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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**
at **MASSEY UNIVERSITY.**

ALISTAIR MARK PINFOLD

1995

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.

ACKNOWLEDGEMENTS

The author wishes to thank the following individuals and organisations for their help in the compiling of this manuscript:

- * Cherie Gordon (External database search), Massey University Library

- * Department of Health

- * Department of Psychology, Massey University

- * Department of Statistics

- * Massey University Library staff

- * Victoria University Library staff

Special thanks go to my supervisor, Kerry Chamberlain, and my mentor, Dr John Podd, for their help, encouragement, and patience.

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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.