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**SOCIO-ECONOMIC STATUS AND PHYSICAL HEALTH  
OUTCOMES:  
THE NEED FOR CHANGE IN THEORETICAL  
FORMULATIONS**

Thesis presented in partial fulfilment of the requirements  
for the degree of **MASTER OF ARTS in PSYCHOLOGY**  
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## ABSTRACT

The Black Report (Department of Health and Social Security, 1980) , which was a seminal publication in the field of health inequality, proposed several possible theoretical explanations for the phenomenon of socio-economic health inequality. To date the models proposed in the Black Report have yet to be improved on, or developed greatly. While research in the field of socio-economic health inequality has been substantial, the state of theoretical formulation which attempts to explain such inequality has remained static. The phenomenon of socio-economic health inequality will be established by producing evidence for how socio-economic status impacts on health from many countries, but especially from the United Kingdom, North America, Australasia, and Europe. Potential pathways for socio-economic status to impact on health outcomes will be assessed, and an illustration of potential pathways will be provided with an application. The theoretical approaches of the Black Report, along with a more recent conceptualisation, will be discussed. The potential contribution of psychological factors to socio-economic health inequality will be considered following the establishment of the relationship, proposal of potential pathways, and theoretical formulation discussions to demonstrate how such factors are involved in socio-economic health inequality. From these first four sections it can be deduced that current theoretical formulations to explain socio-economic health inequality are deficient. To contend with this deficiency it is proposed that a more holistic approach, which includes psychological factors, is necessary. Future research should seek to empirically validate links within the confines of a more holistic framework if our understanding of the relation between socio-economic status and physical health outcomes is to improve.

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## 1.0 ESTABLISHING THE RELATIONSHIP

### **1.1 Introduction**

Socio-economic status (SES) is a multidimensional construct which denotes a persons social standing within a population in terms of that individuals material and social status. Thus, SES is the categorisation of people in terms of their social and economic positions within the context of a wider social framework, usually the society within the confines of a country. Common indexes of SES include income, education, occupation, transport, geographic location, and housing.

Health, like SES, is a deceptively complex concept. Often health is only considered when it is deemed absent or deficient. The biomedical approach to defining health tends to dichotomise the concept into physical and mental health. Psychiatry is that branch of medicine designated to deal with mental health problems, whereas physical health problems are subdivided into several subdisciplines depending on which biological system is deficient or problematic. In the biomedical conceptualisation of mental disorders, the search for a "cure" generally involves the manipulation of biological factors, such as neurotransmitters in the brain, by means of medication. Traditionally the biomedical approach ignores contextual factors in the etiology and production of health outcomes.

Physical health outcomes will be used most of the time as the measure of health because such outcomes are generally easier to operationalise than mental health outcomes. Mental health definitions are subject to almost constant change, whereas physical health definitions seem to remain static.

A more comprehensive means of conceptualising health is known as the biopsychosocial approach. In this approach factors other than purely biological, such as psychological, social, and cultural, are implicated in the health outcomes of the individual. The biopsychosocial approach is at the heart of this research because it embraces the holistic nature of the SES-health relationship. To date the explanation of SE health inequality has been piecemeal at best. The integration of such piecemeal evidence into a more holistic theoretical framework,

to permit a more comprehensive explanation of the SES-health relationship, is the aim of this thesis.

I will focus much of my analysis on the SES-Health relationship on developed nations for several reasons. Firstly, in most developing nations the SE differences in health outcomes are so blatantly obvious that further research is unnecessary to establish a clear gradient of inequality in health as a function of SES (Wilkinson, 1989). Secondly, the type of material deprivation experienced in developing countries compared to developed nations is more extreme, and therefore health outcomes should not really be compared because pooling such information would not give a clear picture of the relationship. Pooled inequality would be an underestimate of the phenomena in developing nations and overstatement of it in developed nations. Thirdly, most of the research carried out on this topic has been done in developed nations, therefore availability of information was a criteria in my decision to limit the geographical context of my analysis. Finally, it is my contention that socio-political ethos has a powerful influence on health outcomes. Patterns of SES are a good approximation of the outcome of socio-political ideology being operationalised in government activity.

Various factors must come into consideration as mediators for the structural features that directly influence health outcomes (Faresjo, 1992). Psychological factors must mediate socio-economic (SE) health inequality otherwise the relationship would be a direct linear one-to-one, which it clearly is not (Wilkinson, 1989). Psychological factors include those external to the individual (that is, environmental), and those internal (that is, dispositional). Psychological factors differentially impact on the relationship due to individuals having different dispositional characteristics and circumstances.

## **1.2 The Evidence**

The organisation of evidence for SE health inequality is a complex issue. Organisation can revolve around key concepts, theoretical approaches, geographic location of research, chronology, or even approaches of various research enterprises. The evidence here will be presented country by country (geographically) and chronologically, and the key concepts will be extrapolated at the conclusion of the chapter.

Probably the best place to start looking for academic research on the topic is the UK. The Black Report (Department of Health and Social Security [DHSS], 1980) was a seminal paper in the research on the SES-Health relationship. Since the publication of the report much academic interest and subsequent publication has resulted. The Black Report was a government sanctioned research initiative instigated to examine the influence of SE factors on health outcomes in the UK. A working party of known experts in the field of health inequality analysed census statistics to assess the existence and extent of SE health inequality in the UK.

The working party did more than simply report on health inequality. They provided four potential theoretical explanations for the phenomenon. The explanations the working party proposed form the basis for much subsequent research and comment, and will be discussed in detail in chapter 3. However, a brief overview of the explanations is in order. Essentially one explanation suggests the phenomena is a function of measurement error (artefact). A second approach (social selection) proposes health leads to SES outcomes, with poor health being synonymous with low SES outcomes. A third approach (cultural/behavioural) argues that individual behaviours are responsible for the phenomenon, whereas the last approach (social causation) proposes that structural factors within a society lead to differential health outcomes which differ systematically according to SES.

### *1.2.1 United Kingdom*

Recent commentary by researchers in the UK and elsewhere simply reiterates what has been promulgated repeatedly since the Black Report, that is, the socio-economic health divide (inequality) continues to predominate, and even appears to be expanding in some places (Black, 1993; Kuh & Wadsworth, 1993).

Black (1993) suggested that in times of conservative political ideology the "gap" widens, and illustrates this with the example of how rates of tuberculosis have increased in New York during the years of the Reagan administration. This is an infectious illness that has been virtually absent from most developed nations for decades. However, during a conservative

approach in an era of economic downturn a very preventable illness is once again a public health problem in one of the most "advanced" of developed nations.

In a study examining causes of mortality in England and Wales between 1979 and 1983 researchers found clear social class based gradients for all major causes of death (Carroll, Bennett, & Davey-Smith, 1993). The steepness of the gradient reflected the severity of inequality, with a steeper gradient reflecting greater inequality, and flatter reflecting less inequality. These researchers assessed standardised mortality ratios (SMR's) to uncover steep gradients for lung cancer, bronchitis, and pneumonia with flatter, but still significant, gradients for ischemic heart disease, cerebrovascular disease, and motor vehicle accidents.

In research examining the SES-health relationship, the Whitehall I study (Marmot, Shipley & Rose, 1984), found an even steeper than expected mortality gradient. A somewhat homogenous sample of British Civil Servants (N=17530), homogenous in that all the work was non-manual in nature, was used for the study. The research was longitudinal in design and examined health outcomes over a period of ten years (1967-1977). The researchers found a strong inverse relationship between SES and health outcomes with those of the lowest occupational category having mortality rates three times greater than those of the highest occupational category. This finding was considerably greater in magnitude than the findings of the Black Report. The authors summarised that due to the size of the differentials that neither social selection, artefact, or cultural/behavioural explanations were sufficient enough to explain the significance of observed differences in health outcomes. They tentatively concluded that social causation was the best explanation for observed results.

In an analysis of other data from the Whitehall I study researchers found that non-ownership of a vehicle (with vehicle ownership as a proxy for SES) was predictive of significantly greater mortality in all occupational groups (Davey-Smith, Shipley & Rose, 1990). Transport status can be thought of as a proxy for SES because of the costs involved with ownership. In fact, the age adjusted mortality differential between non-owners of cars in the lowest SE group and owners in the highest SE group was 4.3, which was substantially larger than previous occupational based class measures such as those used in the Black Report (DHSS, 1980). The findings of greater mortality risk for non-owners is another illustration of the SE

health gradient from a different perspective. What is even more significant is that substantial differences occurred even within SE groups, which indicates transport status may even be an important SE variable.

In the Whitehall II study (Marmot, Davey-Smith, Stansfeld, Patel, North, Head, White, Bruner & Feeney, 1991) researchers examined a new cohort of 10314 British civil servants over a shorter three year period (1985-1988). The threefold difference between highest and lowest SE groups in mortality found in the first study was not replicated, although differences were still substantial. There were no substantial changes in morbidity gradient over the twenty year period from the first sampling in the Whitehall I study to the sampling for the Whitehall II. There was an inverse relationship between SES (in terms of occupational status) and prevalence of angina, ischemia, and chronic bronchitis. Self-reported health status improved with employment grade as did cessation of self "injurious" behaviours such as tobacco consumption or poor diet. As for Whitehall I health behaviours were partly responsible for observed differences. However, the social patterning of such behaviours needed to be taken into account to understand the true nature of the causes of SE health inequality. Structural factors such as social environment, work environment, and income inequality were proposed as key causal agents involved in maintaining SE health inequality.

It has been assumed that because mortality rates have dropped in recent years, and life expectancy has improved over the same period, that the distribution of gains has been evenly distributed by social class (Wilkinson, 1986). Pamuk (1985) provided evidence consistent with the assertion that SES differentially impacts on health outcomes. Further, despite the first two assertions being true, Pamuk demonstrated these gains have not been evenly distributed across SE groups. Pamuk's research used more methodologically sound statistical procedures than much of the prior research, and in doing so overcame some of the methodological criticisms of previous research (eg. Stern, 1983), such as numerator/denominator bias, class reclassification, and changing class size.

Pamuk accepted that health status could be responsible for some of the variance in SES in terms of social mobility, but decried the assertion that such a mechanism (social selection) could account for the size of the discrepancies found in the distribution of health outcomes.

Pamuk accepted health behaviours could be implicated in observed discrepancies, but argued that such behaviours appeared to be patterned according to social class. She concluded that structurally defined social forces interacted to result in differential health outcomes, that is, social causation was operating to maintain SE health inequality.

In a selected review of the literature on SES and disease outcomes researchers provided several very intriguing insights (Marmot, Kogevinas & Elston, 1987). Firstly, they found that while mortality in all the social groups had declined, the rate of decline was not evenly distributed by social class. One potential and interesting explanation proposed for this trend was those in higher SE groups were more likely to adopt healthier behaviours sooner than those in lower SE groups. It seems that a differential diffusion of information and subsequent action effect may be operating. Those in higher SE groups tend to have access to health information sooner than those in lower SE groups and tend to act on such information more readily. This may be a function of those in higher SE groups being better educated, in general, than those in lower SE groups.

Another finding reported by Marmot et al. (1987) was that class inequality in mortality had increased from the 1920's through to the 1970's overall. The analysis also concluded that similar relations between SES and health that occur in Britain also occur in USA, Denmark, Norway, Finland, France, NZ and Japan.

Marmot et al. (1987) found the pattern of SES mortality differentials was different from the normal direction of the relationship for African and Caribbean immigrants. For these groups those in lower occupational groups had better health outcomes than those of individuals in "higher" occupational groups. The authors of this study found that (apart from the unexpected immigrant discrepancy in the SES-health relationship) the differences observed were unlikely to be due to artefact due to the robustness of different measures of social class and concomittant health outcomes. They argued that while social selection does operate, it's contribution was insufficient to explain health differentials. They concluded that social causation was operating to impact negatively on the health outcomes for low SES individuals.

In a study on mortality as a function of social class in the years 1971 to 1985 in the UK it was reported that mortality differences had widened for both men and women in that period (Goldblatt, 1989). Goldblatt's study reiterated much of what had been reported in previous research, that is, artefact was insufficient to explain SE health inequality, as was social selection (although its existence is not contested). Social causation was proposed as the best theoretical explanation for observed SE discrepancy in health outcomes. Goldblatt did advocate something new in this paper, suggesting SE differences existed in occupational terms between two main groups (rather than the five of the OPCS classification system, Black Report etc.), those being manual versus non-manual. The rationale behind the dichotomy was at least two-fold. Non-manual was better off than manual occupations in terms of income and prestige and the reduction in number of groups decreased bias inherent in classifications with more occupational categories.

Richard Wilkinson has been a substantial contributor to the development of explanations for the relationship between SES and health outcomes (Wilkinson, 1986a; 1986b; 1987; 1989; 1990; 1992a; 1992b; 1992c;). In an interesting study exploring the links between class mortality differentials, income distribution, and various trends in poverty Wilkinson (1989) concluded that income distribution rather than absolute values such as average earnings, was critical in explaining mortality differentials. He also argued that relative poverty was an imperative concept in the understanding of why mortality differentials occur. Relative poverty has to do with one's perception of where they fit into a society in social terms, which is strongly influenced by financial circumstances (see section on defining poverty for more detail). Psychological factors such as perception have a contribution to make to understanding the SES-health link.

Wilkinson emphasised the importance of perception but failed to articulate the psychological pathways by which such a construct might impact on health outcomes. While not explicitly stated, it seems factors such as social isolation and its known pathological properties (eg. Berkman & Syme, 1979) are inherent in the link between relative poverty and negative health outcomes.

In a recent review of the literature since the Black Report some familiar conclusions about the relationship and causal factors were generated (Davey-Smith, Bartley & Blane, 1990). These researchers argued that social class differences in mortality had increased and continued to increase. They demonstrated how better measures of SE position had revealed greater inequality in mortality than had previously been reported (eg. Whitehall I and II). The authors of this seminal work (Davey-Smith et al., 1990: titled *The Black Report on socioeconomic inequalities in health* 10 years on) also showed that all countries that collect the relevant data show the same trends in inequality which illustrates how robust, universal, and pervasive the phenomenon of SE health inequality is. These researchers provided evidence to support the social causation explanation for health inequality that was reported ten years earlier in the Black Report. The other explanatory approaches were discounted due to the lack of health outcome variance they explained. It was demonstrated that when indicators for various explanations were controlled for in analyses, the health inequality gradient was still evident.

In a follow-up study from the Davey-Smith et al. (1990) research, further evidence was produced to show the consistency of the mortality gradient across different sub-populations across the UK (Delamothe, 1991). This paper demonstrated a strong and consistent occupational class (ie. proxy for SES) mortality gradient for stillbirths, infant mortality, children aged 1-15 years, men aged 20-64 years, and women aged 20-59 years. While this paper was more of a political comment against the prevalent conservatism in British politics than an academic paper with the aim of analysing data or other research, it served well to illustrate, once again, SE health inequality.

Another key study in the field (Carroll et al., 1993) came to the conclusion that while psychological interventions may be of some use in alleviating some of the problems of SE deprivation, the true remedy to the problem (inequity) requires alteration of structural factors that maintain or increase SE diversity. As Wilkinson (1989) and Williams (1990) also advocated, a smoothing out of the income distribution at the national level is required.

This research considers the problem in terms of multiple levels of analysis. Carroll et al (1993) use the example of the principle of proximity proposed by House (1981) to illustrate this multiple level approach. This principle suggests that macro-structures in society influence

individuals within that society by means of a series of progressively smaller proximal structures within the overall macro-framework. Such proximal structures may include large institutions such as schools, followed by smaller groups such as church or recreational groups, then extended family units, then down to individual units which can be subdivided in terms of micro-level variable such as psychological and physiological mediators. Of course, the actual onset of morbidity or premature mortality involves the linkage of larger proximal components via psychological mediators (eg. stress, control, explanatory style, personal disposition) and ultimately physiological mediators (eg. physiological reactivity, immunity, neuroendocrinology).

The Carroll et al (1993) paper examines each of the explanatory theories first proposed by the Black Report and comes to the inevitable conclusion that artefact and social selection explanations are insufficient to account for health outcome variance. It was argued that while cultural/behavioural explanations have influence, research indicates when such factors are controlled for the relationship is still intact. Social causation was seen as the best explanatory approach, although via a more holistic interpretation which considered the theory of social causation to be mediated by many proximal factors.

Very recent research and comment on the SES-health relationship in the UK serves to reiterate what has already been illustrated by the reviewing of several key papers since the Black Report, that SE health inequality continues and seems to be increasing (Phillimore, Beattie & Townsend, 1994; Carroll, Davey-Smith & Bennett, 1994; Black, 1993; Blackburn, 1993).

### ***1.2.2 North America***

Moving on from the overview of the British literature, research on the relationship between SES and health has been quite substantial in the United States. A seminal paper by Antonovsky (1967) was one of the first to explicitly state the existence of a SE health inequality gradient. The paper was essentially a review of the literature on the topic up to that point and concluded that class differentially impacted on mortality rates (which served as a proxy for health status). While he thought the gap was narrowing and tending to become more

blurred with time, Antonovsky (1967) came to the undeniable conclusion that mortality differential existed as a function of social class. While this was as much an historical account of the early writings on the topic rather than a rigorous scientific critique, it did stimulate much debate and brought the issue into the public forum for more consideration.

Kitigawa & Hauser (1973) found that educational status (as a proxy for SES) was a robust and reliable predictor of subsequent mortality. Those with low education tended to have increased risk of premature death. The work of Kitigawa & Hauser (1973) can be criticised for its inherent reductionist approach. Complex economic theory tends to overlook essential but intangible factors. The overlooking of such factors necessarily reduces the validity of results and generalisability of subsequent assertions. This and other work of health economists has been important in illuminating the health inequality phenomenon, but has been of limited explanatory value.

Other work in the same health economic approach has been forthcoming since the work of Kitigawa & Hauser (1973), such as Grossman (1975). The authors tend to talk about concepts such as human and non-human capital and the accumulation of such capital at the upper tiers of the SE hierarchy, which leads to the somewhat circular conclusion that greater accumulation of such capital is somehow insulating against the negative health outcomes more common amongst those in lower SE groups. While this approach describes the phenomenon, explanation of the phenomena is less than convincing.

The topic went somewhat into recess in the US during the late 1970's and 1980's, but was rejuvenated by researchers (House, Kessler, Herzog, Mero, Kinney & Breslow, 1990) who examined the relationship in addition to a third pertinent variable, age. Their analysis was based on data from the American Changing Lives (ACL) survey (N=3617). Other comparable data was obtained from the 1985 National Health Interview Survey (NHIS; National Centre for Health Statistics, 1986). This research found that morbidity and functional impairment at ages less than 75 years in both samples were disproportionately clustered in lower SE groups. The authors suggested health behaviours seemed to differ by social class, but would not strongly commit behind this assertion.

Probably the next major landmark in American research on the topic was an outstanding critical review of the literature by Williams (1990). While this wasn't primary research in terms of hypothesis testing it brought together much of the literature and synthesised the material in a way that refuted much of the selection literature (eg. Illsley, 1986; Stern, 1983). This work was not only a review but an attempt to build a theory to explain SE health inequality. The conclusions reached reflect a multilevel approach which avoids an overemphasis on any one level of analysis in explanatory terms. That is, no one level (individual, small group [such as family unit], large group [such as work environment], society) is inherently more important than another. Rather the interaction across levels is essential in the explanation of such complex phenomena, for instance, while the individual does have control over behaviour to an extent, it is too much to expect every individual to make logical, rational decisions with regard to health behaviours all the time (Brownell, 1990). Cultural and subcultural values will influence behaviours markedly and the overemphasis on individual accountability has led to the debacle of victim blaming for negative health outcomes (Marantz, 1990).

The Williams (1990) paper examined some of the traditional explanations of SE health inequality such as artefact, drift hypothesis (selection), adequacy and accessibility of health care, and the role of medicine. He argued that while these factors may influence health outcomes, none were sufficient in themselves to explain the SE health variance observed in populations. Williams also examined psychological and psychosocial factors in some detail and concluded that structural factors such as distribution of income within a society, access to preventative care, and general issues of social stratification were more responsible for SE health inequality. It followed from Williams analysis that alleviation of inequality would require structural changes (eg. more egalitarian distribution of income) which were somewhat unlikely given the political ideology prevalent in most developed countries at that time.

In a recent study researchers looked to examine whether improved health outcomes overall had been evenly distributed across SE groups in the period 1960-1986 (Pappas, Queen, Haddon & Fisher, 1993). They found that an inverse relation between SES and health existed, and further, the gap had grown since 1960. This study examined the Matched Record Study of 1960 (N=62400) data, as well as the National Mortality Followback Survey and the

National Health Survey interview (N=18733 and N=55690 respectively) which were both carried out in 1986. Less educated white men had a mortality prevalence 2.5 time greater than the best educated white men. This finding supported their conclusion that improved health outcomes had not been distributed across the SE strata. The comparison of white men only allowed for the avoidance of racial artefact in concluding SES had the influence on health outcomes. The numbers involved in their analyses gives the findings of this study more power and generalisability. This work was somewhat descriptive rather than explanatory in style and did not attempt to invoke theoretical explanation for observed SE differentials in health outcomes.

A group of researchers (Adler, Boyce, Chesney, Folkman & Syme, 1993) approached the problem from a different perspective. They accepted the existence of SE inequality in health outcomes and were looking for how a broad insurance scheme (Universal Health Insurance) might alleviate differences. They came to the conclusion that the problem was considerably larger than had previously been considered. Structural factors beyond individual control were largely responsible for health inequality and accordingly the administration of insurance would not in itself do much to reduce inequality. In fact the new scheme, given the failure of previous similar schemes such as Medicaid and Medicare, was recommended to be abandoned unless other structural changes could be implemented.

This study assessed the linearity of the SES-health relationship and whether a threshold effect was operating or not. The threshold effect assumes beyond a particular income level the health impairments associated with SE inequality would disappear. However this concept was rejected as research cited (eg. Haan, Kaplan & Syme, 1989: cited in Bunker, Gomby & Kehrer, 1989) indicated inequality was incremental across SE groups rather than being some sort of all or none principle. Some research cited (eg. Adelstein, 1980) indicated a linear relationship although other research countered such assertions (eg. House et al, 1990). These authors concluded the linearity concept is still to be finalised. Adler et al. (1993) concluded that health behaviours and access to health care were largely responsible for given health inequality. Although this may be acceptable given the level of analysis the research comes from, it does not take into account structural inequality to explain findings. While implying social causation via emphasising the importance of structural change, these authors de-

emphasised the importance of structural factors by arguing for behaviours at the individual level as causal in the SES-health relationship.

In a recent literature review on the relationship between SES and health new potential theoretical pathways were proposed (Feinstein, 1993). Essentially Feinstein argued that causes of health inequality could be reduced to two causal pathways, which in turn could be further dichotomised. This resulted in what could be visualised as a two by two matrix plotting one of the major causal pathways (personal causes) against the other major pathway (life stage). Personal causes could be further subdivided into resource dependent variables (such as income, home and car ownership, that is material wealth) and non-resource dependent variables (such as psychologic, genetic, and cultural/subcultural make-up). Life stage could be divided into health service use (as a function of age) and life experience (which included health behaviours, educational and occupational factors). It was Feinstein's thesis that the interaction of these pathways led to inferior health outcomes for those in lower SE groups. A more thorough analysis of Feinstein's approach will be undertaken in chapter 3.

This work can be criticised because it underestimates structural causes of inequality which are beyond individual control. The form and structure of large institutions such as health, education, and welfare structures are examples of structural variables. Large structures within a society will influence individuals differentially. For instance, access to education, especially in "user pays" societies, will invariably be less for those in lower SE groups. Also, while an individual is ultimately responsible for one's own behaviour, it is fanciful to expect that individual to act in his own best interests all the time. An individual can only make choices based on the resources available at the time of the decision and for those in low SE groups resources will tend to be less than for those in higher SE groups due perhaps to educational differences according to SES. There are those that argue (eg. Kadushin, 1964) that simple behavioural change will close the SE health inequality gap substantially. While this may to some degree be true it falsely assumes that humans can gain control over their own health outcomes through behavioural change. Human beings, while having the capacity for logical thought, tend to be irrational and do not always make the healthiest decisions. A great example of this phenomenon is the common knowledge that certain foods are healthy and

others are not, yet despite this knowledge most individuals eat far too much saturated fats and then wonder why as a population we have such a high prevalence of heart disease.

Feinstein's (1993) research can be commended for its partial emphasis on temporal factors interacting in the SES-health relationship. Those disproportionately affected by SE health inequality fall into the late middle aged groups (Whitehead, 1987). In younger age groups inequity is virtually nil (West, 1990), and the gradient flattens somewhat in older groups (Townsend & Davidson, 1982), although still persists. The two groups most at risk are the middle-aged group and the newborn (Blane, 1985). Feinstein aptly concluded that for better answers about the SES-health relationship more multidisciplinary work was needed.

Following on from an earlier study researchers (Adler, Boyce, Chesney, Folkman, Kahn & Syme, 1994) argue not simply for a health divide, but for graded differences in health outcomes according to SES. These authors also examine various psychological factors which seem to be involved in mediating the SES-health relationship. These include depression, hostility, psychological stress, and social hierarchies. They also examine various health behaviours such as tobacco and alcohol consumption and physical activity. Both the psychological factors and health behaviours tend to differ systematically with SES. The authors conclude that standard analytic techniques such as multiple regression cannot order the large quantity of divergent information needed to make sense of the complexity of the SES-health relationship. A more holistic emphasis, such as multilevel modelling, may overcome the limitations of multivariate procedures for such complex relationships such as the one under investigation.

Research on the SES-health relationship has been less substantial in Canada than the US. An early study on the relation between SES and cardiovascular disease found for the general relationship between SES and health, that is, those in lower SE groups had disproportionately higher mortality rates from cardiovascular disease (Miller & Wiggle, 1986).

In a more comprehensive analysis of males from the Canada Health Survey (1978; N= approx 2000) further evidence to illustrate the SE health gradient was forthcoming (Hay, 1988). In fact Hay argued that a direct positive relationship between SES and health existed.

Paradoxically, they found that the gradient was inverse for SES and physical activity (as SES increased, physical activity decreased). The physical activity finding did not affect the main finding for the study, which was that SES positively impacts on health outcomes.

Hay (1988) concluded that results could support both social selection and social causation theoretical explanations of the relationship but because of the nature of the research (cross-sectional) firm conclusions with regard to causality were difficult to reach. The problem with arguing both explanations were possibly operating is that they are at somewhat different levels of analysis. Causation is at the group level (ie. class analysis) whereas selection looks at the possible influence of health on social status outcomes (individual). Little consideration is given to various health behaviours, which is understandable given the data source, however more comprehensive analysis may have been necessary. Another criticism of this study was the analysis of males only. It is even more suspect drawing conclusions from cross-sectional data where the subjects represent less than 50% of the population. Despite its limitations this research again underlines the robustness of the relationship between SES and health.

### *1.2.3 Australasia*

In Australia research on the relationship between SES and health outcomes has not been copious, however enough has been carried out to suggest that SE inequality gradients analogous to those found in the UK and North America are prevalent in Australia also.

In research looking at the link between social status and morbidity Broadhead (1985) found men in low SE groups had higher self-report illness, a greater incidence of chronic disability, and had more time where activity was reduced by illness or disability. The relationship for women was less clear although those in work fared significantly better in terms of morbidity outcomes than did those whose primary activity was "home duties". Broadhead did however caution that the trends based on the 1977-1978 Australia Health Survey were not as straightforward as his conclusions may suggest and he did not attempt to elucidate possible causal mechanisms. It was concluded that relative affluence was a significant discriminating variable for morbidity outcomes even when controls for sex and age were imposed.

In a short series of studies on mortality differentials in Brisbane evidence emerged which was consistent with pre-analysis expectations, that is, a SE health gradient would emerge. In a study on SE health inequality Brisbane was subdivided into five areas based on SES which was derived from census data. It was found that mortality rates were consistently higher in lower SE areas, that is, there was an inverse relationship between SES and health outcomes by geographic area (Siskind, Copeman, & Najman, 1987a). Clear SE mortality differentials were reported for accidents, violence, respiratory and circulatory disease in men and for genitourinary disorders in women. Cancer related deaths were inversely related to SES although this relationship was not found in women. However, as the authors duly note, attempting to extrapolate causal explanations from such blunt population based data is problematic. There are many potential sources of confound from the results of such research, including the theoretical proposition of artefactual explanation. Despite these deficiencies the findings were consistent with other similar type research which only serves to reinforce the positive relation between SES and health.

In a second related study on infant mortality and its relation to SES (Siskind, Copeman, & Najman, 1987b) a significant positive relationship was found between SES and infant mortality rates. As for the previous study on overall mortality Brisbane was subdivided geographically into five areas based on SES. Again it was found that those in low SES areas had significantly greater rates of (infant) mortality. The researchers found that those in the lowest SE areas had infant mortality rates some 50% higher than those in the highest SE area. Despite obstetric care being free of charge in the Brisbane area, the authors found 67% of infant deaths occur in the neonatal period (first four weeks). This suggests that individual factors are not necessarily responsible for observed mortality differences. One ventures to suggest that a multitude of factors (most of which are beyond the control of the individual such as housing quality, environmental stressors [such as violence & pollution] and occupational factors) are responsible for SE infant mortality differentials in Brisbane.

In a more recent Australian study, which uses some of the artefactual control strategies advocated by Whitehead (1987), it was found that SES was the primary indicator of premature death in Australia (Black & Lawson, 1993). The analysis compared SES and health outcomes over a twenty year period (1966-1986). When the cause of data is assessed it is not

hard to see how Black & Lawson came to their conclusions. The SES proxy was occupation which, rather than as per the British Registrar's classification system involving five hierarchical groups, was divided into manual versus non-manual. The assumption was that non-manual occupations generally received better income and prestige than manual occupations. In fact occupational prestige, according to the authors, was the primary indicator of SES in Australian males because it incorporated three of the major SE indicators, namely income, occupation, and residence. Data was obtained from the Australian Bureau of Statistics from the 1966, 1971, 1976, 1981, and 1986 censuses. Data was for male only between the ages of 15-59 years, that is, the working years.

One initial criticism of this research is that women, who account for over half the population, are excluded from the analysis. Young and old individuals are also excluded, and therefore the question of representativeness may need addressing. Representativeness has to do with external validity. How well do measures approximate (or represent) the general population? Representativeness is problematic because a large proportion of the population is not considered for this research.

The exclusion of women is also problematic because of the significant contribution women make to the traditional social grouping in developed countries: the family unit. Traditional gender roles within the family unit assign income generation to the male partner and homemaking to the female partner. Within this traditional scenario the work of the homemaker allows the income generator time and energy to devote to that task. The contribution to family earnings is largely intangible, but still vital, especially if the couple have children.

Traditional gender roles have endured a shake-up over the past thirty years in most developed nations (Arber, 1991). Along with an increase in the number of single parent families, most of which comprise the female partner and children, many families now have two income generators. The exclusion of women from the analysis can be seen as doubly problematic here. Firstly, in the case of single parent families, the woman will often be fulfilling both income generator and homemaker roles. This role strain may lead to the compromising of income generation, especially if caring for the children takes precedence over maximising

income generation. Secondly, in cases where both partners are working it is entirely plausible that the woman's contribution is not given the credit due. That is, while the male earnings are included in the analysis, the woman's contribution to earnings and subsequent overall health outcomes is understated.

Even excluding the social unit, the family, there are many more women in the workforce than was the case thirty years ago. Consequently, the contribution of women is again understated by their non-inclusion. Of course, the abovementioned understated contributions of women's income generation to health outcomes overlooks the fact that women generally earn less than men for equivalent work types in all developed nations. Additionally, there tends to be the pooling of women in low paid types of work. There is a relative lack of women represented in professional, managerial, or skilled technical work compared to men. These factors all lead to the understating of the contribution women make to health outcomes as a function of income generation.

When all these factors are considered simultaneously women's contribution to health outcomes at the population level may be understated in all the family scenarios. In the traditional male led family the intangible contribution is understated, and in the single parent or double income situations, repression of earning potential and understating effects, respectively, may be operating. To add to these characteristics of the female contribution to SE health inequality, women tend to earn less for similar work as well as being employed in low income work in disproportionate numbers. When these factors are combined with the known sex differences in population health outcomes, such as more morbidity but less mortality than males in all age groups (Verbrugge, 1989), and greater life expectancy (Waldron, 1983), the exclusion of females from SES-health analyses is problematic.

Despite the drawbacks in Lawson & Black's study, some clear trends were delineated. According to occupational status the lowest SE groups have a lung cancer mortality prevalence four times that of the highest SE group. The total lung cancer incidence was up 20% in the twenty year period, however there may be some systematic mediating variable influencing the lung cancer variance. It is a well established fact that a higher proportion of people in lower SE groups consume tobacco (Berkman & Breslow, 1983). It is likely,

therefore, that the SE patterning of tobacco consumption is inflating the effect SES has on health outcomes in terms of lung cancer.

Mortality by cirrhosis of the liver was up 100% in the twenty year research period, and those in manual occupations had consistently higher mortality rates than those in non-manual occupations (Lawson & Black, 1993). Mortality by ischemic heart disease dropped substantially over the period of the research (45%), however the same pattern of non-manual being better off in terms of health outcomes than manual groups prevails. The same pattern, but to a less substantial degree (25%, with manual > non-manual) occurs with regard to mortality by accidents. Despite significant improvements by some occupational groups within the manual group, other occupational groups within the manual category still have mortality rates by accident some fifteen times greater than most non-manual occupations. The authors of this research concluded that despite significant reductions in mortality overall, marked differences in mortality rates between high and low SES men predominate. Further, improvements in mortality outcomes are mainly due to reductions in deaths due to ischemic heart disease, which has disproportionately favoured men from higher SE groups (Lawson & Black, 1993).

No critical examination of the literature would be complete without a perusal of literature published from within the country of the writer, in this case New Zealand literature on the relationship between SES and health. Like many countries research on the topic in NZ has not been substantial, although compared with some OECD countries such as Canada, Australia, and with several European countries, NZ's contribution has not been light. There has been much interest in the SES-health relationship in recent years (Maskill, 1991; Public Health Association, 1992; Public Health Commission, 1993, 1994).

In the first of a series of studies in response to the Black Report findings, researchers undertook to assess social class differences (as a proxy for SES) in mortality for NZ males (Pearce, Davis, Smith & Foster, 1983a). This analysis revealed substantial class differences in mortality between high and low SE individuals. This disparity was evident despite the use of two different classification systems, firstly in terms of the OPCS's (British Registrars) classification system, and secondly by the Elley-Irving social class categorisation system.

Potential problems for this research include the fact only males were sampled, which as previously mentioned means that over 50% of the population were ineligible for analysis. This, of course, reduces the power for generalisation from the results, especially given that sex differences have been observed in research on health outcomes (Verbrugge, 1989). Another source of potential bias is the analysis of only 15-64 year old males. Assessing premature mortality and avoiding the potential bias of including non-financially active individuals has some rationale, however defies the fact that only about 4% of the population are being assessed ( a sample of 10% was taken of males in the 15-64 year age group, that is, 10% of approximately 1.2 million [the workforce] which is 120,000 over the total population [approximately 3.5 million]). The point of these criticisms is that results and subsequent conclusions may lack the statistical construct known as external or ecological validity, or put more simply, the power of generalisation. Standard problems, such as denominator-numerator bias, may also be a problem here, however the robustness of the results (given two different and comprehensive SES measures - the Elley-Irving scale and the British Registrar Generals occupational class scale) serve to illustrate a replicable magnitude and direction of the SE health inequality gradient observed in many other studies.

In a second and related study by the same researchers (Pearce, Davis, Smith, & Foster, 1983b) mortality outcomes were assessed in terms of cause of death. There was a consistently strong relationship for higher mortality in lower SE groups with the strongest class gradients being observed for death by accident, violence, respiratory disorders, nutritional and metabolic diseases, and diseases of the genitourinary system. Weaker findings were found for death by coronary heart disease (CHD) and digestive system disease.

As for the first paper (Pearce et al., 1983a), both British Registrar General and Elley-Irving SES categorisation scales were used, and again 10% of males aged 15-64 years (at the 1976 census) were analysed. Three disease groupings accounted for 74% of overall mortality variance, those being (in descending order) CHD, cancer, and accidents. All these causes of death have a psychological component (Sarafino, 1990), and all differ systematically according to SES. Weaknesses were essentially the same as for the previous study by the same researchers mainly with respect to representativeness and generalisability. This paper did produce the finding that is often noted by researchers in the field, that being that CHD used

to be (several decades ago) a "disease of affluence" (Townsend & Davidson, 1982) effecting predominantly those in higher SE groups, however now was a problem of predominantly those in lower SE groups. This major change has been attributed to dietary and tobacco consumption changes that have differed according to SES over the last fifty years or so, and these authors supported that hypothesis.

In a third study in this series by the same researchers (Pearce, Davis, Smith, & Foster, 1985), analyses were undertaken to examine the influence of ethnic group as a source of potential bias in findings from previous research. When Maori, Pacific Island, and "other" ethnic groups were analysed separately all showed differences in mortality as a function of SES. However, Maori were consistently worse off than the other two ethnic groups and "other" being consistently better off than the Maori or Pacific Island groups. This left Pacific Island individuals as intermediaries in SE health inequality. Methods were the same as for the previous papers by these researchers (two scales, 10% males aged 15-64 years etc.), as were the potential methodological problems. An unusual observation occurred for both Pacific Island and Maori groups (when measured on the OPCS scale), with non-manual groups (I-III) having higher mortality than manual groups (I-III). This goes against the general trend for higher mortality rates to be found in manual than non-manual workers (Goldblatt, 1990), however, has been noted in other research (Marmot et al, 1987). A possible explanation for this anomaly could be the differing emphasis the differing cultures place on occupation, income, and material circumstances (Henare, 1988).

In a brief commentary one researcher (Davis, 1984) examined the relationship between class, ethnicity, and economic development on health outcomes in NZ men. Davis argued that the sparsity of Polynesian deaths among working men necessitated their removal as a distinct ethnic class and they were subsequently incorporated into the "other" ethnic group (which also constituted Pakeha, Indian, and Asian groups). Health outcomes were measured in terms of mortality, and by cause of death, for the two ethnic groups. In terms of the Registrar Generals classifications group V scored very poorly compared to group I overall by a ratio of 2.3:1. Digestive and respiratory disorders, along with infectious illness were the biggest contributors to SE disparity in health outcomes with ratios of 5.9, 4.4, and 4.1:1 respectively. Davis considered both cultural and lifestyle themes in causality along with material inequality and

deprivation. It was concluded that neither pathway could adequately account for the SE disparity in health outcomes observed. Davis also came to the interesting conclusion that material disparity could only account for 20% of Maori health variance, therefore 80% of Maori inequality was related to Maori cultural and behavioural practices. It may well be that the urbanisation of what were once a rural and tribal population has led to the health demise of many of the Maori people. The dietary and tobacco consumption habits of Maori also contribute substantially to the general ill-health of the Maori ethnic group (Pomare, 1988).

In research that was essentially a synopsis of three previous published papers (Pearce et al, 1983a; 1983b; 1984), researchers examined mortality trends in NZ (1974-1978) in terms of social class and ethnic group (Pearce, Davis, Smith & Foster, 1985). This research, as to be expected, evidenced strong class based mortality differentials for NZ males (aged 15-64 years). The unusual finding was for the extremely large mortality levels in social class V (even compared to the UK!). Patterns of death outcome by disease were similar for UK research with the generalisation that what were once diseases of affluence (eg. CHD) were now concentrated in lower SE groups. Apart from the very large mortality rates for group V individuals, the other crucial observation was that Maori had significantly higher mortality outcomes (up to 50%) than the "other" ethnic group (this held true even when social class was controlled for). Linearity can be used interchangeably with incrementality in the context of this discussion. It refers to the phenomena where as x changes so too does y. The change in y is systematic in terms of the change in x. Linearity in terms of SE health inequality refers to the situation where as SES changes, concomittant changes in health occur. Usually the lowest SE group have the poorest health outcomes, the second lowest SE group the second worst health outcomes, etc. The relationship can be seen as linear when the change in health outcomes is relatively similar across the changing SE groups. Linearity is not occurring when one of the SE groups has health outcomes which violate the usually predictable incrementality of the relationship between SES and health outcomes.

This very large ethnic variation in health outcomes leads to the conclusion that the relationship in this country is not linear, that is, social class differences in health outcomes are not necessarily sequentially hierarchical in terms of SES. This non-linearity leads to at least two conclusions being drawn about the SES-health relationship in this country.

Firstly, British measures may not be appropriate to assess the NZ population; and secondly, the SE health differential is not linear when substantially different cultures are combined for assessment of the relationship. It has been observed in overseas research that homogeneity of populations leads to a stronger relationship between SES and health outcomes (that is, the relationship is stronger in magnitude), as well as the tendency for more linear outcomes in terms of a more stepwise progression from lowest to highest SE group for health outcomes (Adler et al, 1993; Marmot et al, 1984).

In an analysis of the data for cancer mortality for NZ men (aged 15-64 years) by social class between the years 1974-1978, researchers (Pearce & Howard, 1986) found SE differences in outcomes for cancer of the liver, larynx, lung, buccal cavity, and stomach. While the causes for these outcomes might be explained in terms of health behaviours such as the consumption of tobacco, alcohol, and dietary patterns, it is hard to ignore the patterning of such behaviours by SES and the similarities with overseas research (eg. Yeracaris & Kim, 1978). The SE gradient in cancer mortality is similar to the overall mortality gradient, although NZ (especially the Maori population) has some unusual differences. Certain occupations (as for the overseas findings) were at substantially greater risk of premature death by cancer than others, including especially woodworkers, painters, hairdressers, beauticians, and farmers.

A study by Borman, de Boer, & Fraser (1990) examined risk factors for low birthweight in NZ over the period 1981-1983 (low birthweight and infant mortality are often viewed as indexes of a nation's state of health by health researchers here and elsewhere, thus is the rationale for the use of low birthweight risk as a measure of health outcomes in this research). Parental SES in terms of occupational status was assessed as a risk factor, and turned out to be somewhat influential. A large sample (N=8049) of low birthweight babies were assessed in the research to consider various possible causes and inherent risk factors. Although this sample is large it only represents 5.4% of all births in this time period, therefore a different gradient may be observed with a more representative sample of all births. While overseas research has found SES and birthweight to be positively related (Berkman & Breslow, 1983), this study did not replicate that finding.

In a more recent study researchers compared the findings of Pearce et al (1983a; 1983b; 1984) from the 1975-1977 census data, with more recent data 1985-1987 (Pearce, Marshall & Borman, 1991). As for the earlier research, this research assessed 10% of men aged 15-64 years. What had been a relatively uniform and close to linear relationship overall has changed considerably in the period of the comparison. The biggest change was the decrease in inequity for those in the lowest SE groups and slight increases in negative health outcomes for those in higher SE groups. What can be tentatively concluded from this paper is that inequity has flattened out somewhat in the period 1975-1987, especially when compared with the UK where inequality has clearly increased in a similar period (Marmot & McDowall, 1986). The authors of this study concluded that inequality had remained constant overall, and in some instances had increased slightly, despite falls in mortality overall.

The authors (Pearce et al., 1991) saw causation at societal (ie. social causation) rather than the individual level (cultural/behavioural explanations). They argued SE health differentials would result from social policy change, such as abolition of tobacco and alcohol advertising, rather than targeting of high risk individuals in prevention programmes (ie. health education on risky health behaviours such as tobacco and alcohol consumption). Their emphasis was on structural and political change rather than an individual emphasis. They did not ignore the importance of health education and public health prevention programmes, rather they questioned the efficacy with such approaches given the past record with such approaches. The behaviour of target populations for such interventions have historically proved notoriously difficult to influence (Steptoe & Wardle, 1994).

Kawachi, Marshall & Pearce (1991) looked at the relationship between social class and coronary heart disease in NZ men over the period 1975-1987. They found that while the overall rate of CHD in NZ had dropped substantially, the distribution of decrease across social classes was not equal. These authors argued that dietary changes along with class based behavioural changes in tobacco consumption meant CHD was now a dilemma for predominantly those in lower SE groups. While the writers would not imply causality with any degree of authority, they did conclude that decreases in CHD had occurred more in those groups (higher SES) with lower death rates anyway, which resulted in increased CHD mortality disparity between high and low SES individuals in terms of health outcomes.

In ongoing research on the relationship between health (in terms of mortality) and social class, Pearce and his colleagues (Pearce, Pomare, Marshall & Borman, 1993) recently examined changes in mortality between Maori and non-Maori by social class. As for other work by Pearce and his colleagues (Pearce et al., 1983a; 1983b; 1985; 1991) data was from 10% of men aged 15-64 years and from two periods (1975-7 and 1985-7). The researchers were looking to how social and ethnic influences had changed between the mid 1970's and the mid 80'. Overall the researchers found mortality had decreased substantially over the ten year period (Maori 28% and non-Maori 14%), however the Maori mortality rate was still 2.8 times greater than the non-Maori rate.

In comparing Maori with non-Maori and assessing the population (sample) as a whole over the ten year period several trends emerged. Maori mortality in all but the wealthiest (SE or occupational) group has dropped off markedly. Non-Maori mortality has dropped also, but not as substantially. The lowest SE group within the Maori population has substantially higher mortality than all other groups (1975-7 and 1985-7), however it had dropped since the first measure. Many of the problems with this type of population research have already been covered in critiques of previous papers by Pearce and his colleagues.

In another recent NZ study (Marshall, Kawachi, Pearce & Borman, 1993) looking at the SES-health relationship, researchers found social class differences in mortality. This study looked specifically at mortality from diseases which are treatable and preventable by medical intervention. Some illnesses causing mortality are not treatable, that is, palliation is possible but not reversal or prevention of health deterioration (ie. incurable diseases). However, many diseases that can cause death are treatable, and death is preventable, if treatment is administered soon enough. In medical jargon these diseases are said to be amenable to medical intervention. Rather than simply looking at mortality as a proxy for health status, mortality from diseases amenable to medical intervention is more specific in that it gives an indication of the adequacy of health care in a country, region, city, or any geographic area. As for all previous research of this type in NZ the analysis was carried out for men only (aged 15-64 years). Again 10% of the population in the relevant age and sex were assessed in 1976 and 1986 and comparisons made. The Elley-Irving scale was used for SES classification, and mortality for a health proxy.

Age standardised mortality data shows that in the lowest SE group (group 6) mortality decreased markedly over the ten years (-35%), whereas in the next SE group up (group 5) grew somewhat (+22%) in terms of death which was amenable to medical intervention. This was against a backdrop of an overall 29% decrease in such mortality. It seems the decrease in mortality in group 6 may have been to a large degree absorbed by increases in group 5, however there remains a clear mortality gradient in terms of SES. "Men in the lowest socioeconomic group continued to experience a death rate from amenable causes of mortality that was nearly 3.5 times greater than those in the highest socioeconomic group" (Marshall et al., 1993:p259). The authors concluded that despite gains in health outcomes overall (29% decrease in medically amenable mortality and 14% decrease in non-amenable mortality) social class inequality existed in nearly all causes of death examined.

#### *1.2.4 Europe*

Having assessed evidence for SE health inequality in some detail from UK, North America, and Australasia, I will now briefly discuss evidence from several European countries to further solidify the case for SE health inequality from countries with somewhat diverse political and ideological backgrounds.

Diderichson (1990) in an examination of health and social inequalities in Sweden found manual occupations had mortality rates higher than non-manual occupations. During the 1970's the gap between manual and non-manual had widened due to mortality drops in non-manual and increases in manual occupations. In assessing regional mortality differences (ie. geographical location differences) Diderichson found areas with high unemployment (most often rural and northern) experienced higher mortality rates. This is another proxy for SES and concomitant health outcomes. Self-assessed morbidity data also revealed class disparity in health outcomes with manual workers 50% more likely to report recent illness than senior salaried workers.

In a study looking at SE health inequality predominantly in Finland, but in comparison with several other European nations, researchers came to several somewhat predictable conclusions (Lahelma & Valkonen,1990). Non-manual occupations had significantly better health

outcomes than manual occupations. Finland's inequality was comparatively greater than the other Nordic countries (Sweden and Norway), although it was noted that the same occupational groups in all three countries had the highest mortality, those being manual groups. This study also indicated that education as a marker for SES was a robust and reliable predictor of health outcomes in terms of both morbidity and mortality. This relationship was echoed in cross-country analysis not only in Finland, but also in England, Wales, Sweden, Norway, Denmark, and Hungary. Maseide (1990) reported similar findings for health inequality in Norway alone, although this researcher would not argue with any conviction as to the accuracy of his findings. Maseide preferred to avoid overconfidence in extrapolating from various SE or health indicators as to possible causal pathways.

In an analysis of health inequality in Ireland, Cook (1990) found that there were strong differences between SE groups in that country in terms of mortality. This class differential was especially obvious for cardiovascular disease, respiratory disease, and several types of cancer. As Cook himself points out, however, the data on health in terms of social stratification are scarce, especially in comparison to other European nations. Despite reservations Cook concluded from the limited data available, that the phenomena documented elsewhere, SE health inequality, was also occurring in Ireland.

Spain's health inequality was evaluated recently by Rodriguez & Lemkow (1990). These authors found SE health inequality in terms of class and regional differentials. Differentials were large due to the fact that those in higher SE groups has vastly improved health owing to policy intervention by government in the 1960's, while the lower SE groups had stagnated or regressed in this period.

In a study examining SE mortality differences in the Netherlands between 1950 and 1984 researchers (Kunst, Looman, & Mackenback, 1990) came to several predictable conclusions. The strong positive relationship between SES and health outcomes found in 1950 had not narrowed, in fact in some cases it had widened. Diseases which caused mortality in high SE groups in the 1950's (eg. Ischemic heart disease, lung cancer) had reduced their mortality influence in those groups but were now disproportionately exerting their influence on lower

SE groups. This research also found SE disadvantaged geographic regions had populations with worse health outcomes, which is again in accordance with a wealth of prior research (eg. Broadhead, 1985).

Research into SE health inequality in Belgium revealed similar geographic trends to those found in the Netherlands by Kunst et al. (1990), that being that areas of lower SES had populations in poorer health than areas with high SES individuals (Lagasse, Humblet, Lenaerts, Godin & Guido, 1990). These researchers concluded from their population based research that certain geographic locations (low SES), lower SES individuals, and single parent families (assuming less financial resources) were more likely to suffer poor health outcomes.

Lehman, Mamboury & Minder (1990) found the familiar SE health gradient in their research in Switzerland. They found that individuals from manual occupations suffered disproportionately negative health outcomes compared with individuals from non-manual occupations. Again this is not surprising given that income from non-manual work tends to be greater than that from manual work, therefore this is more confirmatory evidence for SE health disparity. The authors go further and provide evidence (from morbidity and disability data) to argue for the hypothesis that those in lower SE groups age "faster" and tend to suffer more at younger ages than those in higher SE groups. This study provides support for Wilkinson's (1992a, 1992b) hypothesis that a nation's income distribution has more influence on the population's overall health status than average or overall wealth. This was reflected in the conclusion that despite one of the highest life expectancy and standard of living ratings in the world, Switzerland had one of the worst records for SE health inequality. Lesser paid manual workers, as appears to be an overarching pattern in most developed countries, bear the brunt of health inequality, particularly those in middle age groups.

### *1.2.5 International Comparisons*

In the final section of my selected analysis of research on the relationship between SES and health outcomes I will examine a sample of international comparative research, that is, comparisons of nations with similar data. Emphasis must be given to caution in interpretation of such studies at this point because there are many factors which are beyond the control of

the researcher which may act or interact to bias results and subsequent assertions and assumptions. The UK, for instance, has an empirically sound database provided by the OPCS for the entire population over a long period of time. Little has changed in the way data is collected since the inception of the database in 1911. What this means is that relatively meaningful assertions can be made on various population trends over time. No other country has access to data of this quality by which to make assessments, therefore comment from the UK is very meaningful (hence the analysis of UK research early in my assessment of the research). My point here is that data will vary across nations and time periods within nations, therefore the ongoing problem of mixing "apples with oranges" remains a valid criticism of international comparisons. Further, most such comparisons are cross sectional and therefore open to all the criticism that has been attached to such research in the past.

Variability in the quality of income data across countries was one of the large methodological problems involved in cross country comparisons of the SES-health relationship according to Le Grand (1987). In this analysis Le Grand also proposes several other potential sources of methodological difficulty. Definitional "vagaries" between different bureaucratic institutional bodies internationally was another potential source of error. For instance, social class is defined differently in different countries with variable class criteria the norm. Le Grand also criticised research with small sample sizes, but more importantly criticised research without an underlying theoretical framework through which to organise findings or extract understanding from. This paper was the synopsis for a much more extensive work and therefore the results provided were only summary in detail. Le Grand using Absolute Mean Difference (AMD), Gini coefficients, and the Atkinson Index (all health economics "tools") organised 32 countries (OECD countries less Turkey, plus Czechoslovakia, Luxemburg, Bulgaria, Hungary, Poland, Romania, and Yugoslavia [with UK subdivided into England, Wales, Northern Ireland, Eire, and Scotland]) into three equivalent sized groups according to health inequality. This paper gave a loose idea of how various developed countries of the world compared in terms of that particular measure. The results were as follows: In the lowest third (in terms of health inequality) were Czechoslovakia, Eire, England, Wales, Finland, West Germany, Luxemburg, Netherlands, Northern Ireland, Scotland and Sweden. The middle third included Australia, Belgium, Bulgaria, Canada, Denmark, East Germany, Iceland, Italy, Japan, Norway, and Switzerland. The highest third, that being the groups with the greatest inequality,

included Austria, France, Greece, Hungary, New Zealand, Poland, Portugal, Romania, Spain, USA, and Yugoslavia (adapted from Le Grand, 1987: p186).

A recent study examined international comparisons of health inequality for four "Eastern Block" countries (Wnuk-Lipinski & Illsley, 1990), Bulgaria, Hungary, Poland, and USSR. A strong positive relationship was noted between parental SES and infant mortality. There was also some limited evidence for adult mortality differentials. Evidence was also produced for geographical (SES proxy) area and health outcomes (ie. geographical mortality gradient) in all the countries, with the worst areas in terms of health outcomes being those which had been subject to heavy industrialisation where pollution was now a public health problem (especially water quality, acid rain, and air pollution).

Two of the main causes of adult mortality were ischemic heart disease and lung cancer, neither of which was amenable to medical intervention (Wnuk-Lipinski & Illsley, 1990). Both diseases are amenable to lifestyle changes which draws one to the "common sense" problem solving strategy of reducing incidence not via treatment but prevention through behavioural change. This can be problematic with the emphasis on individual control and propensity for victim blaming rather than seeing the risk behaviour as being socially patterned. Thus a better option (not proposed by the authors of this research, but having been cited in previous research in this section) may be to reduce incidence of lung cancer (for example) by prohibiting tobacco advertising rather than emphasising individual control of smoking behaviour.

The final important finding of Wnuk-Lipinski & Illsley's (1990) research was that health care facilities and services are somewhat unequally distributed in terms of rural versus urban settings, and in terms of high versus low SES communities. Many of the problems observed in these borderline developed nations are not observed in developed nations. For such nations inequality tends to be absolute in nature rather than relative, where a select few have access to most of the resources, and what is left is divided unequally amongst the masses. Absolute poverty encompasses a whole new myriad of problems not usually encountered in developed countries such as infectious disease and diseases of sanitation.

In a recent study of class differences in infant mortality between England and Wales and Sweden, researchers found Sweden had superior infant health outcomes to England and Wales (Leon, Vagero & Olauson, 1992). Although not specifically stated by the researchers, much of this could be attributed to differing political approaches in the different countries. Sweden has a very much interventionist political approach with strong redistribution of wealth in terms of welfare, health, and education spending by government. England and Wales have far less interventionist governments and income is less evenly distributed.

Therefore Sweden has a reasonably equitable health care system with virtually unrestrained access. Additionally Sweden has a strong welfare state which ensures minimum financial standards and flattens income distribution across the nation. England has considerably less even access to health care services and minimalist efforts at health, welfare, and education, subsequently income is less evenly distributed across the nation. Despite the differences cited between countries Sweden has not been able to eliminate SE health inequality, although the authors argued that if England and Wales could reduce their infant mortality rate to that of Sweden, 63% of postneonatal mortality could be avoided (Leon et al., 1992).

Similar conclusions were reached after more comprehensive research on infant mortality looking at 18 industrialised countries (Wennemo, 1993). This researcher argued that the concept of relative poverty was important in establishing and maintaining inequality and hence implying income distribution as being an important factor in infant mortality outcomes. Wennemo emphasised the importance of the level of poverty, or degree of SE inequality, within a country as being crucial as a predictor of infant mortality rates. In other words, the degree of poorness of the poorest group in a society relative to the rest of the nation is predictive of infant health outcomes. If the lowest SE group is vastly poorer than the rest of the population infant mortality rates will be greater overall.

### **1.3 Summary**

In my analysis of the literature many concepts and trends have been uncovered in an apparently haphazardous and disorderly fashion. The purpose of this section was to illustrate, with pertinent research findings, the existence of the SES-health relationship and examine

various characteristics of the current situation with regard to the relationship. The criteria for the ordering of my discussion of key research findings has been by geographic location and chronologically. This may be somewhat bewildering therefore I will now summarise the major findings from the research in the field to date.

The first issue to emphasise is SES impacts on health outcomes and the direction of the relationship is positive. Thus as SES increases health outcomes improve. It would be erroneous to assume health has no influence on SES, however the magnitude of health inequality negates this as the main direction of the relationship. The positive relationship between SES and health outcomes has received support in terms of mortality and morbidity research, both from self-report data and observational research.

Much of the research carried out on SE health inequality involves the use of census, survey, and epidemiological data and is therefore prone to error from many sources. However the relationship has been found in repeated research, across many countries, and with the use of different indicators for both SES and health outcomes. The direction and magnitude of the relationship is relatively stable across the research which serves to illustrate the robustness of the phenomena of SE health inequality.

While it would be convenient to state that inequality is only pathological beyond a point, or in other words, below a certain SES threshold pathology becomes problematic, that is generally not the case. SE health inequality is an incremental phenomenon (most of the time, exceptions will be discussed shortly) with hierarchical decrements in health as one descends down the SES ladder. The incrementality is reflected by differences even within the SE classes according to one's place within that group.

There was a couple of cases where incrementality in SE health inequality was not observed. Marmot et al. (1987) found a trend for lower SE individuals having better health outcomes

than those in higher SE groups for African and Caribbean immigrants in the UK. Interestingly the same trend was observed for the Maori population in recent NZ research (Pearce et al., 1985). More consideration will be given to this paradoxical anomaly in my conclusions, however it seems that when vastly different cultures are compared the usually predictable linearity and incrementality of the relationship is upset. The specific ethnic groups that appear to defy the relationship (African and Caribbean in the UK, and Maori in NZ, and probably others) have cultural and behavioural practices that differ from the majority of the population. An observation in the UK which makes the situation even more paradoxical is that Indian immigrants have health outcomes similar to the majority of the population (Marmot et al., 1987).

A speculative explanation for this anomaly may be in the observation that heterogeneity may dampen the magnitude of the SES-health relationship. This can be indirectly implied from the findings of the Whitehall studies (Marmot et al., 1984; 1991). These researchers used samples which were more homogenous than those used in other research on the SES-health relationship. The magnitude of the gradient was considerably greater than in most previous research with more random (heterogenous) samples. It may well be that cultures differing from the majority in a particular country are not well served by the measurement of relevant information, however the accuracy of this assertion will be made later.

There appear to be certain stages of the lifespan where SE health inequality is more prevalent than others (Feinstein, 1993), therefore the developmental stage of the cohort under investigation must be taken into consideration when the magnitude of effect of any given research is being assessed. Early periods in life (infancy and early childhood) as well as middle age brackets (40's-late 60's) seem to be at greater risk of negative socioeconomically mediated health outcomes (in terms of having steeper health inequality gradients). The older age groups (60's plus) as well as the more mature young (5-late 30's) have considerably flatter inequality gradients, although they still exist (MacIntyre & West, 1991).

The way income (or more specifically wealth) is distributed in a country seems to have an influence on health outcomes. Countries with more even income distributions, that is, countries with influential welfare sectors (interventionist governments) such as Sweden and Japan (Marmot & Davey-Smith, 1989) have better health outcomes than countries where income is less evenly distributed (Wilkinson, 1992b). Countries like the UK and USA have minimal social and financial support systems set up for their low SES populations. It seems where the minimum income levels are greater for a nation (ie. a more even income distribution) there is a flatter inequality gradient observed.

Another trend that appears from the research is the tendency for health behaviours to vary systematically by SES, culture, and even geographically. These three variables are intimately interrelated and behaviours that are patterned by these variables can be called socially patterned behaviours. It is my contention that social patterning of behaviour explains more variance in health outcomes than individual attempts at behaviour modification, although this assertion needs further research to ascertain validity. The inability of many individually targeted health promotion programmes (eg. smoking cessation) to significantly (especially in low SES individuals) change negative health behaviours is but one reason for this assertion.

Another strand of evidence for the notion of socially patterned behaviours is the observation of major changes in patterns of mortality caused by coronary heart disease (Miller & Wiggle, 1986; Morgan, Heller & Swerdlow, 1989). Not even fifty years ago CHD was a disease of predominantly higher SES individuals. People in high SES groups tended to consume tobacco and alcohol, and had high fat diets. CHD was known as a disease of affluence because of the people in society it tended to afflict most. The position is now different with those in lower SE groups being disproportionately affected by CHD (as well as other diseases that used to be for the affluent mostly such as cancer and accidents). One possible explanation for this major change has been proposed by Bennett, Weinman & Spurgeon (1990) in their diffusion theory. The argument goes that information and action on that information is acted on at different rates by people from different SE groups, with those in higher SES groups knowing and acting faster on new information faster than those in lower SE groups -- that is, information and subsequent action take differential time to diffuse down the SE hierarchy.

The final point that should be taken from this section is the most commonly agreed upon theoretical explanation for SE health inequality by contemporary experts in the field is social causation. An in depth discussion of the reasons for this will be undertaken in chapter 3 of this thesis (Theoretical approaches to the SES-health link). In somewhat oversimplistic terms the argument goes that health inequality occurs due to structural features in the social environment. Unequal access and utilisation of various social institutions such as health, welfare, and education resources serve to reproduce unequal health outcomes by SES within a population.

## 2.0 PATHWAYS FOR SES TO IMPACT ON HEALTH OUTCOMES

### **2.1 Major Pathways**

Health outcomes differ within the population in many ways, some subtle while others are substantial. When health outcomes are assessed across various sociodemographic dimensions one, SES, appears to be playing a major role. I will briefly consider some of these dimensions with the intention of illustrating the role of SES in health outcomes. In no particular order the dimensions of social class, income, education, geographic location, housing, and transport will be considered in relation to health outcomes.

Income, educational status, and social class are major components of SES. Social class is usually assessed in terms of occupational level, although by no means always. Other dimensions of SES include geographic location, transport, and housing. Geographic localities, especially in urban centres, tend to vary according to SES. Most modern cities have affluent suburbs, middle class areas, and poor "slum" areas. The availability of transport, be it private transport or access to public transport, reflects the SES of the individual. Housing quality also reflects on the SES of the individual. Two other factors, gender and ethnicity, are related to SES, but will not be specifically considered in this analysis.

#### **2.1.1 Income**

While it is a widely held view that income is the essential ingredient in the multidisciplinary construct of SES, the magnitude of influence on health outcomes is problematic. Income is but one component which influences the individual's financial standing, and other components such as asset holdings and joint ownership will dilute its efficacy as a valid predictor of health outcomes. The way in which income is assessed may sway outcomes in research. For instance, self-report data may tend to underscore income due to suspiciousness of full disclosure on the subject's part, or official statistics may overscore income owing to such statistics being based on gross (pre-tax) income which is not truly indicative of spending power. It is also likely that those in higher SE groups will have much of their wealth in assets, thus income is but a small component of their total wealth variance. This implies that

income as a proportion of total wealth variance will differ across SE groups and may in fact distort the true picture of inequality. Income has not been used extensively in the literature as a marker for SES in the SES-Health research, although what little has been done confirms a positive correlation between income and health (eg. British Medical Association [BMA], 1987; Public Health Commission [NZ], 1993, 1994; Pappas et al., 1993).

The relationship between income and health is not linear, and recent research indicates absolute income is not a good predictor of health outcomes (Wilkinson, 1986b). Perceptions of income equality are a better indication of health inequality within a society (Wilkinson, 1990). In fact the importance of relativity is a major reason why cross-country or international comparisons of SE health inequality are so problematic. Measures of SES and health will vary between countries, therefore it is likely that the comparison of differing conceptions will reap conclusions that are methodologically problematic such as mixing "apples with oranges". While the relationship between middle-high income and health is not well understood, research in the UK (BMA, 1987; Whitehead, 1987), and Canada (Shah, Kahan & Krauser, 1987) emphasises the importance of the relativity conceptualisation. Given that their research was carried out in relatively wealthy OECD countries, their findings that low income individuals have significantly inferior mortality and morbidity outcomes underlines the importance of perceived SE inequality in inferior health outcomes in developed countries.

The work of Wilkinson has paved the way for psychological interpretations in the SES-Health relationship due to the inclusion of perception into the arena. In countries where income is more evenly distributed like Japan and Sweden, health inequalities are less than countries where wider income distribution occurs such as the UK and USA (Wilkinson, 1989). Subsequent research (Wilkinson, 1990, 1992a) allowed the conclusion that "overall there is clear evidence of a strong relation between a society's income distribution and the average life expectancy of its population"(Wilkinson, 1992a: p167). It seems that more research is required from health researchers (including psychologists) to ascertain how influential various cognitions and perceptions are in health inequality variance.

The physical means by which income influences health are somewhat more straightforward. Lack of income will impact negatively on nutrition, housing quality, access to transport,

health care services, and education. The fact that income influences other factors to be considered, highlights the inter-relatedness of various components of SES and how direct linear relationships between such components and health outcomes are inappropriate as well as unlikely to yield significant explanation of SE health variance.

### *2.1.2 Education*

As for income, the relationship between education and health has received little attention in the literature. Despite the lack of research, available evidence again suggests a positive correlation between education and health (Feldman, Manuk, Kleinman & Cornoni-Huntley, 1989; Valkonen, 1989), although the direction of the relationship has been open to some debate. There are those that argue that poor health leads to subsequent poor educational achievement (Stern, 1983), especially in childhood (Carr-Hill, 1987). The argument goes that poor health not only reduces time at school, but when actually at school learning is impaired because of the ill-health and thus educational outcomes are relatively substandard. This, it follows, leads to frustration on the part of the individual suffering ill-health which tends to culminate in early school leaving and problems with obtaining and maintaining employment. When work is obtained it tends to be low paid, monotonous, sometimes dangerous, and often with low control of immediate environment. Lack of control is known to be associated with poor health outcomes (Totman, 1990). Having low income has been shown to be detrimental to health outcomes and education can be seen as strongly influential of income levels in terms of the type of work attainable given ones education level, and remuneration of differing work types. While the direction of the relationship is open to debate, it exists regardless of the theoretical standpoint, and proponents of both sides suggest this is a phenomenon that is both unsatisfactory and resistant to change.

Alternatively there have been those that argue that education impacts on resultant health outcomes (Jacobson & Thelle, 1988; Leigh, 1983). The relationship is positive, that is, as educational level increases health outcomes also improve. The direction of the relationship is even more pronounced in developing countries (Tresserras, Canela, Alvarez, & Sentis, 1992; Grosse & Auffrey, 1989), which is not surprising given the magnitude of difference

between high and low SES individuals in developing countries. In developed countries educational differences in relative terms are less than developing countries.

Explanations as to how educational differences impact on health are somewhat varied. Although health, especially in childhood, will impact on educational outcomes, it is my contention that the direction of the relationship is from education to health. Accordingly, henceforth in my descriptions of education and health, directionality has health as the dependent variable as a primary assumption.

Education is likely, although by no means inevitably, to influence health behaviours (Matthews, Kelsey, Meilahan, Kuller & Wing, 1989). Lowly educated individuals are more likely to use drugs (both licit and illicit: the latter of which has received scant attention), have poor nutritional status, are less likely to partake in adequate exercise on a regular basis, and are less likely to participate in public health programmes such as immunisation (Berkman & Breslow, 1983). The evidence from the Alameda County study (Berkman & Breslow, 1983) is longitudinal and therefore less likely to be subject to the bias inherent in cross sectional research, such as the phenomenon being an unusual happening at the time of the data collection which confounds results, and subsequent conclusions. Education is an essential component in health outcomes because it allows the individual to understand the consequences of behaviour better and to seek out knowledge and resources within the community to alleviate unhealthy practices. More educated people are better able to seek out and find work, and such work tends to be better paid, thus such individuals are able to secure a better income and in turn superior nutrition, housing, transport, and medical care.

There is known to be a link between education and sex practices, which is not surprising. Better educated individuals are more likely to use birth control, have knowledge of unsafe sexual practices, actually practice safe sex, and participate in family planning than less educated individuals. Those of lower educational status are likely to have more children, born closer together, of which such children are less likely to be as healthy in relative terms as children of better educated individuals (Grosse & Auffrey, 1989). Education is a mediator in many health outcomes even when income is controlled for (Valkonen, 1989). The strength of the relationship (even though based on developing country research) was well articulated

by Grosse and Auffrey (1989) when they stated "that a strong and consistent association of literacy with mortality has been demonstrated to be significant, independent of culture or level of economic development" (p293). A problem with much of the research of the education-health relationship is the use of literacy as a surrogate educational status. In many developed countries the rate of illiteracy is very small therefore profound effects of this problem are limited to very few members of the population. A way around this may be to look at degree of literacy and outcomes to examine progressive health improvements as a function of increases in literacy level.

More research is needed to assess whether such educational differences are predominantly responsible for observed health inequality, or more importantly, to examine just how much variance in health outcomes can be attributed to education level of the individual. It is my contention that such differences form part of a pattern of advantage for those in higher SE groups which is difficult to extrapolate to the one variable. It seems that an interactive combination of many SE variables leads to disproportionately negative outcomes for those of lower SES, and that these differences are mediated by psychosocial constructs such as cognition and perception.

### *2.1.3 Occupation*

The third major contributor to SES differences in health outcomes is social class. Although social class has various forms of measure, occupational group is by far the most popular (Arber, 1987). The Office of Population Censuses and Surveys (OPCS) in Britain has been involved in the collection of occupational data since the start of this century, thus such data are readily of great use for aggregate, time-series, and even longitudinal research. Up until very recently the analysis of such data has involved the comparison of five hierarchical classes in descending order: (I) Professional, (II) Managerial, (III) Skilled manual or nonmanual, (IV) Partly skilled, and (V) Unskilled. Criticisms of measurement (ie. those who advocate measurement error such as Illsley [1986] and Stern [1983]) have led to new approaches to social class analysis. Such approaches for control of measurement criticisms include comparisons of groups I and II (pooled) against groups IV and V (also pooled), as well as comparison of manual against non-manual occupations. Ironically such criticisms have

served to produce research which indicates the gap (health inequality) may actually be wider than first thought (Marmot et al, 1984, 1991). Both Whitehall studies showed that the more homogenous populations tend to exacerbate the health inequality trend. In a large sample of British civil servants those in the bottom SE group had a mortality rate three times that of the top group (Marmot et al., 1984). What this tells us is that heterogeneity tends to dampen the relationship which refutes the assertions of some (eg. Illsley, 1986) that a few groups in a heterogenous population account for substantial variance, that is, the selection approach (Illsley, 1987).

In a selected review of the SE health inequality since the Black Report, researchers using social class measures found that in the succeeding decade, social class differences in mortality had increased. Improved measurement procedures revealed greater mortality inequalities than had previously been found, such health inequalities could be found in all countries that collected the relevant data, differences exist in quality of life for the duration of life (that is, not only at "susceptible" stage of the lifespan), and that selection and artefactual explanations are insufficient to account for mortality differentials (Davey-Smith, Bartley & Blane, 1991). Better operationalisation of social class has resulted in steeper class gradients which indicates the relationship may be more pervasive than previously thought.

#### ***2.1.4 Geographic Location***

Geographic location is another factor affected by SES which seems to impact negatively on health outcomes. Researchers have found that areas of low SES have individuals with poorer health than for those in higher SES areas (Brenner & Mooney, 1983). The trends have been replicated in Australia (Siskind et al., 1987a, 1987b), UK (While, 1988; Balarajan & McDowall, 1988), and the USA (Stockwell, Swanson & Wicks, 1988). Geographic location will be strongly influenced by the three primary determinants of SES (income, education, and social class) and provides another example of how the components of SES interact to produce negative health outcomes for those of low SES.

### **2.1.5 Housing**

The housing status of the individual also has an effect on the health status of that individual (Martin, Platt & Hunt, 1987). Quality of housing can have direct effects on the health of occupants, such as respiratory disorders (Platt, Martin, Hunt & Lewis, 1989). Housing status is affected by the condition of the housing, its location (geographic location, which has just be briefly discussed), and the costs involved to maintain occupancy of the dwelling. Poor health can be implicated in downward mobility in quality of housing (especially if poor health reduces income and mobility) whereas poor quality housing can be implicated in poor health outcomes (Anson, 1988), especially for chronically damp or cold places (Dales, Zanenberg, Burnett & Franklin, 1991; Brunekreef, Dockery, Speizer, & Ware, 1989). A series of publications by Lowry (1989a, 1989b, 1990) offers a good overview of some of the key pathways through which health is affected by housing conditions. At the extreme end of the lower SE grouping the poor very often must deal with overcrowding and its known detrimental health consequences (Meyers, 1988), or even worse, homelessness. These problems are at the extreme end of the SE spectrum, but this should not deter from the importance of housing tenure (as a proxy for SES) and it's impact on health outcomes, for which there is a clear class gradient in health in most developed countries including New Zealand (Kearns, Smith & Abbott, 1991).

### **2.1.6 Transport**

Transport, or rather availability or access to transport, can have a direct impact on health in at least two ways, and therefore can serve as a proxy for SES. Firstly, without transport (whether own private or access to public transport) access to health services is impaired (Haynes, 1991). Secondly, a lack of transport limits access to social support, which is known to have a substantial influence on health outcomes (Herbert & Cohen, 1993).

### **2.1.7 Interrelatedness of Major Pathways**

It should have become obvious through the course of this discussion that components of SES interact to produce differential health outcomes. These separate indicators do not work

independently (that is account for much variance independently), rather they operate in a coordinated, systematic, and somewhat predictable way to produce concomitant health outcomes. For instance, someone who is poorly educated is also likely to have a low income, low paying (or no) employment, live in a run down geographic location in poor quality housing, not own a car and have little, if any, access to public transport. These factors in unison combine to differentially influence health outcomes.

Income, education, and occupation in particular are strongly interrelated. Income is generally a function of occupation which in turn is usually a function of education. Quality and subsequent level of education are usually dependent on parental income level. These three subcomponents are the core of the SES construct and can be called the SES determination triad. This triad of components has a strong influence on variables which carry less influence in the SES-Health relationship, namely housing, transport, and geographic location. It is not surprising that individuals scoring low on the triad also score low on health outcomes.

## 2.2 Poverty

### 2.2.1 Introduction

An inevitable conclusion that can be drawn from the discussion to this point is that those in low SE groups will have more negative health outcomes than those in higher SE groups. Poverty, or more precisely relative poverty, is an essential concept to understand for interpretation of my argument about psychosocial mediators in the SES-health relationship because relative poverty is an outcome of various influences which combine to determine low SES. I will briefly define poverty and recite a very brief history of poverty research at this point.

Conceptualising poverty has been a task of scholars and politicians alike for centuries, although actually trying to measure the concept of poverty has a somewhat shorter history. Even contemporary researchers have difficulty actually defining poverty. Poverty can be generically trichotomised for convenience into absolute, relative, or administrative terms (Townsend, 1979). In attempting to interpret and understand the SES-Health relationship,

especially across cultures, nation states, or across time, it is essential to appreciate which type of poverty is being alluded to in the research. For instance, health patterns in developing countries are likely to be more significant than for industrialised Western nations due largely to the poverty tending to be absolute in such nations rather than a tendency for the less dramatic relative deprivation observed in developed countries.

Dictionary definitions of poverty tend to revolve around absolutist conceptualisations. For instance, the Oxford Concise Dictionary defines poverty in terms of "being poor". In turn, being poor is defined in terms of not having the means to procure comforts and necessities of life, being ill supplied, deficient, or less well supplied than expected.

Initially I will set aside a detailed examination of the phenomenon of administrative poverty as it can be thought of as some arbitrary operationalisation pronounced by a political administration at a given time. Briefly, administrative poverty is an income level below which poverty is said to occur. Therefore the cut-off line for this figure could be thought of as the poverty line. Administrative poverty is often the point where governments will intervene to assure a minimum standard of living. Administrative poverty is an issue that logically should be discussed after the somewhat more theoretical conceptualisations of absolute and relative poverty.

### **2.2.2 *Absolute Poverty***

Absolute poverty, sometimes referred to as subsistence poverty, is defined in terms of having enough resources to subsist (Mead, 1992). This definition is based much on the work of Seebohm Rowntree late last century and in the early parts of this one. Rowntree devised "objective" criteria based on the work of primarily American nutritionist W.O. Atwater.

Making shrewd use of the work of W.O. Atwater, an American nutritionist, reinforced by the findings of Dunlop, who had experimented with the diets of prisoners in Scotland to find out how nutritional intakes were related to the maintenance of body weight, he estimated the average nutritional needs of adults and children, translated

these needs into quantities of food, and hence into the cash equivalent for these foods. (Townsend, 1979; p. 33).

Thus, this conception of poverty involves the physiological needs of the individual, and the minimum financial remunerative materials to acquire those needs. However, this approach is flawed in many ways. For instance individual differences suggest that minimum needs will vary somewhat. Also, who gets to set minimum need parameters? Are such parameters universal? (even sub-cultural differences will lead to different nutritional patterns and needs). Energy requirements will vary depending on how much is expended during the day, and tastes may even influence food intake (e.g. a vegetarian will not consume meat). On top of these many doubts about the subsistence concept, it is known that, especially in developing nations, people are able to live on food intake less than the so-called bottom line subsistence level (Ropers, 1991).

The absolute definition can be further subdivided. Rowntree (1977: cited in Payne, 1991) differentiated between primary and secondary poverty. Primary poverty resulted from an income insufficient to acquire the minimum for maintenance of "physical efficiency." This definition is still fraught with difficulty because maintenance of physical efficiency assumes some arbitrary line of physical functioning below which can be deemed inefficient. Individual differences would prevent such a line existing in reality. It also assumes that nutrition alone is sufficient to achieve physical efficiency, which is blatantly untrue. Many other tangibles such as shelter, clothing, and medical care are necessary for physical efficiency as well as psychosocial intangibles such as social support, stress management, and sense of control over environmental stressors.

Secondary poverty is where income is sufficient to meet the requirements of physical efficiency, but for some reason, the income is unwisely spent. Conventional wisdom would have it that prioritising on income, food, shelter, and fuel is advisable. However poor budgeting skills, overindulgence, and fatalism are thought to result in wasteful spending which may lead to a lack of essentials for "physical efficiency". Essentially this poverty as mismanagement (or wastefulness) can often be explained by poor prioritising skills in budgeting due to lack of education in the lower socioeconomic strata (Townsend, 1979).

Secondary poverty may be seen as a form of victim blaming or scapegoating. Understanding living priorities such as fixed costs (such as rent and power) and flexible costs (such as telephone, food, and transport) is beyond comprehension for some in higher SE groups. Sometimes those in lower SE groups make poor spending decisions on flexible costs which means they can not meet fixed costs. This seems to be becoming more prevalent as societal attitudes in many developed countries are becoming more conservative. Conservatism is associated with individual responsibility and accountability. By this reasoning, the explanations for a person's poverty may be "his own fault", much as, for instance, sporting success is often attributed to natural talent and training.

### *2.2.3 Relative Poverty*

Moving away from the concept of absolute poverty, a newer, more holistic approach to poverty was proposed by Townsend (1979). Relative poverty is a comparative conceptualisation, this is, poverty is seen as being relative given the standard of living that most of a given society enjoy. This divergence from the individualistic approach inherent in absolutist conceptualisation has removed some of the victim-blaming and victim liability components. "Individuals, families and groups in the population can be said to be in poverty when they lack the resources to obtain the type of diet, participate in the activities, and have living conditions and amenities which are customary, or at least widely encouraged, in the society to which they belong". (Townsend, 1979: p. 31).

The advantage of defining poverty in relative rather than absolute terms is that it departs from the moralising that occurs in the latter, as well as certainly being more applicable for developed countries. Poverty is defined in terms of what income is "normal" in the society being assessed rather than looking for scapegoats and victim-blaming which seems inherent in the absolutist approach. More importantly it is a holistic approach in its framing of poverty and involves a necessary move away from the reductionist means of determining absolute poverty (e.g. setting arbitrary subsistence levels, assessing what "physical efficiency" means etc.). It is also holistic in that it includes constructs such as social integration, social support, community participation and social ethos. A thorough examination of poverty is essential to illustrate several important issues in understanding the SES-health relationship.

Firstly, the distinction between absolute and relative poverty is important. Absolute poverty is rarely seen in developed countries. The health consequences for this type of poverty are much more extreme than for relative poverty. It seems the common perception of poverty is this type (Will, 1993; Zucker & Weiner, 1993). Given this common perception and the lack of absolute poverty in developed nations, it is easy to assume poverty does not exist in such countries, therefore the health inequality that is linked with SES is also nonexistent. Nothing, of course, could be further from the truth. Secondly, the concept of relative poverty involves a more holistic conceptualisation of SE deprivation as well as emphasising psychological factors such as social comparisons, social support and embeddedness, and various perceptual and cognitive processes. Thirdly, it allows for the examination of social factors which interact with financial deprivation, such as scapegoating and victim-blaming, to produce disproportionately negative health outcomes for low SES individuals.

## **2.3 Applications**

### **2.3.1 Introduction**

Having briefly considered some of the pathways and associated factors which allow SES to impact on health outcomes, along with a relatively detailed discussion of poverty, I believe these conceptual issues require an application to better appreciate the means by which such factors operate. I will use unemployment as a proxy for low SES and examine various non-material pathways by which this phenomenon may impact on health outcomes. Social support is a key factor which will be considered in the alleviation of high stress levels known to be associated with financial deprivation (Cassell, 1976). I will briefly outline some of the research on unemployment as it provides a reasonable operational definition of financial deprivation that has been officially sanctioned, that is, unemployment benefits are necessarily a government defined operational definition of poverty and therefore a good description of administrative poverty. Unemployed individuals are undoubtedly relatively deprived financially compared with the rest of the workforce-aged population. Additionally, the analysis of such individuals allows one to demonstrate how negative health outcomes are not simply the result of a lack of finance for the purchase of essential needs, but rather some

relationship where psychosocial and structural variables interact to produce negative health outcomes.

### *2.3.2 Unemployment*

It is virtually undisputed that unemployment is positively associated with morbidity (Jackson & Warr, 1982; Jahoda, 1981; Sandford & Mullen, 1985; Warr, 1987; Whelan, 1992; Bethewaite, Baker, Pearce & Kawachi, 1990; Moser, Goldblatt & Jones, 1987; Steffansson, 1991), as well as mortality from many causes (Iverson & Anderson, 1987; Moser & Goldblatt, 1987; Seabrook, 1982). There are degrees of unemployment (rather than the simple dichotomy of employed or not), with some work being permitted before unemployment benefits are cut, which allows slightly greater income than the unemployment benefit for some. Additionally duration of unemployment will render different outcomes depending on various characteristics of the individual (that is, individual differences) as well as social resources available to the individual such as social support, social cohesion, and community integration. Thus the statement that unemployment is positively associated with morbidity and mortality means that unemployment will increase both morbidity and mortality (Linn, Sandifer & Stein, 1985; Smith, 1987), although additional factors such as duration of unemployment and available support will interact to produce differential degrees of negative health outcomes (Leefflang, Klein-Hesselink & Spruit, 1992a, 1992b).

Historically unemployment was seen to lead to increases in morbidity because of a lack of material resources to meet ones needs, hence "impoverishedness" was assumed to be the cause of the negative consequences of being out of work (Totman, 1990). This assumption was invariably based on the work of individuals like Taylor (1911: cited in Landy, 1989) and his scientific management approach to work motivation. His assumption was that money was the primary motivation for work, and it therefore followed that better paid workers would be better motivated whereas, by the same faulty reasoning found in victim blaming, unemployed were unwilling or unmotivated to work. Without doubt Taylor was incorrect in his assumptions because many other job related factors have emerged as key candidates in work motivation research (Landy, 1989). The same is true for the "lack of money" assumption and

negative consequences of unemployment approach that has long been assumed to be "common sense" (Kim & Moody, 1992).

Two key theorists currently dominate the discussion on the issue of how unemployment is pathological: Marie Jahoda and Peter Warr. Jahoda (1981) regards employment as a social institution which besides having obvious economic benefits to the individual, has a number of latent benefits. These include time structure in day to day living, social contact, enforced physical activity, status, purposefulness, and control (although many unskilled jobs lack some of these facets, which might explain why those in lower SE groups suffer poorer health outcomes). According to Jahoda it is these latent benefits that, once removed (and not necessarily the decreases in income), are predominantly responsible for pathogenesis following the onset of unemployment.

Warr (1987) argues along similar lines to Jahoda. He suggests unemployment reduces opportunities for individuals and in doing so leads to reductions in the individuals perceptions of control. Lack of control (which is often conceptualised in terms of locus of control) has often been implicated with pathogenesis (Davison & Neale, 1990; Meyers, 1988; Totman, 1987), and it is the reduction in options that results in perceptions of loss of control over the events in ones life (that is, external LOC). Social contact and status are generally reduced after the onset of unemployment, which according to Warr (1987), along with the control issue, are mainly responsible for pathogenesis. The implication from both Warr and Jahoda's work is that reduced social interaction and perceptions of reduced control over one's life are predominantly responsible for pathogenesis following unemployment onset, not, as "common sense" might advocate simply the reduction in income.

For those who are unemployed, or those who are low in SES the constructs of social support and embeddedness are of crucial importance for the maintenance of good health. While there is little evidence of SE differences in social support (Matarazzo, Weiss, Herd, Miller & Weiss, 1984) there are several good reasons to suggest this might be the case, which may help to underline the role of psychosocial factors in health inequality. Structurally those in lower SE groups are likely to live in run down locations that are distant from many of the amenities in urban areas, tend to have no vehicle and limited access to public transport, and

generally lack the financial resources to afford the social mobility that those in higher SE groups enjoy. The lack of access to public transport can occur in two ways. Firstly, there may be a lack of bus or train routes to the impoverished area, and secondly, there may be greater demand for the service than what has been provided. This is because many people in poor areas have no vehicle and are therefore reliant on public transport, thus their dilemma may be twofold: a lack of supply or too much demand. Those in lower SE groups with good social support obtain this support within the local area, that is within close geographic proximity, whereas higher SE individuals have greater mobility and therefore are not reliant on social support within their immediate geographic location (which is likely to enhance perceptions of control). I will now briefly examine social support and control in the hope of delineating how these psychosocial factors operate in the SES-Health relationship.

### *2.3.3 Social Support*

Social support is essentially a construct denoting social relations between individuals. It can be strictly defined in terms of significant others, or more loosely in terms of all interactions between an individual and anyone else the individual comes into contact with. This concept of the degree of definitional strictness is known as the degree of embeddedness, and has to do with the quality of social support. Intuitively it would be expected that quality of social support (in terms of closeness of association or attachment) may moderate the relation between social support and health outcomes, however it seems that quantity up to a point is a more important determinant of the efficacy of social support avoidance or amelioration (Matarazzo et al., 1984). Regardless of the means by which it exerts its effects there is copious evidence that indicates social support is essential for normal healthy functioning, both psychologically and physiologically (Geiser, 1989; Sarason, Sarason & Pierce, 1990; Berkman, 1984), further, individuals with social support adjust better psychologically to stressful events (of which there would be no shortage of for the low SES individual), recover more quickly from already diagnosed illness, and reduce the risk of mortality from other diseases (Cohen, 1988; Cohen & Wills, 1985; Plotnikoff, Murga, Faith & Wybran, 1991; Taylor, 1990).

There are two main models of how social support exerts its effects: the buffering model, and the direct (or main) effects model (Cohen & Syme, 1985). The buffering model suggests social support will only (or mainly) improve health outcomes during times of stress (which will be most of the time for low SES individuals). Alternatively, the direct effects model advocates improved health outcomes as a consequence of social support regardless of stress or other challenges to health homeostasis. Thus it seems likely the means by which SES exerts its influence will, either way, negatively impact on those in lower SE groups. It is likely that those of low SES will have less social support resources (due to transport based access barriers), therefore both the buffering model and the direct effects model will favour those of higher SES. Additionally, those of higher SES have more social resources to cope with less "objective" stress. While this may be an oversimplification, it is one of a series of non-material factors which result in disproportionately negative health outcomes for those of low SES. This notion is supported by the knowledge that direct effects are generally reported when the support has been measured in terms of how a person is integrated within a social network, whereas buffering effects are generally reported when the support is measured in terms of the availability of psychosocial resources that help an individual respond to stressful events. On both accounts the low SES individual is likely to be worse off.

It is clear that stress in some way impacts negatively on health outcomes (although there are some situations where short term stress can be beneficial to the individual such as deadline in achieving various goals), although the precise mechanisms by which this occurs are somewhat unclear (Plotnikoff et al., 1991). There is evidence that psychosocial variables have substantial effects on human health outcomes (Ader, Felton & Cohen, 1991) and ample research has found for the depreciating effects of a lack of social support, that is loneliness, on health (Baron, Cutrona, Hicklin, Russell & Lubaroff, 1990; Jemmott & Magloire, 1988; Kiecolt-Glaser & Glaser, 1992).

## **2.4 Defining and measuring SES and Health**

### **2.4.1 *Socio-economic Status (SES)***

SES is a multifaceted construct that assesses the relationship between social and economic

situations for individuals. SES is often considered a measure of social class, although social class is really a subset of the components that form SES.

Measures of socio-economic status (SES) are somewhat variable in the literature. The type of measure chosen by the researcher is often dictated by research resources and available official data. The most common measures of SES are income, education, and occupational levels. Other less common SES indicators include geographic location (area), housing conditions, transport availability, family structure, ethnicity, and gender. Although these indicators are a diverse lot, they all tend to co-vary with health outcomes in a familiar pattern, although the magnitude of the relationship is somewhat variable. SES indicators associated with lower socio-economic state often have health outcomes much worse than those indicators of higher SES.

SES indicators are the independent variables in this research and health outcomes are the dependent variables. Although the health markers are somewhat crude, and to a lesser extent so too are the SES indicators, the complexity of the relationship, along with the difficulty in extracting meaningful data to extrapolate understanding from, necessitates over-simplistic but necessary operationalisation of the constructs under investigation in much of the research examined.

#### **2.4.2 Health**

Defining health, as for poverty, is a considerably complex task. Health is often only pondered upon if it is absent, that is when the individual is sick. Health is clearly a conceptual continuum. Health is bad when we are sick and good in the absence of sickness, but such circular reasoning tells us little of the nature of health itself.

Historically health has had its ideological basis in the realms of theology. Up until the middle of the nineteenth century, health was entirely in the realm of the gods, devils, demons or other supernatural phenomena. Victim-blaming, as it is now, was reflected in the sick person being punished by the gods for various improprieties or possessed by demons (often mental illness

such as schizophrenia) by virtue of having a sinful and unrighteous existence on earth (Davis & George, 1993).

Since the middle of the 19th century a more scientific approach has usurped theology as the primary moderator of health status. The biomedical model was based on the concept of external pathogens assailing a healthy victim and reducing the victims health, that is, incurring sickness. Biomedical causality was most often linear, that is, the more pathogens, the greater the chance of more severe health impairment (Davis & George, 1993).

However, in the last thirty years scientists have begun to question the universality of the biomedical model. Certain irregularities began to make the biomedical model somewhat less effective in explaining variance in outcomes of a number of diseases such as cancer, heart disease and asthma. These irregularities in the biomedical approach have led to the development of a new paradigm, the biopsychosocial model (Engell, 1977; Sarafino, 1990). While the biomedical model is still very useful in treating acute medical problems (e.g. heart attack), the same cannot be said for chronic health problems such as arthritis and atherosclerosis (Taylor, 1986). Ironically, the means by which biomedicine has succeeded in alleviating many health problems has had little to do with biomedical research or practice. Vast improvements in our knowledge of nutrition, sanitation, and health hazards (such as smoking and high fat diet), combined with public health initiatives (such as vaccinations) have been responsible for the major health gains of this century (Sheridan & Radmacher, 1992).

Unlike the biomedical approach, the biopsychosocial model promotes a more holistic integration of physical, psychological and social information. This interdisciplinary pooling of information leads to better diagnosis, intervention, and prognosis for individuals with health problems. It also leads to a more expansive definition of health. Rather than simply the absence of presence of disease or illness, health is viewed as the presence of positive health behaviours such as good nutrition, regular exercise, not smoking, being immunised and utilising medical services as necessary (Sarafino, 1990).

Health indicators are usually in terms of death rates (mortality), sickness (morbidity), and premature death among the newborn (infant mortality). Measures include self-reported

morbidity, health service utilisation, and mental health data (Segovia, Bartlett & Edwards, 1989).

## 2.5 Summary

This chapter examined poverty, and in doing so, introduced an important concept, relativity. Relative variability in social constructions necessitates the mediation of perception and various other cognitions. This, of course, brings psychological interpretations into the explanatory arena, and further strengthens the case for more holistic conceptions of the SES-health relationship. Poverty was introduced because it is essentially the definition of low SES, but more importantly, because the low level of SES inherent in poverty status is where negative health outcomes are disproportionately pooled.

To concretise some of the pathways alluded to in this chapter, an application was required. Unemployment was chosen as a good application for two main reasons. Firstly, it served as a good operational definition of administrative poverty. As an indicator of administrative poverty it allowed the more extreme influences of low SES on health to be examined. Secondly, unemployment has been comprehensively examined in the scientific community, and the psychological and social outcomes are widely accepted. Clearly this provides an opportunity to emphasise the non-material and psychosocial pathways by which SES impacts on health.

A brief section on defining and measuring health was included in this chapter to allow the reader to understand how SES and health data are operationalised and collected. This was included to permit an understanding of some of the difficulties in extrapolating meaning from measures of SES and health.

The thrust of this chapter has been to consider pathways by which SES impacts on health outcomes. Describing factors such as income, education, and occupation as pathways may actually overextend the concept of a pathway. A pathway could be considered a sequentially related group of factors that mediate a start and end point in a relationship. The factors assessed are probably better considered in isolation as associations in the SES-health

relationship. Of course, these associations do not stand alone in the real world and are all interrelated. These associations, or pathways, serve to illustrate the proximal factors which mediate SES and health outcomes. This mediation by proximal factors is an important concept for understanding the phenomenon of SE health inequality in terms of a more holistic theoretical framework because it links two factors (SES and health) which have previously lacked credible theoretical explanation in terms of total health variance explained as a function of differential SES.

### 3.0 THEORETICAL APPROACHES TO THE SES-HEALTH LINK

#### **3.1 Introduction**

The purpose of this section is to discuss the key theoretical approaches for the SES-health phenomena proposed in the literature to date. Initially I will outline the theoretical approaches advocated in the Black Report (DHSS, 1980). Following this Feinstein's (1993) model will be considered.

The Black Report proposed four theoretical explanations for SE health inequality. The artefact explanation argues that both social class and health are artificial creations attempting to define complex social phenomena. In the creation of these constructs error in definition, collection of data, and finally extrapolation from findings leads to inflated and misleading conclusions. Simply, artefact was a function of measurement error and proponents of this approach suggest health inequality does not actually exist.

The social selection explanation, which is also known as natural selection, proposes health impacts on SES outcomes. In this formulation health is the independent variable and SES the dependent variable. Individuals who are affected by ill health will tend to deteriorate in SES as their capacity for employment, education, or income generation declines as a function of their poor health.

In the cultural/behavioural approach the level of analysis is necessarily individual. The individual is seen as responsible for his own behaviour and health outcomes are viewed as being caused by the behaviour of the individual. Factors such as cultural affiliation are suspected for shaping various health behaviours such as consumption of tobacco, alcohol, and illicit drugs, as well as nutritional habits and medical service use. In this explanation it is argued that individuals in different SE groups will behave in systematically different ways, with those in lower SE groups behaving in ways which are known to be detrimental to health outcomes. While other SE groups also behave in ways which will have negative health outcomes, the tendency is for more of such behaviours to occur in lower SE groups.

In the social causation approach various societal structures are seen as vital in shaping the environment for the individual. It is this product of societal structural forces which is viewed as causal in health outcomes. This approach is also known as materialist or structuralist. Structures such as health care services, education, government welfare, and even government economic policy can be seen as influencing health outcomes. The relationship between these structures and health outcomes becomes direct when smaller proximal structures such as religious and organised groups, schools, and the workplace are considered in mediating roles.

The difference between the four Black Report explanations are considerable. Social causation is assessed at the group, community, and societal level of analysis, whereas cultural/behavioural explanations assess the link at the individual level of analysis. Both these approaches assume directionality in the relationship with health as the dependent variable. In contrast, social selection sees SES as the dependent variable. Like cultural/behavioural approaches, social selection explanations of SE health inequality are mainly at the individual level, although it is assumed that accumulated selection processes result in health inequality.

Differing disciplinary approaches to the SES-health relationship will also yield differing levels of explanation. Medicine tends to approach the problem at the individual level, which is not surprising given that the unit of analysis is the individual rather than groups. However, from within medicine the sub-disciplines of epidemiology and public health depart from the overall theme of the individual to consider population perspectives in terms of risk factors, incidence, and various other societal level conceptualisations relevant to medicine overall. Sociology by definition explores the relationship from a societal level, whereas (with the exceptions of social and community psychology) psychology assesses the relationship at the individual level. While most people believe economics to be the study of economic factors within nations (macroeconomics) or societies, the dominant arm of economics that attacks health problems (predominantly Health Economics) tends to devise somewhat complex mathematical formulae to predict costs per unit for health care. This is done to enable cost effectiveness analyses and the like to be undertaken to evaluate the "efficiency" of health "enterprises". While there may be other disciplinary approaches (e.g. anthropological, paediatric [which is really a sub-medical discipline and philosophical]) the main approaches in the literature are from medicine and sociology, with psychology in a distant third place.

### 3.2 Artefact

The artefact explanation for socio-economic inequalities in health implies that inequality is not actually present, but rather it is a product of measurement error. The crudity of the measurement process does lend some weight to this argument, although most researchers the field have overlooked this theoretical explanation as commanding anything but minimal SE health variance (eg. Blane, 1985). Pamuk (1985) produced evidence to discredit the artefact explanation arguing that the procedural safeguards in data collection, encoding, and interpretation did allow for potential error, but such error was too small to possibly account for the size of effect involved. Longitudinal research has produced similar discrediting evidence for this explanation (Fox & Goldblatt, 1982; Goldblatt, 1990; Wilkinson, 1986b), usually in terms of suggesting the size of effect was too large to be accounted for by this explanation.

An in depth consideration of measurement bias is essential for the accuracy of conclusions from research to be critically assessed. It is essential to recognise that most artefactual accusations are directed towards the UK OPCS occupational classification methods. Subsequent research has tended to control for such criticisms. From the literature there are several possible artefactual influences that may be operating, to differing degrees depending on the type of research (MacIntyre & West, 1991). Ethical considerations generally mean that the research on the SES-health link is observational, self-report, or an analysis of census-tract statistics. Census-tract analyses invariably involve the analysis of mortality (the health outcome) as a function of occupational status or some other socio-economic measure available in census figures.

The use of such statistics brings us to our first measurement problem, numerator/denominator bias. In the assessment of mortality rates the occupation of the deceased is obtained from death certificates (numerator), while the total number of individuals in each occupational class is extracted from census tract figures (denominator). Mistakes in allocation of occupation (e.g. misreading) from either source (death certificates or census figures) results in error and potential bias (Illsley, 1986). Social class mortality rates may be misleading, especially for class V, unless analysed with care due to the changing size and structure of classes over time

(Whitehead, 1987). This problem has been overcome by various means including comparing manual and non-manual occupations or combining data in classes I & II and comparing with classes IV & V (Wilkinson, 1986b).

Another problem is the reclassification of classes. Occupations have repeatedly been reclassified at each census since 1931 which has made class comparisons over time difficult, unless adjustments are made to account for such reclassifications. One means of overcoming this problem has been to trace occupations that have stayed in the same class for the period under study and discard those that have changed (Whitehead, 1987).

Changing class size presents another measurement problem to the potential artefact arena. There is a smaller percentage of the population in group V and larger percentages in classes I & II since 1931 because of two key factors. Firstly, there has been an upward shift in occupations through the class hierarchy due mainly to more of the population being better educated. Secondly, increases in unemployment, more women in the workforce (and an increasing number of older people in society owing to the ageing population structure), has altered the structure of those "economically active" in the UK. Being economically active is one of the criteria for inclusion in the various analyses carried out on the SES-health relationship by the OPCS. One means of overcoming this problem, as for controlling for numerator-denominator bias, is combining of groups I & II compared with groups IV & V, which compares similar proportions of the economically active population.

Another problem has to do with equivalency of quality of life between occupation groups across time. For instance the quality of life for an individual in group I in 1921 is probably very different from a group I individual in 1981, invariably even in real terms. As for reclassification and class size changes this problem reflects the issue of not comparing like with like, or comparing apples and oranges to decide the best apple. Quality of life is a somewhat qualitative concept and thus requires some degree of subjectivity in assessment. Quality of life should not be confused with the concept of standard of living. Standard of living can be considered the "objective" quantification or operationalisation of the "subjective" concept of quality of life. Differing value systems for different individuals make the concept

of quality of life problematic. Given the subjectivity it is difficult to see how this problem could be controlled for.

Some of the research in this field has taken samples from only a proportion of the population. This has given rise to criticism of the use of a small proportion of the population as a basis for generalisations across the entire population. However, the nature of the research occasionally dictates only assessing a small proportion of the population to make limited generalisations. For instance research into premature mortality by definition will often confine its data collection from 15-64 year age group or sometimes for infant mortality, the newborn only.

### **3.3 Social Selection**

In this approach social class is the dependent variable and health is the independent variable (Strong, 1990; West, 1991). The social selection explanation (also referred to as natural selection in the literature) argues that health impacts on social status, economic status, and mobility (Carr-Hill, 1987; Stern, 1983). One of the larger problems of the health inequality debate has been to establish firmly the direction of the relationship between health and social status. According to the social selection explanation those who are sick during youth or become sick for any length of time during adult life are likely to slip down the SE hierarchy as a consequence of their illness (MacIntyre, 1986). While this may be true for a few (e.g. mobility loss via being or becoming disabled), for the vast majority illness is transitory and is not significant enough to result in a loss of SES. (Wilkinson, 1987). Two health factors may have an influence on income. Firstly, illness in childhood, especially chronic, may impair educational achievement, and thereby reduce future prospects. Secondly, failing health may lead to downward social and economic mobility in the latter part of working life. However, as previously mentioned, mortality gradients are greater in young than old, which indicates this is a crucial period for health, and would tend to counter the downward mobility with age argument proposed by some (Illsley, 1986).

The drift hypothesis for schizophrenics is a good example of how health factors do seem to impact negatively on social status. Schizophrenics withdraw and function less effectively than

before the disorder's onset. This reduces job effectiveness which can lead to job loss, and tends to lead to jobs of less status and pay. For some, unemployment results and can exacerbate the problem. Marriages and relationships tend to flounder and the schizophrenic tends to find him/herself in poor housing or homeless (Wilson, O'Leary, & Nathan, 1992). Chronic bronchitis is a good example of a physical rather than psychological disorder that seems to have similar consequences to schizophrenia (Meadows, 1961, cited in Blane, 1985).

The problem with selection explanations is they do not explain enough variance in health inequality to account for observed discrepancies (Wilkinson, 1986, 1989). While chronic illness is likely to increase in the future, and absorb more variance, at present its levels are insufficient to explain observed differences (Whitehead, 1987). It is accepted that health slowly deteriorates with age. It is also accepted that in most developed countries population structures are changing. Unlike developing countries, where birthrates are high and a large proportion of the population is distributed in the lower age brackets, in developed nations birthrates are relatively low and a large proportion of the population are middle-aged. This ageing population structure is problematic because as the population ages health outcomes for that population are likely to deteriorate, especially in terms of morbidity. If this trend continues, which it is likely to, a future scenario may see selection processes as having more explanatory power in terms of health inequality.

### **3.4 Cultural/Behavioural Approaches**

The third major group of explanatory theories from the Black Report was Cultural/Behavioral explanations. According to this approach different socio-economic groups behave differently with regard to various health behaviours. Behaviours such as the consumption of harmful substances (e.g. alcohol, tobacco, illicit drugs, high sugar or fat diets), regularity of exercise (vs sedentary lifestyles), and utilisation of various health services (e.g. vaccination, ante-natal health services, contraception, and regularity of use, i.e. regular check ups), constitutes the behaviours thought to differ systematically in this conceptualisation (Townsend & Davidson, 1982; Miller & Hunter, 1990). According to this approach the individual is responsible for his own behaviour, and thus altering behaviour on this individual level will alleviate health inequality.

The behaviour of those at most risk from a wide range of negative health outcomes as a consequence of SE deprivation has proven remarkably resistant to persistent efforts by public health agencies to bring about change (Blaxter, 1990). This resistance to change has led to increasingly aggressive efforts by public health agencies to bring about change and the intensity of such initiatives have been the subject of criticism (e.g. Marantz, 1990). Accusations have ranged from victim blaming (Whitehead, 1987), to health fascism from some quarters (Brownell, 1991). Brownell argued succinctly that individuals have limited power over the health outcomes of their own bodies and contended the reasoning behind extreme public health initiatives was flawed. She argued that the idea of behaviour being volitional, an expressive of personal choice, and hence individual responsibility was in some cases being carried too far. The following statement is an example of thinking that Brownell was attempting to decry: "If a person can do as much to improve health, bad health can be perceived as a personal and moral failing." (Brownell, 1991: 306).

### 3.5 Social Causation

Like cultural/behavioural explanations, the social causation theoretical approach sees SES as the independent variable and health as the dependent variable. The social causation approach differs from cultural/behavioural explanations in terms of level of analysis. This approach suggests the overall social and material structure of a society impacts on its low SES (structurally disadvantaged) citizens disproportionately adversely. Those at the lower end of the SE spectrum not only have shorter lifetimes (as reflected by mortality statistics) but are less healthy during their shorter stays in terms of morbidity statistics (Whitehead, 1987; BMA, 1987; Goldblatt, 1989).

Social causation not only implies poor outcomes for low SES members, but a graded trend across all SES groups with those at a higher SE level living longer and enjoying better health than those at lower SE levels in their lifetimes. It is a graded process with those in the OPCS group two fairing better than group 3, and group 3 better than group 4. This grading trend can be conceptualised as a principle of incrementality.

Components that fit into the construct of social structure within a nation are those structures in society that impact on living standards and include income, education, occupation, distribution of wealth, access to health, housing conditions and transport availability, amongst others. These structures impact on individuals within a society differentially. This differential impact, combined with differences in individuals, results in differing health outcomes for individuals. This impact is clearly not direct, but mediated by various psychosocial factors which will be considered later.

One of the problems with the social causation approach is that it tends to be descriptive rather than explanatory. It sets out the social structures in a society that impact on health but does not stipulate how (for instance, via a sequence of proximal mediators) this occurs. Essentially this argument is hard to provide direct support for. Support tends to come indirectly from the inability of other explanatory approaches to account for even minimal proportions of health variance, and the robustness of the phenomena across differing societies and timeframes. Although this approach makes some intuitive sense, the linkages to individual health outcomes have proved far from conclusive (West, 1991). This problem has to do with the difficulties involved in extrapolating causal factors in individual health outcomes from abstract societal structures such as health and education.

It is probably best to look at social causation in tandem with cultural-behavioural explanations. They analyse the SES- Health relationship at different levels (societal and individual respectively), therefore if combined they provide a more comprehensive and plausible model. That is, concepts such as occupation can provide more meaning when considered in terms of individual exposure to hazardous substances in the workplace, sense of control of work environment, and differences in risk of accidents. Psychologic responses to work environment in terms of control, and risk for various negative outcomes, enforce an individual level of analysis to a concept (occupation) which is considered to impact on health outcomes at the societal level of analysis. Clearly this approach would generate research and theory building which would better explain health inequality than current approaches do.

### 3.6 Feinsteins Model

Feinstein's (1993) model for the explanation of SE health inequality will also be considered in my analysis of theoretical approaches in the SES-health link. Feinstein's approach considered material, non-material, individual, and developmental factors in a complex interplay which resulted in health inequality.

In an influential review of the literature Feinstein (1993) proposed a somewhat different theoretical format (from that proposed by the Black Report) for a model to explain socio-economic health inequality. Feinstein proposed causes could emanate from either personal causes or life stage influences. Rather than looking at different levels of analysis, and to a lesser extent directionality, Feinstein conceptualised causation at the individual level and temporally.

Personal causes can be divided into resource dependent (ie. materialistic) characteristics of the individual such as wealth and property ownership, and non-resource dependent characteristics such as genetic attributes, psychological make up, and cultural affiliation. Like personal causes, life stage causes can also be dichotomised. The first component of the dichotomy is the individual's life experiences. With regard to health outcomes, these include diet, drug use, occupation characteristics, and educational background. The second component of the life stage dichotomy is health service utilisation (which includes the access to health care).

According to Feinstein, these explanatory characteristics interact to result in differential health outcomes. Materialistic variables interact with lifespan characteristics to elicit differential outcomes in housing, overcrowding, sanitation, transport, occupational and environmental hazards. Materialistic variables interact with health care utilisation to bring about differential outcomes in capability to purchase health care, pharmaceutical and regular medical and dental care.

Behavioural and lifestage characteristics tended to interact to result in differential diet, drug use, exercise behaviours, educational achievement, leisure activities, and general propensity

for risk-taking. The behavioral/health service use interaction led to differential self diagnosis, adherence and comprehension of medical advice, access to both medical services and information, and awareness of recurrence. The above behaviours will be influenced by lifestage development. For instance, the propensity for drug-taking will be strongly influenced by the age of the individual. Although a generalisation, riskier behaviours are more likely at younger ages (owing to concepts such as adolescent egocentrism) when the consequences of such unhealthy behaviours are unlikely to be expressed in terms of negative health outcomes. Alternatively, the onset of physical decline may be seen as a cue for more physical exercise in a slightly older individual.

Feinstein's emphasis on the individual and temporal issues ignores structural factors which serve to shape access to facilities (e.g. access to health care) and, as many social causation proponents argue, to indirectly influence patterns of morbidity or mortality within that given society. The temporal issue is important. For instance, there will be some who delay financial gratification in their pursuit of academic achievement, for the potential long term gains of educational achievement such as higher income and occupational prestige. Those who do so may accept their relative poverty temporarily for the greater long term gains an education offers, although in the analysis of the SES-health relationships, these individuals are assumed to be part of the lower SE strata (Carr-Hill, 1987). Life stage status clearly will have a positive relationship with resource dependent factors, and to a lesser degree with behavioural (non-resource dependent) in terms of knowledge, experience, and ability to "play the system". Feinstein's model provides an interesting new approach to SE health inequality because it serves to illustrate the complexity of the SES-health relationship. It also attempts to link factors in the relationship in such a way that outcomes are not simply the consequence of one causal pathway but multiple pathways. The importance of multiple pathways is the rationale for a more comprehensive theoretical approach, which is the crux of this thesis.

### **3.7 Conclusion**

It is essential to outline differences in the level of approach to the SES-health relationship. By level of approach I refer to the size of the group (or individual) at which the relationship is being assessed, or at which SES impacts on health outcomes. An appreciation of level of

approach is important because differing level approaches reveal findings which may be incompatible with findings from another level approach. The artefact explanation will vary depending on what type of measure is being blamed for the "misleading" conclusions. The social causation explanation will argue at the macro or societal level of explanation while social selection and cultural/behavioural approaches are generally arguments at the individual level. The rationale for the combining of cultural/behavioural and social causation explanations was primarily for more comprehensiveness because it attacked the problem across more than the one level of analysis. Feinstein's conception was included because it combined factors to explain the relationship in a more comprehensive way than explanations in the Black report.

All the theoretical explanations considered lack sufficient explanatory power or specification for causal inference. Differential level of explanation has a lot to do with this problem. While social causation has received much support in the literature, little has been forthcoming on the means by which this approach actually impacts on individuals. Selection and cultural/behavioural approaches specify the means by which individual level factors will impact on health, but lack the power to explain health discrepancies given the size of effect in SE health inequality. The combination of social causation and cultural/behavioural approaches improves explanatory power but lacks the support of an overarching theoretical framework. Chapter 5 will seek to address this deficiency.

## 4.0 PSYCHOLOGY AND THE SES-HEALTH RELATIONSHIP

### **4.1 Introduction**

The means by which SES impacts on health has been a puzzle for many years. There are those that claim this type of research has been ongoing for centuries (Strong, 1990), although my research focus has been motivated by the findings and recommendations of the Black Report (DHSS, 1980), the Health Divide (Whitehead, 1987), and research subsequent to the Black Report.

Psychological approaches which contribute to the explanation of socio-economic health inequality can be broadly categorised according to the source of the cause of psychological disequilibrium (internal vs external) or how psychological correlates impact on the relationship under examination (direct vs indirect). Essentially the external causes of disequilibrium are those factors operating in the environment of the individual. Stress is generally viewed as an external source of disequilibrium, although this relies on a very simplistic conception of stress. Internal factors generally reflect dispositional characteristics. The degree of directness depends on whether the psychological factor directly impacts on somatic systems (direct) or health behaviours (indirect).

It is my contention that various forms of psychological disequilibrium contribute to negative health outcomes for those in lower SE groups. Understanding the causes of disequilibrium is essential for any potential psychological intervention, because an understanding of how psychological factors interact with both SES and health and the relationship under examination (SES - health) helps explain the nature of that relationship. Understanding how internal, external, direct and indirect psychological factors influence the SES-health relationship enhances our knowledge of that relationship by providing mediators for structural factors to impact on individuals. Further, understanding how psychological factors impact on physiological mechanisms provides the link between thought processes and physiological pathology.

In my examination of psychological factors I will first examine internal factors then external factors. I will conclude this section with an outline of how psychological factors impact on physiological processes. Directness can be evaluated by considering whether the factor under consideration acts directly on physiological systems or on behaviours which predispose the individual to negative health outcomes.

## **4.2 Internal factors**

Internal psychological factors refers to those constructs that are said to reside within the individual, that is, personal dispositions. Personal dispositions are sometimes referred to as personality characteristics or character traits and for the purposes of this discussion are deemed interchangeable. In actuality a detailed examination of traits, personality and disposition reveals subtle definitional differences, however, such differences are insignificant in the context of my analysis.

### **4.2.1 General Mechanisms**

The first part of my examination of internal psychological characteristics falls heavily on the work of Suls & Rittenhouse (1990). It examines three paths by which personal disposition is associated with health outcomes although emphasises that single path causation is unlikely given the complexity of the relationship. This complexity can be demonstrated by the reality that factors will overlap at the individual level to result in differential outcomes (individual differences). In the context of the following discussion personality refers to somewhat uniform ways of thinking and behaving over time that differ across individuals.

The first of the three pathways advocated by Suls & Rittenhouse (1990) is the personality induced hyperreactivity model. This approach suggests that individuals by means of personal disposition differentially respond to various environmental stressors with exaggerated physiological reactivity. Responses can be acute or chronic and are reliant on the individuals perception of environmental stress. Thus the individuals relatively stable personal disposition differentially appraises environmental stress which in turn leads to differential physiological reactivity (in some hyperreactivity) and results in differential health outcomes.

Hyperreactivity has been associated with coronary heart disease in terms of arterial damage, narrowing arteries, and increased risk of cardiac arrhythmias which predisposes the individual to vastly increased risk of sudden cardiac death.

The second pathway is known as the constitutional predisposition model. This approach submits that personal dispositions related to health risk may simply be indicators of some inborn physiological weakness or abnormality that increases disease susceptibility. It is the underlying proposition that these "weaknesses" place the individual at increased risk of negative health outcomes. This approach has two core assumptions: firstly, that personality has a powerful constitutional (genetic) origin, and secondly, that such disposition (personality) increases susceptibility to negative health outcomes. An example of this approach is Eysenck's (1967: cited in Eysenck & Eysenck, 1969) biological theory of personality, in which a dimension known as extraversion-intraversion was thought to vary between individuals. The more introverted the individual scored the more neurotic the person was believed to be. A higher neurotic score was thought to predispose individuals to greater risk of negative health outcomes.

Both the first two models described direct influences on health outcomes. The first suggested individuals will respond differently to environmental stress, and hyperreactors are at high risk of physically pathological outcomes. The second argued that a proportion of the population will have genetically inferred "weaknesses" which also increase the likelihood of pathological physical outcomes. It is possible that such a mechanism (or series of interrelated mechanisms) is responsible for some baseline minimum level of premature mortality or disproportionate morbidity for certain individuals across a population. This would imply a selection explanation for observed SE differences because it suggests those in higher SE groups had selected better outcomes because they are inherently "stronger" or more "stress resistant" individuals. However, this belies the research evidence which suggests the size of effect is too large to be explained in this way, and has been strongly opposed by many researchers in the field (for good recent reviews see Williams, 1990; Davey-Smith et al., 1990; Feinstein, 1993).

The constitutional predisposition approach sees the individual as somewhat passive with his or her genetic "blueprint" as determining health outcomes. Besides the idea of a few genetically transmitted diseases (eg Huntingtons disease and PKU) it is hard to credit major social class differences in health outcomes to genetic predisposition. If such mechanisms were in operation one would expect random variation in inequality (which it clearly is not), although the third proposition (to be discussed shortly) is more plausible.

A third approach labelled the Dangerous Behaviours Model (Suls & Rittenhouse, 1990) argues that personal dispositions may bestow greater risk of negative health outcomes by means of submitting the individual to inherently riskier situations. This model deviates largely from the generally accepted view that stressful life events consist of a variety of risk factors that are clearly separable from the characteristics of a more or less passive individual. That is, the events in an individual's life are intimately bound up in that individual's style of life. Personality differences applied across the population perspective may explain differential health outcome although it is questionable to assume that such personality effects are related to SES. It may be that constitutional and environmental factors for those in lower socioeconomic groups place those individuals in positions where they are more likely to indulge in dangerous behaviours (McLeod & Kessler, 1990). It is not hard to see how taking on work with high risk or noxious environmental circumstances is a better option than unemployment and continued financial deprivation. It is also intuitively appealing from a psychosocial perspective that personality does not operate in a vacuum, that is, individual personality is not a passive agent influenced solely by environment or genetic factors, rather a complex socio-cultural, psychological, physiologic interplay leading the individual to weigh up the odds of success of any given behaviour and act accordingly. Indeed this approach is somewhat synonymous to the Theory of Reasoned action proposed by Ajzen & Fishbein (1980: cited in Bennett et al., 1990). Despite recent evidence to the contrary (Myers, 1988), Ajzin & Fishbein suggest attitudes are predictive of future behaviours. Clearly attitudes will differ somewhat by social class, but the conceptual leap in inferring that a theory at the individual level has the power to explain at the population level is suspect, although an intriguing prospect.

#### 4.2.2 *More Specific Mechanisms*

In departing from a general psychological pathway approach, I will now analyse some internal psychological correlates in more detail. It is undoubted that at the individual level differential personality characteristics will place the individual at variable risk of negative health outcomes. Whether such differences are sufficient to explain the magnitude of health discrepancies by social class found in publications like the Black Report or Health Divide is questionable, but our understanding of the relationship would be somewhat impoverished if such influences were not given some consideration, with a view to incorporating them into a broader understanding of the SES-health relationship.

Depression is known to disproportionately impact on those from lower socioeconomic groups (Davison & Neale, 1990; Wilson et al., 1992). Depression, along with distress and vital exhaustion are known as negative mood or affective states. Vital exhaustion is defined as a mental state characterised by unusual fatigue, a feeling of being dejected or defeated, and increased irritability (Appels & Otten, 1992). Depression (and associated states) has been linked with increased cancer incidence (Shakelle, Raynor, Ostfeld, Garron & Beiliauskas, 1981) as well as cardiovascular disease (Somervell, Kaplan, Heiss, Tyroler, Kleinbaum, & Obrist, 1989).

Depression, like most psychological disorders, is not simply a case of "all or nothing" (ie. a threshold effect), rather is a disposition which fluctuates within and between individuals on a conceptual continuum. It is likely that those in lower SE groups will tend to score higher on this continuum and be at greater risk of negative health outcomes (Link, Lennon & Dohrenwend, 1993). This does not mean that low SES individuals are statistically more likely to be suffering from clinically diagnosed major depression. Rather it infers such individuals will tend to score comparatively worse than higher SES individuals on the depression continuum. It is this tendency for higher depressive scores that may predispose low SES individuals to inferior health outcomes by means of a psychological mediator.

Associated with negative affect is the controversial dimension known as neuroticism. Neuroticism has been defined as an extensive dimension of individual differences

characterised as a tendency to experience a broad spectrum of negative emotions such as anger, anxiety, depression and helplessness (Adler & Matthews, 1994), and is known to have deleterious health consequences. Studies have found neurotic individuals tend to rate health poorly (Watson & Pennebaker, 1989), report more stressful events (Affleck, Tennen, Urrows and Higgins, 1992), report more uncomfortable physical symptoms (Salovey & Birnbaum, 1989), and over-rate the effects of any given stressful event (Bolger, 1990). While direct links between neuroticism and negative health outcomes might be lacking, it has been found that perceived health is predictive of mortality independent of known biological risk factors (Kaplan & Camacho, 1983). The clustering of neuroticism among low SES individuals means this is another psychological pathway which impacts on negative health outcomes and may contribute to the SE health inequality gradient.

Recent research suggests that positive affect in terms of optimism, self esteem, and explanatory style may have been underestimated as a possible causal influence in health outcomes (Taylor & Brown, 1988). Those in lower socioeconomic groups are less likely to be optimistic and thus are more likely to be affected in health outcomes by a lack of this disposition. Optimism has been found to be associated with reporting fewer physical symptoms (Smith, Pope, Rhondewalt, & Poulton, 1989), better coping in stressful circumstances (Aspinwall & Taylor, 1992) and faster recovery from bypass surgery (Scheier, Matthews, Owens, Magovern & Lefebvre, 1989).

Until very recently it was believed the Type A personality construct was causally involved in negative health outcomes such as coronary heart disease (CHD) and various other circulatory disorders (Rosenman, Brand, Jenkins, Friedman, Straus, & Wurm, 1975; Chesney, Eagleston & Rosenman, 1981). Type A behaviour pattern (TABP) can be assumed if one or more of the following characteristics is exhibited: intense striving for achievement, competitiveness, over commitment to work, profound sense of time urgency, impatience and hostility. More recent research has found that TABP is not significantly related to CHD or many other negative health outcomes as was first thought, (Eaker, Pinsky & Castelli, 1992; Mathews, 1988). It appears that the multidimensionality of the TABP was distorting the true picture obtained from the Framington Study (Rosenman et al., 1975), and that a subset of the components of TABP were predominantly responsible for negative health outcomes, those

being hostility, anger, and anger expression (Dembroski, MacDougal, Costa, & Grandis, 1989; Houston, Chesney, Black, Cates & Hecker, 1992). TABP is the only personal disposition thought to act negatively in health that is more predominant in higher than lower socio-economic classes (Adler, Boyce, Chesney, Folkman, & Syme, 1993), which is why it is often known as the "manager's disease".

Recently a new personality construct has been proposed known as the Type C personality. It is characterised by one or more of the following traits: co-operative and appeasing, unassertive, patient, compliant to external authority, unexpressive of negative emotions (especially anger), and often being described as "nice", stoic and self-sacrificing. Indeed the Type C construct has been associated with negative health outcomes including possible cancer causation (Temoshock, 1990). It seems quite possible that components of TABP and TCBP (Type C Behaviour Pattern) work at opposite ends of the socio-economic hierarchy and that personality polarisation to either type is detrimental to health. However much work needs to be undertaken before TABP or TCBP can be linked to the problem of SE health inequality.

Coping and explanatory styles may also explain some of the SES-health variance. Whether or not these personality "traits" differ systematically according to SES is questionable, however like TCBP they offer "personality" explanations for the disproportionately poor health outcomes of those at the lower end of the socio-economic hierarchy relative to those at the upper ends.

#### **4.2.3 Cognitive Processes**

Coping styles involve coping strategies and broadly speaking coping types. Coping types can be broadly subdivided in problem focused coping which involves behavioural and cognitive strategies to directly address the problem, and emotion-focused coping which address the negative affect associated with the problem (Lazarus & Folkman, 1984). Emotion-focused problem solving does not do anything to solve the problem, rather alleviates distress associated with the problem (Lazarus & Folkman, 1984). It is my contention that (due mainly to resources obtained by what is generally a better education) individuals in higher SE groups tend to implement problem focused problem solving strategies whereas low SES individuals

tend to use emotion focused strategies. Indeed this proposition implies various health behaviours may be a by-product of coping strategies (eg. tobacco and alcohol consumption).

Aldwin & Revenson (1987) identified several different coping strategies of which three were problem-focused, namely instrumental action (which involves efforts directed toward solving the problem), support mobilisation (which involves obtaining information, advice and emotional support from others), and negotiation (directed toward others in the problem situation); and four of which were emotion-focused, namely escapism (includes daydreaming, fantasising and drug use), exercised caution (holding back actions that may do more harm than good), minimisation (refusing to dwell on the problem and carrying on as if nothing had happened), and self-blame (directing problem causation inward). It is well known that education and SES are strongly positively correlated (Winkleby, Jatutis, Fran & Fortmann, 1992; Valkonen, 1989; Matthews, Kelsey, Meilahn, Kuller & Wing, 1989). It follows that better educated individuals are more likely to use problem-focused coping methods (due to the positive correlation between education and SES, assuming education and problem solving are also positively correlated), which are more likely to solve the problem than emotion-focused methods.

As already alluded to, education is positively correlated with SES. It stands to reason that education will also be positively correlated with cognitive abilities and processes. It is these cognitive abilities and processes that are said to mediate basic learning processes (that is operant and classical learning) and learned outcomes in Banduras (1977) classic social learning theory conceptualisation.

Most of what we know about our culture, our traditions, the rules of our social group, and our own cognisance of our place in the order of things is learned. According to Bandura learning is not simply a matter of reacting to stimuli, rather individuals apply cognitive processes to the situations they encounter, selecting among the various presented stimuli and organising and transforming them to provide meaning. It seems plausible that those in lower SES groups have less (or less comprehensive) schemata available for comparisons, due to relatively deprived education, thus have a different socialisation pattern to those in higher SES Groups which seems likely to impact negatively on health outcomes. Lower socio-economic

individuals indulge in more health risk behaviours and it seems likely this is due to socially learned cognitions, e.g. A low SES individual, low in education level, cognitive processing, and self-esteem is much more likely than a high SES individual to consume tobacco, alcohol, illicit drugs, have poor nutrition and sanitary habits (Berkman & Breslow, 1983); not because the individual is stupid or careless but because of the social processes of his subculture (low SES). The point here is that SE groups are likely to have somewhat different socialisation patterns, and the pattern for low SES individuals places them at greater risk of negative health outcomes.

In a somewhat evolutionary development a more recent approach builds on social learning theory, especially applied to health outcomes. The Health Belief Model (Becker, 1974; Rosenstock, 1974; cited in Bennett et al., 1990) suggests that several factors will influence the likelihood of behavioural change: the individual's perception of vulnerability to negative health outcomes, perceptions of severity of outcomes, crude cost/benefit analyses in terms of risk reduction (as well as likely efficacy of behavioural change) and the presence of environmental cues to action. In all these components of the HBM young will take greater risks than older individuals. This may be a confound for the finding of greater inequality in younger than the older individuals. It is also suggested that individuals of lesser social status will be more resistant to change than higher SES individuals. While the first two components of the HBM are unlikely to differ greatly between SE groups, inevitably cost-benefit analyses will differ greatly, as will cues to action. Changes in behaviours such as alcohol and tobacco consumption, nutrition, and risk taking behaviours may lead to social isolation especially for lower SE individuals. In turn isolation (because much of the lower SE subculture do indulge regularly in high risk behaviour) will inevitably lead to emotion-focused coping (due to fewer cognitive resources because of inferior education), which may lead back to the old health damaging behaviours. Essentially a negative feedback loop is operating here. If an individual deviates from the norm for the SE group to which he belongs, factors such as social isolation will pressure that individual to change back or that individual may actually change groups.

The reason for the brief outlines and applications of social learning theory and the health belief model to the SE health inequality debate was to emphasise the importance of social structure in the explanation of individual behaviour. Individuals do not exist in a vacuum,

the study of human cognition and behaviour is essentially flawed if social context is not taken into consideration. As an analogy, the study of psychoneuroimmunology needs to take place more *invivo* rather than *invitro*. While *invitro* research is less expensive, faster, and easier to control, it doesn't take into account the "real world" environment of the subcomponents, that is human organisation. Therefore deductions based on such research are inherently flawed. The importance of social structure bring us to the next section in my examination of psychological factors in SE health inequality, namely external (mainly indirect) environmental influences.

#### *4.2.4 Summary of Internal factors*

This section on internal psychological factors has covered a great deal of material in a very small space. Considerably more could have been added for virtually every factor considered, however, space and time constraints prevent such an undertaking. The general mechanisms considered included the personality induced hyperreactivity model, the constitutional predisposition model, and the dangerous behaviours models. These models are all deduced from Suls & Ruttenhouse (1991), and serve to illustrate theoretical explanations for how various personality factors could influence health outcomes. The implication here is that personality factors, especially in terms of the dangerous behaviours model, may vary systematically according to SES.

More specific psychological factors such as depression, neuroticism, optimism, and personality constructs were also proposed as potential mediators in SE health inequality. Various cognitive processes were considered in the mediation process, especially perception in terms of explanatory styles. Cognitive processes invariably have a part to play in the SES-health relationship, especially in terms of explaining how relative poverty seems to disproportionately impact on negative health outcomes. As for personality factors, some of these specific mechanisms and cognitive processes may vary according to SES.

### 4.3 External factors

#### 4.3.1 *Concepts of Stress*

According to Newbury, Jaikens-Madden & Gerstenberger (1991) defining stress can occur in at least three ways. Firstly, stress can be thought of as an organism's response to its environment. Clearly environment circumstances can influence perceptions of stress in an individual. For instance, an unemployed individual obtaining manual work is likely to perceive his or her new situation as stressful in more ways than is obvious. Potential sources of stress may be the change in lifestyle (from unemployment - work), personality differences in the workplace, and the desire to make an impression, that is, to look for the employment to be long term. Secondly, stress can be defined in terms of a stimulus definition, that is (objectively speaking) the situation may legitimately elicit a stress-response. Going back to the above example, if the job involved moving radioactive waste, rather than simply labouring, then the situation itself may warrant a stress response due to risks such as leakage and contamination. Caution must be taken in this component of the definition because it is the individual's response that is important in the stress - negative outcome relationship. The point is that what is clearly stressful for one individual may not be for another, regardless of whether the source of the stress response is "legitimate" or not. Finally stress can be defined in terms of a set of interactions and relations, that is, a relational definition. Clearly to state that "stress" resides in the individual or the environment exclusively is not only reductionist but naive. The stress-response involves a complex set of interactions which combine to challenge the individuals coping resources. The stress-response becomes pathological when the individual's resources for coping are perceived (by the individual) to be overcome by the "stressful" circumstances.

The third approach is probably the best way of conceptualising how stress impacts on health outcomes in the relationship under examination. However this conception can be seen as oversimplistic for several reasons. Stress does not always lead to negative health outcomes and may lead to positive outcomes eg. an individual being harassed by friends to see a physician over some physical complaint may feel he is being "hassled" by those friends and may feel "stressed". Visiting the doctor may relieve the "stress" as well as help the individual

overcome the health problem for which he sought help in the first place. Many components within this conception vary within and between individuals (coping resources, perception of environmental stress, pre-existing perceptions prior to new challenges etc.), but it is my belief that those in lower SE groups will be relatively disadvantaged in most areas of this conception.

#### *4.3.2 Variations in the impact of stress*

There are several other issues that should be considered with regard to how stress impacts on health. The severity of the stressor must be assessed to understand how much detriment it will impact on the individual. Severity will often be couched in "objective" terms, however the reality is that stress severity is purely arbitrary. Witnessing a death is objectively very stressful, although if you are a soldier, police officer, or doctor the stress may be relatively less than for the "normal" population. When placed in subjective terms however, the severity of stress becomes less arbitrary. The death of a loved one will inevitably elicit various stress-responses, as will divorce, being jailed, or getting married. In relation to the SES health inequality debate, financial stress will be considerably more severe on low SES individuals than high SES people. I would argue that perceptions of relative financial deprivation via psychophysiological pathways have a large impact on health outcomes of low SES individuals. Similar arguments could be made about marriage, death, and divorce.

Predictability and controllability are two factors thought to mediate the effect of stress severity on pathogenesis. In terms of predictability the premature death of a loved one is thought to have more serious outcomes than death at the expected time of life, e.g. the death of a sister in a car accident aged 30 years is likely to be more stressful than death of a sister aged 82 years. Controllability has particular applications to the SES-healthy relationship, especially in terms of work environment. Research has demonstrated low control of environment can lead to negative health outcomes (Friedman, 1991). Many individuals in lower SE groups who are not unemployed are involved in low paid, sometimes dangerous, manual work. This kind of work typically avails little or no control yet high demands on individual performance. This is but one of many potential links between material deprivation (low SES) and negative health outcomes.

Another important facet of the stress - health outcomes relationship is that of duration of stress - response. While it is not always the case, severe stressors tend to be short in duration, whereas more minor stressors tend to be chronic. Minor stressor, individually, may not be chronic, but the accumulation of minor stressors sequentially may be perceived as ongoing chronic hassles. A severe stressor of chronic duration is believed to be particularly pathogenic (Newbury et al., 1991), although even minor level stressors when accumulated over time can be seen as problematic in terms of health outcomes.

### *4.3.3 Stress and SES*

These images of "stress" and its subcomponents and some of its moderators promulgate a somewhat dim view of prospects for those in lower socioeconomic groups. Those in such groups are likely to be near the problem end of the variables mentioned and are therefore at much greater risk for negative health outcomes as a consequence. Severity of stressors is likely to be greater in lower SE groups, predictability less, controllability less and duration will tend to be chronic rather than acute. The pathogenic extent of these factors is unknown, however a pure linear relationship is unlikely because of individual differences in disposition and environmental circumstances. The consequence of interaction between factors may in some cases be linear (that is, additive), but many also be synergistic (multiplicative) or some combination thereof (Marmot, 1986).

One of the main sources of stress for lower SE individuals is financial or material (Catalano, 1991; George, 1992). Individuals in low SE groups often encounter difficulty supplying the basic necessities in life such as adequate diet, clothing, and fuel (in winter) along with adequate housing, transportation and access to health services. They tend to have difficulty in obtaining regular employment and have a relative lack of mobility in that they lack for the resources to pack up and move on if the area they reside in becomes too depressed economically to sustain enough employment for the local population (Colledge, 1982). In most developed nations the notion of poverty amongst more affluent groups (i.e. higher SE groups) is that of the absolute poverty endured in many developing nations (Zucker & Weiner, 1993). However in developed nations poverty tends to be relative, which is important when considering possible causes for SE differentials in health outcomes.

Perceptions of poverty are important in explaining outcomes. Perceptions of poverty are important in explaining how health is disproportionately shared amongst members of a population, and indeed this is another instance where psychology fits into the explanatory process.

To this point, in this section on psychological influences on the SES-health relationship, I have examined several intriguing potential possibilities for factors impacting on differential health outcomes. I have considered internal constructs such as depression, negative affect, neuroticism and behaviour patterns such as Type A and Type C. These constructs may act directly on health outcomes in that they themselves have a differential effect on health outcomes. In contrast constructs such as explanatory style, coping strategies, social learning theory and the health belief model all act indirectly on health outcomes in that they influence health behaviours which in turn impact on health outcomes. Social learning theory and the health belief model are theoretical frameworks which serve to illustrate how social factors beyond individual control influence health outcomes.

Attempts were made to conceptualise the complex construct of stress. It is fair to say SE differentials in stress are probably largely responsible in explaining SE health outcome variance. However, actually defining and operationalising stress is problematic. Several different possibilities were considered, such as stress being an organismic response to the environment, stress being a definition of the actual stress stimulus, and stress being a set of interactions between relations. Other factors in the stress-response relationship were also considered including the severity and duration of the stressor, and control of the stressful environment by the individual.

The three pathways proposed by Suls & Rittenhouse (1991) offer a reasonable framework for the stress-response relationship. The Dangerous behaviours model is the classic conceptualisation of indirect psychological influences, while the constitutional disposition model encompasses the direct effects approach succinctly. The personality induced hyperreactivity model can be applied to the stress literature in terms of linking stress to negative health outcomes.

#### 4.4 Psychological and physiological linkages

The next section is provided to delineate potential links between psychological constructs and physiological outcomes. This is aptly described as micro-level relations in that it outlines how social and psychological factors impact on human physiology to elicit various health outcomes and thus forms a crucial link between disciplinary approaches.

Psychological and social factors impact on physiological outcomes by means of two key pathways, both of which can be subdivided for more comprehensive analysis. The human body can be subdivided into several interrelated systems which interact to produce homeostasis. Homeostasis is a medical term used to denote normalcy. Homeostatic parameters are, in essence, a confidence interval outside which homeostasis is said to have been violated. In reality, homeostatic parameters are somewhat arbitrary given the remarkable diversity in variation accounted for by individual differences. Despite the arbitrariness of parameters, population pressure produces statistical norms for homeostasis (beyond which health is said to be compromised), which allow clinicians to make judgements at the individual level based on the normal curve, which has been based on population pressure.

##### 4.4.1 *Neuroendocrine Pathways*

The first pathway by which psychosocial influences interact to influence various health outcomes is through the neuroendocrine system. The neuroendocrine system comprises part of the nervous system and part of the circulatory system. It involves the exchange or sharing of information between nervous system (NS) and endocrine subsystems. The nervous "system" involves primarily activity of the brain in terms of electrochemical neurotransmission, while the endocrine system involves chemical transmission of messenger substances (hormones) via the circulatory system. Despite the known specificity of various chemical substances in the human body, there is also great redundancy and sharing of some of these substances. Several substances are known to share roles, e.g. epinephrine (adrenalin) and norepinephrine are known as neurotransmitters and known hormones which have somewhat different effects depending upon the location and type of receptor they act on.

The neuroendocrine system involves interaction between the endocrine system and the brain. There are many subsystems operating, but the most influential of these systems can be reduced to two: namely, the pituitary-adrenocortical system and the sympathoadrenomedullary (SAM) system. The pituitary-adrenocortical system involves the interaction between the pituitary (which is influenced by, and influences the hypothalamus) and adrenal glands. It involves bodily responses caused by the activity of hormones, predominantly steroids released by the adrenal glands. These steroids include estrogens, progesterones, androgens, and mineralocorticoids but predominantly a group of naturally occurring steroids known as glucocorticoids. The two main glucocorticoids are cortisol (hydrocortisole) and corticosterone. These substances are known to alter the metabolic activity of various tissue types by interaction with specific receptors in that tissue. This specificity of action for hormones and neurotransmitters is essential in regulatory processes. An example is probably necessary to point out how this system seems to operate. An individual suddenly becomes unemployed due to no fault of that individual. Unemployment is known to have serious psychological and physical consequences (War, 1987; Whelan, 1992; Iversen & Andersen, 1987; Jackson & Warr, 1982), which at the individual level may be manifested in terms of increased and chronic nervous arousal. For arousal to be maintained (i.e. chronic) the hypothalamus (well known for its regulatory activities) influences the pituitary which in turn influences the adrenal glands to secrete various substances including glucocorticoids to maintain the increased metabolic rate that may have been initiated by the psychological anguish created by the loss of employment. Chronic over-activation of the nervous system involves the subsequent over-release of various substances, chief amongst them being glucocorticoids (especially cortisol) which is known to have deleterious effects on the body in terms of tissue damage by over-activation. When the amount of tissue damaged exceeds the body's healing capacity overall health begins to deteriorate. The application of the personality induced hyperreactivity model seems apparent here. While this is over simplistic and reductionist, it serves to illustrate the mechanisms by which pituitary-adrenocortical activity serves to impair health outcomes at the individual level.

The sympathoadrenomedullary (SAM) system involves the activity of the sympathetic nervous system which is one component of the conveniently dichotomised central nervous system, with the other component being the parasympathetic nervous system. The parasympathetic

nervous system can be further subdivided into somatic (under voluntary control) and autonomic (involuntary) subcomponents.

The SAM system involves interaction between the brain (via the hypothalamopituitary axis) and the adrenal glands, or more specifically the adrenal medulla. The adrenal medulla is known to secrete the cholinergic chemical acetylcholine. Acetylcholine which is also a neurotransmitter, is known to be active at many receptor sites throughout the body, but is particularly active at muscle junctions near or around locomotive joints. This is not surprising given that the primary role of the SNS is for urgent action, which is invariably going to involve major limb movement such as running, fleeing or engaging in combat (hence the notion commonly involved with SNS activity - "fight or flight"). When the SAM system is chronically overactive physical health problems can ensue, due invariably to problems of habitual response and changes at the cellular level of the site of action. SAM dysfunction is somewhat less pronounced than pituitary-adrenocortical dysfunction and tends to act in terms of overall changes induced rather than specific problems which can be incurred by the latter (Newbury et al, 1991).

#### *4.4.2 Immunological Pathways*

The second major pathway by which psychosocial factors interact with physical dimensions in humans is by means of reducing immune system efficiency, thus indirectly increasing likelihood of tumours and infections. As for my brief coverage of neuroendocrine pathways this description is necessarily brief and somewhat reductionist, but serves the purpose of illustrating how psychosocial factors can influence physical health outcomes.

Essentially the immune system can be seen as a surveillance-defence system that identifies foreign intruders at the cellular level and mobilises the body's defence system to eliminate the intruders. The immune system is subdivided into cellular responses which involve lymphocytes that act directly on invading pathogens, and humeral response, which involves lymphocytes which produce antibodies that circulate systemically (Calabrese, Kling & Gold, 1987; Matarazzo et al., 1984). It is clear that stress in some way reduces immunocompetence, although the mechanisms by which this occurs are still unclear (Plotnikoff et al., 1991).

There is evidence that psychosocial variables have important effects on human immunocompetence. (Ader, Felton & Cohen, 1991).

#### *4.4.3 Conclusion*

Culling out how psychosocial factors impact on immune function involves analysis of how thought patterns (cognitive) influence physiological changes in the brain, particularly the hypothalamus, which in turn impacts on cellular activity in the immune system. There is ample evidence for SE deprivation having adverse psychological impact.

This impact can be reflected in adverse neuroendocrine response and immunosuppression. There is also little doubt that neuroendocrine and immune function are interrelated (as are all systems and subsystems in the human organism), thus impairment in either is likely to impact on the other. For the relatively deprived in modern developed nations there are problems that many better off, cannot conceive, such as real financial stress, low quality (often damp) housing conditions, poor education, lack of adequate nutrition, transport, and health service access. These structural factors impact on the individual psyche (via proximal structures such as work and home environment) which in turn impact on brain processes and subsequent neuroendocrine and immune activity. These factors serve to illustrate the multicausality and multilevel nature of the problem of SE deprivation, and indeed underlie the rationale for my call for the need for multilevel theoretical approaches to conceptualise and address the problem.

Evidence has been provided to establish the mediating role of psychological factors in the SES-health relationship. It appears that psychosocial factors have both direct and indirect influences on health. Additionally, it appears that SES impacts on psychological and sociocultural aspects of individuals.

Direct mediation is evidenced by, for example, immunosuppression or excessive neuroendocrine response to a stressor, as a function of psychological processes. Cognitive processes, such as health beliefs, provide evidence for the indirect influence of psychological

factors. The action of direct and indirect psychological influences tends to result in differential health outcomes which seems to vary systematically to SES as well.

The brief section on physiological pathways for the direct influence of psychological factors on health outcomes was provided for two main reasons. Firstly, it demonstrated how psychological factors could have a direct influence on physiological outcomes. That is, it showed that some physiological factors varied according to psychological precursors. Secondly, it provided the second part of the construct I have denoted intraindividual factors. This "construct" is the bottom rung of a collection of hierarchically organised factors which form the basis for a multilevel model approach to examining complex phenomena. Multilevel modelling is described in more detail in the next chapter, but alludes to the assessment of phenomena across "levels" of approach. In the health related example of multilevel modelling, the health transition model, phenomena can be assessed at the (from top to bottom) systemic, societal, institutional/household, and individual level of analysis. Accordingly, explanation for various phenomena may differ as a function of level of analysis.

In summary, psychological factors form the vital link between environmental influences and health outcomes. The influence of the family, which has been largely overlooked in this analysis, will probably interact with psychological factors in health outcomes because of the emotional importance of the family, and the amount of contact during the lifespan (especially the formative years) members of families tend to have with one another. The family forms part of a complex interplay of factors in the SES-health relationship which require a more comprehensive theoretical framework for interpretation. The next chapter will endeavour to achieve the more comprehensive approach alluded to here.

## 5.0 THE NEED FOR A MORE COMPREHENSIVE APPROACH

### **5.1 Introduction**

The purpose of this final chapter is to draw together the threads of evidence, the exploratory approaches, the causal pathways, and the psychological factors together in a meaningful way to better understand, and hopefully, explain the SES-health link.

In chapter 1 (Establishing the relationship) the SES-health relationship was briefly described and copious, divergent evidence from around the world was produced to establish health inequality as a function of SES. The volume of evidence produced served to illustrate the consistency of the relationship across differing countries and scenarios.

Chapter 2 (Pathways for SES to impact on health outcomes) proposed several potential pathways by which SES could impact on health outcomes. SES and health were further defined in this chapter and measures for each were outlined. A detailed analysis of poverty was undertaken in this chapter, and a potential application of relative poverty (unemployment) was carried out to demonstrate potential material and non-material pathways for SES to impact on health outcomes.

Chapter 3 (Theoretical approaches to the SES-health link) examined the key theoretical explanations for health inequality known to exist as a consequence of SES. Explanations from the Black Report (DHSS, 1980) were assessed (Artefact, social selection, cultural/behavioural, and social causation approaches), along with a more recent conception advocated by Feinstein (1993).

Chapter 4 (Psychology and the SES-health relationship) considered the role of psychology in the SES-health relationship. In this chapter both internal and external psychological factors were considered. Internal factors were assessed in terms of general mechanisms, more specific mechanisms, and cognitive processes. This chapter also examined micro-level physiological links to psychological processes in terms of potential neuroendocrine and immunological pathways.

The purpose of this chapter will be to discuss the state of current knowledge on the SES-health relationship, then exceptions to general principles will be considered. Knowledge and exceptions will then be integrated by analysing proximal pathways such as occupation, home environment, access to medical care, and psychosocial resources of low SES individuals. Pathways and key concepts in the SES-health link will then be integrated into a holistic multilevel theoretical framework. The health transition model will be used as an example of a multilevel approach. Following this further research in key areas will be advocated, and all preceding information synthesised into the final conclusions.

## **5.2 The Nature of the Relationship**

There is much evidence (See chapter 1) that indicates SES impacts negatively on health outcomes. This section will review information on the current state of the knowledge on this topic, as well as considering exceptions to the general findings linking SES to health. The final part of this section will attempt to integrate current knowledge and exceptions by examining potential mediating pathways.

### **5.2.1 Current Knowledge**

The state of current knowledge on the SES-health relationship can be reduced to several main conclusions. Firstly, ample evidence suggests that SES has a positive relationship with health outcomes. As SES increases, health improves. That is, as SES increases, concomittant negative health outcomes tend to decrease. This positive relationship has been observed repeatedly in many differing contexts, which reinforces the pervasiveness of the relationship.

The second conclusion that can be reached from the research literature is that the SES-health relationship is very robust, irrespective of how variables of either SES or health are assessed. It is found when SES is assessed by income, education, occupation, geographic location, housing conditions, or transport status, and when health is assessed by mortality, morbidity (both self-report and "objective"), or mobility (eg. days off work), that health outcomes vary

as a function of SES. The positive relationship between SES and health is a robust phenomenon, and very few exceptions violate this general rule.

Thirdly, gradients are substantial and they tend to be too large to be explained at the individual level. Essentially, what this means is that the size of health outcome differentials at the population level (ie. country or group being assessed, such as city or suburb) as a function of SES are too large to be explained by individual differences or factors under individual control, such as health behaviours. While the impact of individual level factors, such as health behaviours including risk taking, sociocultural affiliation, and consumption of harmful substances, may explain some health outcome variance, the contribution of such factors falls well short of explaining observed health outcome differences between SE groups. Gradients are substantial in terms of the disproportionate aggregation of negative health outcomes in lower SE groups. The degree of health inequality in a scenario can be deduced from the slope of the health gradient, with a steeper gradient being indicative of greater inequality.

Fourthly, health inequality gradients illustrate the next conclusion that can be extracted from the literature, namely the incrementality and linearity of the relationship. SE differences in health outcomes generally follow a hierarchical graded pattern, with low SE groups having the worst outcomes, the next SE group up having outcomes better than outcomes in the lowest group, but inferior to the next SE group up the SE hierarchy. This process continues up the SE hierarchy to the highest SE group who have the best health outcomes in a given society.

The fifth conclusion drawn from the literature has to do with temporal factors and explanatory approaches to the SES-health relationship. Currently, selection approaches are not sufficient to explain the magnitude of effect observed in many studies. Selection approaches suggest poor health tends to select poor SES outcomes. While this perspective may contribute to observed health outcomes, researchers have demonstrated that such a contribution is insignificant (eg. Wilkinson, 1986a, 1987; Goldman, 1994). The work of Feinstein (1993) suggests temporal factors have been largely overlooked. Probably the greatest contribution Feinstein made to SES-health research was his observation that SE health problems tend to

occur mainly in two areas of the developmental lifespan: the very young (newborn and infant), and the middle years (approximately 35-65 years).

When Feinsteins temporal contribution is combined with information drawn from the human geography literature, that being the phenomenon of the ageing population structure in many developed nations, the potential for selection processes to be more influential in explaining health inequality becomes more apparent.

The existence of an ageing population structure means that selection processes may have greater influence on health outcomes at some future date because increasing numbers of individuals are reaching the high risk stage of development (advocated by Feinstein and others) where SES tends to impact more on health outcomes than at other developmental stages. SE factors impact on health, especially in terms of morbidity, in middle-aged and very young individuals.

With increasingly large sections of the population in the middle age bracket, this country, along with many other developed nations, must come to terms with increasing proportions of negative health outcomes for the population as a whole. Negative outcomes can be expected because SES impacts on health in middle aged individuals (which are growing), and because negative health outcomes may "select" downward SE mobility and concomittent increases in negative health outcomes which are associated with low SES.

Sixth, of the theoretical explanations proposed to date, social causation appears to be the most powerful approach in explanatory terms, because it accounts for more variance in health outcomes than do other approaches from the Black Report. All the explanations have limitations (many of which were discussed in chapter 3), and causation explanations are no exception. While this approach does explain substantial health outcome variance, the actual means by which outcomes differ by this approach are shortcoming. Social causation argues that social structures within a society impact on health by shaping the environment for the individual. The impact of income distribution makes more sense when considered within the framework of a social causation approach because income distribution is so directly affected

by large structures in a society. That is, government action in terms of education, health, and social spending (not to mention taxation and other monetary controls), along with private investment in the economy has a powerful influence on health outcomes which is beyond the direct link between individual accountability, ability to pay for health, and health outcomes.

Seventh, repeated studies have indicated that income per capita is not a great predictor of health outcomes at the national level (Marmot & McDowall, 1986; Robine & Ritchie, 1991). The same can be said for many commonly accepted indicators of a nation's wealth such as average income, median income, or average weekly wage. Therefore, the simple assumption of "wealth makes health" is not well grounded (Kim & Moody, 1992). The degree of equality in income distribution appears to be more predictive of health outcomes. More importantly the degree of impoverishedness of the lowest SE groups compared to other SE groups is more predictive of population health outcomes. If the lowest SE groups are greatly disadvantaged then health problems are greater overall. Income distribution is mediated by factors such as the government's fiscal and monetary policy in terms of taxation and monetary controls, not to mention the degree to which the government subsidises health and education, and the level to which a government is prepared to supply social security in terms of benefits and pensions.

The final conclusion has to do with the causes of mortality. Mortality is one of the key health measure used in the literature to illustrate health inequality. Mortality differences, like other health indicators, are yet another strand of evidence which indicate health outcomes vary as a function of SES. The nature of illness factors has changed markedly over the course of this century in most developed nations. Up until the start of this century the major cause of negative health outcomes was infectious disease. Improved sanitation and hygiene, along with public health initiatives in developed countries has essentially eliminated the impact of infectious disease. This century has seen the rise of degenerative diseases such as CHD and cancer as holding dominance in the health variance of populations of developed countries at large. Many degenerative diseases have psychosocial components and tend to vary systematically with SES. While infectious illness may vary according to SES, such disease processes tend to be more problematic in developing countries where absolute poverty causes more extreme problems than those observed in developed nations where relative poverty and degenerative illness dominate health outcome variance. The point here is, that despite

increased overall health in developed nations over the last fifty years or so, and major changes in causes of mortality (ie. from infectious disease to degenerative disorders), SE health inequality has remained, and even worsened in some places.

In summation, the state of current knowledge on the SES-health relationship can be reduced to a series of conclusions. SES has a positive relationship with health outcomes which is consistent across a wide variety of situations and locations. This consistency can be construed as a contributor to an important factors in the SES-health relationship, such as the robustness of the relationship. Not only is the relationship positive and robust, it is also incremental (by social class) and linear (as SES increases, so to does health). The linearity of the relationship gives rise to the SE health gradient, with the steepness of the gradient being indicative of the degree of inequality (the steeper the gradient, the greater the inequality).

Average and per capita indicators of wealth are not good predictors of health outcomes. It seems that the extent of poverty in the lowest SE groups, relative to the other SE groups, is more predictive of the population's overall state of health. While social causation and cultural/behavioural approaches can explain some of the SE health variance, their contributions are far from conclusive. While social causation has emerged as the most complete theoretical explanation of SE health inequality, it is far from complete.

There has been a fundamental change in the causes of mortality in recent decades. CHD used to be known as a "disease of affluence" due to the individuals who were predominantly afflicted by it. In contrast, contemporary health patterns in developed nations indicate CHD is now a problem of lower SE groups, that is, a "disease of poverty". Despite these major changes in causes of mortality the phenomenon of SE health inequality continues unabated.

### *5.2.2 Exceptions*

Although there are several clear conclusions that can be accepted about the SES-health relationship, there are also some exceptions to these. At present these exceptions do not occur extensively enough to draw any clear conclusions about their causes.

Firstly, the predictability of the relationship tends to degenerate when cultural and ethnic groups which have value systems which are vastly different from the value system of the dominant culture within a country are assessed. This exception has been demonstrated in research in the UK with Caribbean and African immigrants, as well as in New Zealand with Maori and Pacific Island populations. It seems plausible that cultural values have a direct influence on the importance individuals place on, for instance, material gain. Indifference to material gain, not to mention other issues such as the use of modern medical services (and other health practices) are potential explanations for this anomaly. Further research is needed to assess the validity of these potential explanations.

Another possible explanation for cultural differences may be in terms of the observed differences in research with samples differing in homogeneity. The Whitehall studies undertaken by Marmot and his colleagues demonstrated that more homogenous samples have steeper and more linear health inequality gradients. It stands to reason that for more heterogeneous samples the opposite may occur. That is, with the heterogeneity created by cultural differences, gradients may be less linear and not as steep. The size of discrepancies may be a function of sample size and characteristics. Smaller samples with larger proportions of the cultural minority will probably display outcomes the most divergent from commonly observed outcomes in SES-health research.

Secondly, although the association between SES and health is usually strong within countries, the same can not be said about the relationship between countries. It is clear that the psychosocial and cultural make-up of countries will vary considerably, and this heterogeneity will influence any cross-country comparison. Another important factor which influences this exception is the type of data collected, and how such data is collected. Different countries have different approaches to both these issues, and therefore international comparisons must be viewed with some degree of caution. At the very least, conclusions from such research must be conservative.

Thirdly, there are some causes of mortality, and types of morbidity, which defy the usual trends for negative health outcomes which occur as a function of SES. Higher SE groups tend to have higher prevalence rates for certain types of morbidity such as myopia, obesity,

allergies, and melanoma (Gomby et al., 1989). Factors such as diet, exercise, and available leisure time (and use of this time e.g. sunbathing) have been postulated as possible explanations for this anomaly, although the true causes for such discrepancies should be the subject of future research.

In summation, there are not many exceptions to the conclusions proposed in the current knowledge section. Heterogeneity of the population appears to reduce both the magnitude and linearity of the SES-health relationship, which is succinctly illustrated by the differences observed in different ethnic and cultural groups. International comparisons are problematic for examining SE health inequality due to several factors such as differential criteria for data selection, collection of data, and subsequent analyses performed. As with virtually every rule there are exceptions. There are some types of morbidity and causes of mortality which are more problematic for higher than lower SE groups, but these are few.

### *5.2.3 Integration of Knowledge and Exceptions*

Possibly the biggest problem for the attempted explanations of SE health inequality is a lack of generality. Explanations from the Black Report seem to be rooted in one level of analysis, and in doing so, are too reductionist to provide a comprehensive explanation of the phenomena. Levels of analysis are to do with the magnitude of social activity at which the relationship is being assessed. The SES-health relationship can be assessed at the population level (macro), various group levels (eg. country, city, suburb), and at the individual level. Population level explanations (eg. social causation) tend to explain more health outcome variance but lack the causal specifics, whereas individual level approaches (eg. cultural/behavioural approaches) tend to provide better potential causal explanations for inequality but lack accountability for health outcome variance. There are several mediating factors that are involved in the means by which social causation impacts on health outcomes.

Without doubt occupation and living environment play a crucial role in the health outcomes of individuals. Work environments in lower SE groups tend to be more dangerous, more polluted, and offer lower levels of control at the individual level. Work strain, comprising a lack of control combined with high performance demands is known to be a powerful stressor

(Landy, 1989). It is likely that those in lower SE groups will be at higher risk of encountering work strain, not to mention the financial strain inherent in lower SES. It is also likely that environmental stress levels (in terms of work strain, financial strain, environmental toxins, and general psychological "hassles") will be comparatively higher in lower SE groups, which overlooks the strong likelihood that many of those in lower SE groups will be unemployed. In such cases occupational environment as a stressor would be invalidated, but financial strain must increase due to relative financial deprivation (administrative poverty) inherent in unemployment.

Living environment also will have a major impact on health. This does not simply allude to the domicile of the individual, but the area of the domicile. Low SES areas tend to be more run down, polluted, and relatively lacking in essential services, such as accessible transport and medical care, compared to higher SE groups. Actual domicile standards are also important in mediating health outcomes. Certain housing conditions, such as damp, cold, and noisiness, are known to be predominantly a problem for those in lower SES groups, and are also known to relate to negative health outcomes.

Another way in which SES may impact on differential health outcomes is through creating differential access to health care (Haynes, 1991). Not only do individuals of higher SES have more access, but they have access to a better quality of health care (Balarajan et al, 1987). This issue is less problematic in countries with good public medical facilities and free health care, such as Sweden, but is much more influential where health care has been privatised and medical facilities must "compete" for "customers". In countries where health is privatised or semi-privatised, there is a tendency for great fluctuation between the quality of medical facilities available to the public, and private ("user pays") health care users. Those from low SES groups use public health facilities because private facilities, which tend to be of a higher quality and, more often than not, are used by high SES individuals, are beyond financial viability for low SES individuals. Private "customers" are better able to obtain higher quality health care due to the superior ability to pay rather than the need for medical care. The same higher SES individuals obtain better medical treatment and thus tend to be ill for shorter periods. Cumulatively, lower SES individuals can be substantially disadvantaged in terms of access to health care: they get ill more often; obtain inferior prevention knowledge; once they

obtain treatment it is often of inferior quality; and once ill they tend to stay so for longer periods. Low SES individuals tend to live shorter lives then, and during their shorter lifetimes they endure more morbidity, at least partly as a function of their inability to fund health maintenance.

Individual personality characteristics seem to interact with SES to influence health outcomes. While I do not imply SES causally influences personality outcomes, it is likely that many individuals of lower SES have various common personality characteristics. Individuals of low SES tend to have lower self-esteem and lower self-efficacy compared to their higher SES peers (Adler & Matthews, 1994). These characteristics when carried to their extremes are manifested in psychological pathology, often in terms of depression, neuroticism, general negative affect, heightened anxiety and underachievement in both academic and occupational endeavour. Underachievement directly impacts on SES (in terms of educational and occupational outcomes), while neuroticism, negative affect, and anxiety all are known to have negative psychophysiological consequences. All the theoretical approaches from the Black Report appear to view individuals as passive recipients for the causal influences of SE health inequality. Evidence from research examining the potential for personality characteristics influencing health outcomes tends to counteract the idea of the passive recipient.

My examination of current knowledge and exceptions was presented as a series of conclusions drawn from the literature. These conclusions were common themes drawn by experts in the fields of SES, health, and various related subject areas. The problem, of course, is to draw these conclusions and themes together in some meaningful way. It stands to reason that any potential theoretical formulation will not only integrate accepted "facts" in the SES-health relationship, but also exceptions.

While it is generally accepted that social causation is the best theoretical explanation to date, I have also shown how this approach is not comprehensive enough. Combining social causation and cultural/behavioural approaches increases the explanatory value for the SES-health relationship because it assesses it over more than one level of analysis. This dual level approach is closer to the multiple level approach which I advocate is necessary for the understanding of SE health inequality.

Individual differences, in terms of psychological characteristics, amongst members of the general population, is a key factor in health outcome variance which has generally been overlooked by researchers in the field up until now. Psychological factors serve to mediate the SE circumstances which act on health outcomes. The impact of psychological mediators can be both direct (such as hyperreactivity as a function of stressful circumstances) and indirect (such as psychological factors influencing health behaviours).

The purpose of integrating knowledge and exceptions was not simply to regurgitate the contents of the preceding sections, but rather to orient the reader to the conclusion that a more comprehensive approach is necessary to explain SE health inequality. If a single level of approach was going to be a panacea for our understanding of health inequality then the individual level, common sense would dictate, would be the most promising. One would expect individual level success because individual input and outputs are easier to control and extract potential causal relationships than higher level efforts such as small group or community or even societal. However, repeated efforts at individual level explanation and intervention (in terms of public health initiatives) have been of limited success. Clearly factors beyond individual control are involved in health outcomes, which is not to say better health behaviours would not be beneficial for overall health outcomes.

### ***5.3 Integrating the Links into a Theoretical Framework***

Explanations for SE health inequality, as already alluded to, tend to lack external validity. That is, the explanations to date lack the ability to generalise. This is because current theoretical explanations only attempt elucidation of potential causal mechanisms at one level of analysis, and for this reason they can not reasonably explain the phenomenon beyond the level at which it is assessed. Explanations at the individual level of analysis do not account for any significant amount of variance in health outcomes, while explanations at group levels of analysis tend to explain more variance but can not specify the means by which variance is accounted for. Thus, individual level approaches are more specific about possible causes of outcomes but lack overall explanatory power, whereas group level approaches explain more variance but lack specifics about potential causal mechanisms.

The social causation explanation receives the most support in the literature, and is, the best of the explanatory approaches proposed by the working group in the Black Report. Despite being the best approach, greater specification of mediating factors must be included to improve explanatory power. I propose that social causation operates through a series of proximal (in terms of level of analysis) mediating factors that allow structural factors within a society to impact on individuals. Structural factors and health outcomes are very distant from one another, which explains why it has been so difficult to link the two in the past. The impact that structural factors have on individuals is operationalised through a chain of events which occur at proximal levels to allow a "flow of effect" from SES to health outcomes.

One way by which comprehensiveness of explanation can be vastly improved, is by combining social causation and cultural/behavioural approaches. This would permit specific individual level explanations to be linked to the structural factors that help shape the environment for cultural/behavioural factors to influence individual outcomes. However, even the combination of these two approaches leaves gaps in our knowledge of the relationship, which serves to illustrate the thrust of this thesis. Neither social causation or cultural/behavioural approaches specify the mediating factors between structural and individual factors, or between individual and intra-individual psychophysiological factors which serve as pathways for SE factors to impact on health outcomes.

A more holistic approach is necessary to overcome the limitations of present approaches to explain SE health inequality. Such an approach must take into account all levels of analysis to ensure a more comprehensive and plausible explanation of observed SE differences in health outcomes. The reason for this is simple. The type of phenomena which tends to defy analytic approaches is usually very complex, tends to be composed of many interrelated factors, and tends to involve scenarios which can not be operationalised in an experimental manner. The complexity of the relationship under examination falls into this category.

An approach which fits the holistic requirements needed to better explain SE health inequality, and which has received support in recent commentary (eg. Spicer & Chamberlain, 1994), is known as multilevel modelling. Such an approach suggests that for complex social phenomena, such as the current topic of discussion, assessment at all levels of analysis, not

of analysis, not to mention making the connections between levels, is essential to comprehensive understanding and explanation. In this conception no one level is more important than another in explanatory terms, each is equally important in contributing to the complex chain of tangible and intangible events that ultimately impact on health outcomes.

Rather than continuing this discussion of holism and multilevel modelling in abstract theoretical terms, I will illustrate this type of approach with an example of multilevel modelling that is applied to health outcomes. While this model does not necessarily explain SE health inequality, it does provide a workable theoretical framework which, serves to illustrate how complex phenomena may be explained. It also provides a sound theoretical base for future research.

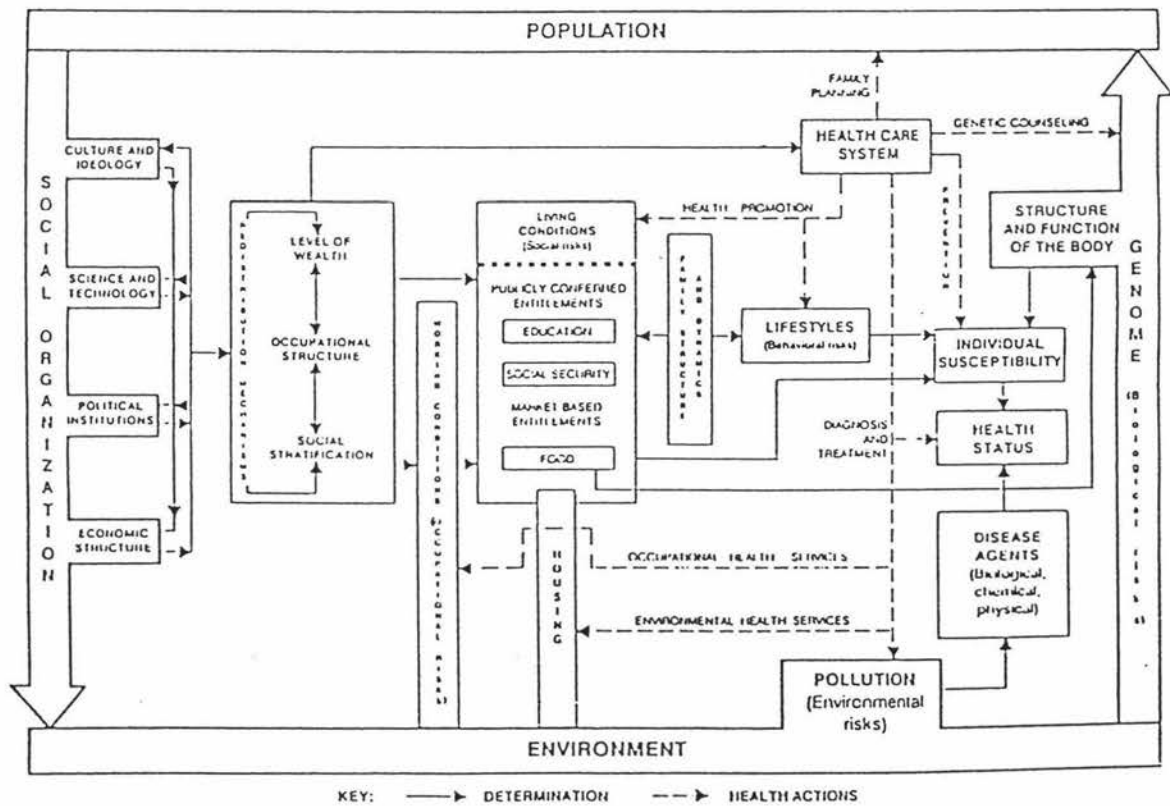


Figure 1: Determinants of health: an illustration of the complexity of the SES-health relationship (from Frenk, Bobadilla, Stern, Frejka & Lozano, 1991).

The theoretical framework which will serve as an example of a multilevel approach is known as the health transition model (Frenk et al., 1991). This model can be described in at least two ways. The first is in terms of level of analysis, while the second is a more comprehensive approach looking at many proximal mediating factors and how these interact to result in differential health outcomes. The first approach is more conceptual, whereas the second is more generic and considers many factors, how these factors interact, and conveniently provides an excellent framework for future empirical research to be based on because of the specificity of this conceptualisation.

The specifics of the second approach (See Fig 1) will be left to the original authors (Frenk et al., 1991), that is, they will not be considered further here. The figure from the original work is reproduced to provide the reader with an appreciation of the level of approach I wish to endorse as being most effective in the explanation of SE health inequality.

The first approach serves to meld together much of the information provided to date. This approach considers the determinants of health to exist at four interrelated and hierarchical levels of analysis. In descending order these are systemic, societal, institutional/household, and individual levels. No one level is more important, or carries more explanatory weight, than another. The hierarchical nature indicates the size of the social environment from which the analysis is being made, that is, level of analysis and potential effect.

The systemic level involves dimensions such as population genomes (that is, genetic factors which tend to fluctuate at the population level eg., the tendency for many Asian individuals to be lactose intolerant), and environmental social organisation which includes concepts such as culture and ideology, economic structure, and political institutions. Societal level factors are defined as structural determinants and include factors such as social stratification, level of wealth, occupational structure, and redistribution mechanisms such as welfare and tax systems. The institutional/household level of analysis is subdivided into various determinants such as working and living conditions, lifestyle and use of the health care system. The individual level response in this conception is simply the individual's behaviour.

Despite its comprehensiveness, it is my belief that this model lacks a crucial level which is involved in the SES-health relationship. My own conception would include another level, that being intra-individual, which would describe psychological and physiological factors in the determination of health outcomes, and their interrelationships. It is the interaction of these factors which finally determines the health outcome of the individual, although such outcomes are the consequence of all the levels of influence. Health determination will be shared differentially across the levels of analysis according to the individual, and the psychophysiological (intra-individual) level is simply the means by which health outcomes finally impact on that individual.

Factors which will influence that individual's health outcomes will impact across all the levels. At the systemic level government policy, common genetic characteristics of the ethnic group to which the individual belongs, and cultural values will influence outcomes via mediation of several factors. At the societal level factors such as the degree of class division within the society of the individual will influence outcomes, not to mention distribution of wealth (which will be related to the class division issue) and redistribution mechanisms such as taxation and welfare payments (which will be reflections of government policy at the systemic level of analysis). The institutional/household level will include factors which impact on health outcomes such as working and living conditions, family structure, and interaction with institutions such as health and education systems. At the individual level psychophysiological factors will mediate the above external influences on health outcomes.

Psychological factors (and related psychophysiological outcomes) are one of the crucial under-examined links between SE factors and health outcomes. The importance of the contribution of psychological factors is illustrated by the diversity of health outcomes accounted for by individual differences. Despite many similarities in the general population in terms of environmental and dispositional factors, individual differences in psychological profiles results in great diversity in health outcomes.

Despite the noted individual differences in health outcomes that occur, there is growing agreement that an overarching causal relationship is operating, although the specifics of the relationship continue to defy elucidation. This lack of causal specification between SE factors

and health outcomes has been brought about by a theoretical framework that lacks the comprehensiveness necessary to encapsulate the enormous complexity of the SES-health relationship.

The framework I have advocated is more holistic than any proposed to explain SE health inequality so far. It builds on Feinstein's (1993) effort by including structural and intra-individual factors which serve to improve comprehensiveness in explanatory terms by increasing our assessment of the SES-health relationship by two or more levels of analysis. This more holistic emphasis has the advantage over less comprehensive approaches of integrating both general conclusions and exceptions in the SES-health relationship. One would expect that this more complete theoretical approach will draw together previously unrelated factors and provide improved understanding of how SE factors impact on health outcomes. I have no doubt that the utilisation of more holistic approaches, such as the multilevel approach advocated, would result in more fruitful research on the SES-health relationship, and more importantly, elucidate the multicausal nature of the means by which SES impacts on the health of the nation.

#### **5.4 Future research and conclusions**

Future research should seek to establish empirically the links between various components within a multilevel framework. In recent years research being undertaken to establish the link between SES and health outcomes has been driven by the theoretical framework provided in the Black Report. Although this research has been empirical, the theoretical framework on which it is based has been deficient. Research should be theory driven, and studying the SES-health link is no exception. Various components of the multilevel approach will need to be examined from differing disciplinary perspectives, therefore research from within a multilevel approach is inherently multidisciplinary.

Various exceptions and anomalies in the SES-health relationship need urgent attention to ensure such phenomena are not simply unusual cases rather than some factor which lacks explanation by means of the current method of scientific investigation and explanation.

Research needs to be undertaken to answer why African and Caribbean immigrants in the UK have health outcomes which violate the normally predictable relationship between SES and health. The fact that those in higher SE groups tend to suffer more from a few types of morbidity and cause of mortality (eg. acne and melanoma) also needs further investigation. The impact of government policy on health outcomes also needs investigation. It is unlikely that the impact of government policy could be examined in anything but a multilevel approach because proximal mediators would need identification and the links would require empirical verification.

I have established four basic tenets quite clearly. Firstly, the SES-health relationship is an extremely complex one which involves multidisciplinary dissemination to provide a true understanding of it's nature and prevalence in developed nations. Secondly, current theoretical explanations that account for the relationship are deficient. Consequently our understanding of causal factors has stagnated due to theoretical approaches which, because of the reductionism in each approach alone, have descriptive rather than explanatory power. Thirdly, the most effective means of overcoming the current theoretical deficiencies are to expand the theoretical approach into a more holistic orientation. Multilevel modelling is one means by which a more holistic approach could be achieved. Fourthly, future research should seek to explain discrepancies in the SES-health relationship, as well as empirically verifying the links within the multilevel framework, such as the multilevel modelling approach.

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