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The Psychological, Social and Biological Determinants of Health: A Review of the Evidence

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Socioeconomic status and health

Socioeconomic inequalities in health are essentially universal: poorer health is more common in the disadvantaged. In all countries where data are available, mortality has been shown to be higher among those in less-advantaged socioeconomic positions, regardless of whether socioeconomic position is indicated by education level, occupational class or income level (Mackenbach *et al*., 2003) and this is evident for both men and women. These variables are interrelated, but represent different dimensions of socioeconomic status (Kristenson *et al*., 2004). Compared to present occupational status, education relates more to social status in early life, whereas income describes the availability of material resources but also a level of status. For measures of education, occupation and income, people with low socioeconomic status in general tend to suffer poorer health.

In a number of large scale studies a gradient appears across the social spectrum, rather than a threshold effect, indicating that it is the position within the social hierarchy that is important for health (Marmot & Wilkinson, 1999). Studies examining the associations of each socioeconomic indicator with mortality and morbidity have repeatedly shown consistent gradients. These gradients have been shown for all cause mortality, but also for a wide range of diseases, especially coronary heart disease, diabetes, respiratory diseases, arthritis, poor birth outcomes, and for accidents and violent deaths (Marmot & Wilkinson, 1999).

The inverse relationship between socioeconomic position or status and health is one of the most consistent epidemiological findings. The social distribution of physiological risk is partly a reflection of the social patterning of unhealthy behaviours. Unhealthy diet, lack of exercise, tobacco and drug use, have now become strongly associated with social disadvantage.

However, an extensive volume of research identifies social factors as being at the root of these inequalities in health. For example, in studies of Scottish men (MacLeod *et al*., 2005), British civil servants in the Whitehall II study (Marmot *et al*., 1991) and participants of the Helsinki Health Study (Lahelma *et al*., 2004), individual social disadvantage has been consistently associated with poorer health and higher rates of mortality regardless of the measure of social position used and even after controlling for other risk factors. Area-based socioeconomic measures have also been shown to be independently associated with higher risk factors for morbidity and mortality in a number of studies (Davey Smith *et al*., 1998; Riva *et al*., 2007; Vescio *et al*., 2009). These studies have shown that there is an increased risk of mortality in deprived areas compared to more affluent ones.

Impact of socioeconomic status on health over the life course

Socioeconomic circumstances at different stages of the life course can influence specific adulthood health outcomes. Increasing evidence indicates that socioeconomic circumstances during the early years of life are important determinants of later health outcomes and disease risk in adult and older life, with the propensity for poor health in adulthood being greatest among those from disadvantaged backgrounds. It has been

claimed that the risk of mortality accumulates during the life course (Power *et al*., 2007; Ben-Shlomo & Kuh, 2002) and that exposure to risk factors may occur many years before the development of the outcome (Davey Smith *et al*., 1998). Whether increased morbidity and mortality in adulthood are the result of biological programming due to critical events *in utero*, the accumulation and interaction of harmful exposures along the pathway between infancy and adulthood, or a combination of both remains unclear for most diseases.

A number of studies to date have emphasised the importance of childhood social circumstances for adult mortality (Davey Smith *et al*., 1998; Ben-Shlomo & Kuh, 2002; Galobarades *et al*., 2006) and recently also for general health, which has been suggested to reflect ageing processes and the chronic conditions accumulated over the lifecourse (Ben-Shlomo & Kuh, 2002; Osler *et al*., 2009). To date most (Vagero & Leon, 1994; Davey Smith *et al*., 1997, 1998; Brunner *et al*., 1999; Galobardes *et al*., 2006; Power *et al*., 2007), but not all (Lynch *et al*., 1994), suggest that childhood conditions are important predictors of risk regardless of social class destination in adulthood.

Coronary heart disease is a good example of an adult disease that develops throughout the life course. Although coronary heart disease manifests itself in adulthood, atherosclerosis, an important underlying process leading to the disease, may begin at a much earlier age. An increasing number of studies have examined the link between childhood socioeconomic circumstances and cardiovascular disease in later life. Adverse childhood socioeconomic position has been reported to be associated with a poorer health profile in mid adulthood (45 years of age), independent of adult social position and across diverse measures of disease risk and physical and mental functioning (Power *et al*., 2007). Individuals with the most disadvantaged backgrounds had poorer health profiles across multiple measures of disease risk and health function. At mid adulthood associations with childhood social class were identified for blood pressure, body mass index, high density lipoprotein, triglycerides, lung function, depressive symptoms and chronic widespread pain, with a general trend of deteriorating risk as quantified by participants' father's occupation from class I (professional occupations) to V (unskilled occupations). These findings are in line with previous studies showing associations with both child and adult socioeconomic status and position for cardio-respiratory risk in adult life.

Results from a small number of studies have also shown links between high levels of risk factors early in life and atherosclerosis in later life. Blood pressure, low density lipoprotein cholesterol levels, smoking and body mass index (BMI) measured between 12 to 18 years of age in the Cardiovascular Risk in Young Finns Study was associated with greater adult carotid intima-media thickness (cIMT), independent of adult levels of these risk factors (Raitakari *et al*., 2003). Likewise the Bogalusa Heart Study of young adults reported greater cIMT in participants who had higher levels of low-density lipoprotein cholesterol and BMI during childhood (Li *et al*., 2003). A systematic review of forty individual level studies reported a robust inverse association between childhood circumstances and cardiovascular risk in thirty-one of the reviewed studies (Galobardes *et al*., 2006). This review confirmed that the evidence supported the position that those who experienced worse socioeconomic conditions in their childhood, independent of their circumstances during adult life, generally were at greater risk of developing and dying from cardiovascular disease.

It is also well recognised that early life and childhood environment and diet are important in determining rate of growth, timing of maturation, final stature and health outcomes as an adult. Low birthweight is associated with cardiovascular disease in adulthood. Short adult height is also known to be a risk for cardiovascular and cancer mortality and for poor adult health (Wadsworth *et al*., 2002) and leg length in childhood is a marker for cardiovascular disease and cancer (Gunnell *et al*., 1998; Davey Smith *et al*., 2001). Longer leg length is associated with advantaged socioeconomic circumstances in childhood. Adult leg length is a useful indicator of adverse circumstances and poor nutrition in infancy and childhood as confirmed by data from the 1946 British Birth Cohort (Gunnell *et al*., 1998) and by the British Women's Heart and Health Study (Lawlor *et al*., 2003). Studies which have investigated the association between leg length and cardiovascular disease risk and mortality have shown an increased risk of mortality with decreasing leg length measured in both childhood (Gunnell *et al*., 1998) and adulthood (Davey Smith *et al*., 2001). These findings suggest that adverse diet and living conditions in childhood, for which leg length seems to be a sensitive indicator, are associated with an increased risk of coronary heart disease in adulthood. This association adds additional support to the evidence that pre-adult influences are important in the aetiology of coronary heart disease.

An inverse association with coronary heart disease risk is not reported for trunk length or sitting height in adults (Wadsworth *et al*., 2001; Davey Smith *et al*., 2001). Dental status is affected by oral health as well as general diseases over the life course and has also been shown to be strongly related to mortality (Thompson *et al*., 2004; Osler *et al*., 2009).

However despite this growing evidence that early life socioeconomic position contributes to morbidity and mortality in adulthood via a number of pathways, little is known about the biological mechanisms responsible for this observed relationship and the processes responsible for this accumulation of risk. Given the evidence that both childhood and adult socioeconomic position are associated with morbidity and mortality from specific causes, it follows that they are also likely to be associated with the biological and behavioural risk factors for those outcomes.

Coronary heart disease and socioeconomic status

Coronary heart disease (CHD) is a leading cause of mortality and morbidity in developed countries and in many populations shows an inverse social gradient as demonstrated by a higher incidence in areas of socioeconomic deprivation compared with socioeconomically advantaged areas (Davey Smith *et al*., 1998; Lawlor *et al*., 2005; Singh-Manoux *et al*., 2008a). Over the last two decades Scotland has seen a halving of mortality from coronary heart disease, although cardiovascular mortality in Scotland is still among the highest in Europe and globally (Muller-Nordhorn *et al*., 2008). Moreover, the rate of decline appears to be slowing in young adults in deprived groups and has been attributed to poor lifestyle choices and behaviours rather than a decline in medical management of coronary heart disease (O'Flaherty *et al*., 2009).

Traditional cardiovascular risk factors, including smoking, high blood pressure, high cholesterol and diabetes, do not fully account for or explain the excess burden of cardiovascular diseases in the population (Everson-Rose & Lewis, 2005). It is increasingly accepted that variation in the prevalence of classical risk factors only partially accounts for the gradient in CHD (Shrewy *et al*., 1992; Tunstall-Pedoe *et al*., 1997; Capewell *et al*., 1999) and there is a need to uncover other potential explanatory variables.

One candidate is chronic 'low grade' activation of the innate immune system which may start early in life (Atabek, 2008) and be influenced by cumulative effects of socioeconomic status over the life course (Koster *et al*., 2006; Tabassum *et al*., 2008). Other novel possible contributors to increased CHD risk are insulin resistance and endothelial dysfunction (Yudkin *et al*., 1999; Pollitt *et al*., 2008).

Low socioeconomic status has been related to higher levels of inflammatory markers. Inflammation, a biological response of the immune system, has been associated with increased morbidity and mortality across the life course, from childhood and adolescence to old age (Pollitt *et al*., 2008). Meta-analyses of results from prospective studies suggest that inflammatory markers such as fibrinogen, C-reactive protein (CRP) and interleukin-6 (IL-6), acute phases proteins induced as a part of the immune response to acute infection or injury, and haemostatic markers such as von Willebrand factor (vWF) and tissue plasminogen activator antigen, are all part of the evolving understanding of cardiovascular disease, including atherosclerosis, stroke and myocardial infarction (Danesh *et al*., 2005).

Evidence to date supports the suggestion that inflammation plays an important part in the process of atherosclerosis and the development of coronary heart disease (Jousilahti *et al*., 2003). It has also been suggested that atherosclerosis is primarily an inflammatory disease (Ross, 1999). CRP has been associated with the presence and severity of atherosclerosis, and has been found to predict acute cardiovascular events in middle-aged men in the Monica Study (Koening *et al*., 1999) and older adults in the Heart and Soul Study (Lubbock *et al*., 2005). The association between plasma fibrinogen concentration and the risk of cardiovascular disease has also been demonstrated in a number of studies (Danesh *et al*., 2005).

The relationship between socioeconomic status throughout life and chronic inflammation was assessed in a recent publication by Tabassum *et al*., (2008) who reported accumulative effects of socioeconomic status on CRP and fibrinogen. Likewise, in a large cohort of middle aged white and African-American adults the accumulation of adverse socioeconomic conditions (at both the individual and neighbourhood level) throughout life was associated with elevated systematic inflammation in adulthood (Pollitt *et al*., 2008). A similar association was reported by Jousilhti *et al*., (2003) in 1500 middle aged Finnish men, where the inverse associations between socioeconomic status and markers of inflammation were particularly strong in men below 60 years of age. In the Whitehall II study of civil servants, social position was reported to be inversely associated with IL-6 and CRP and participants who had mild depression also had impaired endothelial function (Hemingway *et al*., 2003). In a study of over 3000 American adults over 70 years of age, low socioeconomic status was

also associated with significantly elevated levels of IL-6, CRP and tumour necrosis factor- α (TNF-α) (Koster *et al*., 2006).

Multi-ethnic studies have also shown an association between low socioeconomic status and the incidence and prevalence of cardiovascular disease and elevated inflammatory markers but highlight significant differences between racial and ethnic groups. A systematic review of thirty-two relevant studies concluded that poverty and non-white race were associated with elevated CRP levels amongst adults (Nazmi & Victora, 2007). Compared to white populations, blacks, Hispanics and South Asians had higher recorded CRP levels. The impact of socioeconomic status on inflammation was found to be the same as in white populations. The Multi-Ethnic Study of Atherosclerosis, involving more than 6000 adults aged over 45 years, reported that in multi-ethnic populations persons of lower socioeconomic position had a greater inflammatory burden than those of high socioeconomic position (Ranjit *et al*., 2007). An analysis of the data from the US National Health and Nutrition Examination Survey of nearly 80000 American adults reported that African Americans, Hispanics and women were more likely to have high levels of CRP, compared to the white American population (Alley *et al*., 2006). Socioeconomic status was related to higher CRP levels, but this effect was greatest at very high CRP levels (>10mg/L), with the authors suggesting that differences in very high CRP may be due to factors beyond acute ill health and may reflect chronic health, behavioural and disease processes associated with low socioeconomic status.

Early life socioeconomic status has also been linked with raised levels of inflammatory markers in a number of studies (Kivimaki *et al*., 2005; Pollitt *et al*., 2007; Miller & Chen, 2007) but not in others (Gimeno *et al*., 2008a). Low childhood social class and education levels were associated with elevated levels of CRP, fibrinogen, white blood cell count and von Willebrand factor in white children (Pollitt *et al*., 2007). The findings were less consistent among African-American children. However in a recent study which took a life course perspective, the association between socioeconomic position and CRP concentrations was seen in early adulthood, 24-39 years, but not in childhood or adolescence in the Cardiovascular Risk in Young Finns Study (Gimeno *et al*., 2008a). The authors commented that the socioeconomic differences in CRP seen in adult life do not appear to be determined at conception or to be a pathway starting early in life and tracking in adulthood. This cohort study has also reported a direct correlation between socioeconomic status, CRP levels and cIMT (Kivimaki *et al*., 2005), a non-invasive measure of atherosclerosis and a reliable indicator of future risk of coronary heart disease. The authors reported that the observed interrelations were driven by the effects of adiposity.

The association between low level inflammation and cardiovascular risk, although quite a recent finding, is well established. It remains to be established, however, if the risk of cardiovascular disease could be reduced by reducing levels of inflammation markers in individuals.

The endothelium plays a key role as the signal transducer that regulates vascular inflammation. Endothelial dysfunction and increased arterial stiffness (loss of arterial

elasticity) have also been reported to be early markers of accelerated vascular ageing in young adults and in children with a family history of hypertension. Low childhood birth weight has also been associated with an increased incidence of adult cardiovascular disease, insulin resistance and high blood pressure (Barker *et al.*, 1993). In a study of 44 nine year old children, half of whom had a low birthweight, impaired endothelial function and a trend towards increased carotid stiffness was identified in the low birth weight group (Martin *et al.*, 2000).

Carotid ultrasound is an efficient validated tool for assessing the degree of atherosclerosis in an individual. Measurement of the carotid artery wall intima-media thickness is a commonly used marker of atherosclerosis and a valid presymptomatic predictor of coronary heart disease. The ultrasound detection of carotid plaques is also highly informative. Plaque score has been shown to be associated with risk of myocardial infarction and stroke (van der Meer *et al.*, 2004). Several studies have examined the relationship between socioeconomic status and ultrasound markers of atherosclerosis. Most studies have investigated individual level measures of socioeconomic position and their relation to cIMT (Ebrahim *et al.*, 1999; Lamont *et al.*, 2000; Kivimaki *et al.*, 2006). Associations between community level socioeconomic status and cIMT and carotid plaque score were reported in a study of middle aged American adults (Petersen *et al.*, 2006). This study showed community disadvantage to be associated with greater intima-media thickness irrespective of individual income or educational attainment level. Similarly, in a study of over 4000 Swedish adults, significant associations between area level deprivation and carotid plaque score were also reported (Rosvall *et al.*, 2007). These associations were slightly reduced on adjusting for individual level markers of socioeconomic position. In the Rotterdam study, the non-invasive measures of atherosclerosis, carotid plaque score and cIMT, were strong predictors of myocardial infarction (van der Meer *et al.*, 2004). The authors concluded that the relatively crude measures directly assessing plaques in the carotid artery predicted myocardial infarction equally as well as the more precisely measured and technical cIMT. However, a number of studies have now reported plaque presence to be more strongly predictive than cIMT of future cardiovascular events (Belcaro *et al.*, 2001; Stork *et al.*, 2004). Likewise, the Tromso Study of over 6000 adults aged 28 to 84 years found carotid plaque area to be a stronger predictor than cIMT of first myocardial infarction, particularly in women (Johnsen *et al.*, 2007).

Cognitive function, socioeconomic status and health

Research into cognitive function in prospective cohort studies reveals that there is likely to be multiple determinants of cognitive decline. Besides age, the other two general factors most widely implicated and accepted are biology and socioeconomic factors.

Accumulating evidence shows that low socioeconomic status is related to poor mental and physical function and that this association has a basis in both social and biological factors. A number of studies have examined the association between educational attainment level and cognitive change (Farmer *et al.*, 1995; Lee *et al.*, 2003) and found it to be strong. However the evidence of an association between other indicators of socioeconomic status – such as

occupational level, income and area of residence – and cognitive function remains limited. In a large community study of American older adults aged 70-79 years, people with low socioeconomic status had an increased risk of cognitive decline compared to people with a high socioeconomic status. This was found across a range of indicators of socioeconomic status, the strongest association being with education (Koster *et al*., 2005). As expected, disease prevalence and risk factors were elevated in the population of low socioeconomic status. However, biomedical factors could only explain 5% of the socioeconomic differentials in cognitive decline. Neighbourhood deprivation in urban areas has also been shown to be associated with poorer cognitive function in older adults, independent of the effects of individual and household socioeconomic factors (Lang *et al*., 2008). A similar study has also reported a significantly higher prevalence of cognitive and functional impairment in elderly individuals living in socioeconomically deprived areas regardless of their own socioeconomic status (Basta *et al*., 2007). A small number of research studies have shown that ethnic minority status is also a risk factor for poorer cognitive function in older age (Zsembik & Peek, 2001; Masel & Peek, 2009) but the impact of ethnicity on the rates of cognitive decline in old age remain unclear.

Several studies have also suggested an association between lifestyle and cognitive decline. Cognition is affected by the classical lifestyle risk factors which are associated with deprivation related morbidity and which often exert their biological effects via harmful health behaviours including smoking, excessive alcohol intake and obesity (Elwood *et al*., 1999; Kalmijn *et al*., 2002; Sabia *et al*., 2008). Education; physical activity including walking; mentally demanding work and managerial positions; leisure and intellectual activities; and living with a partner have all been reported to predict a more favourable cognitive status in middle and later life (Weuve *et al*., 2004; Hakansson *et al*., 2009). However a recent study investigating the association between long working hours (more than 55 hours per week) and cognitive function identified that long working hours may be one of the risk factors having a negative effect on cognitive performance in middle age (Virtanen *et al*., 2009).

Childhood socioeconomic status is associated with cognitive ability and achievement during childhood, including IQ, literacy, achievement tests and grade retention. These associations are well established and are observed throughout development, from infancy to adolescence and into adulthood (Noble *et al*., 2005, 2007). Cognitive function in childhood highly correlates with cognitive function in adulthood and is influenced by biological and social conditions in early life which then influence adulthood circumstances.

Another novel hypothesis which has been posed as a 'fundamental cause of social inequalities in health' is intelligence, assessed by a measure of individual IQ (Gottfredson, 2004). Observations have shown that low IQ scores ascertained in childhood, mid-life and older ages are associated with elevated rates of mortality and morbidity (Hart *et al*., 2003; Batty *et al*., 2006, 2007). IQ scores are socially patterned and a link has also been reported between literacy and health related behaviours, injuries and the self-management of ill health (Gottfredson, 2004). In a systematic review of individual level studies linking early IQ with later mortality, higher IQ in the first two decades of life was consistently related to lower rates of total mortality in middle to late adulthood (Batty *et al*., 2007). In the West of

Scotland Twenty-07 study, indices of socioeconomic position were significantly associated with health outcomes in the expected direction. Scores from a test of IQ did not explain the socioeconomic gradients in health of these participants but did lead to a reduction in the magnitude of the gradients (Batty *et al*., 2006). Lower childhood IQ was shown to be related to higher mortality risk for coronary heart disease in a prospective observational study linking the Midspan Studies and the Scottish Mental Survey 1932 (Hart *et al*., 2003). However, in a recently published population based longitudinal study of children followed from age 10 to 75 years in Sweden, mortality differences by own educational attainment in adulthood were not explained by childhood IQ (Lager *et al*., 2009).

Socioeconomic conditions across all stages of the life course do appear to make a unique contribution to cognitive function in later life. Evidence also suggests that in terms of cognitive function, origin is not necessarily destiny, as disadvantaged socioeconomic circumstances in childhood may be overcome to a degree by upward mobility in later in life.

Biomedical factors, including common diseases and their risk factors, are important mechanisms that help to explain the potential association between low socioeconomic status and cognitive decline. Evidence is now well established that vascular risk factors, such as blood pressure (Singh-Manoux & Marmot, 2005) and indicators of vascular disease are associated with cognitive impairment and dementia (Muller *et al*., 2007). Vascular disease has been reported to be predictive of poor cognitive function in the general population. The presence of vascular disease was associated with diminished cognitive function in a large cohort of middle aged adults (Singh-Manoux *et al*., 2003). The common carotid artery intima-media thickness (cIMT) is one of the more recently identified vascular risk factors. As a marker of the structural and functional properties of the vessel wall and an early indicator of atherosclerosis, it has been shown to have a robust association with cardiovascular disease. An association between cIMT and cognitive impairment has also been reported in a number of studies (Breteler *et al*., 1994; Cerhan *et al*., 1998; Muller *et al*., 2007). This association was also reported in analysis of the Whitehall II study. This research identified an overall association between cIMT and a number of measures of cognitive performance in middle aged adults in a low socioeconomic group (Singh-Manoux *et al*., 2008). The authors commented that individuals with high socioeconomic position in their study appear to have a high cognitive reserve which is preventing functional manifestations of atherosclerosis.

Several studies have also suggested that raised levels of inflammatory markers are associated with cognitive decline in dementia and normal ageing. To date the most frequently investigated markers of systemic inflammation in relation to cognitive decline are C-reactive protein (CRP), intercellular adhesion molecule (ICAM) and interleukin-6 (IL-6). In an investigation of inflammatory marker concentrations and cognitive performance in a healthy ageing population, high concentrations of CRP were found to be indicative of impaired cognitive function (Teunissen *et al*., 2003). Gimeno *et al*., (2008b), in a study of middle aged adults found that raised levels of the inflammatory marker IL-6 were moderately associated with lower cognitive performance status but there was little evidence of an association with cognitive decline in midlife. This association was more evident in men than women. In the prospective Edinburgh Artery Study, Raffnsson *et al*., (2007) found that systematic markers of inflammation (IL-6 and ICAM) were associated with progressive decline in cognitive abilities in older people.

Evidence appears to support a clear link between socioeconomic status and cognitive function. The impact of individual indicators of socioeconomic status, namely education and income, appear to be important. Increasing evidence also supports the biological link between impaired cognitive ability and increased levels of systemic inflammation and vascular damage. The relationship between high inflammation and impaired cognitive function appears to be reduced but not abolished by individual-level markers of low socioeconomic status. Current evidence appears to support that lower cognitive performance in deprived individuals at present appears to be better explained by the disadvantages of poor education and low income than biological factors.

The impact of personality on health

Among the psychological factors that impact on health, personality, that is stable individual differences in thinking, feeling and behaving, plays a pivotal role. It underpins the consistency with which we think, act and feel across different situations over time. Adult personality traits are thought to be derived from early life differences in temperament which are partly genetically determined and shape exposure to social experiences (Steptoe & Molloy, 2007). These personality traits predict a range of outcomes with some consistency, including the quality of family and social relationships, marital status and satisfaction, occupational choices, political attitudes and criminality (Ozer & Benet-Martinez, 2006). Longitudinal studies have also demonstrated that personality traits identified in childhood are able to predict health outcomes occurring in later life such as overweight and obesity, unintentional injuries, metabolic syndrome and longevity (Vollrath, 2006).

The association between personality factors or traits and a range of both positive and maladaptive (negative) health behaviours is now well established and known to influence morbidity and mortality. In a study of 716 men and women aged over 58 years, positive affect or outlook was associated with greater social connectedness, optimism, adaptive coping responses, lower depression and favourable physiological functioning (Steptoe *et al*., 2008). On the other hand negative affect was associated with negative relationships, greater exposure to chronic disease, depressed mood, poorer mental health and pessimism. Positive affect was related to protective social and psychological resources, but not to chronic stress exposure. Happier individuals do not experience lower levels of chronic adversity in their lives, but have greater protective resources that enable them to handle problems flexibly and effectively, together with better mental health (Steptoe *et al*., 2008).

People who demonstrate higher levels of hostility and anger are at greater risk for heart disease and atherosclerosis (Whiteman, 2006). Extraverted and neurotic characteristics have both been shown to be predictive of mortality. High neuroticism is associated with poor subjective health status and also predicts clinically-defined chronic illness (Hudek-Knezeviç & Kardum, 2009). Personality attributes have been reported to be associated with increased risk of hypertension (Barefoot *et al*., 1983; Carroll *et al*., 1997), coronary heart disease and atherosclerosis (Barefoot *et al*., 1995), myocardial infarction (Everson *et al*., 1997) and all-cause mortality (Everson *et al*., 1997; Nabi *et al*., 2008a). In the Baltimore Longitudinal Study

of Aging, where participants were followed for up to fifty years, longevity was associated with being conscientious, emotionally stable (low neurotism) and active (Terracciano *et al*., 2008). The association of personality traits with longevity was reported to be independent from the influence of smoking and obesity. Higher levels of neuroticism have also been shown to be predictive of shorter survival in an elderly North American male sample (Mroczek & Spiro, 2007), while Nabi *et al.,* (2008a) reported that the personality feature 'neurotic hostility' (traits of negativism, resentment and hostility), clearly predicted all cause and cause-specific mortality in a large French cohort. Coronary heart disease-prone personality types (individuals who experience anger, aggression and lack of autonomy) and anti-social personality types (individuals who exhibit psychopathic, impulsive, rebellious and hostile behaviours) were also associated with cardiovascular and external causes of mortality (accidents and suicides) respectively. Optimism was reported to be associated with a reduced incidence of CHD and total mortality in the Women's Health Initiative, a large cohort study of over 95,000 post-menopausal women in America. Cynical hostility (pessimism) was associated with an increased risk of total mortality and cancer-related mortality across the cohort whereas the trait of optimism (positive future expectations) was associated with a reduced incidence of coronary heart disease (Tindle *et al*., 2009). These features were independently associated with these important health outcomes in black and white women.

Morbidity and subjective wellbeing are also influenced by the interactions between personality and health behaviours. Smokers have been shown to score more highly on the personality factor of neuroticism, and lower on characteristics of agreeableness and conscientiousness than those who have never smoked (Terracciano & Costa, 2004). Openness to experience (a facet of extraversion) and low neuroticism have been associated with a more active decision-making style with respect to self-health care (Flynn & Smith, 2007), while high extraversion predicts a greater propensity to access health care resources, which in turn may have significant implications for morbidity and mortality (Chapman *et al*., 2009). The relationship between personality and the increasingly important problem of obesity is unclear. High neuroticism has been associated both with being underweight (Terracciano *et al*., 2009) and with obesity (Chapman *et al*., 2008) in adults. However in a large study of over 1000 adolescents, dimensions of personality were associated with fruit and vegetable consumption and sports-related physical activity (de Bruijn *et al*., 2005). Adolescents who consumed more fruit and vegetables were more agreeable and more open to experience. Extraversion was also reported to be positively associated with sport-related physical activity, a finding in line with previous studies in adults (Rhodes *et al*., 2003).

Whilst the influence of personality in these studies is likely to be mediated by positive or maladaptive health behaviours, an intriguing explanation for some personality-based differences in health may lie in inflammatory processes. Positive associations have recently been reported between hostility and emotional negativity and levels of interleukin-6 (IL-6) and C-reactive protein (CRP) (Coccaro, 2006; Marsland *et al*., 2008). Similarly, CRP has been positively associated with higher scores on "pessimistic worry" (a feature of neuroticism) in a large sample of 42 year old females (Henningsson *et al*., 2008). High neuroticism and low conscientiousness have also been associated with increased levels of IL-6 and CRP (Sutin *et al*., 2009).

The evidence that personality factors are associated with health-related behaviours that influence health status may have important implications for understanding why certain sub-groups within the population experience significantly better, or worse, health than others. Personality factors (such as hostility) have been found to be associated with lower socioeconomic status (as assessed by indicators of occupation, education and income) among adult men and women (Carroll *et al*., 1997; Christensen *et al*., 2004). In a large French cohort study, personality factors explained all-cause and cardiovascular mortality gradients observed for measures of adult socioeconomic position in men, but did not explain mortality in women (Nabi *et al*., 2008b). As described earlier, the marked gradient in health as a function of socioeconomic status is clearly demonstrated, with people living in deprived circumstances being significantly more prone than their affluent peers to ill health. Given the evidence here that neuroticism is associated with harmful health behaviours, it would be important to establish whether neuroticism tends to exacerbate the health problems of those living in deprivation while extraversion may offer a protective function.

Biological aging, telomere length and socioeconomic status

It is generally accepted that psychological stress leads to premature ageing and the earlier onset of disease. The evidence presented here has demonstrated links between disadvantaged socioeconomic circumstances, stress and indices of poor health, including risk factors for CHD and poor cognitive function. Researchers to date have tried to understand how 'stress gets under the skin' to give rise to this elevated disease risk. Access to resources, health behaviours and psychological characteristics explain some, although by no means all, of the socioeconomic gradient. One further mechanistic possibility put forward is that cardiovascular disease and cancers are in part age-related diseases, whereby socioeconomic disadvantage increases mortality risk by accelerating the ageing process (Batty *et al*., 2009).

The exact mechanisms of how such stress exerts this effect, including whether stress accelerates ageing at a cellular level and how cellular ageing translates to the ageing of the individual, is the subject of much discussion. Recent research points to the crucial role of telomeres and telomerase in cellular ageing and potentially in disease. Telomeres are DNA-protein complexes that cap chromosomal ends, promoting chromosomal stability (Epel *et al*., 2004). In people, telomeres shorten with age in all replicating somatic cells; therefore telomere dynamics (length, attrition) capture biological ageing above and beyond chronological ageing, such that shorter telomeres represent increased biological senescence. In a study of fifty-eight healthy women, mothers to either a healthy child or chronically ill child, the mothers of ill children were found to have the highest levels of perceived stress and to have telomeres shorter on average by the equivalent of at least one decade of additional ageing compared to low stress women (Epel *et al*., 2004), implicating shorter telomeres in the adverse sequelae of prolonged psychological stress.

To date a small number of studies have considered the impact of socioeconomic adversity on telomere length. The evidence presented in these studies at present is mixed, with a positive association reported by Cherkas *et al*., (2003) in a large cross section study of

female twins, and no association reported by Harris *et al*., (2006) in Scottish 1921 Lothian Birth Cohort, by Adams *et al*., (2007) in the Newcastle Thousand Families Study and by Batty *et al*., (2009) in the West of Scotland Coronary Prevention Study. In this last, most recent study, the largest cross sectional study to examine this relationship to date, no strong evidence was reported that any of four indices of socioeconomic status (educational attainment, employment status, area-based deprivation and physical stature) was robustly related to telomere length in this study of over 1500 men (Batty *et al*., 2009). The current balance of evidence does not provide clear support for a strong and consistent socioeconomic-telomere gradient. Further large scale research is required to confirm or refute this relationship.

Conclusion

In conclusion, the evidence presented here is complex, multi- disciplinary and dimensional, and rapidly expanding and yet demonstrates the significant impact of poor socioeconomic status and area level deprivation in creating and exacerbating ill health. This review further reinforces the evidence that people in our poorer communities at present have poorer health compared to those who live in more affluent areas, and explores some of the pathways through which this association is expressed.

This review carried out across population based and community studies, national and international research and investigations from varied scientific disciplines further highlights the complex and multifaceted nature of the interactions between the social, psychological and biological determinants of ill health. Taken together, the breadth and diversity of the research considered makes the relative consistency of the findings notable.

The evidence here has shown the impact of poor early life circumstances and low socioeconomic childhood status on the accumulation and development of risk factors for poor health outcomes as an adult; and the clear and well established associations between socioeconomic status and CHD and cognitive performance – and has tried to highlight some of the potential explanatory variables for these correlations. The review also considered the emerging fields of research which are assessing the influence of an individual's personality on their risk of disease and possible future health outcomes and the impact of accelerated biological ageing on stress and elevated disease risk. These aetiological links continue to need further exploration.

Inequalities in health matter. The relationship between measures of socioeconomic position and mortality is a strikingly consistent finding. Despite the well documented simplicity of the social position:health outcome association, more complex questions as to the mechanisms whereby such associations arise and how amenable they are to change through intervention remain unanswered. We need to continue to work across and integrate diverse disciplines that have usually worked independently to build a more sophisticated understanding of the determinants of health and health inequalities. We must also continue to recognise the conditions, in which people are born, grow, live and work, and age as being fundamental to both understanding the causes and taking action to redress health inequalities.

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