Introduction to clinical health psychology

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Why a book on clinical health psychology?

The term ‘clinical health psychology’ stems from two strands of psychology, each with a different history and focus. Clinical psychology has moved from its historical roots of the 1950s, when practitioners worked almost exclusively in psychiatric settings, to the provision of therapy in a wide variety of settings and with a range of patients, including those who are physically ill. Underlying this move has been a more subtle shift in therapeutic approach. The focus of clinical interventions is no longer solely the remediation of mental health problems. Many clinical psychologists work in settings such as pain clinics or on cardiac rehabilitation programmes, where the majority of patients cannot be considered ‘mentally ill’. The rationale for this shift is that the behavioural changes required by participants in such programmes can be facilitated by practitioners with a theoretical understanding of factors that influence behaviour change and the skills to work with individuals or groups to facilitate this process. Clinical psychologists have also moved from an almost exclusively patient focus to one that encompasses a wide variety of roles, including teaching and training, the supervision of others engaged in psychological therapy, and working at an organizational level in a variety of ways.

Those that have made the move to working with physically ill patients have necessarily had to encounter and use a different set of theories and principles from those that guide practice in psychiatry or other specialisms. They have encountered the scientific discipline of health psychology.

Health psychology is an applied discipline. As such, it draws predominantly on theoretical models developed in a wide variety of psychological disciplines, including cognitive psychology, social psychology, and the study of emotions. In the UK, for example, its development began in the mid-1980s, when it was first recognized as an area of academic interest by the British Psychological Society (BPS), which in 1986 established a health psychology section, led by Professor Marie Johnston. Since then, this group has gained divisional status within the BPS. This change in
status is not trivial. It indicates that the status of health psychology has shifted from one of a shared scientific interest to that of an applied profession. Health psychologists, with appropriate training, can now achieve chartered status and work autonomously within the health service and other settings. Health psychologists are already working in areas such as health promotion, health-related research, and as consultants to a variety of organizations. An increasing number are also more directly involved in the provision of health care.

The delineation of the roles of the two professional groups in this setting has already resulted in forests of paper being consumed in consultation documents and lively debate. At present the boundaries between the two professions are blurred. A crude position statement would indicate that clinical psychologists will maintain the patient as their primary focus, while health psychologists work at other levels: teaching, training, working at an organizational level, and so on. Those who have both clinical skills and the knowledge and practice base of health psychology, and who may truly be called clinical health psychologists, may adopt any or all these roles. However, this simple categorization fails to take account of the training in non-patient issues that clinical psychologists receive, and will surely be challenged by clinicians who are skilled in working at an organizational level, by health psychologists who prove to have excellent therapeutic skills, and so on.

Whatever the final, probably overlapping roles, adopted by each profession, what is clear is that psychologists working in medical settings need to be aware of both health and clinical psychology theory and how it can be applied to maximize the effectiveness of health care delivery. This book provides an introduction to the knowledge base, theory, and the practice of both health and clinical psychology as applied to health, and is relevant to professionals, trainees or students wishing to gain an understanding of health and clinical psychology as applied to the care of the physically ill. It is divided into four parts:

- Part I: Behaviour, stress and health
- Part II: Understanding health-related behaviour
- Part III: Applied health psychology
- Part IV: Clinical interventions

**Part I: Behaviour, stress and health**

The two chapters in Part I consider the relationship between behaviour, stress and health. Chapter 1 firstly considers the risk for disease associated with a number of individual behaviours such as smoking or poor diet, before moving to consider more ‘social’ causes of ill health such as low socio-economic status or poor working conditions. It also considers how the effects of gender on health may be behaviourally mediated and
not simply a function of biology. Each section considers some of the controversies that have been associated with the relevant area of research as well as what we know about the associations between each risk factor and disease. Chapter 2 focuses on social, psychological and physiological theories of stress. It considers the relationship between theories that consider stress to be ‘in the eye of the beholder’ (for example Lazarus and Folkman 1984) and those that consider stress to be a more direct function of environmental demands and resources (for example Hobfoll (1989)). It then describes the physiological processes that underlie the stress response, considering the cortical, sympathetic and immune systems.

Part II: Understanding health-related behaviour

The second part also comprises two chapters. The first provides a critical overview of some of the most influential theories of behavioural decision making used and developed by health psychologists. These social cognition theories attempt to identify key variables (attitudes, social norms, cost/benefit analyses and so on) that underpin choices related to health behaviours. They have not been without their critics, however, and the chapter explores some of the strengths of other approaches to the study of health-related behavioural choices. The second chapter in Part II examines children’s and adults’ concepts of both health and illness and how these concepts influence their response to illness. It also considers how cognitions form an important part of our response to one particular symptom: pain.

Part III: Applied health psychology

This part has three chapters. The first identifies a number of elements of the care system that impact on how people cope with illness and react to hospitalization. The second considers how these may be influenced by psychologists and other health professionals to maximize the effectiveness of the health care system and minimize its adverse psychological consequences on the individuals who enter it. The chapters cover issues such as the experience of hospitalization, how people cope with a diagnosis of severe or chronic illness, staff/patient interactions, adherence to medication and behavioural programmes, factors that influence medical decision making, and stress and the hospital system. The second chapter includes examples of the work currently conducted by health psychologists within the health care system.

The third chapter in Part III adopts a critical stance in relation to health promotion. It argues that while educational and community-wide programmes based on psychological principles have proven effective in changing behaviour in the past, future initiatives should focus on changing
new risk factors and social and structural moderators of disease, including socio-economic inequalities and work factors, rather than focusing exclusively on changing individual behaviour. In doing so, the chapter refers back to some of the risk factors discussed in the Chapter 1 and social cognition theories discussed in Chapter 3.

Part IV: Clinical interventions

The final part has four chapters. The first provides a brief description of the clinical interventions that are conducted with patients who are physically ill. The types of intervention selected for inclusion in this chapter are those most commonly used with patients in acute medical settings. They include interventions that have been developed specifically for use with patients with chronic health problems, and others that are in more general use. The effectiveness of these approaches in the management of disease states, reducing risk for disease progression, and helping people to cope with the emotional sequelae to their illness is considered in the final two chapters. Sitting between these chapters is one that focuses on the assessment of health and psychological status in physically ill patients. This interrupts the flow between the chapters describing therapeutic approaches and their application in health care settings. This was done for two reasons. First, to emphasize the importance of assessment and not to make it the final, ‘add-on’, chapter. Secondly, some of the assessment instruments described in this chapter contribute to the evaluative research reported in the following two chapters.

And finally . . .

Writing this text has encouraged (nay, forced!) me to read in areas of health and clinical psychology about which I previously knew very little. It has instilled an interest in areas previously hidden from me, some of which I am now actively involved in researching. I hope the book is able to provoke such an interest in you, and that you enjoy reading it.

Paul Bennett
Part I
Behaviour, stress and health
Psychosocial correlates of health

The assumption that behaviour or personality is linked to health is not new. The Ancient Greeks and Romans described associations between personality and health, and this presumptive link has since continued through medical folklore and even psychoanalytic theory. However, the scientific exploration of links between behaviour and health is a relatively young venture. Even ‘classic’ studies identifying a link between smoking and disease were conducted only in the 1950s and much relevant research is more recent. This research has had three primary foci. The first has been the link between behaviours, such as smoking or eating habits, that confer risk of disease indirectly. Smoking, for example, may cause disease as a result of the carcinogens inhaled. The behavioural repertoire associated with smoking is not in itself harmful. A second set of research has focused on behaviours that directly moderate risk of disease. This includes exercise, but perhaps more excitingly from a psychological perspective has also included individual differences, including Type A and C behaviour. A third strand of research has focused on elements of the social or psychological environment that influence disease rates, including social support and socio-economic status. The latter, in particular, has recently emerged as an extremely important area of research.

Rather than simply report the relationship between these variables and disease rates, this chapter not only reports such data but also looks at some of the issues or controversies raised by each set of research. The chapter considers:

- Individual risk factors and disease
- Personality and disease
- Social and environmental influences on health
Investigating the behaviour/disease relationship

Two differing methodologies have been used to measure the association between psychosocial factors and health. The simplest is known as a case-control design, and involves comparing individuals who have a disease with controls matched on important variables such as socio-economic status (SES) and age. Any between-group differences found on other variables of interest are thought to imply causality. This type of approach is fairly cost-effective but has a number of weaknesses. First, it considers only differences between the survivors of illness and controls. Those who die of their disease are excluded from such an analysis, potentially weakening the magnitude of any observed behaviour/disease relationship. Secondly, the method allows associations between variables to be identified but the directions of such relationships have to be assumed. In some cases the direction of any relationship may be obvious: people with lung cancer, for example, are unlikely to start smoking as a consequence of their disease. However, causality can be more difficult to disentangle in other cases. Findings of high levels of stress in individuals with a debilitating disease, for example, may raise the question as to whether the disease or the stress came first.

An alternative approach, known as a longitudinal design, involves measurement of behaviour in a cohort of (typically) healthy individuals prior to disease onset. As the study progresses, those that develop disease are identified and comparisons are made between the baseline characteristics of these individuals and those who do not develop the disease. Again, any differences on such measures are thought to imply causality. This method has the benefit that the behavioural variables are measured prior to disease onset, so causality can be assured. However, there are a number of problems inherent in this approach. First, the cohort of individuals has to be sufficiently large to ensure that a statistically significant number of individuals will develop the diseases under investigation. Secondly, the method is based on the assumption that baseline levels of behaviour will remain constant over the period of the study. The long duration of such studies makes this assumption questionable. It is possible that many individuals within the cohort will make considerable lifestyle changes over follow-up periods that may last up to twenty years or even more. Any such changes may attenuate the relationship between behaviour and the initiation of disease. That such studies still find relationships between behaviour and disease attests to the strength of such relationships.

Behaviour and health

Some of the first evidence to substantiate a link between behaviour and health came from the Alameda County Study (Berkman and Syme 1979).
This longitudinal study has followed nearly seven thousand initially healthy individuals for a period of more than 20 years and identified which factors measured at baseline were associated with health or ill health over this period. One of their earlier reports was the first to highlight an association between 'lifestyle factors' and increased longevity. The behaviours, now known as the Alameda Seven, were: sleeping 7–8 hours a day, having breakfast every day, not smoking, rarely eating between meals, being near or at prescribed weight, moderate consumption of alcohol, and regular exercise. Cross-cultural comparisons have also shown an association between behaviour and health. The longevity of the Abraskians, a people who live in a remote part of Russia and who reputedly live to extreme ages, for example, has been attributed to genetics and a variety of behavioural factors, including a low animal fat and high vegetable diet, high levels of social support, no consumption of alcohol or nicotine and vigorous work activity.

Evidence linking behaviour and health is now overwhelming. Peto and Lopez (1990), for example, estimated that 75 per cent of all cancer-related deaths are attributable, at least in part, to behaviour. Others, including the World Bank (1993), have stated that a significant number of chronic diseases and up to half of all premature deaths can be attributed to behavioural factors. Five behaviours in particular are associated with risk for disease: smoking, alcohol misuse, poor nutrition, low levels of exercise and unprotected sexual intercourse.

**Smoking**

Smoking doubles the risk of premature death. Approximately 3 million people die of tobacco use each year across the world (Peto and Lopez 1990). It is responsible for approximately 30 per cent of cases of coronary heart disease (CHD), 75 per cent of cases of cancer, 80 per cent of cases of chronic obstructive airways disease, and 90 per cent of deaths associated with lung cancer. The risks attributable to passive smoking are also substantial. It is estimated that about 25 per cent of lung cancers that occur in non-smokers are attributable to passive smoking. In Greece, the risk for cancer attributable to passive smoking is considered comparable to that of smoking itself.

Present morbidity levels associated with smoking reflect the cumulative risk of smoking over many years, and historical processes of some decades ago. Increased lung cancer rates in women over the past two decades are thought to be the result of a rapid increase in the numbers of women smoking during and after the Second World War. Reductions in lung cancer rates amongst men may reflect the introduction of cigarette filters at about the same time. In contrast, childhood illnesses represent the more immediate impact of smoking. In the USA, an estimated half million cases of childhood pneumonia and bronchitis are attributed to parental smoking. Smoking cessation decreases risk for all smoking related diseases: former smokers live longer than persistent smokers do.
In the West, smokers are now a minority in every age and social group. In the UK, for example, adult smoking rates between 1974 and 1996 fell by 26 per cent among men and 22 per cent in women, to 29 and 28 per cent of the population, respectively (see Figure 1.1). Smoking rates among young people fell consistently between 1974 and 1992. More recently this decline has slowed and there is evidence of increasing smoking rates in this group, particularly among young women, who may soon prove the majority of smokers in a number of countries.

**Excessive alcohol consumption**

Excessive alcohol consumption may impact adversely on both short- and long-term health. It is thought to contribute to 3 per cent of all cancers. Alcohol also contributes to conditions such as cirrhosis of the liver and hypertension. However, the most damaging effect of alcohol may be behavioural. It is estimated that 20 per cent of psychiatric admissions, 60 per cent of suicide attempts, 30 per cent of divorces, and 40 per cent of incidences of domestic violence are associated to some degree with alcohol misuse.

In general, there is a linear relationship between level of alcohol consumption and disease rates. The one exception to this is that relating to CHD. A number of cross-sectional studies in the 1970s reported a J-shaped relationship between consumption and disease rates. This suggested that moderate consumption of alcohol is associated with lower risk for CHD than total abstinence, while higher levels of consumption increase risk. This unexpected relationship was met initially with some caution, with some suggesting that it may have been an artefact of the populations studied. It was suggested that the higher than expected rates
of CHD among the non-drinkers may have reflected the inclusion of individuals who had stopped drinking as a consequence of drink-related health problems. However, a number of longitudinal studies (see, for example, Shaper et al. 1994) have found the J-shaped relationship between alcohol and CHD after following cohorts of individuals free from disease at baseline. A mechanism through which cholesterol reduces risk of CHD has now also been found: moderate consumption appears to reduce harmful cholesterol levels.

Defining what is meant by excess alcohol consumption has proven far from simple. This confusion is illustrated by changes to health advice made by the UK government in 1995. Between 1986 and 1995 the recommended limits for weekly consumption were 21 units of alcohol or less for men, and 14 units or less for women. In 1995, a government committee established to review these guidelines recommended they be increased to 28 and 21 units per week, respectively. These changes caused a furore and much criticism among alcohol experts, particularly as they were not based on any new evidence (see, for example, British Medical Journal, vol. 293). Consequently, a number of health promotion and alcohol agencies have been reluctant to adopt these guidelines and there is a lack of clear advice concerning the recommended limits to consumption.

The percentage of the population to exceed the 21/14 unit limits has remained quite stable throughout the past decade, although there has been a slight increase among women: 27 per cent of men and 12 per cent of women exceeded these limits in 1996 (see Figure 1.2). Consumption declines with age: 40 per cent of men aged 18–24 and 18 per cent of those over 64 years report drinking over the recommended limits. The same pattern is found among women, although consumption is lower, with rates of 24 and 7 per cent respectively. Those in the lower socio-economic groups tend to drink more than the more socially advantaged.
Cholesterol

Raised serum cholesterol levels increase risk for CHD. The Multiple Risk Factor Intervention Trial (MRFIT), for example, followed over 350,000 adults for six years and found a linear relationship between baseline cholesterol level and the incidence of CHD or stroke (Neaton et al. 1992). Individuals within the top third of cholesterol levels were three and a half times more likely to develop cardiovascular disease than those in the lowest third. While there is no threshold level below which there is no risk for CHD, risk is significantly increased by cholesterol levels above 5.2 mmol/litre for those aged over 30 years, and above 4.7 mmol/litre for younger people. These margins place about two-thirds of the UK population at some risk for CHD as a consequence of their serum cholesterol levels (Lewis et al. 1986).

Cholesterol is essential to life. It is a constituent of every cell in the body, and is implicated in a variety of bodily functions, including the production of sex hormones and the bile necessary for digestion. A significant percentage of our cholesterol is synthesized by the liver; the rest is absorbed from food. Circulating levels of cholesterol are also mediated by stress (see Chapter 2) and exercise levels. Nevertheless, the most frequent method by which public health authorities have tried to control cholesterol is through dietary means. Recommended levels of intake are frequently substantially lower than actual levels. In the USA, for example, approximately 44 per cent of calories are consumed as fat, contrasting with the recommended level of 30 per cent. Despite these figures, there is some evidence that the British diet is becoming more healthy (see Figure 1.3).

The unexpected twist in the cholesterol story is that low cholesterol levels also confers risk of premature mortality. The MRFIT study found that individuals with low cholesterol levels carried a risk of suicide or trauma-related death 1.4 times greater than that of men in the mid-range. Even more dramatically, a longitudinal study of 52,000 Swedish adults reported that participants in the low cholesterol range evidenced a rate of non-illness-related mortality 2.8 times higher than those in the mid-range: risk for suicide was 4.2 times greater (Lindberg et al. 1992).

The link between low levels of cholesterol, suicide and accident rates may appear, at first consideration, somewhat surprising. However, there is considerable evidence from forensic studies that low levels of cholesterol are associated with aggression, personality disorder and low mood. With these findings in mind, the excess mortality following cholesterol reduction is perhaps not so surprising. More problematic is finding an explanation. Current explanations are focusing on a link between cholesterol and serotonin levels. Low levels of cholesterol are associated with low serotonergic activity, which, in turn, is linked to aggression and disinhibition of behaviour. While there is reasonable evidence to support both these links, the mechanisms through which the effects are mediated are unclear. One possibility is that low cholesterol levels mediate changes
in cell membrane function resulting in alterations to the serotonergic neurotransmission processes.

**Exercise**

Those who are physically active throughout their adult life live longer than those who are sedentary. One of the earliest studies to report such a relationship compared CHD rates among bus drivers and bus conductors who shared a similar working environment, but engaged in significantly different levels of exercise. Bus conductors evidenced significantly lower rates of CHD. In retrospect, some of these differences may be attributable to differences in stress levels associated with the differing job types (see below). However, these findings have been supported by a number of longitudinal studies. Paffenbarger *et al.* (1986), for example, monitored leisure time activity in a cohort of Harvard graduates for a period of 16 years. Those who expended more than 2000 kcal of energy in active leisure activities per week lived, on average, two and a half years longer than those who expended less than 500 calories in exercise. How this protection is achieved, whether through short, intense periods of exercise or longer, less intense periods, appears unimportant and no additional health gain is achieved by exceeding these limits. Uptake of exercise is protective against CHD, leading to reductions in resting blood pressure, cholesterol and triglyceride levels. Exercise is also an important aspect of weight control and, particularly in women, is protective against osteoporosis. As a function of the relatively low levels of fat in those who exercise regularly, it may also protect against some cancers.
Forty-five per cent of the UK population report engaging in some form of leisure exercise at least once a month, with the figure rising to 64 per cent if walking is included in these activities. A lesser number of individuals achieve the levels of exercise considered necessary to protect against CHD: here, the figure is nearer to 25 per cent of the adult population (Norman et al. 1998). These figures, however, represent a significant rise in exercise participation: in 1985, only 20 per cent of British men and 2 per cent of women engaged in such levels of exercise. Those who engage in exercise are more likely to be young, male, and members of higher socio-economic groups. Participation in leisure exercise among professionals, for example, is virtually double that among unskilled manual workers (80 versus 45 per cent), although the latter may engage in more physically demanding work activity. Those who participated in some form of sport in their youth are almost three times more likely to exercise in adulthood.

Unsafe sex

Estimates of the prevalence of HIV in 1997 suggested that a total of 30.6 million people were infected with HIV worldwide. In the UK, the primary route of infection has been through sex between men, accounting for 72 per cent of all AIDS cases reported by 1998. Eighteen per cent of cases resulted from heterosexual sex. However, the incidence of new cases among gay men is falling slowly while the incidence of HIV infection within the heterosexual community is rising. In addition, young people are at increasing risk of infection: adolescent heterosexuals account for about 20 per cent of all newly reported cases in the USA (Stiff et al. 1990).

Nearly half of British adolescents aged between 16 and 17 report having had at least one sexual partner during the previous year. However, they are unlikely to plan intercourse and those using a condom are in the minority. The findings of a large-scale British survey conducted by Wellings et al. (1994) indicated that only about half of those whose first sexual experience occurred between the ages of 16 and 24 years used a condom at first intercourse: 31 per cent of men and 24 per cent of women report using no form of contraception. Younger people were less likely to take precautions: over 60 per cent of respondents who had their first sexual experience at the age of 13 reported not using any form of contraception.

Heterosexuals in the general adult population use condoms consistently only about 10–15 per cent of the time with primary partners and 15 per cent of the time with secondary partners (Dolcini et al. 1995). Even more concerning from a disease prevention perspective is the choice made by those who are known to be HIV positive not to use a condom. Sobo (1993), for example, was able to identify a group of HIV positive women who did not use a condom with their lover or husband, but did
Table 1.1  The percentage of respondents engaging in one or more key health-related behaviours

<table>
<thead>
<tr>
<th>No. of health behaviours</th>
<th>Percentage of sample</th>
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<tr>
<td>0</td>
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</tr>
<tr>
<td>1</td>
<td>20.6</td>
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<tr>
<td>2</td>
<td>34.3</td>
</tr>
<tr>
<td>3</td>
<td>30.9</td>
</tr>
<tr>
<td>4</td>
<td>8.1</td>
</tr>
</tbody>
</table>

Source: Norman et al. 1998

so with more casual sexual partners (see Chapter 3). The likelihood of an increase in the rate of non-protected intercourse appears to be rising, perhaps because of the wide prevalence of beliefs that new treatments for HIV will be curative (Kalichman et al. 1998).

Is there a healthy lifestyle?

In an attempt to answer this question, Norman et al. (1998) categorized over 13,000 people in a British survey as either engaging or not engaging in up to four health-related behaviours: not smoking, moderate alcohol use, exercising three or more times a week, and eating fruit or vegetables regularly. While 93 per cent of the sample reported engaging in at least one of these behaviours, only 8 per cent reported that they did them all (Table 1.1).

Personality and disease

Type A behaviour and hostility

First identified by two cardiologists, Rosenman and Friedman, Type A behaviour (TAB) was defined as an excess of competitiveness, time urgency and easily aroused hostility. The absence of such characteristics is referred to as Type B behaviour. Early case-control and longitudinal studies showed significant associations between TAB and CHD. Such was the research consensus achieved that, by the mid-1980s, TAB was considered to confer the same degree of risk for CHD as the more traditional risk factors of high blood pressure and cholesterol. Subsequent, and frequently methodologically flawed, studies failed to find a relationship between TAB and disease progression in men either at high risk of CHD or who had already experienced a myocardial infarction (MI). As a consequence, this degree of consensus is no longer evident. However, one component of TAB, hostility, appears to convey the degree of risk
previously ascribed to TAB and this has formed the primary focus of more recent research.

A number of case-control studies have reported significant associations between hostility and CHD, with the strongest relationship being found among those under 50 years of age. Such a finding is not unique: the predictive power of other biological and behavioural risk factors is also strongest among younger people. However, the most convincing evidence linking hostility to CHD has stemmed from longitudinal studies. In one of the earliest such studies, Barefoot *et al.* (1983) followed 255 physicians for 25 years and found that those who scored above the median on measures of hostility taken while in training were nearly five times more likely to experience an MI over this period than those who scored below the median. These data have been supported by findings in older populations. European studies (for example Everson *et al.* 1997) following middle-aged men for up to 25 years have found an association between hostility and coronary events. Hostility may also contribute to the development of CHD in women (Lahad *et al.* 1997). Data from some of these and other studies combined into meta-analysis suggested a significant association between hostility and CHD (Miller *et al.* 1996).

A second set of studies has measured the association between hostility and the degree of atheroma of the cardiac arteries, measured using angiography. Although there have been a number of positive findings, several studies have failed to find any relationship between hostility and atheroma. One explanation for these disparate findings may be biased sampling. Angiograms are invasive procedures usually conducted only on patients who report some degree of symptomatology. Accordingly, the samples used are likely to be non-representative, and the results of most of these studies should be considered with caution. Two types of study are the exception to this rule: those that track the progression of atheroma over time, and those involving angiograms in representative populations. In the first type, Julkunen *et al.* (1994) measured the relationship between hostility and the progression of atherosclerosis over a two-year period in a sample of 119 middle-aged men. The progression of atheroma was almost twice as fast in highly hostile participants as in those who were low in hostility. Utilizing the second methodology, Barefoot *et al.* (1994) took advantage of the need for airline pilots to have routine angiograms to ensure their fitness to fly. They compared the hostility scores of pilots with angiographic evidence of CHD with those having none. Among non-smokers only, pilots found to have evidence of CHD had higher hostility scores than did those in the comparison group.

**Type C behaviour**

Type C behaviour has been independently described by Greer and Morris (1975) and Temoshok (1987). It is defined as an aggregate of several
coping styles, in particular being stoic, cooperative, appeasing, unassertive and inexpressive of negative emotions, particularly anger. It is thought to be linked to the development of cancer.

A number of studies have shown an association between Type C personality and the incidence or progression of cancer. Using a case-control design, Kune et al. (1991), for example, compared patients newly diagnosed with colorectal cancer and community controls matched for age and gender. Patients with cancer were significantly more likely to report histories of unhappiness in childhood and recent adult life and to have strong feelings of discomfort when experiencing feelings of anger. These differences may have been influenced by knowledge of a diagnosis of cancer. Accordingly, more weight should be ascribed to the findings of studies where a diagnosis was not known at the time of psychological assessment. A number of such studies (for example Greer and Morris 1975) have found that women who suppressed their anger or had a conforming personality were more likely to have malignant changes than those without these characteristics. Longitudinal studies have also shown Type C characteristics to predict cancer. Shaffer et al. (1987), for example, followed 972 physicians for a 30-year period and found that participants characterized by high levels of ‘acting out’ and emotional expression had a less than 1 per cent risk of developing cancer. Participants characterized as ‘loners’ and thought to inhibit emotional expression were 16 times more likely to develop cancer than those in this group.

Concordant with the Type C hypothesis is work examining the impact of ‘fighting spirit’ on cancer progression. This constellation of behaviours is considered to be the opposite of Type C behaviour, and has been associated with longer survival following diagnosis. In the first study to identify this characteristic as a prognostic factor, Derogatis et al. (1979) found that women who showed ‘fighting spirit’ during treatment for metastatic breast cancer lived significantly longer than those who did not. However, many of these women also received less chemotherapy, suggesting that disease severity may have been worse in the poor survival group or that their higher levels of fighting spirit could have been a consequence of being subjected to lower levels of a physically and mentally debilitating treatment. Greer (1991) also found fighting spirit to be an important determinant of survival. In a longitudinal study following 62 women with early non-metastatic breast cancer, he identified five reaction types: denial, fighting spirit, stoic acceptance, helplessness and hopelessness, and anxious preoccupation. Breast cancer recurrences and mortality were recorded 5, 10 and 15 years later. By 15-year follow-up, 43 per cent of the women who were categorized in the fighting spirit group were alive without recurrences; this compared with 17 per cent in all the other groups. Similar evidence has been published in a variety of cancers, including melanoma and lung cancer. However, there have also been some negative findings. In a larger replication study of the original Greer study, for example, Dean and Surtees (1989) found denial measured...
after surgery was associated with a *favourable* outcome in a group of 125 women with non-metastatic breast cancer followed for up to eight years.

Such contradictory findings have meant that the relationship between Type C behaviour, ‘fighting spirit’, and cancer remains controversial and far from proven. A related strand of research has focused on the health outcomes following attempts to facilitate active coping with disease and the expression of emotions. These studies are considered in Chapters 7 and 8.

Social and environmental influences on health

**Socio-economic status and health**

There is strong historical evidence that the more affluent members of society have lived longer than the less well off. More recent evidence of this health gradient can be found in a study of nineteenth century obelisks in Glasgow graveyards. In an imaginative study, Davey Smith *et al.* (1992) measured the height of obelisks in Glasgow graveyards as a proxy for the wealth of the individual buried beneath them. They compared these with the ages of the first generation buried below and found a strong linear relationship between the height of the obelisks and the age of their first occupant, suggesting that the more wealthy lived longer. What is important is not just that this relationship existed, but that the families buried in the graves represented a small and wealthy fraction of the Glasgow population. This is not evidence that the very poor and immiserated did not live as long as the rich did: rather, that the relatively rich did not live as long as the very rich. Such a gradient still exists. It is progressive, and throughout the social classes. It holds for women as well as men and is characteristic of all western countries (Wilkinson, 1992).

A number of explanations have been proposed to account for these differences. People in lower socio-economic groups may be exposed to more environmental insults, low quality and damp accommodation, and air pollution. An alternative explanation may be that less well off individuals engage in more health-damaging behaviours, such as smoking or excessive alcohol consumption. While both these explanations may account for some of the differences in some of the studies, they cannot explain them all. Marmot *et al.* (1984), for example, explored the impact of a number of these variables on the health of British civil servants working in London over a period of ten years. Their findings indicated that while those in the more deprived social groups did engage in more health-damaging behaviours, these did not fully explain the health/SES relationship. When variations in smoking, obesity, plasma cholesterol and blood pressure were statistically partialled out of the risk
equation, occupational-status-related differentials in health still remained. Mortality was three times higher among men in the lowest grade than those in the highest.

While people who occupy the lower socio-economic groups may engage in more health-damaging behaviours, the adverse health effects of these behaviours may be overwhelmed by factors associated with their economic position. Hein et al. (1992), for example, reported data from a 17-year prospective study of CHD in Danish men. Adjusting for a variety of confounding factors, they found that men who smoked were three and a half times more likely to develop CHD than non-smokers. However, when these data were analysed according to SES, white-collar smokers were six and a half times more likely to experience a cardiac event than the equivalent non-smokers. Amongst blue-collar workers, smoking status conferred no additional risk for CHD. Fewer middle-class people may smoke, but those that do may be particularly vulnerable to its health-damaging effects. Conversely, the impact of smoking on the health of the less well off is seemingly overwhelmed by social factors.

Comparisons of life expectancy across different western countries suggest some intriguing explanations of the relationship between social class and health. Wilkinson (1992) provided powerful evidence that it is not absolute wealth that determines health. He drew on evidence that showed only a weak relationship between the absolute wealth of the society and overall life expectancy. More predictive is the distribution of wealth within a society. The narrower the distribution, whatever its absolute level, the better the overall health of the nation. Accordingly, although Japan and Cuba differ substantially on measures of economic wealth, both have relatively equitable distributions of income and long life expectancies throughout their populations. Of particular interest is evidence from Scotland, which tracked average age of mortality and income distribution over the life of the Thatcher government. As earning differentials rose, so premature mortality among the less well off increased despite their access to material goods, food, clothing and so on remaining relatively constant. These data led Wilkinson to suggest that, for the majority of people in western countries, health hinges on relatively more than absolute living standards.

Wilkinson’s explanation of health differentials suggests that we engage in some form of comparison of our living conditions with others in society, and that knowledge of a relative deprivation in some way increases risk of disease. Three different psychological processes may also be implicated in the health gradient. Individuals in the lower socio-economic groups report more stressors than those in higher groups and that these stressors are frequently linked directly to their material conditions. In addition, the less well off have less control over their environment and fewer personal resources to moderate the impact of such stressors than the better off. Finally, social support, a powerful mediator of health status, is less available to those in lower socio-economic groups (Adler et al. 1994).
Social isolation and health

There is substantial evidence that both men and women who have a small number of social contacts are more likely to die earlier than those who have more extended networks. Data from the Alameda County Study (Berkman and Syme 1979), for example, showed increased longevity to be associated with relatively high numbers of social ties as a consequence of marriage, contacts with close friends and relatives, church membership and membership of other organizations. The most isolated were the most prone to premature death even after controlling for factors such as smoking, alcohol use and levels of physical activity. In a later study, Reynolds and Kaplan (1990) found that women who had few social contacts and were socially isolated were at double the risk of developing hormone-related cancers and evidenced an almost fivefold increase in risk of dying from them than less isolated women.

Similar results have been reported in European samples. Orth-Gomer and Johnsson (1987), for example, followed a cohort of 17,400 men and women for a period of six years and found that both men and women who had a restricted social network evidenced a 50 per cent greater risk of CHD than those who were socially embedded. However, the relationship between social contact and health was not always linear: those with many social contacts did not always benefit in terms of health. Older women, for example, who had many social contacts evidenced higher mortality than those with medium-sized networks. To explain these apparently anomalous findings, Orth-Gomer and Johnsson analysed their data not simply according to the absolute number of contacts, but taking into account the nature of the contacts. When they did this, they found the strongest predictor of mortality was a lack of social integration, which the authors considered to provide guidance, practical help and a feeling of belonging. They found only a low association between the provision of emotional support and CHD, although a later study by the same group found this to be an important protective factor. In an alternative interpretation of the Type A hypothesis, Orth-Gomer and Unden (1990) suggested that TAB or hostility might confer risk of CHD as a consequence of associated social isolation. In a longitudinal study, following a cohort of men for ten years, they found no differences in mortality between Type A and Type B men. However, over this period, 69 per cent of the socially isolated Type A men in the cohort had experienced an MI, in contrast to the 17 per cent incidence among those who were socially integrated.

Further evidence of the impact of social isolation can be found in studies of populations already experiencing disease. Williams et al. (1992), for example, found that patients with CHD who were unmarried and without a confidant experienced a threefold higher risk of mortality over a five-year follow-up period than those who were. Reflecting the subjective nature of social support, some studies that have failed to find a
The relationship between marital status and mortality following diagnosis of illness have found the quality of these relationships and the emotional support within them to be associated with survival.

The mechanisms through which social isolation confers risk are yet to be fully understood. Two primary processes are thought to be through depressed mood and health-compromising behaviours, both of which are associated with social isolation. Broman (1993), for example, reported that a decrease in social ties predicted an increase in health-damaging behaviours, while gains in social ties were associated with increases in health-protective behaviours. Of course, social ties may also serve to maintain unhealthy behaviours: friends may offer cigarettes to fellow smokers and put pressure on individuals not to quit smoking so as to maintain group cohesion. The impact of depression on risk of disease is considered in the next chapter.

**Gender and health**

Women, on average, live longer than men do. In the UK, life expectancy rates in 1996 were 75 years for men and 80 years for women. The most obvious explanations for these differences are biological. Oestrogen, for example, delays the onset of CHD by reducing clotting tendency and blood cholesterol levels. Not only is there a marked increase in the prevalence of CHD in postmenopausal women, they also become affected by the same influences as men. Lahad et al. (1997), for example, found hostility scores were linearly associated with increased risk of MI in postmenopausal women.

Studies of the relationship between disease and factors such as work strain, social support and hostility have rarely been conducted with women. Those that have suggest that men typically evidence greater stress hormone, blood pressure and cholesterol rises in response to stressors than women. However, while there may be some differences in the process of response between the sexes, these differences may not simply reflect biological differences. Work by Lundberg et al. (1981), for example, suggested that women in traditionally male occupations exhibit the same level of stress hormones as do men in similar jobs. In addition, where women feel equally or more threatened by the stressor than men their physiological response matches that of men. These findings suggest that social and cultural processes may drive some of these differences.

Societal processes also influence levels of engagement in health-related behaviours. Women exercise less frequently than men, partly as a consequence of family responsibilities and joint home/work responsibilities. In addition, women are frequently disadvantaged in behavioural negotiations. Nowhere is this more stark than in the negotiation of sexual behaviour: nearly 40 per cent of a sample of young Australian women
reported having engaged in sexual intercourse at some time when they did not want to do so (Abbott 1988). Whatever the cause, men behave differently from women: they are more likely to smoke cigarettes, and smoke higher nicotine and tar cigarettes than women: they typically eat less healthily, and drink more alcohol than women (Reddy et al. 1992). They may also encounter adverse working conditions more frequently than women.

Together, these data suggest that while biological factors may contribute to some of the differentials in health status between men and women, others are behaviourally or societally mediated. Gender differentials in life expectancy arise, to a significant degree, from the cumulative effects of different social worlds that men and women experience from the moment of their birth. As socio-political conditions change and more women enter the workforce, an emerging question is whether they will also take on the excess mortality associated with men. This does not seem to be the case. Indeed, there is increasing evidence that occupying multiple roles, including paid employment, is associated with better health and lower rates of premature mortality (Vagero and Lahelma 1998). However, there may be some important exceptions to this general finding: women who occupy demanding roles both at work and in the home appear at particular risk of disease (see below).

Minority status and health

There is a strong association between ethnicity and health status. In the UK, rates of ill health and premature mortality amongst ethnic minorities are typically higher than those of the white population, although there are also differences in mortality patterns between ethnic minorities. Rates of CHD amongst British men from the Indian sub-continent are 36 per cent higher than the national average and among young people are two to three times higher than that of whites. The Afro-Caribbean population has particularly high rates of hypertension and strokes, while levels of diabetes are high among Asians. In contrast, both groups have lower rates of cancer than the national average. A similar picture is found in the USA, where black people have higher age-adjusted mortality rates than whites for a number of diseases including various cancers, heart disease, liver disease, diabetes and pneumonia.

Some of these variations in health outcomes may be explained by differences in behaviour across ethnic groups. In the UK, for example, alcohol-related morbidity is high amongst African Caribbean men and Asian males of Punjabi origin, while a high dietary fat intake is common among Asians. Ethnicity may also confer different sexual norms and behaviours that may impact on health. The most common exposure route for HIV infection among whites is through sexual intercourse between
men: for blacks it is through heterosexual intercourse, whilst for Asians it is mixture of both.

A significant proportion of members of ethnic minorities occupy lower socio-economic groups. Accordingly, explanations of health or behavioural differences between ethnic groups have to take socio-economic factors into account. Stress specifically associated with minority status may also contribute to high disease rates. Ethnic minorities experience wider sources of stress than whites as a consequence of discrimination and racial harassment and the demands of maintaining or shifting culture. They may also experience more problems in gaining access to health services such as cancer screening and antenatal care.

Minority status may also be conferred by behaviour. A number of studies have shown that isolation experienced as a consequence of sexual orientation may impact significantly on health. Cole et al. (1996) found that healthy gay men who concealed their sexual identity were three times more likely to develop cancer or infectious diseases than men who were able to express their sexuality. The same research group found social rejection to influence disease progression in HIV-infected men. Those who experienced social rejection evidenced a significant acceleration towards a critically low CD4+ lymphocyte level (see Chapter 2) and time to diagnosis of AIDS.

**Working conditions and health**

One of the most widely accepted models of work stress is that of Karasek and Theorell (1990). Their job strain model identified three key influences on work stress: the demands of the job, the latitude the worker has in dealing with these demands, and the support available to them. They suggested that these interact to predict stress and stress-related disease. In contrast to previous theories of work stress, they noted that high job demands are not necessarily stressful; it is when these combine with low job autonomy and low levels of support that the individual is likely to experience stress. Their model suggests that rather than the stereotypical 'stressed executive', those who experience stress are likely to hold blue-collar or supervisory posts. In a review of studies examining the strength of the Karasek model, Kristensen (1995) considered 16 studies measuring the association between job strain and mental and physical health outcomes. Fourteen reported significant associations between conditions of high job strain and the incidence of either CHD or poor mental health. An alternative model has been proposed by Siegrist et al. (1990). This suggests that work stress is a consequence of an imbalance between perceived efforts and rewards. High effort and low reward are thought to result in emotional distress and adverse health effects. In a five-year longitudinal tracking over 10,000 British civil servants both theories received some support (Stansfeld et al.
Age-adjusted analyses showed low decisional latitude, low work social support, and effort/reward imbalance to predict poor physical health.

For men, it appears that job strain is a function of the working environment alone. For women, who may frequently have significant responsibilities beyond the workplace, work strain appears to combine with other areas of stress to confer risk of disease. Haynes and Feinleib (1980), for example, showed working women with three or more children to be more likely to experience CHD than those with no children. Alfredsson et al. (1985) compared the risk conferred by work strain and working overtime on men and women in a sample of 100,000 Swedish men and women. As predicted by Karasek’s model, higher rates of MI were associated with increased work strain in both men and women. However, working overtime decreased risk for MI among men, while it was associated with an increased risk in women. For women, working overtime of ten hours or more per week was associated with a 30 per cent increase in risk for CHD. One explanation for these contradictory findings is that men may compensate for their increase in working hours by a decrease in demands elsewhere in life. For women, such increases may not be so compensated and simply increase the total demands made on them. This increase in overall demands may constitute the main risk for stress and disease. Support for such a hypothesis can be found in the results of a study by Lundberg et al. (1981), who found that female managers’ stress hormone levels remained raised following work, while those of male managers typically fell; this effect was particularly marked where the female managers had children.

Summary and conclusions

Since the 1950s there has been irrefutable evidence that certain behaviours contribute to the development of disease. This has led, in part, to a movement towards increasing personal responsibility for health care and significant health promotion programmes and numerous self-help books aimed at promoting individual behaviour change. In the wake of this personal responsibility movement has followed a new wave of research which has emphasized that health is a consequence not simply of one’s own behaviour but also of the social, economic and political context in which we live. This has fed criticism of the self-help movement, claiming that this approach leads to a risk of blaming individuals for their own poor health. It has also highlighted the need for public health initiatives to target environmental factors as well as individuals if the health of the population is to be enhanced. Such an agenda has been established by the World Health Organization and is returned to in Chapter 7.
Further reading