figure 6

An illustration of confounding in which a third variable or confound (III) effects both the trait (T) and health (H).

Figure 1

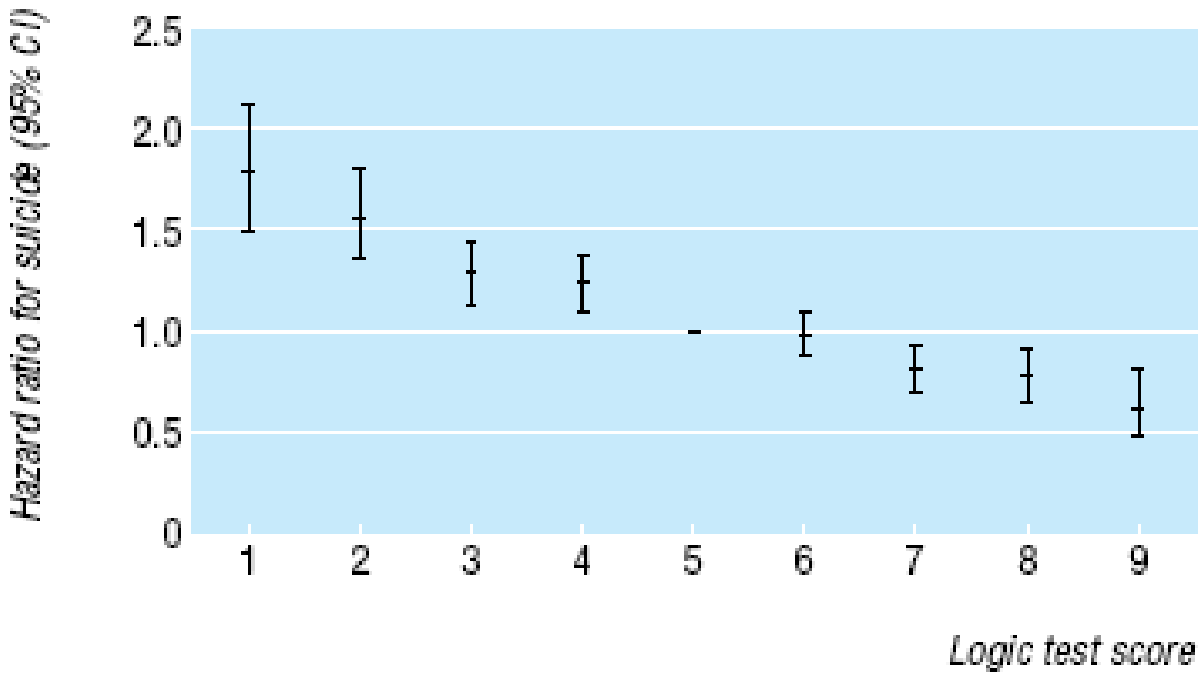
Hazard ratio for suicide (95% CI)
2.5
2.0
1.5
1.0
0.5
0
1
2
3
4
5
6
7
8
9
Logic test score

Figure 2

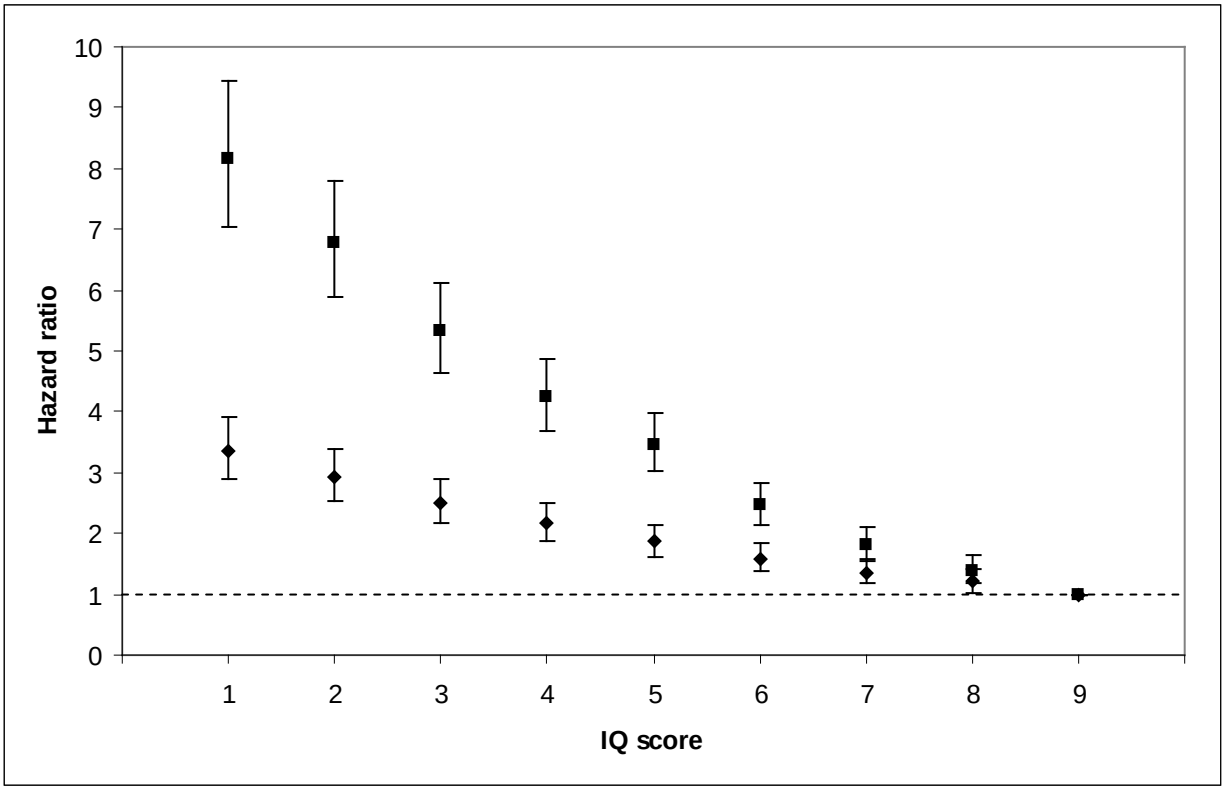

Figure 3

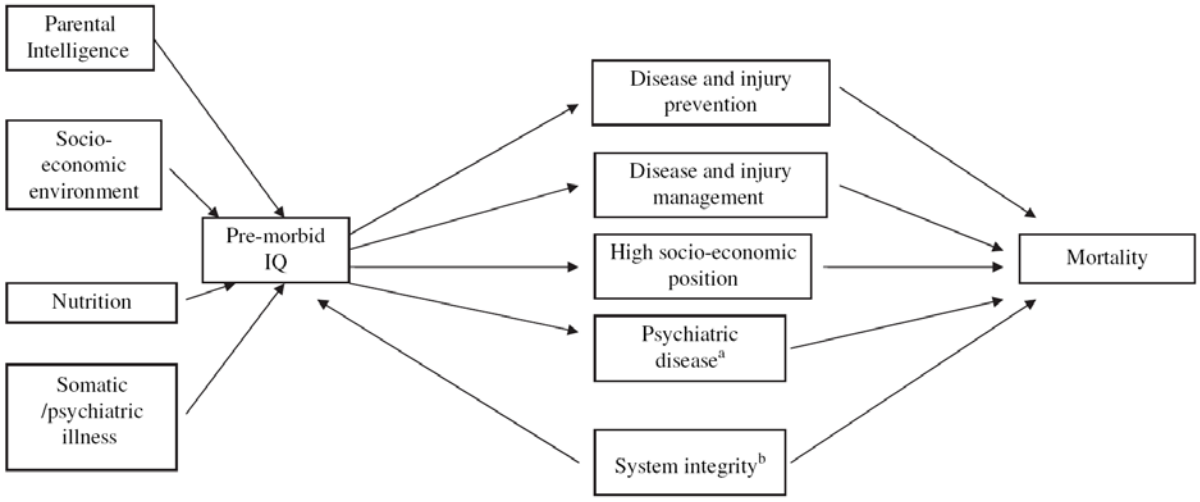
Parental Intelligence
Socio-economic environment
Nutrition
Somatic /psychiatric illness
Pre-morbid IQ
Disease and injury prevention
Disease and injury management
High socio-economic position
Psychiatric disease[a]
System integrity[b]
Mortality

Figure 4

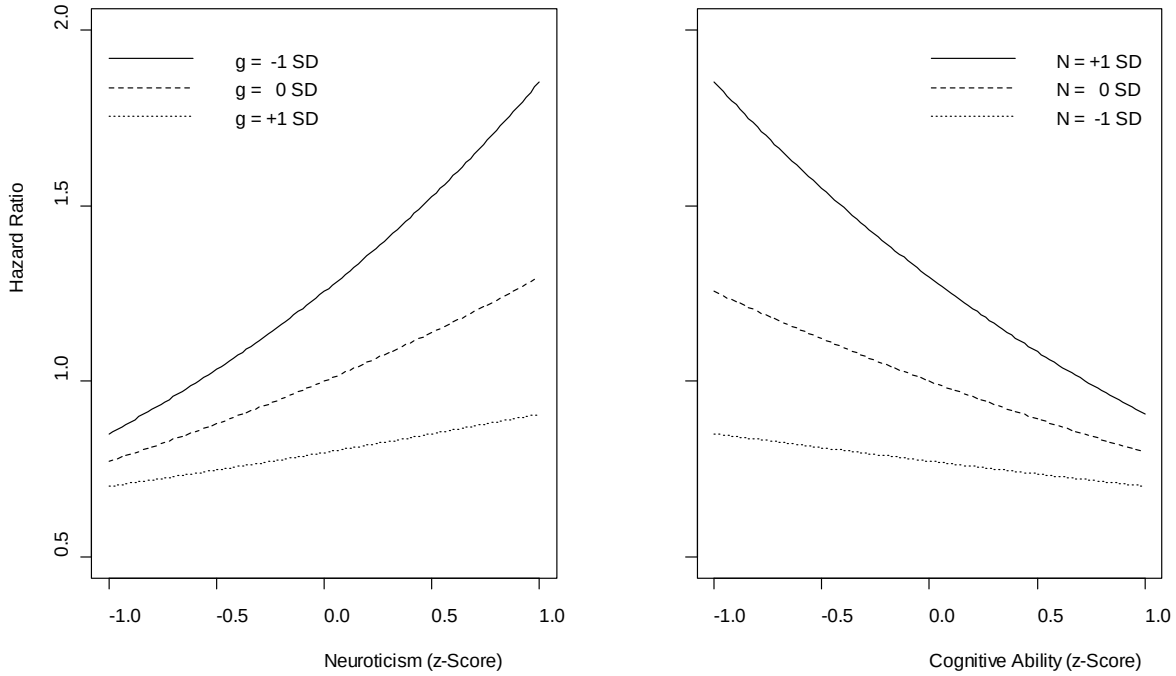

Figure 1 from Weiss et al. *Psychosomatic Medicine, 71*, 385-394

Figure 5a

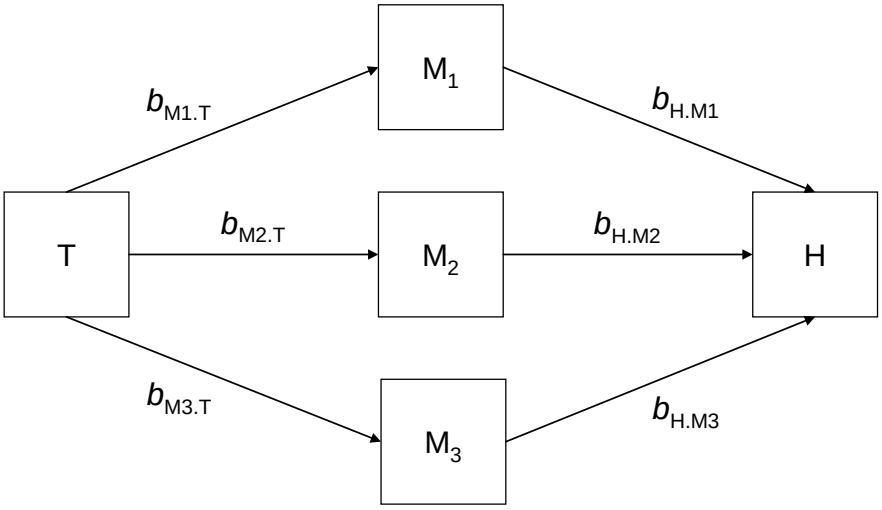

Figure 5b

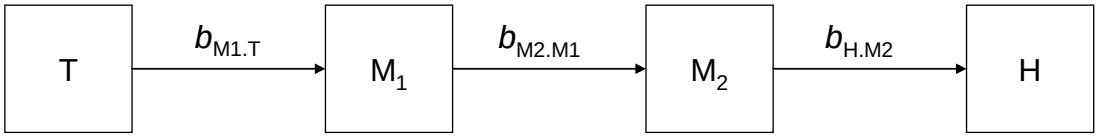

Figure 5c

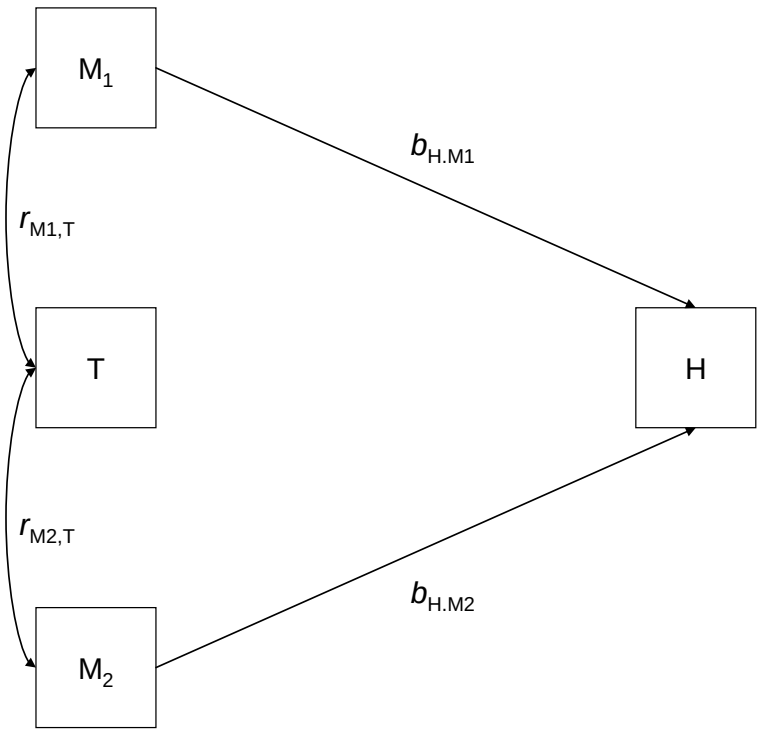

Figure 5d

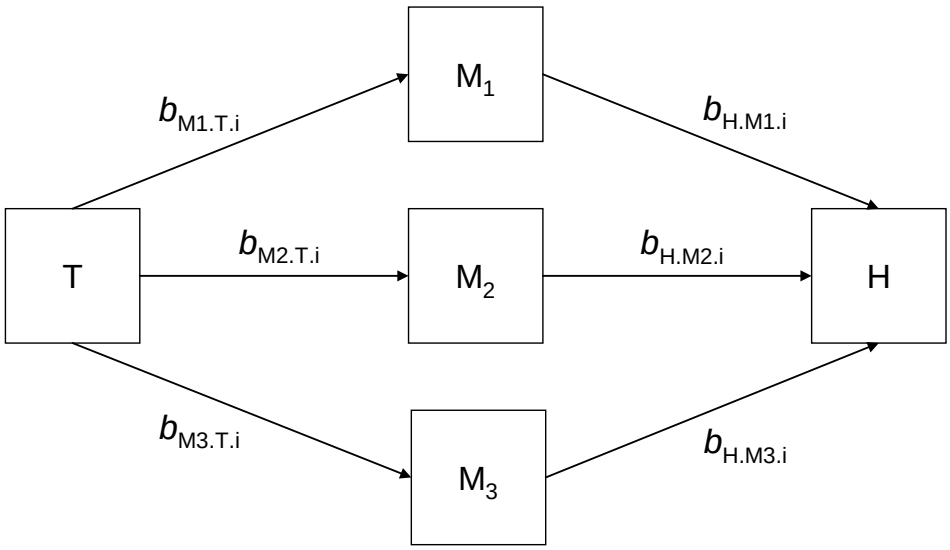

Figure 5e

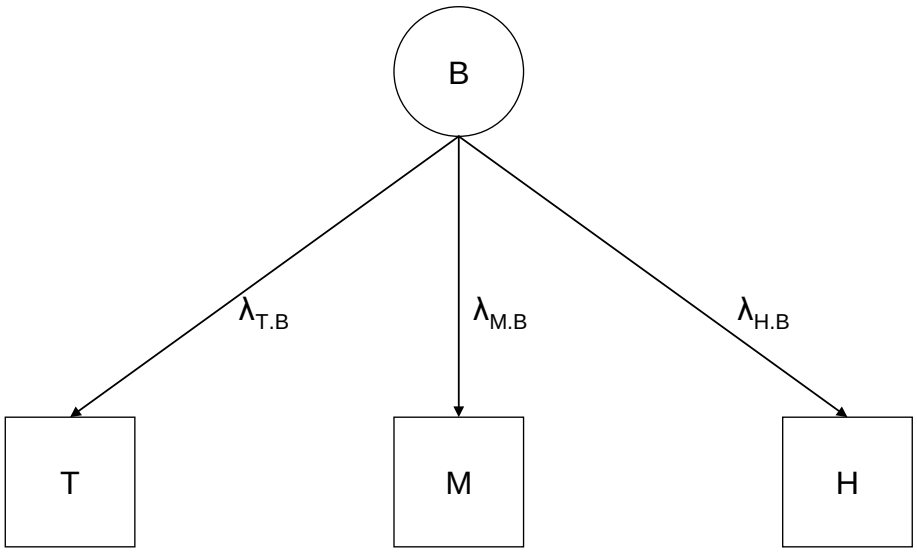
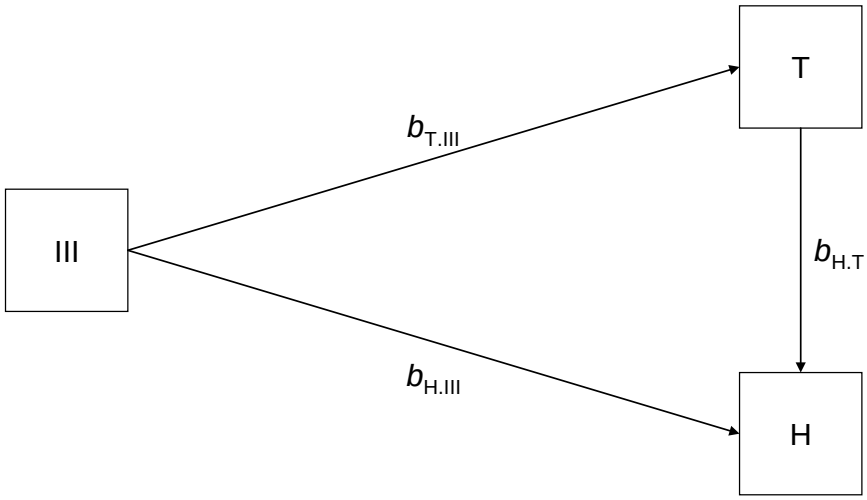
Figure 6