"The Role of Perceived Stress and Personality in Symptom Experience of Chronic Inflammatory Bowel Disease."

Thesis submitted in partial fulfilment of the requirements for the degree of Master of Philosophy in the discipline of Psychology.

This research programme was carried out in collaboration with the Gastrointestinal Unit at The Western General Hospital, Edinburgh.

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Abstract.

Two studies were performed with participants suffering from one of two inflammatory bowel diseases (Crohn’s Disease or Ulcerative Colitis). One of the main thrusts of the research was to compare two theoretical models of the stress and illness relationship (namely the Transactional model and the negative affectivity/latent variable model). The first study, a cross-sectional design was intended to elucidate some of the demographic, personological, and behavioural factors involved in experience of disease activity. Disease differences and gender differences are also discussed. Personological, behavioural, and medical factors were used to attempt to predict disease experience outcome – perceived stress, neuroticism, and perceived disease severity being notable successes in predicting disease experience outcome over and above clinical predictors used. The second study, a pilot of a longitudinal study, investigated the temporal relationship between these psychological factors and the daily course of the disease over a 28-day period. The results from both studies support the theory of a link between perceived stress and disease activity experience. The results from both studies are also used to attempt to evaluate two models of stress and illness (Transactional and latent variable). The success of this support for either theory is limited and is discussed in detail. Both studies do, however, show the importance of considering psychological factors in these disease populations.

Key words: Crohn’s Disease, Ulcerative Colitis, perceived stress, neuroticism, hierarchical multiple regression, daily stress, and daily disease activity.
CHAPTER 1: Introduction.

1.1. Psychology and health

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1. Introduction.

1.1. Psychology and health

The field of health psychology is concerned with the relationships between behaviour, health and illness. This includes attempts to reliably assess levels of health and ill-health as well as attempting to isolate behavioural/psychosocial factors that predict health status. Health psychology, then, is concerned with research on these issues and the application of findings of research to help to change people’s behaviour in order to prevent or cure disease / illness, and to promote wellness and good health.

The progress of twentieth-century science, and its success in diagnosing and treating certain illnesses/diseases, has changed the pattern of disease experience and thus is changing the principal problems faced by most modern medical practitioners. In 1900, the three most common causes of death were influenza, pneumonia, and tuberculosis. More recently, however, the three leading causes of death are heart disease, cancer and strokes (Sexton, 1979). Thus, while doctors at the turn of the century struggled with contagious diseases, today’s major medical problems may be more closely related to behaviours, such as smoking, eating the right foods, and dealing with stress. As a result health professionals are becoming increasingly involved in trying to change the way people live rather than focussing on how they die. Thus psychology may have an integral role to play in augmenting modern medicine, in the search for treatments / causes / preventions of these modern ailments.
1.1.1. Stress and health.

Living in the twentieth-century is stressful. Indeed it can, and has been, argued that all life is stressful. Hans Selye (1974), an acknowledged early authority on stress, claimed that in humans stress is always present to some degree, and that some level of stress is actually desirable. He went so far as to claim that an absence of stress in people is called death. It may be the case that some stress is desirable to increase arousal and performance, but we are all aware that prolonged and/or severe stress may be detrimental to our psychological or even physiological well being.

At first, human stress-illness research focussed on a group of illnesses known as psychosomatic disorders. These include ulcers, high blood pressure, migraine or tension headaches, asthma, and a variety of skin complaints such as eczema and psoriasis. Researchers quickly realised, however, that stress may not be limited to experiences of acute illness such as these, but may also be involved in exacerbation of chronic illnesses or in the causes of diseases with unknown aetiology. Before considering the role of stress in illness it is necessary to consider what is actually meant by the term stress. It will become apparent that the term is not quite as simple as it may at first seem (Seyle, 1974).

1.1.1.1. Stress/stressors/stressful?

The Penguin dictionary of psychology (1985) gives two definitions of stress:
1. “Generally, any force that when applied to a system causes some significant
modification of its form, usually with the connotation that the modification is a
defformation or a distortion. The term is used with respect to physical, psychological and
social forces and pressures. Note that stress in this sense refers to a cause; stress is
the antecedent of some effect.

2. A state of psychological tension produced by the kinds of forces or pressures alluded to
in 1 above. Note that stress in this sense is an effect; stress is the result of other
pressures. When meaning 2 is intended, the term stressor is typically used for the
causal agent.” (Reber, 1985, pgs 736 & 737)

Without wanting to bore the reader with yet another introductory section
dedicated to definitions, and rigorous semantics, this dichotomous view of an
apparently simple term is important (I will attempt to be as brief as possible).

The first of these two definitions, i.e. that stress is exemplified by specific
events/pressures/troubles/etc., is the definition that a lot of readers will be familiar
with. These events/pressures/troubles/etc., may consist of severe life events (deaths,
divorce, changing jobs or schooling, moving, etc.), or ongoing smaller pressures (high
workload, work politics, caring for ill people, etc.). This assumes that stress can be
treated, in terms of research design, as an independent variable, which we may
observe or even manipulate with a view to analysing its effects on an organism. A
large body of research has attempted to do just this. It is clear that manipulating
events, and purposefully applying stress (in this sense) to an organism, has ethical
implications, and the extremes of this sort of research have been restricted to animal
studies or older experiments with humans when ethical constraints appear to have been less stringent. Thus research into stress (in this sense) is now more or less totally observational.

Considering stress as an independent variable, however, has one serious drawback. It ignores the important point that different people may react very differently to exactly the same proposed stressful event. Can we say from this that one person is stressed and the other is not? Or that they have different reactions to stress? To do either of these acknowledges that stress has differential effects on different people, and therefore, that stress, to a certain extent, exists within that person, and is not solely external to the individual. This is the construct that is alluded to in the second of the definitions mentioned above – i.e. given the same situation (or stressor) one individual may experience stress and another may not. Thus, from this perspective, stress is experiential and exists in the perceptions and experiences of the individual. Some situations, however, may be considered as universally stressful, e.g. the death of a friend/spouse/family member, and for an individual to not react by experiencing stress (albeit in differing magnitudes) may be seen as indicative of a problem. Thus it appears that ‘stress’, the umbrella term, needs to be considered as both events that may, or may not, be experienced as stressful – potential stressors, and also as the individual reactions to those stressors – experienced/perceived stress.
1.1.1.2. Life events stress and health.

As mentioned above, one way of considering stress is to observe the life change events and the stress that they produce, this is known as life events research, because it concentrates on the life events that precipitate stress. The kind of event that is being referred to here are things like the death of a spouse, divorce, marriage, major injury, being fired from work, etc. Note that these events are not necessarily considered as negative experiences, but they may also be positive and still cause stress (e.g. marriage, pregnancy, new job, etc.). Several groups of researchers have attempted to arrange these events on a scale so that relative stress values can be ascertained (Holmes & Rahe, 1967; Sarason & Spielberger, 1979). One such arrangement of these events can be seen in table 1.1.1.2.1

Table 1.1.1.2.1. The relative stress values of various life change events.

<table>
<thead>
<tr>
<th>Life Change Events</th>
<th>Life Stress Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Family</td>
<td></td>
</tr>
<tr>
<td>Death of a spouse</td>
<td>100</td>
</tr>
<tr>
<td>Divorce</td>
<td>73</td>
</tr>
<tr>
<td>Marital separation</td>
<td>65</td>
</tr>
<tr>
<td>Death of a close family member</td>
<td>63</td>
</tr>
<tr>
<td>Marriage</td>
<td>50</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>40</td>
</tr>
<tr>
<td>Son or daughter leaving home</td>
<td>29</td>
</tr>
<tr>
<td>Personal</td>
<td></td>
</tr>
<tr>
<td>Detention in jail</td>
<td>63</td>
</tr>
<tr>
<td>Major personal injury</td>
<td>53</td>
</tr>
<tr>
<td>Sexual difficulties</td>
<td>39</td>
</tr>
<tr>
<td>Death of a close friend</td>
<td>37</td>
</tr>
<tr>
<td>Outstanding personal achievement</td>
<td>28</td>
</tr>
<tr>
<td>Start or end of formal schooling</td>
<td>26</td>
</tr>
<tr>
<td>Changing to a new school</td>
<td>20</td>
</tr>
<tr>
<td>Change in residence</td>
<td>20</td>
</tr>
<tr>
<td>Major change in eating habits</td>
<td>15</td>
</tr>
<tr>
<td>Vacation</td>
<td>13</td>
</tr>
<tr>
<td>Work</td>
<td></td>
</tr>
<tr>
<td>Being fired from work</td>
<td>47</td>
</tr>
<tr>
<td>Retirement from work</td>
<td>45</td>
</tr>
<tr>
<td>Changing to different line of work</td>
<td>36</td>
</tr>
<tr>
<td>Trouble with boss</td>
<td>23</td>
</tr>
</tbody>
</table>
In the last 15 years, one important thrust in illness aetiology research has been that of the potential negative causal effect of stressful life events like those listed above (Tijhuis et al, 1995; Craig et al, 1994; Schwartz et al, 1994; Servant & Parquet, 1994; Chorot & Sandin, 1994; Justice 1994). Life events scales are typically generated by having many individuals rate on a limited scale (e.g. 1-100) how stressful events chosen from a large list are, and then amalgamating those ratings. One of the most widely used scales was the Readjustment Rating Scale (Holmes and Rahe, 1967 see above table 1.1.1.2.1.). These scales have then been used to predict illness reporting. The theory is that high scores on life events scales (from the number of events or the sum of weighted events) are related positively to illness. This outcome variable, here named 'illness', may be many things like psychiatric symptoms, depression (Brown and Harris, 1978; Paykel, 1974), suicide attempts, heart disease (Theorell, 1982), leukaemia (Wold 1968), rheumatoid arthritis, colds and influenza (Evans et al, 1988; Stone et al, 1988), asthma, etc. The basic finding of all this body of work is that stressful life events do appear to predict illness reporting, but the effect size of this prediction is generally small – typically correlations reported are between .20 and .29, and thus account for relatively little variance in illness reporting (Lin et al, 1979).

This relatively small overall finding may be due to various methodological limitations of the studies including retrospective data collection or because of individual
variation in reactions to these stressful events, among other things (specifics of the limitations of life events research and inflammatory bowel disease will be discussed later). Therefore, because of the possible limitations to this area of research and the problems surrounding it, attention has also been focussed on the potential effects of perceived minor stressors/hassles on illness, including how these stressors may exacerbate already existing illnesses.

1.1.1.3. Minor stressors (hassles)/ daily stress and health.

One of the main reasons why research into the stress-illness link using major life events measures is difficult is because of the relative infrequent occurrence of these major life events. Conversely minor stressors / daily stress / hassles can be conceptualised as frequently occurring events that are perceived as stressful, annoying and unpleasant. Thus someone may score very low on major life events style measures, but still report feeling stressed, and they themselves might be unable to point to any specific ‘events’ that caused their stress. It is more likely that little annoyances or hassles have accumulated to produce stress. Imagine a day as follows: You wake up and in the shower you find that your flatmate has used the last of your shower gel, on your way to work you get delayed for a meeting by a traffic jam, at lunch they put gravy on your food when you told them not to, you get stuck behind a tractor driving home, and when finally home, your flatmate/partner has left a mess in the kitchen. It may seem silly to claim that any one of these hassles, individually, could affect health - people do not get heart disease because they have no shower gel in the morning - but cumulatively the effects of all the little hassles during the day may be profound. Delongis and his colleagues (1982) developed a questionnaire to
measure how stressful such everyday stressors are. The 117 items on the Hassles scale included having unexpected company, owing money to someone, worrying about weight, misplacing things, and being too busy. They found that the more hassles a person reported, the more likely the person was to have some health problems. Hassles scores were found to be more highly correlated with physical and mental health than were major life events measures (Kanner et al, 1981). They also measured ‘uplifts’, such as relating well to family members, eating out, and meeting responsibilities. Though they had expected to find that these uplifts counteracted the effects of stress, there was little evidence that positive events had any impact on health (Lazarus, 1981). Hassles measures like this (a one-off large measure), employing retrospective remembering for hassles over the last month, however, appear to ignore the essential temporal dynamics of this type of stressor. That is, these stressors are not necessarily well remembered because they are relatively minor, it is the cumulative effect of these hassles over time that is important, not the amount over the last month. Take for example, two individuals: individual A has had five hassles on every other day for the previous month; individual B, however, has had the same number of hassles, but they were all concentrated on the last week of the month. It is very likely that these individuals will have differing experiences of stress and therefore its knock-on effect on health may be different. This difference in experiences of stress will not be picked up by the large one-off style hassles measures. For this reason daily measures of minor stressors were devised and the attempt was made to compare scores on these to daily fluctuations in health.
The overall finding for research into the links between daily stress and health, does suggest that measures of daily stress can predict daily health fluctuations (Brantley and Jones, 1993). This link has been found in asthma (Jemmot and Locke, 1984; Goreczny, 1989), in headaches (Levor et al, 1986), and in diabetes (Goetsch et al, 1990), amongst others. Unfortunately, however, once again the effect size of this relationship between daily stress and health appears small, and variations across different illnesses in the size of this relationship can also be seen. There may be many reasons for this, some of which are outlined below in the section on methodological issues (see section 1.1.3.). More discussion on minor stressors and health, specific to inflammatory bowel diseases is provided in section 1.2.3.2.

1.1.2. Models of stress and health.

Various attempts have been made to model this stress – health relationship, given that it exists, with varying success, two of which will be discussed here. The first is a model of stress that has been very influential in stress research for a long time – this is the Transactional model of stress (Lazarus, 1966). The other is a more recent attempt to model stress, and relies on the use of more modern statistical techniques, and at present is quite prominent in research into stress and health – that is the Latent variable/negative affectivity model (Watson & Pennbaker, 1989).
1.1.2.1. Transactional model.

One particularly influential model of stress has been that proposed by Lazarus (1966, 1990). His cognitive transactional theory of stress, views the person and the environment in a dynamic, reciprocal, multidimensional relationship. Lazarus and Folkman (1984) state that stress is defined as a relationship between person and environment which is appraised as taxing or exceeding the individuals' resources and endangering the well being experienced by that person. Although there are variations of transactional models of stress, they tend to have at least three basic elements, these are: a) antecedents to stress, b) mediators of stress and c) outcomes of stress (Jerusalem, 1993). This model (see figure 1.1.2.1.1), which has been widely used in the explanation and development of theory for the stress-illness relationship, assumes that antecedent variables such as personality indicators or environmental stressors act via mediating variables, such as coping, to produce stress- or illness-related outcomes. These three conceptual black boxes will be considered separately.

Figure 1.1.2.1.1. Representation of the transactional model of stress (Lazarus & Folkman, 1984).

1.1.2.1. Antecedents.

The antecedents of the stress – outcome process tend to be divided into two distinct groups, these are variables relating to the external environment, and variables that are more personal or internal in nature.
a) Environment.

Environmental variables considered to be antecedents to this stress process are many and various. Out of these the characteristics of the stressor are important. The ‘size’ of the stressor is obviously important (in occupational terms this may be workload), e.g. an increase in expected output - writing an Mphil thesis may be considerably more stressful to an individual than writing a short essay. The nature of the stressor may also be important – that is although the size of a stressor may be the same, the stressor may be perceived as a positive thing like getting married as opposed to a negative stressor like the death of a friend/spouse/family member. Level of social support can be seen as another environmental antecedent to the stress process, in that if individuals believe they are part of a strong social support network then they may react differently to stressors (Aaronson, 1989). This, however, highlights the possible duality of this construct as social support is also thought to be an important mediator of the stress process (Abdel-Halim, 1982; McGee, Serey & Graen, 1983; Wilcox, 1981 – see section 1.1.2.1.2.c.), and it may be that ‘true’ social support is the mediator, and believed/perceived social support is the antecedent – implying that this may be more of a personal type of variable.

b) Personal.

As has already been mentioned, it may well be that perceived social support is a personal variable that may have effects on the stress process, by influencing a person's perceptions of, or reactions to, stressful events. Other variables thought to
act in the same way are often to be found on the left-hand side of the stress-outcome equation. These include certain personality traits, such as neuroticism, Type A behaviour, locus of control and cognitive hardiness. The latter two of these, locus of control and cognitive hardiness, may also, to a certain extent, be considered as mediating the stress – outcome relationship and therefore will be discussed along with the other mediating factors (see sections 1.1.2.1.2.b. & d. for discussion of locus of control and cognitive hardiness respectively).

The role of personality in disease and illness and illness reporting has a long history, and there are several good reviews which tell this tale (for a recent review see Adler & Mathews, 1994). Friedman and Booth-Kewley (1988) have tried to show that coronary arteriosclerosis is related to a personality type called ‘Type A’. Type A behaviour consists of excessive achievement striving, competitiveness, impatience, hostility, vigorous speech and motor mannerisms, and hostility. Eysenck (1985) proposed that the personality traits that are associated with cardiovascular disease and cancer are at opposite ends of particular personality dimensions. Thus an individual who is at risk of cardiovascular disease would score high on the personality dimensions of psychoticism and neuroticism, and the person who is at risk of cancer would score low on these same dimensions. More recently the personality dimension of neuroticism has been associated with, and implicated as a major antecedent of, many members of the ‘psychosomatic family’ of illnesses. N.B. ‘Psychosomatic family’ is a loose umbrella term used to denote all those illnesses thought to be related to psychosocial stress or major life events – e.g. facial neuralgia, non cardiac chest pain, peptic ulcers, irritable bowel syndrome, globus
pharynges, dysphonia, chronic fatigue syndrome, etc. It has been suggested in the literature that there may be certain personality characteristics that predispose individuals to react negatively (in terms of somatizing their stress) to stressful situations. The most pervasive of these personality characteristics hypothesised to be involved in the stress-illness link is the personality trait of neuroticism.

Neuroticism is defined as "a broad dimension of individual differences in the tendency to experience negative, distressing emotions and to possess associated behavioural and cognitive traits" (Costa and McCrae, 1987). Individuals with high scores on neuroticism report more illnesses and more frequent and severe physical symptoms, though they have a normal life expectancy (Smith and Williams, 1992). Watson and Pennebaker's (1989) highly influential construct of 'somatopsychic distress' or 'negative affectivity' can almost be seen as synonymous with neuroticism (Deary et al, 1997). Similarly, Mashall et al (1994) showed that many health related psychological indices were strongly related to neuroticism, in a large scale empirical study.

It is also possible, however, that personality's influence on the outcomes of the stress process is through its effect on the mediating variables within this process. For example particular coping styles / strategies employed by the individual may be associated with that individuals' personality. These potential mediating variables will be considered now, and the role of personality will be considered further in discussion of the alternative model of stress – illness, namely the Latent variable model.
1.1.2.1.2. Mediating Factors.

a) Coping

Throughout the history of stress research, various conceptualisations of the process of coping have been put forward. These date back at least as far as Freud (1933), who saw coping as an unconscious mechanism of 'ego defence' that individuals use to deal with internal threat and conflict. These included repression, rationalisation, and projection, and Freud conceptualised all defence mechanisms as pathological. Since then defence mechanisms have been divided into primary (e.g. repression) and secondary (e.g. projection) (White, 1948), or more recently into adaptive and non-adaptive strategies (e.g. Haan, 1977; Hilgard, 1949; Miller & Swanson, 1960; Vaillant, 1977).

During the 1970s and 1980s coping was more commonly seen not as an unconscious process that needed to be uncovered by the clinician, rather as a conscious response or reaction to external stressful or negative events. Thus, as these coping responses are believed to be conscious and may involve cognitive aspects of strategy formation and/or behaviour, the use of self-report measures is justified (Folkman & Lazarus, 1988; McCrae, 1984; Endler & Parker 1994). Since this conceptual change in the 1970s, now regarding coping as a conscious response to stressors, most research has taken the approach of using self-report measures of coping styles/strategies.

One useful distinction in the literature on coping is the difference between research performed from an intra-individual approach and that from an inter-individual
approach. This distinction is analogous to the difference in state and trait measures respectively. The intra-individual approach may seek to assess behaviours and cognitions of the same individual or the same group of individuals across different types of situation (i.e. the impact of various specific stressful situations on coping behaviours). Conversely the inter-individual approach would seek to acquire stable indices of individuals' coping styles/strategies for comparison with others. The majority of work on coping has taken an inter-individual approach focusing on the individual differences of coping styles/strategies, although some work has taken an intra-individual approach (see Taylor, 1990).

Table 1.1.2.1.2.a.1. (adapted from Endler & Parker, 1992) shows a number of studies that have identified varying amounts of coping dimensions within this inter-individual approach. Although the number of dimensions identified by these studies varies from as little as two to as many as twelve, there does appear to be some consensus on the distinction between emotion-focused coping (person-oriented) and problem-focused (task-oriented) coping dimensions (Endler & Parker, 1992). In addition to these two major dimensions, a third is identified by many of the studies shown in table 1.1.2.1.2.a.1. This is avoidance-oriented coping. This dimension can include person-oriented and task-oriented factors, i.e. one can avoid the stressful situation by being with other people (social distraction - e.g. at a party), or by substituting the stressful situation by engaging in another distracting task (distraction - e.g. watching television).
### Table 1.1.2.1.a.l: Summary of coping dimensions assessed in recently developed coping scales

<table>
<thead>
<tr>
<th>Reference</th>
<th>Coping Dimensions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amirkhan (1990)</td>
<td>1. Problem solving</td>
</tr>
<tr>
<td>Coping Strategy Indicator (C.S.I.)</td>
<td>2. Seeking social support</td>
</tr>
<tr>
<td>Billings and Moos (1981)</td>
<td>3. Avoidance</td>
</tr>
<tr>
<td>Billings and Moos (1984)</td>
<td>1. Appraisal focused</td>
</tr>
<tr>
<td>Carver, Scheier and Weintraub (1989)</td>
<td>2. Problem-focused</td>
</tr>
<tr>
<td>COPE</td>
<td>3. Emotion-focused</td>
</tr>
<tr>
<td>The Life Events and Coping Inventory</td>
<td>2. Self-recognition</td>
</tr>
<tr>
<td>L.E.C.I.</td>
<td>3. Distraction</td>
</tr>
<tr>
<td>Endler and Parker (1990a,b)</td>
<td>4. Self distraction</td>
</tr>
<tr>
<td>Coping Inventory for Stressful Situations</td>
<td>5. Endurance</td>
</tr>
<tr>
<td>C.I.S.S.</td>
<td></td>
</tr>
<tr>
<td>Epstein and Meier (1989)</td>
<td>1. Emotional</td>
</tr>
<tr>
<td>Constructive Thinking Inventory</td>
<td>2. Behavioural</td>
</tr>
<tr>
<td>C.T.I.</td>
<td>3. Categorical thinking</td>
</tr>
<tr>
<td>Feifel and Strack (1989)</td>
<td>4. Superstitious thinking</td>
</tr>
<tr>
<td>Life Situations Inventory</td>
<td>5. Naive optimism</td>
</tr>
<tr>
<td>L.S.I.</td>
<td>6. Negative thinking</td>
</tr>
<tr>
<td>Folkman and Lazarus (1980)</td>
<td>1. Problem-focused</td>
</tr>
<tr>
<td></td>
<td>2. Emotion-focused</td>
</tr>
<tr>
<td>Folkman and Lazarus (1985)</td>
<td>1. Problem-focused</td>
</tr>
<tr>
<td></td>
<td>2. Wishful thinking</td>
</tr>
<tr>
<td></td>
<td>3. Distancing</td>
</tr>
<tr>
<td></td>
<td>4. Emphasising the positive</td>
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<tr>
<td></td>
<td>5. Self-blame</td>
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<td></td>
<td>6. Tension-reduction</td>
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<tr>
<td></td>
<td>7. Self-isolation</td>
</tr>
<tr>
<td></td>
<td>8. Seeking social support</td>
</tr>
<tr>
<td>Ways of Coping Questionnaire</td>
<td>2. Distancing</td>
</tr>
<tr>
<td></td>
<td>3. Self-controlling</td>
</tr>
<tr>
<td></td>
<td>4. Seeking social support</td>
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<tr>
<td></td>
<td>5. Accepting responsibility</td>
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<tr>
<td></td>
<td>6. Escape-avoidance</td>
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<tr>
<td></td>
<td>7. Planful problem-solving</td>
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<tr>
<td></td>
<td>8. Positive reappraisal</td>
</tr>
<tr>
<td>Miller (1980, 1987)</td>
<td>1. Information-seeking</td>
</tr>
<tr>
<td>Miller Behavioural Style Scale</td>
<td>(monitoring)</td>
</tr>
<tr>
<td>M.B.S.S.</td>
<td>2. Information-distracting</td>
</tr>
<tr>
<td>(blunters)</td>
<td></td>
</tr>
<tr>
<td>Nowack (1989)</td>
<td>1. Intrusive positive thoughts</td>
</tr>
<tr>
<td>Coping Style Scale</td>
<td>2. Intrusive negative thoughts</td>
</tr>
<tr>
<td></td>
<td>3. Avoidance</td>
</tr>
<tr>
<td>Patterson and McCubbin (1987)</td>
<td>4. Problem-focused</td>
</tr>
<tr>
<td>Adolescent Coping Orientation for Problem Experiences A. - C.O.P.E.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1. Ventilating feelings</td>
</tr>
<tr>
<td></td>
<td>2. Seeking diversion</td>
</tr>
<tr>
<td></td>
<td>3. Developing self-reliance</td>
</tr>
<tr>
<td></td>
<td>4. Developing social support</td>
</tr>
<tr>
<td></td>
<td>5. Solving family problems</td>
</tr>
<tr>
<td></td>
<td>6. Avoiding problems</td>
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<tr>
<td></td>
<td>7. Seeking spiritual support</td>
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<tr>
<td></td>
<td>8. Investing in close friends</td>
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<tr>
<td></td>
<td>9. Seeking professional support</td>
</tr>
<tr>
<td></td>
<td>10. Engaging in demanding activities</td>
</tr>
<tr>
<td></td>
<td>11. Being humorous</td>
</tr>
<tr>
<td></td>
<td>12. Relaxing</td>
</tr>
</tbody>
</table>
Thus the consensus of the three main dimensions of coping are as follows:-

**Task-oriented:** Strategies used to solve the problem, or at least reduce the effects of the problem.

**Emotion-oriented:** Dealing with the problem by an emotional response. Getting angry or upset are common responses.

**Avoidance-oriented:** Avoiding the problem by distraction or social diversion.

Within the process-oriented transactional model of stress, personological and situational factors interact serving to increase state anxiety and threat perceptions as a result of external stressors. However, it has been argued that an individual's particular coping strategy will affect the very situations that are interacting to produce negative outcomes of stress, and not just the individual's perceptions of the threat of the stressful situation. Thus coping strategies appear to mediate between antecedent stressful events and such consequences as anxiety, depression, psychological distress and somatic complaints (e.g. Auerbach, 1989; Billings & Moos, 1981; Coyne, Aldwin & Lazarus, 1981; Endler, 1988; Endler & Parker, 1990b,c; Perlin & Schooler, 1978).

b) **Locus of Control**

Like coping styles, the idea of locus of control (LOC), whether or not the control for phenomena experienced by the individual occur from within that person or from outside, has been reported to be related to self-reports of health status /
symptomatology. Rotter (1966) originally proposed a curvilinear relationship between LOC and reported symptoms, although research since has shown this not to be so (Rotter, 1975). Rather a number of studies have found LOC to be related to self-reported psychopathology in a linear fashion (Joe, 1971).

The division made by Levenson (1973b) in his setting up of the Internal, Powerful others, and Chance (I, P, & C) locus of control scales has proved useful in separating scores for different clinical groups (see Levenson, 1973a, 1973b; Wallston & Wallston, 1981, 1982). Levenson divided the external locus of control into two facets, that due to chance and that due to the influence of 'powerful others'.

Wallston & Wallston (1982) have reported a positive correlation between beliefs in chance and powerful others (i.e. an external locus of control) and scores on a depression inventory for epileptic persons. Wallston & Wallston (1982) have also reported a positive correlation between chance LOC and depression in chemotherapy patients. Levenson (1973b) has himself reported that "neurotics were significantly less likely to believe in control by powerful others and chance forces than schizophrenics, while depressives scored between the two" (p. 403).

Thus in general it is considered that individuals with an internal locus of control in their lives feel less impact from stress than those with an external locus of control or a belief that luck or fate controls their lives (Krause & Stryker, 1984; Revicki & May, 1985; Stern, McCants, & Pettine, 1982; Matheny & Cupp, 1983; Johnson & Sarason, 1978; Lefcourt, Martin & Saleh, 1984; Sandler & Lakey, 1982).

The role of control appraisal in adapting to chronic diseases is especially important in respect to cognitive adaptation theory. Living with a serious chronic illness creates
unusual psychological burdens for patients as they try to balance their need to
maintain a sense of mastery over their lives with their need to surrender treatment
of their disease to their health care providers (Reid, 1984). It is common in many
chronic illnesses for the course to follow a period of general remission with
occasional acute flare-ups of the symptoms of the disease (this can be seen in
Rheumatoid arthritis, multiple sclerosis, inflammatory bowel disease, and asthma,
amongst other diseases). In these uncertain conditions the importance of the
perceptions of control patients have over their disease becomes obvious. Those
that feel they have greater confidence in their own ability to control their illness
(which could also be seen as being a higher level of self efficacy) are less depressed
and anxious and exhibit less impairment in daily living (Nicassio et al, 1985). There
are two current views concerning the control beliefs of patients adapting to having a
chronic illness (Affleck et al, 1987). The first of these is for the patient to feel that
where the opportunities for personal control are perceived to be small, the most
adaptive approach would be to surrender that control to powerful others, this can
be seen as a sort of vicarious control (Rothbaum et al, 1982). The second view is that
individuals can exert some control over some areas of their disease, no matter how
small, and that this will have adaptive benefits for those that can do this (Miller,
1980). This latter view leads to the prediction that patients’ perceptions of personal
control over their disease may be especially harmful and maladaptive, when the
disease is most severe, e.g. in a period of flare up, or where treatment options are
non-existent or limited.
One area in which patients suffering from a chronic illness can realistically and adaptively exercise personal control is in treatment decision making (Reid, 1984). It has been found that in one chronic illness group (Rheumatoid arthritis) patients report greater control over their symptoms than their disease course and that the health care providers had greater control over their disease course than they did (Affleck et al, 1987). Thus it can be seen that patient control beliefs would be very useful in relation to their perceptions of their disease severity, and it is in this respect that the current research looks at the perceptions of locus of control and their relationships to the patients’ health.

c) Social Support

Social support can also exert its effect on health by promoting activity in healthy behaviours. Individuals who report receiving more support for health promoting behaviours (like quitting smoking, exercise, avoiding alcohol) are reported to be more likely to engage and persist in those behaviours (Aaronson, 1989; Treiber, Baronowski, Braden, Strong, Levy, & Knox, 1991). Many other studies have found a buffering effect of social support on the stress-strain relationship (see Abdel-Halim, 1982; Gore, 1978; House, McMichael, Wells, Kaplan, & Landerman, 1979; Karasek, Triantis & Chaudry, 1982; Kobasa & Puccetti, 1983; Lefcourt et al, 1984; Sandler & Lakey, 1982; Seers, McGee, Serey & Graen, 1983; Wilcox, 1981).

In a recent review Adler & Mathews (1994) state that "some individuals may offset stress-induced emotional distress by engaging in behaviours that have health
damaging consequences" (p.243) – they also claim that with the addition of social support individuals may be less likely to engage in these health-damaging behaviours. Another example of this can be seen in the buffering effect of social support on the relationship between recent loss of a loved one and alcohol use among older adults (Jennison, 1992). Social support buffered the effects of loss - the relationship of loss to drinking was reduced for those with more support.

However, many of these investigations did not find consistent effects across different a) stressors and indexes of strain – e.g. work / relationship problems, b) sources of support – e.g. friends / loved ones / colleague, and c) personal characteristics of the subjects – e.g. high/low in neuroticism.

Indeed there is also research that suggests that social support plays no buffering role in stress-strain relationships. Ganster, Mayes, & Fusilier (1986) found no buffering effect of social support, or indeed any higher order interaction effect with other variables in the stress process, for 326 employees of a contracting firm. Likewise both LaRocco & Jones (1978) and Etzion (1984) found no buffering effect of social support for the stress-strain relationship in 3725 Navy men and 657 Israeli managers respectively.

Clearly the research findings for the effect of social support on the stress-strain or stress-illness relationship are ambiguous. One problem that continually re-emerges
is the problem of construct specificity, with many constructs of social support including other constructs, for example the related Adlerian construct of social interest, which has been established as a buffer (Crandall, 1984; Zarski, Bubenzer & West, 1986; Crandall & Kytonen, 1980). Social interest may well encompass both locus of control and social support. More simply, many of the social support concepts may include aspects of the social diversion factor of coping strategies/styles.

d) Cognitive Hardiness

If the stress-illness relationship is mediated by aspects of personality, it seems logical to talk about different groups of individuals who are differentially susceptible to illness following the same stressors or stressful events. That is that a) stress cannot be conceived in terms of an external event independent of the individual’s appraisal of the event, and b) some individuals are more likely than others to appraise events in such a way that they evoke a stressful response. Kobasa (1979) has proposed the concept of psychological hardiness to distinguish between individuals that are less susceptible to illness following stressful situations and those that are not. Namely those that are ‘psychologically hardy’ are less likely to report somatic complaints faced with the same stressful situations as others. Kobasa identifies three components of the hardy personality. Firstly hardy individuals are high in commitment: a “tendency to involve oneself in (rather than experience alienation from) whatever one is doing or encounters” (Kobasa, Maddi, & Kahn, 1982, p. 169) [c.f. task-oriented coping as opposed to avoidance-oriented coping]. Secondly, hardy individuals are high in challenge: a “belief that change rather than stability is normal in
life and that the anticipation of changes are interesting incentives to growth rather than threats to security” (Kobasa et al, 1982, pp. 169-170). Thirdly hardy individuals are thought to be high in perceived control: a “tendency to feel and act as if one is influential (rather than helpless) in the face of the varied contingencies of life” (Kobasa et al, 1982, p. 169).

Studies conducted by Kobasa and her colleagues have used both retrospective and prospective designs to support the notion that cognitive hardiness plays a predictive role in health status (Kobasa, 1979; Kobasa, Maddi, & Courington, 1981; Kobasa et al, 1982), even when other health predictors are treated as independent (prior illness, Kobasa et al, 1982; Type-A behaviour, Kobasa, Maddi, & Zola, 1983; constitutional predisposition, Kobasa et al, 1981; and social support, Kobasa & Puccetti, 1983). Given a relationship between cognitive hardiness and health, the potential mechanism for the relationship still remains unclear. To what extent, as mentioned above, do aspects of cognitive hardiness affect individuals’ health by the indirect mechanism of coping strategies, and to what extent are the effects direct? That is, are there conceptual and construct overlaps between cognitive hardiness and other constructs in the realm of health psychology. The two different roles of hardiness, 1) as an indirect buffer by acting on the individual’s coping mechanism, and 2) as a direct influence on the experience of strain, can be seen in figures 1.1.2.1.2.d.1. and 1.1.2.1.2.d.2. respectively.
Four studies have been reported that tested for hardiness main effects (the direct effect) and hardiness by stressful life event interactions (the buffering effect) on self reported illness (Kobasa et al, 1981; Kobasa et al, 1982; Kobasa et al, 1983; Kobasa & Puccetti, 1983). It is worth noting at this point that the dependent variable in these studies is self-reported illness and not illness per se. This raises the question whether hardy individuals are not simply less ill than their non-hardy counterparts, but rather hardy individuals may be less willing to acknowledge their illnesses (either
to others or indeed to themselves) as this would result in cognitive dissonance bearing in mind their high levels of perceived control. Despite this, along with other criticisms it can be concluded from the results of these studies that: "a) hardiness does have a direct effect on self-reported health, although we reserve judgement as to its effects on actual health; b) the buffering effect of hardiness on stress is substantially weaker than its direct effect; and c) these effects are more apparent for some hardiness sub-components than for others" (most notably Control and Commitment) (Hull, Treuren, & Virnelli, 1987, p.520).

1.1.2.1.3. Summary

We have seen just some of the variables that have been postulated as mediating the stress-health relationship acting in the middle section of the transactional model of stress (see figure 1.1.2.1.1.) - coping styles/strategies, cognitive hardiness, locus of control, and social support. Some of the constructs not mentioned above that might also play some sort of mediating role are self efficacy, alexithymia (problems of expressing emotion), coherence (feelings of congruence, fitting in), life values (long-range goals), etc. Hopefully it is clear to see that there is no lack of theoretical constructs for health psychologists to use when considering this question. This does, however, generate the problem of comparability between studies. If studies are measuring different constructs it becomes difficult to see where the common ground is between all the mediating variables. Is it possible that all these constructs might be tapping into just a small number of 'true' mediators or underlying factors? This problem shall be addressed again.
One other problem that has been noted concerning a vast majority of the work into this area is the lack of objective antecedents and outcomes to which mediating variables can be compared. Research into Life Events research has been criticised on the grounds that the temporal measurement may be too grand in scale to pick up on the factors playing a role in causing stress outcomes (Fava & Pavan. 1976/77; Hislop, 1974). Life Events research has also been criticised for relying too heavily on retrospective experimental design. The majority of outcome measurement relies on self-reports of symptomatology or distress, which begs the question - are certain individuals only reporting greater symptomatology not actually experiencing it? Likewise the vast majority of mediating variables like social support and coping styles also depend on self-reports, as objective measures may prove difficult to obtain. Finally, if there are problems concerning the starting and end points of one's model then how can the connection via mediating variables be easily made? This is akin to trying to build a bridge when it is unknown where on either riverbank to start construction, and hoping to meet somewhere in the middle; similarly, the bridge itself may be badly engineered.

1.1.2.2. Latent variable model.

In contrast to the concept of a transactional model of stress (antecedents leading to outcomes by means of mediating variables), is the theoretical latent variable / negative affectivity model of stress and health. This model suggests that many self-reports - related to emotions and bodily states - might have a common source in a very broad
dimension of 'somatopsychic distress' or 'negative affectivity' (Watson & Pennebaker, 1989). Thus, instead of the linear transactional model, the negative affectivity model depends on the concept of a latent variable that is central to the plethora of constructs open to health psychologists. A diagrammatical representation of the latent variable model of stress and illness can be seen in figure 1.1.2.2.1.

Figure 1.1.2.2.1. A schematic representation of a latent variable model of stress-like negative affectivity.

F1 is a latent variable that could represent what has become known as negative affectivity - closely related to neuroticism.

Thus all the factors that are associated with the stress-illness relationship, are connected through a central underlying trait which can be called anything but appears to closely resemble the personality trait neuroticism.

1.1.2.3. Comparison of models.

In research that has attempted to compare these two models in specific illness groups, it has been concluded that “one may neither be so complacent about the independence of constructs as might be entailed in some transactional models nor so
cynical as a full blown negative affectivity model would suggest" (Deary, Clyde & Frier, 1997). Therefore it appears that some form of hybrid model would best describe the actual processes going on in the stress-illness relationship. The use of adequate modelling of the stress process might also go some way to help establish what it is that determines the extent to which individuals somatise their stress. The vast quantity of constructs available leaves us confused as to the independence and specificity of the measures we are using. To what extent do these measures all tap into the same underlying construct? The answer to this question appears to be “not totally”, otherwise the predominant theory of stress would be that of negative affectivity. It is true, however, that many of the measures used do intercorrelate, be this possibly through antecedent personality variables or whatever, thus to identify one construct as the single mediator between stressors and illness or occupational strain is possibly theoretically unsound. The health psychologist is faced with the proverbial walk on Antarctica’s frozen ground. One is unsure whether or not one is walking on frozen-over sea or independent separate land masses. Burrowing down through the ice might provide some insight into the state of the land/sea underneath but might not be constructive as to how it connects with other land masses which may be separate or attached by promontories. It is not until the ice melts that one can truly assess what one is, and has been, walking on. To say that one mediating variable connects independent antecedents and outcomes of the stress process is difficult if one is employing measures that continue to intercorrelate. This is analogous to claiming that a certain bit of ice is above land, and the adjacent patch of ice is over water, without having identified the boundaries of the land.
1.1.3. Methodological issues.

There are almost as many possible methodological issues to be considered when performing any area of research, as there are pebbles on the beach. Many of these are generally understood and are usually dealt with quite well. I will discuss two issues for this field of research that are particularly pertinent.

1.1.3.1. Individual differences.

There may be individual differences in the extent to which different individuals exhibit the stress-illness relationship, given that it exists. Some individuals when faced with stressors may indeed somatise that stress in terms of physical symptoms — these could be called ‘stress responders’. Other individuals, however, may not react to the same stressor in the same physical way — ‘stress non-responders’. If these individuals were grouped together in the analysis, it is possible that the net results might dilute or even negate a true stress-illness relationship. This may explain why findings to date on the stress-illness relationship have been slightly meagre, inconsistant or equivocal.

1.1.3.2. Reporting bias.

One problem that has arisen out of the search to find the personological link between stress and illness is the lack of hard evidence between personality and objective disease measures (Deary et al, 1995). Due to this, the emphasis has been shifted towards the search for a link between personality and illness/disease.
reporting, which has been notably more successful. This shift to people’s perceptions of disease is not unimportant especially in relation to health-related behaviours as it is their perceptions of their bodily states that lead people to take or avoid health-related behaviours (Deary et al, 1995). This shift in emphasis, however, also generates some problems which must be considered. When self-reports are being used to measure perceptions of stress and illness, there may be some level of reporting bias at play. For example, the reason neuroticism is quite predictive of health status, may be due to selective encoding / recalling of either stress or illness status. That is, individuals who score higher on neuroticism may not be objectively experiencing greater physical symptoms, but may be attending to them more and thus encoding them as a symptom, and thus remember them better. Alternatively some combination of these may be true. Thus part of the relation between neuroticism and elevated levels of self-reported symptoms may be due to actual differences in objective health related to neuroticism. Another part, however, may be due to subjectively inflated perceptions of illness on the part of the high neuroticism scoring individuals. Therefore if a positive link is found between self-reported perceived stress and self-reported illness, it makes it increasingly difficult to be able to interpret this finding meaningfully.
1.2. The psychology of inflammatory bowel disease.

1.2.1. What is IBD?

Inflammatory Bowel Disease (IBD) is a group of chronic disorders that cause inflammation or ulceration in the small and large intestines. Most often IBD is classified as Ulcerative Colitis (UC) or Crohn’s Disease (CD) but may sometimes be referred to as colitis, enteritis, ileitis, and proctitis.

Ulcerative colitis causes ulceration and inflammation of the inner lining of the colon and rectum, while Crohn’s disease is an inflammation that extends into the deeper layers of the intestinal wall (see figure 1.2.1.1). Ulcerative colitis and Crohn’s disease cause similar symptoms that often resemble other conditions such as irritable bowel syndrome (spastic colitis). Because both of these diseases cause similar symptoms differential diagnosis may be difficult, although the accuracy of diagnosis is improving greatly.
Crohn’s disease usually involves the small intestine, most often the lower part (the ileum). In some cases, both the small and large intestine (colon or bowel) are affected. In other cases, only the colon is involved. Inflammation may also affect the mouth, oesophagus, stomach, duodenum, appendix, or anus. Crohn’s disease is a chronic condition and may recur at various times over a lifetime. People may have long periods of remission, sometimes for years, when they are free of symptoms.
A recent prevalence study for the whole of Europe by Shivananda et al (1996), found the overall incidence per 100,000 at ages 15-64 years was 10.4 for Ulcerative Colitis, and 5.6 for Crohn’s Disease. However, the same large research study noted large regional differences across Europe. For example, the incidence rates in northern European centres were 40% and 80% higher than that for southern European centres for Ulcerative Colitis and Crohn’s Disease respectively (Shivananda et al, 1996). In their study the incidence rate ranged from 24.5 per 100,000 in Iceland, to 1.6 per 100,000 in Almada (southern Portugal) for Ulcerative Colitis, and from 9.2 per 100,000 in both Amiens (north west France) and Maastricht (the Netherlands) to 0.9 per 100,000 in Ionnina (north west Greece) for Crohn’s Disease. The incidence of these diseases in the UK, as reported by these researchers, was 10.0 and 3.8 per 100,000 for Ulcerative Colitis and Crohn’s Disease respectively. It is emphasised, therefore, that incidence rates do differ depending on geographic location. It is also noted that incidence rates also appear to be increasing – Kyle (1992) found a prevalence rate of 147 per 100,000 in the Grampian region of Scotland in 1988.

The symptoms of both of these disorders include abdominal pain, diarrhea, fever, vomiting associated with weight loss, and gastrointestinal bleeding. Both diseases are typically punctuated by periods of remission and exacerbation of these symptoms, with patients often being symptom free for long periods of time (Jewell et al, 1992). It is because of the similarity these two disorders show in their symptomatology that they are often considered together as inflammatory bowel disease (Whitehead &
Schuster, 1985), and some researchers have thought the two to be manifestations of the same disease (Kirsner & Shorter, 1982). There are, however, several differences between the two disease diagnosis which, to begin with shall be considered separately.

1.2.1.1. Disease differences – psychological.

Although some researchers in the field believe Crohn’s disease and Ulcerative Colitis to be manifestations of the same disease (Kirsner & Shorter, 1982; Whitehead & Schuster, 1985), there is some evidence that appears to point to psychological differences between the two disease populations. It is not an uncommon finding in the literature for Crohn’s disease patients to show higher rates of lifetime psychiatric symptoms than healthy controls, Ulcerative Colitis patients and non-gastrointestinal controls (Helzer et al, 1982; Helzer et al, 1984; Tarter et al, 1987). These symptoms included state and trait anxiety, depression, hyperchondriasis, phobia, panic, and somatization disorder. Other research, however, has shown there to be no differences between the disease populations on current psychiatric disorders (Andrews et al, 1987). Therefore the research on disease differences in terms of psychiatric / psychological criteria is equivocal.

1.2.1.2. Gender differences in prevalence of IBD.

The epidemiological literature on possible gender differences between men and women in inflammatory bowel disease does suggest some differences in prevalence
across gender. In Crohn's Disease the ratio of men to women for the whole of Europe according to Shivananda et al (1996) is 1:1.22. In Ulcerative Colitis the equivalent ratio is 1:0.81 (Shivananda et al, 1996). Thus there are more women with Crohn's Disease than men, and more men than women with Ulcerative Colitis.

1.2.2. Inflammatory bowel disease and smoking.

There is some evidence to suggest that smoking tobacco is linked to some aspects of the disease course in inflammatory bowel disease.

In Crohn's Disease more patients have been found to smoke or have been a smoker than an age/community matched control group [n=280] (Tobin et al, 1987). Smoking has been recognised as a risk factor for needing surgery (Lindberg et al, 1992), and recurrence of Crohn's Disease after surgery (Sutherland et al, 1990; Cottone et al, 1994), particularly in women and heavy smokers. In one study (Kurata et al, 1992), Crohn's Disease sufferers who currently smoked reported more days troubled by symptoms per month than non-smokers (15.4 days compared with 5.0 days – p < 0.001; n = 169), but the average number of hospitalisations per year was not significantly higher. One recent study (Cosnes et al, 1996), found that Crohn's Disease patients who smoked, again particularly women and heavy smokers, run a higher risk of developing severe disease involving the need for immunosuppressive therapy, than non-smokers (e.g. for female smokers, the 10 year risk of immunosuppressive therapy was 52% ± 11% compared with 24% ± 10% for non-
smokers, \( p < 0.001 \)). They also found that the use of immunosuppressive treatment neutralised the influence of smoking on surgery rates (Cosnes et al, 1996).

It has also been found that smoking tobacco (and in particular the nicotine intake that this involves) can affect disease experience in Ulcerative Colitis. Many studies have found that nicotine intake appears to have some protective function against Ulcerative Colitis yet a detrimental affect on disease course in Crohn's disease patients (see Thomas et al, 1998 for a good review of this literature). In 160 inflammatory bowel disease patients Fraga et al (1997) found that giving up smoking was a risk factor to developing Ulcerative Colitis (odds ratio: 3.2, \( p = 0.02 \)) and that in this disease group, non-smokers and, especially ex-smokers, need surgery more frequently than smokers (\( p < 0.01 \)) – they did not find this to be the case with Crohn's disease. Russell et al (1996), testing 1105 patients, found that in Crohn's disease smoking females reported a lower quality of life than non-smoking females (on all four dimensions of the IBDQ – inflammatory bowel disease questionnaire), but in keeping with Fraga et al's findings, that moderately smoking male ulcerative colitis patients reported fewer bowel complaints compared with non-smoking male ulcerative colitis patients (\( p < 0.001 \)). This is interesting in that smoking trends in the UK would predict, if anything, a decrease in Crohn's Disease and an increase in Ulcerative Colitis – which is not in line with the epidemiological evidence.

Thus in summary smoking appears to have a protective effect from Ulcerative Colitis and a possible detrimental effect in Crohn's Disease. Despite most of the research confirming these relationships, the nature of causation or the mechanisms of the relationship still remain unclear. First, it is entirely possible that nicotine intake...
affects disease activity in a purely physiological way - e.g. Nicotine may alter adherent surface mucus secretion and production of mucosal eicosanoids (Cope et al, 1986; Zijlstra et al, 1994) and smoking has been shown to have profound effects on cell-mediated immunity (Tollerud et al, 1989). It is also possible that there is something about experiencing the diseases that leads people to smoke or to quit, and that this relationship is what is being measured. For example, in Crohn’s Disease the experience of having severe disease activity (which might lead to surgery) might make a smoker smoke more or might initiate smoking - this hypothesis, however, seems unlikely. Thirdly, it is possible that smoking and disease activity are both related to a third variable (maybe personality) that affects both smoking behaviour and disease activity. Thus someone with a certain personality is more likely to smoke as well as being more likely to have severe disease activity – in the case of Crohn’s disease. In the case of Ulcerative colitis this would be reversed and may depend on a different personological profile. With regard to this an interesting recent study was performed by Barrett et al (1996). They assessed 82 inflammatory bowel disease patients on three self-report scales - the Eysenck Personality Questionnaire (E.P.Q.), the General Health Questionnaire (G.H.Q.) and the Hospital Anxiety and Depression Scale (H.A.D.S.) - primarily to establish whether Crohn’s disease patients’ personalities differed significantly from those with ulcerative colitis. They found that there were some differences in personality between Crohn’s disease patients and Ulcerative colitis patients (namely Crohn’s disease patients were significantly more extroverted ($P = 0.04$) than Ulcerative Colitis patients and tended to have higher psychoticism scores ($P = 0.06$)) but that these differences tended to be associated with smoking behaviour. Thus in their logistic regression models that adjusted for age, sex and smoking the associations
with extroversion and psychoticism scores persisted but were no longer significant at the 5% level.

1.2.3. Stress IBD research.

Akin to research in other areas of stress and illness, the three main research paradigms in this field investigate the physiological effect of stress due to major ‘life events’, the physiological effect of stress due to minor ‘hassles’, and potential physiological benefits from psychological therapeutic intervention. It is noted here that the term physiological effects may over emphasise the objectivity of physiological measures. In actuality these are normally measured by assessing symptom reporting which may be considerably less objective.

1.2.3.1. Stressful life events and IBD research.

A number of studies have addressed the issue of the precipitation and/or exacerbation of either or both ulcerative colitis and Crohn’s disease by stress due to major life events. Fava and Pavan (1976/77) compared 60 patients suffering from large bowel disorders including inflammatory bowel diseases, appendicitis patients, and irritable bowel syndrome). They found that the inflammatory bowel disease patients showed significantly more life events, especially more ‘exits’ and ‘undesirable events’, 6 months prior to the onset of illness than did the control appendicitis group. This finding was replicated for ulcerative colitis patients alone by Hislop
(1974) who also observed more life events in the six month period prior to onset of the illness for these patients compared with a control group.

Conversely to these findings Helzer et al (1982) found no correlation between the severity of ulcerative colitis symptoms and the frequency of potentially stressful life events within the 6 months prior to the interview. Helzer et al (1984) also found that Crohn's disease patients did not differ from patients suffering from other chronic illnesses when compared on a life events questionnaire. North et al (1990) reviewed 15 controlled ulcerative colitis studies, and reported that eight (53%) found a relationship between life events and the illness, and seven (47%) did not. Therefore the evidence for a stable relationship between stressful life events and ulcerative colitis is far from conclusive. This may be due to various methodological flaws used in life events research, which will be mentioned in greater detail below.

Similarly Gerbert (1980) reviewed several studies on the psychological aspects of Crohn's disease and reported that in 9 of 11 (82%) an important connection between stressful life events and the beginning of a relapse could be ascertained. Again, however, the majority of these 9 studies showing this link can be criticised on some of the methodological grounds mentioned below.

As stated in section 1.1.1.2., life events research tends to focus on the kinds of events that cause major life change such as redundancy, pregnancy, divorce, death of a friend or spouse, etc. Fortunately, in any 'normal' individual's life these events tend to happen with relative infrequency. Therefore one of the only practical ways
to study the effects these events have on a specific disease such as Crohn's disease, which in itself is relatively rare (between 0.9 – 9.2 per 100,000; Shivananda et al, 1996), is to use retrospective data collection of life events (Duffy et al, 1991; von Weitersheim et al, 1992; Monk et al, 1970). Such a methodology may be replete with flaws when causal inferences are to be made.

First, individual variation in the responses to life events questionnaires, or indeed at interview, questions the use of life events as a measure of stress for small sample sizes and within-subject analysis. A second problem is that with the use of retrospective data collection, patients with these diseases might indeed express greater levels of life events compared to controls, for to do so might serve to provide them with some explanation of the course of their own illnesses (von Wietersheim, 1992) i.e. an external, unstable attribution. Retrospective studies also often tend to disregard the individual temporal nature of the illness - the change of the disease activity over an extended period of time. Knowledge of such changes would enable researchers to examine whether an important increase or decrease in disease activity is linked to the proposed precipitating factors, like stress, particularly in clarifying temporal relationships.

Some more methodologically sound prospective studies have been carried out using life events measures in order to negate some of the problems mentioned above. Campbell et al (1986) using continuous assessment of stressful life events and symptoms of abdominal pain and diarrhoea found no significant correlation between
the occurrence of these variables in Ulcerative Colitis patients. On a study of 32 patients with relapsing inflammatory bowel disease using within-case regression slopes, North et al (1991) found no temporal association between life events and changes in intestinal symptoms. Riley et al (1990) tested 100 patients over a 48 week testing period and found no difference between patients with an acute relapse of ulcerative colitis and those patients in remission on a modified Paykel Life Event Scale. It must be noted however, that although they measured a sizeable sample on a standardised measure over a long period of time, all improvements on previous research, they only collected data every 12 weeks which might generate some problems drawing inferences from the results.

As is common in many areas of research, there is a problem in drawing comparisons between all these different studies. This is due to a lack of standardised measures of stress due to life events and of the putative outcome, disease activity. Differing sampling characteristics, methods of analysis and the lack of appropriate control groups only serve to compound this problem, leading to an equivocal, even contradictory, picture of the stress-illness relationship in these inflammatory bowel diseases.

A further problem with life events studies is that of timing and more specifically time lapse. Even in longitudinal studies, when the subject is asked to report the life events occurring in the previous month or two, the chances are that the resultant immunologically mediated disease activity might not be recorded accurately. For
example, it has been reported that the time lag for the effect of minor life events on
the common cold is approximately four days (Evans & Edgerton, 1991), thus to
analyse this by means of assessment in the order of months may miss the subtle,
possibly short lived, relationship between stress and disease. This error could be
corrected by collecting data on a shorter term basis, and thus due to the infrequency
of major life events, a measure of daily minor stressors/perceived stress may be
more appropriate to this finer temporal analysis.

1.2.3.2. Daily stress/hassles and IBD research.

The evidence stated above supporting a relationship between stressful life events and
inflammatory bowel diseases must be treated cautiously. The reason for this can be
seen in the problems of comparison between studies as well as the various
methodological flaws associated with life events studies (both mentioned previously).
The use of daily stress measures along with prospective study designs has to be
greatly emphasised in order to avoid these pitfalls. Therefore, researchers
interested in the stress-disorder relationship have focused attention on minor
stressful events and daily hassles as opposed to the problematic major life events
(Brantley et al, 1987). Indeed it has been suggested that measures of daily stress may
serve to account for a larger percentage of the variance in prediction of physical
symptoms than major life events (DeLongis et al, 1982).

To date, however, few studies have been conducted that use daily measures of stress
when considering the stress-illness relationship in inflammatory bowel diseases. One
study that has used a psychometrically appropriate instrument to measure daily stress and its relation to Crohn's disease was conducted by Garrett and colleagues (1991). They recorded the perceived impact of minor stressful events using the Daily Stress Inventory (Brantley & Jones, 1989) along with both the signs and symptoms of the disease for 28 consecutive days for 10 subjects. They found a direct relationship between daily stress and Crohn's disease activity when the effects of previous major life events were controlled for. This was not reflected in the findings for the within-subject analyses, which may reflect the limited sampling period of 28 days, during which substantial change in either disease activity or daily hassles may not have occurred. The inconsistent within-subject correlations also advocate the importance of taking into consideration individual differences in possible mediating or antecedent personological factors of the stress-illness process.

Another interesting study that measured daily psychosocial stress and symptomatology was performed by Greene et al (1994). They assessed 11 subjects on the IBD symptom diary and the psychosocial stress diary for 7 consecutive days each month for a year – thus generating 12 weeks worth of daily data. Despite the obvious criticism of their study being the small number of subjects involved, they did report some interesting results. They found a positive concurrent (zero time lag) relationship between both daily and monthly stress and inflammatory bowel disease activity. Their results also suggested a monthly rebound effect, whereby IBD symptom severity was negatively responsive to the previous month's stress. One criticism of this study, other than its use of only 11 participants, is its lack of personological data to assess any possible individual differences that may have
existed in the participants' responses to stress. They did use a coping styles measure which was not found to influence the stress-symptom relationship. This study (Greene et al, 1994) raises a number of methodological worries. Unlike the other 'prospective' studies, which used modified forms of the Crohn’s Disease Activity Index (C.D.A.I.), this study employed its own measure of 6 symptoms weighted equally to give a Crohn’s Disease only IBD Symptom complex score! No attempt appears to have been made to validate it against the CDAI. The stress measure was also 'novel'. The demands on subjects were considerable in terms of discriminations required and thus the reliability/validity of the stress measure must be questionable. Similarly, no reference to the validation of the Coping styles measure is offered.

1.2.3.3. Stress Intervention/therapy and IBD research.

Whether or not stress is an antecedent to inflammatory bowel diseases, the symptoms of them are clearly stress producing. Mallett et al (1978) have examined the effects of these diseases on the lifestyle of the individuals. They found that two thirds of their participants had to change their work routines and half of the patients reported generalised irritability with their families to be a problem during an attack. If, however, stress is an antecedent to these diseases, or at least serves to precipitate a relapse, then a course of therapy aimed at reducing the stress should have a reducing effect on disease activity.
Barbara Milne and colleagues (1986) conducted just such a study. They took two groups of 40 Crohn’s Disease patients, and administered the intervention to one of the groups and not to the other. This intervention took the form of a 6 month stress management course. They then followed up the two groups 4, 8, and 12 months down the line. The intervention group showed marked reduction on the Crohn’s disease activity index (CDAI: Best et al, 1976) and the inflammatory bowel disease stress index at all three points of follow up. A similar study performed by Schwarz & Blanchard (1991) attempted to evaluate a psycho-behavioural treatment for inflammatory bowel disease. They compared the effectiveness of a multi-component behavioural treatment package (including IBD education, progressive muscle relaxation, thermal biofeedback, and coping strategy training) on 11 test subjects with IBD, to the effectiveness of the control condition of solely symptom monitoring in 8 IBD patients. They found that at post treatment, the treatment group showed greater mean reductions on 5 symptoms (most notably abdominal pain ratings) than the control group. On Total Symptomatic change, however, the control group showed more improvement than the treatment group, although the treatment group perceived themselves as coping better with IBD, as feeling less IBD-related stress, and as experiencing less anxiety and depression when compared with the control group. Both these studies go some way to imply that stress does indeed have some role in the severity of disease activity.

1.2.3.4. Is there a link between stress and illness in IBD?

This research is not unequivocal. For example some research on life events in inflammatory bowel disease has reported higher levels of major life events in
inflammatory bowel disease patients than in either disease control groups or the
genral population (Fava & Pavan, 1976/77; Hislop, 1974). When looking at daily
stress and inflammatory bowel disease research, evidence can be found to support
the stress-illness link (Garrett et al, 1991; Greene et al, 1994). Similarly, intervention
studies have also found evidence to suggest a stress-illness link in inflammatory
bowel disease (Milne et al, 1986).

Evidence supporting the null-hypothesis (i.e. of there being no link between stress
and illness activity in inflammatory bowel disease) is also present, however (see
above). Methodological problems of measurement, including retrospective design
and data collection, lack of standardisation of measures of stress, illness activity and
mediating variables like coping or control, may contribute to these overall
inconclusive findings. Differing sample characteristics, methods of analysis and small
sample sizes only serve to compound this problem, possibly leading to an equivocal,
even contradictory, picture of the stress-illness relationship in these inflammatory
bowel disease diseases. Clearly the evidence provides us with a muddled picture as
to the role stress plays in inflammatory bowel disease.

1.2.4. The current research.

Clearly the ideal piece of research to answer the questions posed about the stress-
illness relationship would involve a great deal of work. One would need to assess
many participants (say at least 100) on a series of validated standardised measures
(including daily stress, mood, and disease activity, and periodical measures of coping,
personality, locus of control, etc.), for a long period of time or many regular smaller periods of time. The logistics of carrying out such a research study are outwith the constraints of time, money, effort, demand on subjects, etc., which most researchers (MPhil students included) need to comply with. Researchers have, therefore, compromised this ideal and attempted to assess the 'true' relationship between stress and illness through less onerous methods. We have already seen several attempts to do this. One study may be a cross-sectional study attempting to capture the relationship in one moment of time. We have already heard some criticisms of this type of method, however performing such a study does enable the research team to assess a greater number of individuals on more measures (the greater demand on the subject being relatively short lived). The alternative it seems is to measure fewer individuals on fewer measures over a longer period of time, which we have also seen. The cross-sectional method is akin to taking a slice through a tree trunk and attempting to draw inferences about the whole tree, from ground to utmost branches, from what is seen in that slice. Conversely the longitudinal research method is akin to making a long plank out of the length of the tree and trying to draw inferences about the trees width. To complicate things further, the measuring devices that are being used to look at either the tree slice or the plank may be very different and may have problems of their own. Therefore comparison between two trunk slices is difficult let alone between the tree slice and the plank.

Given that to 'see' the whole tree is virtually impossible, one can quite quickly see that a combination of these two methods may in fact be the best (although not perfect) way of 'looking' at the tree, and that this should be done with good standardised tools. This is what the current research attempts to do. Two studies are presented here, the main cross-sectional study, and a smaller pilot of a
longitudinal study – see chapter 5. Both studies employ the use of measures that are generally accepted and are quite well validated, and that are also comparable, if not identical, from one study to the other. Both studies assess the individual's personality, a factor that as we have already seen may be of great importance. The author is aware that both studies have their limitations, the pilot longitudinal study possibly more so than the cross-sectional, but the attempt is made to address some of the limitations of previous research in this area.

Bearing these limitations in mind, the current research attempts to examine the stress-illness relationship in Crohn's Disease and Ulcerative Colitis populations, firstly to establish whether it exists or not. Given that it does exist, the attempt is made to delineate which of the factors that are usually associated with the stress-illness relationship are most predictive of disease activity. To do this indexes of the following factors are gathered from the participants: demographic details, perceived disease severity, disease length, beliefs about health behaviours, perceived stress, disease activity, coping styles and strategies (palliative, instrumental, distraction, and negative emotion), trait personality (neuroticism, extroversion, openness, agreeableness, and conscientiousness), locus of control (internal, chance, and powerful others). Regression analysis of these indexes is used to try to provide a tentative comparison between the Transactional and Latent Variable models of stress and illness by looking at their respective predictive power. Thus with the particular indices being assessed here applied to the more generic model diagrams represented earlier (see figures 1.1.2.1.1 and 1.1.2.2.1) the comparison would be as follows:-
Figure 1.2.4.1. A schematic representation of the transaction model of stress-illness vs. a latent variable model with the particular indices assessed in the current research applied to the conceptual boxes.

Transactional Model

- Antecedents
  - perceived stress
  - locus of control
  - neuroticism
  - coping strategies / styles

- Mediators
  - locus of control
  - neuroticism
  - coping strategies / styles
  - Perceived Severity

- Outcomes
  - perceived disease severity
  - CDAI
  - Perceived Stress

vs. Latent Variable Model

- Illness Outcomes
  - perceived severity
  - CDAI

- Coping Style/strategy
  - palliative
  - instrumental
  - distraction
  - neg emotional

- Locus of Control
  - Internal
  - Chance
  - Powerful others

- Perceived stress
  - PSS-10

negative affectivity
neuroticism

NOTE: The concept is in Red, the application of that to the current study is in green, and any doubling up of index (possibly pointing out the non-specificity of the constructs involved) i.e. where the index belongs in more than one construct box, is in blue italic.

For an explicit more detailed list of the research questions and aims for the current research see section 2.5.

[Note: Separate introduction, justification, aims, methods, results and discussion for the pilot longitudinal study can be seen in chapter 5.]
CHAPTER 2: Methods.

2.1. Design.

2.2. Participants.

2.2.1. Exclusion Criteria.

2.3. Procedure.

2.4. Measures.

2.5. Aims and research questions.


2.1. Design.
A cross-sectional one off design was implemented. This was performed by post. Inflammatory bowel disease patients were assessed on a series of self report measures.

2.2. Participants.
Participants were chosen from lists of inflammatory bowel disease patients attending a gastrointestinal out-patient clinic. From a list of 234 patients, 190 (81%) met our inclusion criteria (see section 2.2.1. below for exclusion criteria), and were still present at the addresses given on the lists.

2.2.1. Exclusion Criteria.
Participants were excluded if:-

a). They had received major gastrointestinal surgery, i.e. a colostomy or ileostomy.

b). They were pregnant – pregnancy, and the hormonal changes associated with it, is thought to have an effect on the symptoms of inflammatory bowel disease (Castiglione et al, 1996).

c). They possessed concomitant physical or psychiatric illnesses (like cancer or depression) – medical records were accessed and scrutinised to establish concomitant illnesses.
Of this 190 who were invited to participate, 112 (59%) responded that they wished to participate in the study, 36 (19%) responded that they did not wish to participate in the study, 42 (22%) did not respond and 2 affirmative consent responses were received too late to be included in the assessments. Of these 110 positive consents received by the cut off date, 99 (90%) completed usable questionnaire packs were returned. The mean age of this total sample was 50.2 yrs (SD = 16; range = 19-78).

The break down of these 99 into their component gender and disease diagnoses groups is as follows (see figure 2.2.1):

<table>
<thead>
<tr>
<th></th>
<th>Crohn's Disease</th>
<th>Ulcerative Colitis</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td>15</td>
<td>24</td>
<td>39</td>
</tr>
<tr>
<td>Women</td>
<td>31</td>
<td>29</td>
<td>60</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>46</strong></td>
<td><strong>53</strong></td>
<td><strong>99</strong></td>
</tr>
</tbody>
</table>

In Crohn's Disease the ratio of men to women in this study is 1:2.07; the comparative figure for the sex ratio of men to women in Ulcerative Colitis is 1:1.21. This is similar to previous research showing a Europe-wide ratio of men to women for Crohn's Disease of 1:1.22 and Ulcerative Colitis of 1:0.81 (Shivananda et al, 1996). The predominance of women among inflammatory bowel disease sufferers is
greater for Crohn’s disease and may in some part be attributable to the greater longevity of women.

No response bias was detected between sexes or disease cohorts; although more women responded than men, and more Ulcerative Colitis patients responded than Crohn’s Disease patients, these differences were not statistically significant. No significant interaction effect between these two independent variables (gender and disease diagnosis) was noted for responses made.

2.3. Procedure.

Patients were sent an information sheet/informed consent form (see appendix 8), and were given approximately one month to respond to this. If responses, either positive or negative, were not received within this month a reminder letter was sent with a replacement informed consent form. If responses were still not received a month from this reminder letter being sent, then it was assumed that the patients did not wish, or were unable, to participate in the study. Similarly, on receipt of consent to participate in the study, subjects were sent a questionnaire pack. If this was not returned within a month a second reminder pack was sent. Analysis of the returned questionnaires began one month after these reminder questionnaires had been sent, thus any questionnaires received after this date were excluded from the analysis. A diagrammatical representation of this procedure can be seen in figure 2.3.1.
Figure 2.3.1. Representation of the procedure of this study.
2.4. Measures.

Subjects were assessed on a series of 8 measures:

a). Demographic details. (see appendix I)

Participants' gender, age, employment and marital status were assessed.

b). Brief disease history. (see appendix I)

Participants were asked to rate their current perceived disease severity on a scale of 1-10 of increasing severity. They were also asked to recall how long ago they had been diagnosed as having inflammatory bowel disease. Patients' medical records were accessed to extract information on medication levels and treatment regimes, and clinical indices (like white blood cell counts, ESR, and haemoglobin levels).

c). Health behaviours. (see appendix I)

Participants were asked whether or not they thought they had a healthy diet; and if they were a non-smoker, an ex-smoker (if so how long ago did they stop smoking), or a current smoker (if so how many they smoked per day), as it is reported that smoking is related in various ways to inflammatory bowel disease experience (Tobin et al, 1987) (see section 1.2.2.). They were also instructed to categorise themselves
into one of six categories of exercise ranging from ‘never’ exercising to taking exercise ‘at least once every day’.

d). Perceived stress / minor life events (see appendix 2)

The 10 item Perceived Stress Scale (PSS-10, Cohen et al, 1983) indexes perceptions of stress experienced over the last month. The scale has been validated in a random stratified sample (n=2387) of healthy ‘normal’ individuals, and internal consistency was satisfactory (Cronbach’s alpha = 0.75) (Cohen and Willamson, 1988). A two day test-retest reliability as assessed in college students was 0.85 (Cohen et al., 1983), although longer test-retest correlations are lower due to the short-term nature of minor stress/stressors. This scale has also been shown to possess reasonable predictive validity; in the same college student samples it correlated with indices of depressive symptomatology significantly higher than life event measures (0.65 vs. 0.18 respectively).

e). Inflammatory Bowel Disease Activity (see appendix 6)

To assess the disease activity, an index was used that is solely based on those data in The Crohn’s Disease Activity Index (CDAI - Best et al, 1976) which can be recorded directly from the patients (frequency of diarrhoea, abdominal pain, general well being). This index (called DA – for Disease Activity) (von Weitersheim et al, 1992) can be used for patients with Ulcerative Colitis as well as for patients with Crohn’s Disease. This index has been found to correlate highly with the full Crohn’s Disease
Activity Index – $r = 0.93$ (von Weitersheim et al, 1992), thus indicating that the modified index supplies very similar information.

f). Coping strategies/styles (see appendix 5)

Subjects were asked to complete the Coping with Health, Injuries and Problems (CHIP - Endler et al, 1992, 1993) questionnaire of coping strategies/styles. This multidimensional index, which was developed specifically for coping with health problems, is divided into four factors each with 8 items (Palliative, Instrumental, Distraction, and Negative Emotion). This measure has been validated in various populations including college student, adult, and medical patient groups. With particular relevance to the present study the medical patient population included 13.0% described as having severe chronic illnesses (diabetes, cancer, etc.), and 5.9% with gastrointestinal problems. Internal Alpha reliabilities for these three groups separately (students/adults/patients) for the four factors were satisfactory, ranging from 0.75 to 0.85. The CHIP also showed construct validity in correlations with other existing coping scales, e.g. with the Coping Inventory for Stressful Situations (CISS - Endler & Parker, 1990a) and the Coping Strategy Indicator (CSI - Amirkhan, 1990) (see Endler et al, 1992).

g). Trait personality. (see appendix 3)

Trait personality was assessed using the NEO-Five Factor Inventory (NEO-FFI - Costa & McCrae, 1992). This 60 item personality inventory indexes five relatively
stable and independent factors of trait personality each with 12 items: these are neuroticism; extraversion; openness; agreeableness; and conscientiousness. The reliability of these factors as measured by Cronbach’s alpha are good, ranging from .89 to .95 (Costa & McCrae, 1992). Various factors from this index (in particular the variable neuroticism) have also been shown to have significant predictive validity for stress outcomes – For example, Deary et al (1996) found that the personality trait of neuroticism was significantly related to levels of job stress and occupational burnout (Maslach Burnout Inventory) in a representative sample of 39 psychiatrists from 500 consultant doctors.

h). Locus of control (see appendix 4)

This was assessed using the Multidimensional Health Locus of Control Scale (MHLC) as developed by Wallston et al (1978). This 18 item scale indexes three dimensions of health locus of control: Internality (IHLC); Chance (CHLC); and Powerful Others (PHLC), which may be used separately or combined to identify different subgroups or ‘types’. For example, individuals scoring high on internal and low on powerful others loci of control may be described as a ‘type’. The MHLC has been validated in college students, chronic patients (e.g. Arthritis), healthy adults, and persons engaged in preventive health behaviours, in which the alpha reliabilities range from 0.67 to 0.77 for each of the subscales (Wallston, et al., 1978). The scale also has construct validity when compared and correlated with Levenson’s Multidimensional Locus of Control Scale (Levenson, 1973b), which is also based on three scales measuring beliefs in internality, powerful others, and other/externality.
2.5. Aims and Research Questions.

The present study had various aims and research questions to be addressed (also see introduction for justification of research aims and questions).

These were as follows:

Research questions.

1. What are the characteristics of the two inflammatory bowel disease diagnostic groups, Crohn’s Disease and Ulcerative Colitis, in terms of demographic, behavioural or psychological factors, or experiences of disease activity? What are the similarities and differences between these two disease cohorts for these factors?

2. Are there any gender differences in these disease populations in terms of demographic, behavioural or psychological factors, or experiences of disease activity?

3. Are there any interaction effects between disease cohort and gender in terms of demographic, behavioural or psychological factors, or experiences of disease activity?

4. Is there a link between perceived stress and illness experience in the inflammatory bowel diseases Ulcerative Colitis and Crohn's disease?

5. What are the relationships between potential predictors of stress outcomes, other psychological/behavioural/medical independent variables, and the outcome
variables? Do they support a transactional, a latent variable, a combination of the
two, or another, model of the stress and illness relationship?

6. What factors (demographic, behavioural, psychological, clinical) are useful in
predicting disease activity? What roles do these factors play in predicting disease
outcome?

Aims

1. To investigate the relationship between perceived stress and symptom
   experience in subjects with Crohn's Disease and Ulcerative Colitis.

2. To explore the potential influences of personality, locus of control, coping
   behaviour, diet, exercise, and smoking, – in isolation or in interaction – on the
   stress symptom relationship.

3. To assess the ‘fit’ of findings within alternative models of the stress-illness
   relationship.

All statistical analyses were carried out on a Macintosh® Powerbook 165 personal
computer utilising SPSS.X® and Statview® software. For some participants small
amounts of data were missing from the whole set of questionnaires, thus the number
of subjects ranged from 90 to 99. Sample sizes are indicated in the results section
where missing data was obtained. Similarly this sparse missing data did not appear to
come from particular questionnaires, or particular questions on a particular
questionnaire, and did appear to be randomly distributed between the questionnaires and between the items within the questionnaires.

Research questions 1 and 2 (section 2.5.) were addressed by performing paired t-tests between either the two disease groups or the two sexes. Some variables did not suit this form of analysis (they were either discontinuous, or categorical), and were therefore compared across disease cohort or gender appropriately (by Mann-Whitney test or by Chi Squared for discontinuous or categorical data respectively). The question of interaction effects (research question 3) was assessed by performing a two-way analysis of variance between disease group and gender, for the variables for which this could be performed (see above). Correlational evidence was gathered to assess research questions 4 and 5. Although this does not, of course, address direction of causality, further analyses may help to clarify this issue. To assess research question 6, a series of univariate and hierarchical multivariate regression equations were employed.
CHAPTER 3: Results.

3.1. Disease group and gender differences.

3.1.1. Group characteristics and health behaviours.

3.1.1.1. Crohn’s Disease and Ulcerative Colitis.

3.1.1.2. Males and females.

3.1.2. Perceived stress, disease activity, coping, locus of control and personality.

3.1.2.1. Crohn’s Disease and Ulcerative Colitis.

3.1.2.2. Males and females.

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3.4. Perceived stress and Locus of Control.

3.5. Personality and disease activity.

3.6. Personality and coping.

3.7. Personality and locus of control.

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3.10.1. Univariate regressions.


3.10.2.1. Predicting overall disease activity.

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3.10.3.1. Predicting overall disease activity.

3.10.3.2. Predicting abdominal pain.

3.10.3.3. Predicting experiences of well being.

3.10.3.4. Latent variable vs. transaction model of stress and illness.
3. Results.

3.1. Disease group, and gender characteristics and health behaviours.
Overall between group differences, i.e. Crohn's Disease vs. Ulcerative Colitis, and male vs. female, for all the variables, are presented in tables 3.1. – 3.6. These results will be considered throughout this section.

<table>
<thead>
<tr>
<th>Description of difference</th>
<th>D.F.</th>
<th>Total Chi Squared</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1). No differences between CD and UC on employment status</td>
<td>5</td>
<td>7.9</td>
<td>.16</td>
</tr>
<tr>
<td>2). No differences between CD and UC on marital status</td>
<td>3</td>
<td>1.4</td>
<td>.69</td>
</tr>
<tr>
<td>3). No differences between CD and UC on exercise status</td>
<td>4</td>
<td>4.3</td>
<td>.37</td>
</tr>
<tr>
<td>4). CD patients were more likely to be smokers than UC patients</td>
<td>2</td>
<td>6.6</td>
<td>.0376</td>
</tr>
<tr>
<td>5). No differences between CD and UC on perceived balance of Diet</td>
<td>1</td>
<td>1.6</td>
<td>.21</td>
</tr>
</tbody>
</table>

\( n = 97 \); bolded denotes significant results

<table>
<thead>
<tr>
<th>Variable</th>
<th>Crohn's Disease ((n=46))</th>
<th>Ulcerative Colitis ((n=53))</th>
<th>Z-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abdominal Pain ((0-3))</td>
<td>51.3</td>
<td>47.0</td>
<td>-0.80</td>
<td>.43</td>
</tr>
<tr>
<td>Well Being ((0-5))</td>
<td>54.9</td>
<td>43.9</td>
<td>-2.11</td>
<td>.0352</td>
</tr>
</tbody>
</table>

\( n = 99 \); bolded denotes significant results
Table 3.1.3. Means (SD) and the results of two-tailed Students t-tests for the main variables between Crohn's disease and Ulcerative Colitis patients.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Crohn's Disease (n=46)</th>
<th>Ulcerative Colitis (n=53)</th>
<th>t-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>50.3 (16.6)</td>
<td>50.1 (15.6)</td>
<td>0.05</td>
<td>.96</td>
</tr>
<tr>
<td>Disease Length</td>
<td>11.8 (11.8)</td>
<td>11.0 (10.4)</td>
<td>0.34</td>
<td>.73</td>
</tr>
<tr>
<td>Perceived Disease Severity</td>
<td>5.0 (2.5)</td>
<td>4.0 (2.6)</td>
<td>1.90</td>
<td>.06</td>
</tr>
<tr>
<td>IHLC</td>
<td>21.8 (5.3)</td>
<td>22.4 (5.5)</td>
<td>-0.57</td>
<td>.57</td>
</tr>
<tr>
<td>CHLC</td>
<td>19.1 (4.9)</td>
<td>18.9 (5.5)</td>
<td>0.22</td>
<td>.83</td>
</tr>
<tr>
<td>PHLC</td>
<td>20.5 (5.3)</td>
<td>18.9 (6.2)</td>
<td>1.30</td>
<td>.18</td>
</tr>
<tr>
<td>Perceived Stress</td>
<td>15.5 (7.3)</td>
<td>14.8 (8.1)</td>
<td>0.46</td>
<td>.65</td>
</tr>
<tr>
<td>Palliative Coping</td>
<td>23.7 (5.3)</td>
<td>23.3 (4.4)</td>
<td>0.41</td>
<td>.69</td>
</tr>
<tr>
<td>Instrumental Coping</td>
<td>31.5 (4.8)</td>
<td>30.8 (5.6)</td>
<td>0.65</td>
<td>.51</td>
</tr>
<tr>
<td>Distraction Coping</td>
<td>23.8 (4.7)</td>
<td>23.8 (5.1)</td>
<td>0.00</td>
<td>.99</td>
</tr>
<tr>
<td>Negative Emotion Coping</td>
<td>10.9 (2.8)</td>
<td>11.5 (4.2)</td>
<td>-0.74</td>
<td>.46</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>22.3 (8.9)</td>
<td>22.1 (9.0)</td>
<td>0.15</td>
<td>.88</td>
</tr>
<tr>
<td>Extraversion</td>
<td>25.6 (4.8)</td>
<td>25.1 (5.6)</td>
<td>0.48</td>
<td>.63</td>
</tr>
<tr>
<td>Openness</td>
<td>24.6 (6.4)</td>
<td>25.5 (5.1)</td>
<td>-0.79</td>
<td>.43</td>
</tr>
<tr>
<td>Agreeableness</td>
<td>32.0 (5.1)</td>
<td>31.4 (4.7)</td>
<td>0.67</td>
<td>.51</td>
</tr>
<tr>
<td>Conscientiousness</td>
<td>33.4 (6.1)</td>
<td>32.6 (5.4)</td>
<td>0.73</td>
<td>.47</td>
</tr>
<tr>
<td>Overall disease activity</td>
<td>34.4 (36.5)</td>
<td>27.9 (28.6)</td>
<td>0.97</td>
<td>.33</td>
</tr>
<tr>
<td>No. of soft stools last week.</td>
<td>11.3 (15.0)</td>
<td>10.2 (13.0)</td>
<td>0.37</td>
<td>.71</td>
</tr>
</tbody>
</table>

n = 99, bolded denotes significant results
### Table 3.1.4. Results of the Chi-squared tests for disease gender differences for the categorical variables.

<table>
<thead>
<tr>
<th>Description of difference</th>
<th>D.F.</th>
<th>Total Chi Squared</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1). Men are more likely to be in full time employment, and less likely to be retired or unemployed than women.</td>
<td>5</td>
<td>15.3</td>
<td>.0092</td>
</tr>
<tr>
<td>2). No differences between men and women on marital status</td>
<td>3</td>
<td>7.6</td>
<td>.05</td>
</tr>
<tr>
<td>3). Men were more likely to take exercise more regularly than women</td>
<td>4</td>
<td>12.6</td>
<td>.0135</td>
</tr>
<tr>
<td>4). No differences between men and women on smoking status</td>
<td>2</td>
<td>0.4</td>
<td>.82</td>
</tr>
<tr>
<td>5). No differences between men and women on Diet</td>
<td>1</td>
<td>0.7</td>
<td>.41</td>
</tr>
<tr>
<td>6). No differences between men and women on disease diagnosis</td>
<td>1</td>
<td>1.3</td>
<td>.26</td>
</tr>
</tbody>
</table>

n = 98; bolded denotes significant results

### Table 3.1.5. Mean Ranks and the results of Mann-Whitney tests for the non-parametric variables between male and female patients.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Male (n=39)</th>
<th>Female (n=60)</th>
<th>Mean Rank</th>
<th>Mean Rank</th>
<th>Z-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abdominal Pain (0-3)</td>
<td>40.0</td>
<td>54.8</td>
<td>-2.68</td>
<td>.0074</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Well Being (0-5)</td>
<td>40.8</td>
<td>54.3</td>
<td>-2.56</td>
<td>.0105</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

n = 99; bolded denotes significant results
Table 3.1.6. Means (SD) and the results of two-tailed Students t-tests for the main variables between male and female patients.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Male (n=39)</th>
<th>Female (n=60)</th>
<th>t-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>49.6 (13.0)</td>
<td>50.6 (17.8)</td>
<td>-0.32</td>
<td>.75</td>
</tr>
<tr>
<td>Disease Length</td>
<td>11.2 (8.4)</td>
<td>11.5 (12.5)</td>
<td>-0.10</td>
<td>.93</td>
</tr>
<tr>
<td>Perceived Disease Severity</td>
<td>4.2 (3.0)</td>
<td>4.6 (2.3)</td>
<td>-0.78</td>
<td>.44</td>
</tr>
<tr>
<td>IHLC</td>
<td>22.0 (4.7)</td>
<td>22.2 (5.8)</td>
<td>-0.21</td>
<td>.83</td>
</tr>
<tr>
<td>CHLC</td>
<td>18.6 (4.6)</td>
<td>19.3 (5.6)</td>
<td>-0.58</td>
<td>.56</td>
</tr>
<tr>
<td>PHLC</td>
<td>18.4 (4.9)</td>
<td>20.4 (6.3)</td>
<td>-1.64</td>
<td>.10</td>
</tr>
<tr>
<td>Perceived Stress</td>
<td>12.5 (7.6)</td>
<td>16.9 (7.2)</td>
<td>-2.92</td>
<td>.004</td>
</tr>
<tr>
<td>Palliative Coping</td>
<td>21.2 (4.7)</td>
<td>23.8 (4.9)</td>
<td>-0.60</td>
<td>.55</td>
</tr>
<tr>
<td>Instrumental Coping</td>
<td>31.5 (5.8)</td>
<td>30.9 (5.0)</td>
<td>-0.51</td>
<td>.61</td>
</tr>
<tr>
<td>Distraction Coping</td>
<td>22.6 (4.9)</td>
<td>24.6 (4.8)</td>
<td>-2.01</td>
<td>.048</td>
</tr>
<tr>
<td>Negative Emotion Coping</td>
<td>10.5 (3.3)</td>
<td>11.7 (3.7)</td>
<td>-1.62</td>
<td>.11</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>18.2 (7.8)</td>
<td>24.9 (8.5)</td>
<td>-3.94</td>
<td>.0002</td>
</tr>
<tr>
<td>Extraversion</td>
<td>25.7 (5.8)</td>
<td>25.1 (4.8)</td>
<td>0.49</td>
<td>.62</td>
</tr>
<tr>
<td>Openness</td>
<td>25.0 (4.4)</td>
<td>25.1 (6.5)</td>
<td>-0.10</td>
<td>.92</td>
</tr>
<tr>
<td>Agreeableness</td>
<td>31.1 (4.9)</td>
<td>32.1 (4.9)</td>
<td>-0.95</td>
<td>.34</td>
</tr>
<tr>
<td>Conscientiousness</td>
<td>33.2 (5.6)</td>
<td>32.8 (5.8)</td>
<td>0.29</td>
<td>.77</td>
</tr>
<tr>
<td>Overall disease activity</td>
<td>21.9 (26.4)</td>
<td>36.7 (34.9)</td>
<td>-2.21</td>
<td>.0295</td>
</tr>
<tr>
<td>No. of soft stools last week.</td>
<td>7.9 (12.1)</td>
<td>12.5 (14.7)</td>
<td>1.61</td>
<td>.11</td>
</tr>
</tbody>
</table>

\( n = 99 \); bolded denotes significant results
3.1.1. Group Characteristics and Health Behaviours.

3.1.1.1. Crohn’s Disease and Ulcerative Colitis.

Neither disease group differed significantly from the mean age of the total sample (50.2 yrs, SD = 16; range = 19-78), nor was the difference between the disease groups statistically significant (50.3 yrs, SD = 16.6, range = 19-78; 50.1 yrs, SD = 15.6, range = 22-75, for the Crohn’s disease and Ulcerative colitis patients respectively). No significant differences were noted between the disease groups on any of the demographic groupings (like employment status). In terms of health behaviours (like exercise taken) the only significant difference was in smoking, whereby Crohn’s Disease patients were significantly more likely to be smokers than Ulcerative Colitis patients ($\chi^2 = 6.6; df = 1; p < .05$) – see table 3.1.1. Thus because of the almost negligible differences between these two disease cohorts, they will be considered together when addressing whether any gender differences exist.

3.1.1.2. Males and Females.

The mean age of the total sample was not significantly different from that of either men or women, nor were there any significant differences between gender in age (49.6 yrs, SD = 13.0, range = 20-69; 50.6 yrs, SD = 17.8, range = 19-78, for the male and female patients respectively). Independent of disease grouping, several gender differences did exist, however, on the demographic groupings, whereby men were more likely to be in full-time employment, and less likely to be retired or unemployed than women ($\chi^2 = 15.3; df = 5; p < .01$) – see table 3.1.4. Men were also more likely to take exercise more regularly than women ($\chi^2 = 12.6; df = 4; p <$

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.01) – see table 3.1.4. There were, however, no differences between men and women on smoking, diet, marital status or diagnosis of disease.

3.1.2. Perceived stress, disease activity, coping, locus of control, and personality.

3.1.2.1. Crohn’s Disease and Ulcerative Colitis.

No significant differences were noted between the two disease cohorts on perceived stress, overall disease activity, coping styles, locus of control or personality (see table 3.1.3.). However, the Crohn’s Disease patients expressed significantly higher (i.e. worse) scores on the Well being sub-scale of the disease activity index ($Z = -2.11; p < .05$ – see table 3.1.2.). i.e. because the Well being score is scored in the direction that higher scores mean lower well being, Crohn’s Disease patients are expressing lower well being. (Sub-components of the Disease activity index and their relation to these variables can be seen in tables 3.1.2. & 3.1.3.)

3.1.2.2. Males and Females.

Women scored significantly higher than men on: perceived stress ($t = -2.92; p < .01$ - 2-tailed); overall disease activity ($t = -2.21; p < .05$ – 2-tailed); abdominal pain rating from the Disease Activity Index ($Z = -2.68; p < .01$); higher on the (negative) Well being rating from the Disease Activity Index ($Z = -2.56; p < .05$), again meaning that women are expressing lower well being; and neuroticism ($t = -3.94; p < .0005$). (Sub-components of the Disease activity index and their relation to these variables can be seen in tables 3.1.5. & 3.1.6.)
3.2. Perceived stress and disease activity.
Perceived stress was related to disease activity and perceived disease severity. In the total patient group, significant Spearman correlations were found between perceived stress scores and abdominal pain rating and (negative) Well being rating of .39 (p<.05) and .42 (p<.005) respectively (thus as perceived stress increases experiences of pain increase and well being decrease). The correlation between perceived stress and perceived disease severity was .39 (p<.05). The number of soft/liquid stools factor from the Disease Activity Index was not significantly related to perceived stress, either when the disease cohorts were considered together or separately. Perceived stress was also correlated with abdominal pain and well being ratings from the Disease Activity Index in the two separate disease groups.
Perceived stress and abdominal pain ratings were correlated at .33 (p<.05) and .44 (p<.005) for Crohn’s disease and Ulcerative Colitis respectively. Perceived stress and Well being ratings were correlated at .48 (p<.005) and .34 (p<.05) for Crohn’s disease and Ulcerative Colitis respectively (similarly, as perceived stress increases experiences of well being decrease). Perceived stress and perceived disease severity were correlated significantly at .37 (p<.05) in the Ulcerative colitis patients only. These correlations can be seen in table 3.2.1.
Table 3.2.1. Spearman correlations between perceived stress and disease activity/perceived disease severity.

<table>
<thead>
<tr>
<th>Perceived Stress (PSS-10)</th>
<th>Disease Activity Index - Abdominal Pain</th>
<th>Disease Activity Index - Well Being</th>
<th>Perceived Disease Severity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crohn's Disease</td>
<td>.33*</td>
<td>-.48**</td>
<td>-.17**</td>
</tr>
<tr>
<td>Ulcerative Colitis</td>
<td>.44**</td>
<td>-.34*</td>
<td>.37*</td>
</tr>
<tr>
<td>Together</td>
<td>.39*</td>
<td>-.42**</td>
<td>.39*</td>
</tr>
</tbody>
</table>

* p<0.05; ** p<0.005, No. of soft/liquid stools factor from Disease Activity Index was not significantly related to perceived stress, n ≥ 94.

3.3. Perceived stress and coping.
Perceived stress and negative emotion coping were positively correlated for the total group at .38 (p<.01) [this was significant for ulcerative colitis patients .53 (p<.01) but not for the Crohn's disease patient group]. Perceived stress was not significantly correlated with the other factors of the CHIP when the disease cohorts were considered together, nor when they were considered separately.

3.4. Perceived stress and Locus of Control.
The results of the relationships between perceived stress and factors of Locus of control can be seen in table 3.4.1. Perceived stress and internal health locus of control were significantly correlated in the negative direction at -.23 (p < .05), when the disease cohorts were considered together. Thus as perceived stress increases, the belief that one's health is in the control of oneself decreases. Clearly as this is correlational evidence, interpretation of causal direction is problematic. When the
disease cohorts are considered individually, however, the picture changes somewhat. In Crohn’s Disease patients perceived stress is not significantly correlated with any of the factors of the locus of control measure (internal, chance, or powerful others). In Ulcerative Colitis patients, however, perceived stress is significantly negatively correlated with internal health locus of control (as was seen for the total sample) at \(-.32\) (\(p < .05\)). In Ulcerative Colitis patients, perceived stress is also significantly positively correlated with powerful others locus of control at \(.27\) (\(p < .05\)). Thus in Ulcerative Colitis patients as perceived stress increases, the belief that your health is in your own control decreases and the belief that your health is in the control of powerful others increases (again no causal direction is implied).

<table>
<thead>
<tr>
<th></th>
<th>IHLC</th>
<th>CHCL</th>
<th>PHLC</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Perceived Stress (PSS-10)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Crohn’s Disease</td>
<td>-.10</td>
<td>.03</td>
<td>-.10</td>
</tr>
<tr>
<td>Ulcerative Colitis</td>
<td>-.32*</td>
<td>.09</td>
<td>.27*</td>
</tr>
<tr>
<td>Together</td>
<td>-.23*</td>
<td>.07</td>
<td>.13</td>
</tr>
</tbody>
</table>

\* = \(p<.05\), ** = \(p<.005\); No. of soft/liquid stools factor from Disease Activity Index was not significantly related to perceived stress; \(n=94\).

3.5. Personality and Disease Activity.

When the total sample was assessed (see table 3.5.1.), trait neuroticism was significantly correlated with experiences of abdominal pain (\(r = .33; p < .01\)) and (negative) Well being (\(r = .34; p < .01\)), but not with overall disease activity or number of soft stools. Thus individuals who expressed higher levels of neuroticism also expressed higher levels of abdominal pain and lower levels of well being.
Extroversion was also significantly correlated with levels of abdominal pain ($r = -0.21; p < 0.05$) and (negative) Well being ($r = -0.27; p < 0.05$), but in the negative direction. Thus individuals expressing higher levels of extroversion are expressing lower levels of abdominal pain and higher levels of well being. There is a concern that this may be due to the factors of the NEO-FFI not being totally independent and exhibiting internal correlations (see below). Openness significantly correlated with overall disease activity ($r = 0.29; p < 0.05$) and number of soft stools ($r = 0.25; p < 0.05$), both in the positive direction, but not with abdominal pain or well being. Agreeableness did not correlate significantly with any of the disease activity scores. Conscientiousness correlated significantly with (negative) Well being ratings ($r = -0.21; p < 0.05$), in the negative direction. Thus as conscientiousness increases so does well being (and negative Well being scores decrease). Again this correlation may be due to intercorrelations between the factors of the NEO-FFI (see below).

Table 3.5.1. Pearson correlations between personality and disease activity for the total sample.

<table>
<thead>
<tr>
<th>Overall Disease Activity</th>
<th>Number of Soft Stools</th>
<th>Abdominal Pain</th>
<th>Well Being (negative)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>0.10</td>
<td>0.00</td>
<td>0.33**</td>
</tr>
<tr>
<td>E</td>
<td>-0.20</td>
<td>-0.15</td>
<td>-0.21*</td>
</tr>
<tr>
<td>O</td>
<td>0.29*</td>
<td>0.25*</td>
<td>0.09</td>
</tr>
<tr>
<td>A</td>
<td>-0.02</td>
<td>-0.01</td>
<td>-0.11</td>
</tr>
<tr>
<td>C</td>
<td>0.01</td>
<td>0.08</td>
<td>-0.17</td>
</tr>
</tbody>
</table>

* = $p<0.05$, ** = $p<0.01$; N = neuroticism; E = extroversion; O = openness; A = agreeableness; C = conscientiousness; $n = 94$.

In Crohn’s disease patients, personality was significantly related to the (negative) Well being ratings but not to overall disease activity, abdominal pain, or number of soft stools. There was also a significant positive correlation between trait openness...
and overall disease activity ($r = .37; p < .05$). Neuroticism, extroversion, and conscientiousness scores were correlated with (negative) Well being scores ($r = .31, p < .05; r = -.40, p < .01; \text{and } r = -.34, p < .05$ respectively). Once more some of these correlations may be spurious due to inter-correlations of the NEO-FFI (see below).

In Ulcerative Colitis patients, personality was not related to overall disease activity, or number of soft stools. Trait neuroticism was the only personality factor to correlate with any of the disease activity sub-scales. It significantly correlated positively with both abdominal pain ($r = .46, p < .01$) and (negative) Well being ($r = .38; p < .05$) ratings. Thus as neuroticism increases, abdominal pain experiences decrease, and well being decreases.

It must be noted, however, that neuroticism itself was significantly correlated with extroversion at -.40 ($p<.01$) [-.43 ($p<.01$); -.36 ($p<.01$) for Crohn’s disease and ulcerative colitis patients respectively] and conscientiousness at -.47 ($p<.01$) [-.50 ($p<.01$); -.44 ($p<.01$) for Crohn’s disease and ulcerative colitis patients respectively]. Similarly extroversion and conscientiousness measures were also significantly correlated at $.35 (p<.05)$ [.41 ($p<.01$); .29 ($p<.05$) for Crohn’s disease and ulcerative colitis patients respectively]. This means that they share variance, which may make interpretations of these findings difficult (see discussion – section 4.5.).
3.6. Personality and coping.
Trait neuroticism was significantly correlated with negative emotion coping at .54 (p<.01) [.56 (p<.01); .53 (p<.01) for Crohn's disease and ulcerative colitis patients respectively]. Thus as neuroticism scores increase so do negative emotion coping scores. In the Ulcerative colitis patients extroversion was significantly positively correlated with distraction coping at .29 (p<.05) – i.e. high scoring extroverts are more likely to use distraction to cope. In the same disease cohort agreeableness was significantly negatively correlated with palliative coping at -.29 (p<.05) – as agreeableness increases levels of palliative coping decrease - and conscientiousness was significantly positively correlated with instrumental coping at .34 (p<.05).

In the Crohn's disease patient cohort, agreeableness and negative emotion coping were significantly correlated at -.46 (p<.01) in the negative direction – meaning that as scores on agreeableness increase scores on negative emotion coping decrease, and openness and distraction coping were significantly correlated positively at .29 (p<.05).

3.7. Personality and Locus of Control.
Several significant correlations were noted between trait personality and health locus of control for the total sample (see table 3.7.1.). Trait neuroticism was significantly correlated with Chance locus of control at .27 (p<.01). Openness was also correlated with chance locus of control at -.23 (p<.05) but in the negative direction. Powerful others locus of control was significantly positively correlated with trait neuroticism at .20 (p<.05) and negatively with openness at -.40 (p<.01).
In the Crohn's disease sample (see table 3.7.2) trait neuroticism, agreeableness, and conscientiousness all correlated significantly with Chance health locus of control ($r = .29, p < .05; r = -.29, p < .05; and r = -.29, p < .05$ respectively). Please note while the neuroticism-chance health locus of control was in the positive direction, the agreeableness and conscientiousness correlations with chance health locus of control were in the negative direction. Trait openness was significantly negatively correlated with powerful others locus of control at -.48 ($p<.01$).

In the Ulcerative colitis sample (also see table 3.7.2) trait neuroticism was significantly positively correlated with Chance locus of control at .27 ($p<.05$) and powerful others locus of control at .33 ($p<.05$). Extroversion was significantly positively correlated with internal locus of control at .33 ($p<.05$). And openness was
significantly negatively correlated with powerful others locus of control at -.33 (p < .05).

Table 3.7.2. Pearson correlations between personality and health locus of control for the Crohn's disease and ulcerative colitis populations considered separately (the correlations for Crohn's disease patients are before the slash and for ulcerative colitis patients after the slash).

<table>
<thead>
<tr>
<th></th>
<th>IHLC</th>
<th>CHLC</th>
<th>PHLC</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>-.08 / -.24</td>
<td>.29* / .27*</td>
<td>.01 / .33*</td>
</tr>
<tr>
<td>E</td>
<td>-.04 / .33*</td>
<td>-.02 / -.09</td>
<td>-.05 / -.14</td>
</tr>
<tr>
<td>O</td>
<td>-.01 / -.19</td>
<td>-.09 / -.34*</td>
<td>-.48** / -.33*</td>
</tr>
<tr>
<td>A</td>
<td>.02 / .09</td>
<td>-.29* / .05</td>
<td>-.11 / -.06</td>
</tr>
<tr>
<td>C</td>
<td>.19 / .01</td>
<td>-.29* / .04</td>
<td>.03 / -.01</td>
</tr>
</tbody>
</table>

* = p < .05; ** = p < .01; N = neuroticism; E = extroversion; O = openness; A = agreeableness; C = conscientiousness; IHLC = internal health locus of control; CHLC = chance health locus of control; PHLC = powerful others health locus of control; n > 94.

3.8. Perceived stress and personality.

Various significant correlations were noted between perceived stress and trait personality (see table 3.8.1). Perceived stress and trait neuroticism were positively correlated at .74 (p<.01) [.69 (p<.01); .79 (p<.01) for Crohn's disease and ulcerative colitis patients respectively]. Perceived stress and extroversion were negatively correlated at -.40 (p<.01) [-.43 (p<.01); -.39 (p<.01) for Crohn's disease and ulcerative colitis patients respectively]. Perceived stress and conscientiousness were correlated negatively at -.42 (p<.01) [-.48 (p<.01); -.37 (p<.01) for Crohn's disease and ulcerative colitis patients respectively]. In Crohn's Disease patients only, perceived stress was also significantly positively correlated with trait openness at .31 (p < .05).
As is mentioned above neuroticism is correlated with extroversion and conscientiousness, i.e. these factors are not orthogonal, and some of the correlations between perceived stress and extroversion and conscientiousness may well be due to the variance that these variables share with neuroticism.

Table 3.8.1. Pearson correlations between perceived stress and personality.

<table>
<thead>
<tr>
<th>NEO-FFI</th>
<th>Perceived stress</th>
<th>Perceived stress</th>
<th>Perceived stress</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PSS-10 (both disease groups)</td>
<td>PSS-10 (Crohn's disease)</td>
<td>PSS-10 (Ulcerative Colitis)</td>
</tr>
<tr>
<td>N</td>
<td>.74**</td>
<td>.69**</td>
<td>.79**</td>
</tr>
<tr>
<td>E</td>
<td>-.40**</td>
<td>-.43**</td>
<td>-.39**</td>
</tr>
<tr>
<td>O</td>
<td>.15</td>
<td>.31*</td>
<td>.02</td>
</tr>
<tr>
<td>A</td>
<td>-.13</td>
<td>-.06</td>
<td>-.20</td>
</tr>
<tr>
<td>C</td>
<td>-.42**</td>
<td>-.48**</td>
<td>-.37**</td>
</tr>
</tbody>
</table>

* = p < .05; ** = p < .01; N = neuroticism; E = extroversion; O = openness; A = agreeableness; C = conscientiousness; n > 92.


The results from a two-way analysis of variance also showed no significant interaction effects between gender and disease cohort on any of the dependent variables mentioned above. For example, female Crohn’s disease patients did not show significantly different scores from male ulcerative colitis patients.

Crohn’s disease patients were significantly more likely to be smokers than Ulcerative Colitis patients (p < .05), as mentioned above, and smokers reported significantly
higher abdominal pain ratings ($p<.05$). However, there was only a non-significant tendency for Crohn's disease patients to report higher levels of abdominal pain than Ulcerative colitis patients.

3.10. Predictors of Disease Activity.
The prerequisites for an independent variable to be chosen to be entered into the regression equations, being used to try to predict the dependent variables of disease activity were as follows. The independent variables entered had to correlate significantly (statistically and in terms of effect size) with the dependent variable being used for that particular regression equation; or were theory led. [N.B. Due to the large numbers of variables measured in this research several statistically significant correlations were expected by chance. Thus statistically significant correlations that were not associated with the models of stress and illness being examined were not included in the regressions. For example – some of the correlations found for extroversion were not included in the regression equations as they may well be statistical artefacts.]

3.10.1. Univariate regressions.
[see table 3.10.1] The overall disease activity index was not singularly predicted significantly by levels of neuroticism or by levels of perceived stress. When the outcome variable was separated into its component factors of i) number of soft stools, ii) Abdominal pain and iii) Well being, several significant predictions could be observed. Similar to the overall disease activity index, number of soft stools was not
predicted by either levels of trait neuroticism or of perceived stress. However, abdominal pain and well being were both significantly predicted by levels of neuroticism and by levels of perceived stress. 14% and 09% of the variance in abdominal pain was predicted by the variables of neuroticism and perceived stress respectively. Similarly 14% and 09% of the variance in well being was predicted by the variables of neuroticism and perceived stress respectively.

Table 3.10.1. Individual regressions of perceived stress and neuroticism on the outcome variables.

<table>
<thead>
<tr>
<th>Independent Variable</th>
<th>Dependent Variable</th>
<th>R²</th>
<th>R² Change</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>PSS-10</td>
<td>Disease Activity Index</td>
<td>.03</td>
<td>-</td>
<td>.07</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>Disease Activity Index</td>
<td>.01</td>
<td>-</td>
<td>.33</td>
</tr>
<tr>
<td>PSS-10</td>
<td>No.Soft Stools</td>
<td>.00</td>
<td>-</td>
<td>.51</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>No.Soft Stools</td>
<td>.00</td>
<td>-</td>
<td>.99</td>
</tr>
<tr>
<td>PSS-10</td>
<td>Abdominal Pain</td>
<td>.14</td>
<td>-</td>
<td>.0001</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>Abdominal Pain</td>
<td>.09</td>
<td>-</td>
<td>.0027</td>
</tr>
<tr>
<td>PSS-10</td>
<td>Well Being</td>
<td>.14</td>
<td>-</td>
<td>.0001</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>Well Being</td>
<td>.09</td>
<td>-</td>
<td>.0025</td>
</tr>
</tbody>
</table>

Bolded denotes significant results, n > 90.


The main aims of the regression analyses were to differentiate between the transactional and the latent variable models of stress and illness, and to identify those variables that are most predictive of outcome. Trying to find variables that predict the outcomes will have obvious interest when trying to find applications for the research. For example, if it is found that dysfunctional coping mechanisms are associated with negative disease experiences, like pain, then in order to reduce pain, more adaptive functional coping strategies could be taught.
Thus variables were chosen that correlate with the outcome variables, or were thought to be associated with either the transactional or latent variable models of stress and illness. Therefore most of the analyses were comparing two regression equations, these were:

1). Trying to predict outcome using the variables age, disease length, 
   **perceived stress**, perceived disease severity, negative emotion focused 
   coping, internal, powerful others and chance health loci of control.

2). Trying to predict outcome using the variables age, disease length, trait 
   **neuroticism**, perceived disease severity, negative emotion focused 
   coping, internal, powerful others and chance health loci of control.

The interesting difference will be to compare these two regression equations. This should help us establish whether the results support a transactional or latent variable model of stress and illness in this disease population. Thus if the actual situation is best modelled by a latent variable theory, one would expect to see the vast majority of variance explained by neuroticism alone (given that it closely relates to the conceptual idea of negative affectivity). This would mean that relatively little or no additional variance would be explained by the other variables. If a transactional model more accurately modelled the real situation, then one would expect to find a lot of the variables adding their own proportion of the total variance explained. One would expect the variables that did this to be those associated with this model of stress and illness, like perceived stress, loci of control, and coping mechanisms.
Similar to the individual regressions seen in section 3.10.1 above, the most explained variance was obtained for the more subjective indices of disease activity experience (namely abdominal pain and well being) as opposed to the more objective measure of number of soft stools in the last week. Neuroticism and perceived stress were not entered into the same regression equations due to their obvious covariance.

The results of the regression equations can be seen in table 3.10.2.1.
### Table 3.10.2.1 Hierarchical multiple regression results.

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Independent Variable</th>
<th>$R^2$</th>
<th>Change $R^2$</th>
<th>Beta</th>
<th>Sig. In total equation</th>
<th>Overall Sig. (F.)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Disease Activity</strong></td>
<td><strong>Age</strong></td>
<td>.00</td>
<td>.00</td>
<td>.137</td>
<td>.33</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td><strong>Disease length</strong></td>
<td>.03</td>
<td>.03</td>
<td>.007</td>
<td>.96</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td><strong>Perceived Stress</strong></td>
<td>.05</td>
<td>.02</td>
<td>.130</td>
<td>.30</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td><strong>Perceived Severity</strong></td>
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<td>.342</td>
<td>.0066</td>
<td>-</td>
</tr>
<tr>
<td></td>
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<td>.00</td>
<td>-.023</td>
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<td>-</td>
</tr>
<tr>
<td></td>
<td><strong>IHLC</strong></td>
<td>.17</td>
<td>.01</td>
<td>-.115</td>
<td>.32</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td><strong>PHLC</strong></td>
<td>.18</td>
<td>.01</td>
<td>-.125</td>
<td>.33</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td><strong>CHLC</strong></td>
<td>.18</td>
<td>.00</td>
<td>.017</td>
<td>.90</td>
<td>.06</td>
</tr>
<tr>
<td><strong>No. Soft Stools</strong></td>
<td><strong>Age</strong></td>
<td>.01</td>
<td>.01</td>
<td>.140</td>
<td>.34</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td><strong>Disease length</strong></td>
<td>.03</td>
<td>.02</td>
<td>.025</td>
<td>.85</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td><strong>Perceived Stress</strong></td>
<td>.03</td>
<td>.00</td>
<td>.028</td>
<td>.83</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td><strong>Perceived Severity</strong></td>
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<td>.08</td>
<td>.281</td>
<td>.0293</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td><strong>Negative Emotion</strong></td>
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<td>.00</td>
<td>.005</td>
<td>.97</td>
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</tr>
<tr>
<td></td>
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<td>.00</td>
<td>-.097</td>
<td>.47</td>
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</tr>
<tr>
<td></td>
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</tr>
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<td><strong>Abdominal Pain</strong></td>
<td><strong>Age</strong></td>
<td>.04</td>
<td>.04</td>
<td>-.139</td>
<td>.30</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td><strong>Disease length</strong></td>
<td>.06</td>
<td>.02</td>
<td>.044</td>
<td>.72</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td><strong>Perceived Stress</strong></td>
<td>.14</td>
<td>.08</td>
<td>.238</td>
<td>.0489</td>
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<td></td>
<td><strong>Perceived Severity</strong></td>
<td>.22</td>
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<td>.279</td>
<td>.0200</td>
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<tr>
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<td>-.072</td>
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</tr>
<tr>
<td></td>
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<td>.24</td>
<td>.02</td>
<td>-.147</td>
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<td>-</td>
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<td>.012</td>
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<td><strong>Well Being</strong></td>
<td><strong>Age</strong></td>
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<td>.00</td>
<td>.218</td>
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</tr>
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<td></td>
<td><strong>Perceived Stress</strong></td>
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<td>.13</td>
<td>.392</td>
<td>.0012</td>
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Bolded denotes significant results; n > 90.
### Table 3.10.2.1 (continued) Hierarchical multiple regression results.

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Independent Variable</th>
<th>R²</th>
<th>R² Change</th>
<th>Beta</th>
<th>Sig. In total equation</th>
<th>Overall Sig. (F.)</th>
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<tbody>
<tr>
<td><strong>Disease Activity</strong></td>
<td></td>
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<td><strong>No. Soft Stools</strong></td>
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<td>.097</td>
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<td>-</td>
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<td>.00</td>
<td>.031</td>
<td>.82</td>
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<td><strong>Abdominal Pain</strong></td>
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*Bolded denotes significant results, n ≥ 90.*
3.10.2.1. Predicting Overall Disease Activity.

The regression equations trying to predict overall disease activity and number of soft stools were not statistically significant.

3.10.2.2. Predicting Abdominal Pain.

Twenty four percent of the variance in abdominal pain was explained when eight variables (age, disease length, perceived stress, perceived disease severity, negative emotion focused coping, internal health locus of control, chance locus of control, and powerful others locus of control) were entered into the equation in this order. Roughly equivalent levels of explained variance was explained by the two equations, one with perceived stress and one with neuroticism. The overall equation with perceived stress explained 24% compared with 22% for the overall regression equation with neuroticism replacing perceived stress. Individually perceived stress and neuroticism contribute 8% and 7% to these two overall equations respectively, when trying to predict abdominal pain.

3.10.2.3. Predicting experiences of well being.

[N.B. The measure of well being is scored such that when it is said for example that perceived stress predicts well being it means that high levels of stress are associated with lower experiences of well being.] Similarly to predicting abdominal pain eight variables (age, disease length, perceived stress, perceived disease severity, negative emotion focused coping, internal health locus of control, chance locus of control,
and powerful others locus of control) explained 28% of the overall variance in well being when entered in this order. When perceived stress was replaced by neuroticism a little less total variance was explained (25%) for the overall equation, and neuroticism itself explained only 9% of the variance compared with 13% explained by perceived stress. Similar conclusions can be drawn from this result as were drawn from the regressions trying to predict abdominal pain. The regression equations to predict well being differed from those trying to predict abdominal pain in an interesting way, however; a significant additional amount of variance (5% and 4% for the equations including perceived stress and neuroticism respectively) was explained by the variable Powerful others health locus of control.

3.10.2.4. Latent variable vs. transactional model of stress and illness.

From the results trying to predict abdominal pain and well being experiences, it appears that the most predictive variables are perceived stress, neuroticism (possibly because of its covariance with perceived stress), perceived disease severity, and powerful others health locus of control (in predicting well being), in that order from most predictive downwards.

If a latent variable model of disease activity experience were the ‘true’ situation, one would expect to see a greater percentage of variance explained by trait neuroticism (as it is expected that the latent variable would be most closely related to this variable) which would not be added to significantly by variance in variables like locus of control and coping mechanisms. This, however, was not the case, and it appears
that these results do not suggest the existence of a latent trait explaining all, or indeed most, of the variance in the outcome variables. Therefore it seems that these results are more likely to confirm the transactional model of stress and illness; i.e. that a certain amount of variance in the outcome is explained by perceived stress, but then additional variance is explained by mediating factors like cognitions of control and perceptions of disease severity. Or indeed it may be that some combination of the two models may fit the data set best. This will be discussed in a later section.

3.10.3. Hierarchical regressions – psychological and clinical variables.

Clearly when predicting outcomes of disease activity, more clinically oriented indices relating to disease activity are extremely useful, and it is important to consider them to be able to comment on the predictive power of psychological indices over and above the usefulness of these clinical measures. To this end, three clinical measures were included in the regression equations. These were E.S.R. (erythrocyte sedimentation rate), total white blood cell count, and haemoglobin levels. The first two were chosen as indices of inflammation and infection, the third was chosen as an index of malabsorption, as both Crohn’s disease and ulcerative colitis may cause problems of malabsorption. These variables were entered into the regression equations in the first three steps, to establish their significance in predicting outcomes, before entering additional demographic or psychological variables.
Similarly to the previous hierarchical regressions, two equations were tested against each other, one with perceived stress and the other with trait neuroticism replacing perceived stress. Thus the independent variables entered were as follows:

1. ESR
2. Total WBC
3. Haemoglobin
4. Age
5. Disease length
6. **Perceived stress**
7. Perceived severity
8. Negative emotion coping
9. IHLC
10. PHLC
11. CHLC

1. ESR
2. Total WBC
3. Haemoglobin
4. Age
5. Disease length
6. **Neuroticism**
7. Perceived severity
8. Negative emotion coping
9. IHLC
10. PHLC
11. CHLC

These two lists of variables were used to predict the overall disease activity and the components of number of soft stools, abdominal pain ratings and well being ratings. The results of these equations can be seen in table 3.10.3.1.
### Table 3.10.3.1. Hierarchical multiple regression results with clinical indices.

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Independent Variable</th>
<th>$R^2$</th>
<th>$R^2$ Change</th>
<th>Beta</th>
<th>Sig. In total equation</th>
<th>Overall Sig. (F.)</th>
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Bolded denotes significant results, $n > 90$. 
**Table 3.10.3.1. (continued) Hierarchical multiple regression results with clinical indices.**

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<th>Dependent Variable</th>
<th>Independent Variable</th>
<th>$R^2$</th>
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<td><strong>Well Being</strong></td>
<td>E.S.R.</td>
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<td>.00</td>
<td>-.232</td>
<td>.08</td>
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<td>.00</td>
<td>-.050</td>
<td>.65</td>
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<tr>
<td></td>
<td>Haemoglobin</td>
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<td>.10</td>
<td>-.233</td>
<td>.10</td>
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<tr>
<td></td>
<td>Age</td>
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<td>.00</td>
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<td>.18</td>
<td>.</td>
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<td></td>
<td>Disease length</td>
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<td>.00</td>
<td>-.065</td>
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<td>.07</td>
<td>.436</td>
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<td>Perceived Severity</td>
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<td>.316</td>
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<td>.51</td>
<td>.0067</td>
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Bolded denotes significant results; $n \geq 90$.  

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When clinical indices are taken into account the regression equations trying to predict overall disease activity (for the equation with perceived stress only), abdominal pain, and well being were statistically significant. Number of soft stools was still not significantly predicted by the variables chosen in either equation. The clinical indices of haemoglobin and E.S.R. appeared to be the most predictive of the three clinical indices, predicting sizable proportions of the overall variance in outcome (from 5-16%, and from 0-9% for haemoglobin and E.S.R. respectively). Of these haemoglobin appeared to predict more overall variance more reliably across the differing outcome variables than E.S.R. did.

The inclusion of clinical indices in the first three steps, generally increases the overall variance explained by the equations. More specifically, perceived stress, neuroticism, and perceived severity appear to be adding slightly less to the overall equation, in terms of explained variance, and powerful others health locus of control appears to be adding slightly more.

3.10.3.1. Predicting overall disease activity.

With this inclusion of clinical indices the previously non-significant equation attempting to predict overall disease activity utilising perceived stress, became significant. Twenty eight percent of the variance in disease activity was explained when eleven variables (ESR, total WBC, haemoglobin, age, disease length, perceived stress, perceived disease severity, negative emotion focused coping, internal health locus of control, chance locus of control, and powerful others locus of control) were
entered into the equation in this order. Roughly equivalent levels of variance were explained by the other equation, trying to predict overall disease activity with neuroticism, although this was not significant. The overall equation with perceived stress explained 28% compared with 26% for the overall regression equation with neuroticism replacing perceived stress. Individually both perceived stress and neuroticism contributed low percentages to these two overall equations (2% and 0% respectively). Out of the psychological variables perceived severity contributed the greatest percentages – 7% and 9% for the equations with perceived stress and neuroticism respectively.

3.10.3.2. Predicting abdominal pain.

Thirty-five percent of the variance in abdominal pain was explained when eleven variables (ESR, total WBC, haemoglobin, age, disease length, perceived stress, perceived disease severity, negative emotion focused coping, internal health locus of control, chance locus of control, and powerful others locus of control) were entered into the equation in this order. Roughly equivalent levels of variance were explained by the two equations, one with perceived stress and one with neuroticism. The overall equation with perceived stress explained 35% compared with 34% for the overall regression equation with neuroticism replacing perceived stress. Individually both perceived stress and neuroticism contributed low percentages to these two overall equations (5% and 4% respectively). Out of the psychological variables perceived severity contributed the greatest percentages – 6% for both of the equations.
3.10.3.3. Predicting experiences of well being.

Thirty three percent of the variance in disease activity was explained when eleven variables (ESR, total WBC, haemoglobin, age, disease length, perceived stress, perceived disease severity, negative emotion focused coping, internal health locus of control, chance locus of control, and powerful others locus of control) were entered into the equation in this order. Roughly equivalent levels of explained variance were explained by the two equations, one with perceived stress and one with neuroticism. The overall equation with perceived stress explained 33% compared with 32% for the overall regression equation with neuroticism replacing perceived stress. Individually both perceived stress and neuroticism contributed to these two overall equations (9% and 7% respectively).

3.10.3.4. Latent variable vs. transactional model of stress and illness.

The psychological variables that are most predictive of outcome, in addition to variance explained by the clinical indices, appear to be perceived stress, neuroticism (possibly because of its covariance with perceived stress), perceived disease severity, and powerful others health locus of control (in predicting well being), in that order from most predictive downwards.

Similarly to the regression equations when clinical indices were not entered, it appears that these results do not suggest the existence of a latent trait explaining all,
or indeed most, of the variance in the outcome variables. Therefore it seems that these results are more likely to confirm the transactional model of stress and illness – variance in the outcome being explained by perceived stress, then additional variance explained by mediating factors like cognitions of control (in particular powerful others locus of control) and perceptions of disease severity. Or indeed it may be that some combination of the two models may fit the data set best. Again further discussion of this can be seen in a later section.
CHAPTER 4: Discussion – Main study.

4.1. Disease group differences.

4.2. Gender differences.

4.3. Interaction effects – analysis of variance.

4.4. Perceived stress and disease activity.

4.5. Personality and disease activity.

4.6. Other factors – coping, LOC, personality.

4.7. Predicting disease outcome.
   4.7.1. Univariate regressions.
   4.7.3. Hierarchical multiple regressions – psychological and clinical variables.
   4.7.4. Latent variable vs. transactional model of stress and illness.

4. Discussion – Main Study.

4.1. Disease group differences.

As is reported in the results section, Crohn’s Disease patients were significantly more likely to be smokers than Ulcerative Colitis patients. This supports a similar finding by Tobin et al (1987), who also noted this group difference in smoking behaviour. The fact that the disease groups did not differ on the other demographic or health behaviour variables suggests the homogeneity of this inflammatory bowel disease population.

Previous research, however, has found there to be various psychological differences between the two main diagnoses of inflammatory bowel disease (despite some researchers believing Ulcerative Colitis and Crohn’s disease to be manifestations of the same disease - Kirsner & Shorter, 1982; Whitehead & Schuster, 1985). It is not an uncommon finding in the literature for Crohn’s disease patients to show higher rates of lifetime psychiatric symptoms than both controls and Ulcerative Colitis patients (Helzer et al, 1982; Helzer et al, 1984; Tarter et al, 1987). Other research, however, has shown there to be no differences between Crohn’s disease and Ulcerative Colitis patient groups on current psychiatric disorders (e.g. Whitehead & Schuster, 1985). Therefore the research is equivocal. The current research tentatively supports the view that there are little or no differences between the two disease cohorts when considering variables singularly (i.e. matched comparisons between scores on one variable and scores on another – e.g. t-tests and Mann-Whitney). The only difference to be found between the disease cohorts on the
'psychological' variables (i.e. not demographic or health behaviour variables), was found on the Well Being factor of the disease activity index, with Crohn's disease patients reporting lower well being. The 'p' value for this finding only just reached significance, and it may be that this was just a statistical artefact, generated from performing multiple comparison tests. In other words, if one performs enough statistical tests on a given data set, then some statistically significant results will be obtained by chance alone. The reason the current research only tentatively supports the view that there are little or no differences between the two disease groups, is because it is difficult and possibly inaccurate to use a non-finding to support a null-hypothesis. There may be many reasons why differences were not noted, including measurement errors, methodological errors, etc., so to claim support of a null-hypothesis from the lack of a finding is not unproblematic. This does not mean, however, that findings based on small effect sizes should be discounted, it may be that in the overall population being assessed, a mixture of individual differences exist - some showing large effect sizes some showing none or in the opposite direction, thus when grouped together small, or no findings may be reported. This will be discussed in greater detail later when discussing methodological considerations.

The two disease cohorts did differ, however, when it came to assessing the correlations between the main psychological variables. The links between perceived stress and objective disease activity (symptom reporting) were mirrored across the two disease populations, but the link found in the total sample between perceived stress and subjective disease severity only emerged in the Ulcerative Colitis population. Thus a perceived stress - illness activity link was shown in Crohn's
disease patients more objective indices of disease severity (symptom reporting), although this is not reflected in the Crohn's disease patients' perceptions of their disease severity. The correlations between perceived stress and personality traits were very similar when the diseases were considered together or separately. The notable exception to this being a small correlation between perceived stress and openness in the Crohn's disease patients that is not mirrored either in the total group or in the Ulcerative Colitis patients, and could be due to another explanation, or may be a statistical artefact. In terms of control beliefs the correlations seen in the total sample were generally reflected in the correlations for the two disease groups considered separately. The notable exception to this is the existence of negative correlations between chance health locus of control and agreeableness and conscientiousness (both at -.29) in the Crohn's disease patient group, which is not shown in the Ulcerative Colitis group or in the total sample. This may represent a genuine connection between the belief that one's health is controlled by chance factors and low scores on agreeableness and conscientiousness in Crohn's disease patients, and thus may represent a real difference between the two disease groups.

4.2. Gender differences.

The results pointed to several differences between men and women. First, in terms of demographics and health behaviours, men were more likely to be in full-time employment, and more likely to exercise regularly, than women. The former of these two findings may be so because of a limitation in the demographic questionnaire. The employment status section of the demographic questionnaire (see appendix 1) does not contain an entry for 'housewife' (or 'homemaker'), which
could be seen as full-time employment, and therefore, had this been included this may have redressed this imbalance. Possibly more interesting than these two differences, may be the very lack of differences shown. Men and women did not differ on their scores for smoking cigarettes, their perceptions of the balance of their diet, marital status, or diagnostic classification of disease. The finding of no differences in smoking behaviour across the sexes in our populations does appear to be in line with recent research on gender and smoking (see Prescott et al, 1998; Robinson & Klesges, 1997; Audrain et al, 1997). Once again, it should be noted that to draw conclusions from the lack of a finding is problematic – it may be that the results support the null-hypotheses, but there are many other reasons why a statistically significant result may not be reached. Similarly although there were more women than men in the overall population, this difference was not statistically significant. The ratios of men to women for disease incidence were similar to previously published epidemiological papers (see Shivananda et al, 1996).

There were several other significant gender differences, whereby women reported greater disease activity (except for the number of soft stools reported), perceived stress, more distraction oriented coping, and higher levels of the personality trait of neuroticism. There could be several reasons why this may be. They may actually be correctly reporting the ‘true’ levels of these measures, and the women in this study may be more neurotic, more stressed, be coping by distraction more, and have worse disease activity, than men. There is some evidence to suggest that women consistently score higher than men do on neuroticism (Deary et al, 1996), although little or no differences between the genders on perceived stress, distraction oriented
coping and disease activity, are reported in the literature. One possible explanation of these differences, is that there is some form of reporting bias across the genders. That is, men and women in this population are, in fact, experiencing similar levels of these factors but that women are ‘reporting’ their feelings more honestly. It should be noted, however, that women’s scores on openness (not significantly higher than men’s) do not reflect this. This is not to say that there is a conscious effect on the male side to respond dishonestly, but simply that women may be more in touch with themselves, and their feelings and thoughts, and may therefore be expressing themselves in these self-report measures in a manner that is more true to themselves than the men. Differential responding due to social desirability factors may be an additional problem in the interpretation of these gender differences.

4.3. Interaction effects – analysis of variance.

In order to address research question 3 (see section 2.5) – Are there any interaction effects between disease cohort and gender in terms of demographic, behavioural, or psychological factors, or experiences of disease activity? – a two-way analysis of variance was performed. As is reported in the results section (see section 3.9), no significant interaction effects between disease group and gender were found.

One interesting point, is noted, however. As is mentioned above, Crohn’s disease patients were more likely to be smokers, which supports research by Tobin et al (1987), and smokers reported higher levels of abdominal pain, which supports smoking as a risk factor in certain inflammatory bowel diseases (Lindberg et al,
1992). However, the interaction of these two, i.e. for Crohn’s disease patients to report higher levels of abdominal pain, was not significant.

4.4. Perceived stress and disease activity.

The main hypothesis, predicting a relationship between perceived stress and disease activity, was generally supported by the findings. The significant correlations between the PSS-10 and abdominal pain (positive) and well being (negative) ratings offer support for the hypothesis that perceived stress is linked to disease activity (or perceived disease activity) in these disease populations. Thus, in these disease populations, it appears that as individuals experience greater levels of perceived stress, they also experience higher levels of the more subjective side of disease activity (experiences of pain, and perceptions of well being). This finding supports other studies of these disease populations (Fava & Pavan, 1976/77; Hislop, 1974; Gerbert, 1980 - see section 1.2.3.). The current finding of a link between stress and illness in these populations also reiterates the importance of considering psychological factors, like perceived stress, when attempting to understand individuals’ experiences of illness. In other words, living with an illness (in this case inflammatory bowel disease) involves a lot more than just the physiological aspects - management of the psychological side may improve the individuals’ experiences of the disease activity. This may be especially evident in the aspects of the disease activity experience that are more subjective in nature, like pain and well being experiences, although it may have a reducing effect on experiences of the more objective/physiological aspects of the disease experience also. Thus if stress and disease activity, experiences of pain, and well being are linked, then this has serious
implications for the care of individuals with conditions where these are the primary symptoms – like chronic arthritis, IBD, chronic back ache, chronic fatigue syndrome, etc. This line of thought carries with it the implication that a causal link between stress and illness exists; this may, or may not be the case.

As we have seen, stress and certain illness experiences are linked in this disease population. However, the type of analysis used to assess this (at this stage), namely correlational analysis, does not enable causal direction interpretation. It may be that increased levels of perceived stress do cause experiences of disease activity to worsen, but this assertion cannot be made from correlational evidence. This is because from this evidence it is just as likely that worsening experiences of disease activity cause the individual to experience greater levels of perceived stress. Alternatively both perceived stress and disease activity experiences may be linked via a third variable that affects them both. Correlational evidence cannot distinguish between these three possibilities. This is one limitation of the use of a cross-sectional research design (see section 4.8, below, for a fuller account of problems with this main study). Further discussion of the role of perceived stress and disease activity experiences can be seen in later sections of this chapter (see section 4.7).

4.5. Personality and disease activity.

As seen in the introduction to this thesis, personological variables have been strongly implicated in the experience of illness (see sections 1.1.2.1.1.a., & 1.1.2.2.). The results from this cross-sectional study do suggest a need to at least consider
personological factors in the stress-illness relationship – many previous studies have not. The results from the main study show links between personality variables and illness experience. The most important of these personality variables (based on effect size from the current results, and also based on theoretical standpoints in the literature) is trait neuroticism. In the introduction to this thesis it is argued that trait neuroticism may be involved as an antecedent to the Transactional model of stress and illness (Lazarus, 1990), or may be very closely related to a Latent variable that affects many self-reports relating to emotions and bodily states (Watson & Pennebaker, 1989). Discussion of this argument can be seen later (see section 4.7.). When not considering the role of personality in models of stress and illness, the importance of personality in other areas of health psychology can still be seen (see section 3.5). For example, similar to the results for perceived stress, trait neuroticism was found to be related to the more subjective aspects of disease activity, namely abdominal pain and well being. Thus individuals who score highly on trait neuroticism express greater disease activity, and (vice versa) those who score low on neuroticism tend to report lower disease activity. This finding is consistent with a large body of work reporting the importance of neuroticism in health related outcomes (see Adler & Mathews, 1994, for a recent review). This also implies that individual differences in reactions to stressful situations, as defined by trait neuroticism, may be more telling in the link between stress and illness than the originating stressors themselves. This generates a problem of interpretation. Is it just that the more neurotic individuals are perceiving their stressful situations as more stressful, and also perceiving their disease activity as worse? This will be discussed further later on (see sections 4.7 & 4.8).
Trait neuroticism was not the only personality variable to be related to disease activity, however. Relationships between extroversion and openness and disease activity were also noted, although these correlations (between extroversion and disease activity and between openness and disease activity) may only be artefacts of the intercorrelations between the different factors of the personality questionnaire. In other words, neuroticism and extroversion (for example) are not totally independent and did negatively correlate, thus the correlation between extroversion and disease activity may be as a result of extroversion’s shared variance with neuroticism. The same may be true for openness.

4.6. Other factors – coping / LOC / personality.

As is mentioned above, when lots of analyses are performed resulting in many statistical results, several statistically significant results will occur by chance alone. This is known as the omnibus effect (Hooker et al, 1992; Cohen & Cohen, 1983), and is particularly common when looking at many correlations calculated from the same data set. This is mentioned to remind the reader that to look at one isolated correlation and assign particular significance to it, in terms of supporting/refuting hypotheses, may over emphasise the importance of the result (effect size may be a lot more telling). It is important to bear this in mind when interpreting results of multiple correlations. Clearly, it is easier to interpret a correlation that supports the research hypothesis and that has a large effect size, than a correlation between two variables that may, or may not, in fact be linked, that is not based on hypotheses from previous research, and may have become significant through chance. This does not mean that important findings do not occur by accident, it is only intended to
emphasise the tentative approach that has to be taken when interpreting some of the results. With that said, various interesting findings discussed here, may need to be treated with a degree of caution.

Perceived stress was correlated with negative emotion focussed coping, such that subjects who expressed higher levels of perceived stress, also reported using more negative emotion focussed coping styles/strategies. If the stress-illness process were best modelled by the Transactional Model of stress and illness, then one would expect the antecedent perceived stress levels to be related to mediating negative emotion focussed coping (often described as a dysfunctional coping strategy). Similarly one would expect this coping strategy to be related to disease outcome. Thus, theoretically, individuals who experienced greater levels of stress, who coped in terms of greater negative emotion, would then express greater levels of disease activity. Thus if negative emotion focused coping was related to disease outcome then this could provide support for the Transactional Model of stress and illness. This was not the case. In the overall sample (both diseases considered together), negative emotion focussed coping was not related to the disease outcome. It may be that coping strategies are not so 'mediating' as they are expressed in the theory, and that they are performing more as antecedent variables more akin to a personality trait than to a state coping strategy. That is to say that individuals may repeatedly use similar strategies to cope, even if these are thought to be dysfunctional in style. This would mean that coping strategies involved in the stress-illness process would be correlated to trait personality variables. In the current study, negative emotion focussed coping was quite strongly related to trait
neuroticism (see section 3.6.), such that the individuals reporting higher neuroticism were also more likely to be using more negative emotion focussed coping strategies. This could mean two things: First, it could mean that the current study provides support for the Transactional Model of stress and illness, but that the variable negative emotion focussed coping is behaving more as an antecedent than as a mediator; OR, secondly, that the current study actually provides evidence for the Latent variable model of stress and illness, and coping, stress, and disease outcome are all related to the putative latent variable negative affectivity – negative emotion focussed coping being correlated to neuroticism because of shared variance in negative affectivity. Thus the picture still remains unclear (this will be discussed further below – section 4.7.4).

For locus of control, the expectation is that the factors associated with a stronger stress-illness relationship, such as higher levels of perceived stress, higher levels of trait neuroticism, more dysfunctional coping styles,strategies, will be linked to a belief that one’s illness/disease is outwith one’s control. Thus it would be hypothesised that higher scores on neuroticism, negative emotion focussed coping, and perceived stress are likely to be related to high scores on external control (chance and powerful others) and lower scores on the internal locus of control. The current study found several correlations that support this expectation. In the total sample, perceived stress and internal health locus of control were correlated negatively, i.e. high perceived stress scorers had less internal control beliefs. In Ulcerative Colitis patients, perceived stress was also positively correlated with powerful others locus of control, such that as perceived stress increases, the belief
that your health is in the control of powerful other increases. Neuroticism was positively correlated with chance and powerful others loci of control, thus as individuals express greater neuroticism, the belief that external factors (chance and powerful others) control your health also increases. These correlations are as expected, and also support other literature which claims to show that individuals with an internal locus of control in their lives, feel less impact from stress than those with an external locus of control or a belief that luck or fate controls their lives (Krause & Stryker, 1984; Revicki & May, 1985; Stern, McCants, & Pettine, 1982; Matheny & Cupp, 1983; Johnson & Sarason, 1978; Lefcourt, Martin & Saleh, 1984; Sandler & Lakey, 1982). There were a couple of unexpected correlations, including significant negative correlations between openness and external control (chance and powerful others). In other words, individuals who are more ‘open to new ideas’ (Costa & McCrae, 1992) believe their health to be less dependent on chance factors or powerful others. Interestingly, however, no correlation was noted between openness and internal health locus of control (one might expect a positive correlation). Thus, although individuals who score higher on openness are expressing that control of their health is less dependent on external factors, they are not claiming that it is dependent on internal factors.

4.7. Predicting disease outcome.

4.7.1. Univariate regressions.

The results showed that the overall disease activity index – consisting of number of soft stools over the last week, experiences of abdominal pain, and well-being rating –
was not singularly predicted by trait neuroticism or perceived stress levels (section 3.10.1). This may mean that neither perceived stress nor neuroticism are good predictors of disease activity. It may also mean that the measures are not very good, either the measures doing the predicting (perceived stress and neuroticism) or the measures being predicted (overall disease activity). In this case it would be difficult to claim support for either the Transactional Model or the Latent variable model of stress and illness. It has been claimed, however, that it may be more likely to be the subjective experience of disease activity that can be predicted by (or at least is related to) psychological factors, than the objective physiological disease activity (Watson & Pennebaker, 1989). This claim was supported by the current research - abdominal pain and well being were predicted much better by perceived stress and neuroticism, than number of soft stools. Between neuroticism and perceived stress, it appears that perceived stress (over the last month) is a greater predictor of pain and well being than trait neuroticism. This may indicate that in this population, state, and not trait, stress/anxiety/neuroticism is more telling of disease experience. However, it is reiterated that the state and trait measures of perceived stress and neuroticism do correlate very highly, possibly suggesting that they are to a certain extent measuring the same phenomenon. It is clear, however, that if these variables predict somewhere around 10% of the variance in experiences of pain and well being then they are important and need to be considered by all parties involved in caring for these patients – and their implications may be great (see section 6.3).

It has hopefully become clear from the results to date that several variables have emerged as important in the overall stress-illness process and also in experiences of disease activity. Hopefully, it also clear that these variables are unlikely to have their effects on disease activity in isolation, and that they are much more likely to share certain amounts of variance in disease activity, and work in conjunction with each other through whatever way (Transactional or latent variable). As stated earlier (section 3.10.2.), the main aim of the hierarchical regressions was to compare two regression equations, one involving perceived stress, the other neuroticism. This was attempting to distinguish between the Transactional and the Latent variable models of stress and illness. Thus if the actual 'true' situation were best modelled by a latent variable theory, one would expect to have found that the vast majority of explained variance in the outcome variables, to be explained by neuroticism alone (given that it closely relates to the concept of negative affectivity), and that relatively little or no additional variance would be explained by the other variables included in the regression equations. If a transactional model more accurately explained the 'true' situation, then one would expect to find a lot of the variables adding their own proportion of the total variance explained.

Overall disease activity was not significantly predicted by either of the two regression equations. The reason for this may be the same reason why overall disease activity was not significantly predicted in the univariate regression equations (see the previous section). That is, it is more likely to be the more subjective aspects of disease experience that can be predicted by psychological variables.
Therefore, presumably for the same reasons, the number of soft stools factor of the disease activity index, which is more objective in nature (although not totally — there may be aspects of selective recall, remembering bias, estimation bias, etc., at play here), was also not significantly predicted by either of the hierarchical regression equations. However, both regression equations attempting to predict the more subjective outcomes of abdominal pain experiences and well-being ratings, were statistically significant. Thus the four hierarchical multiple regression equations that significantly predicted disease experience were as follows:

1. Predicting **abdominal pain** experiences using variables age, disease length, *perceived stress*, perceived disease severity, negative emotion focussed coping, internal, powerful others, and chance health loci of control, entered in that order.

2. Predicting **well being** experiences using variables age, disease length, *perceived stress*, perceived disease severity, negative emotion focussed coping, internal, powerful others, and chance health loci of control, entered in that order.

3. Predicting **abdominal pain** experiences using variables age, disease length, *neuroticism*, perceived disease severity, negative emotion focussed coping, internal, powerful others, and chance health loci of control, entered in that order.

4. Predicting **well being** experiences using variables age, disease length, *neuroticism*, perceived disease severity, negative emotion focussed coping, internal, powerful others, and chance health loci of control, entered in that order.
The difference between the equations using perceived stress to predict disease experience (equations 1 & 2 – above), and those using neuroticism (equations 3 & 4 – above), was small. Both pairs of equations showed similar patterns of variance explained by the variables used, excepting that perceived stress predicted a little bit more of the explained variance in most of the outcome measure than did neuroticism. This can be seen in figures 4.7.2.1. & 4.7.2.2. for the equations trying to predict abdominal pain and well being, respectively.

Figure 4.7.2.1. Graph of the additional percentage variance explained by each of the psychological variables in the regression equations trying to predict abdominal pain experiences – equation including perceived stress vs. equation including neuroticism.
From looking at each graph it can clearly be seen that the patterns for the variance explained by the variables used are very similar – i.e. the dark columns follow approximately the same shape as the light columns – with only a few differences between the equation using perceived stress and the equation using neuroticism. The most notable thing about both graphs is this similarity between the variance explained in the equations using perceived stress and that explained in the equations using neuroticism. As has been mentioned before these two variables (perceived stress and neuroticism) do correlate highly together and therefore share a lot of variance with each other. They may even to a certain extent be measuring the same thing – despite perceived stress being more of a state measure and neuroticism being a trait measure. This state/trait difference, may go some way to explain why perceived stress is, generally speaking, a better predictor of the outcome measures than neuroticism. From looking at the graphs, one can see that the best predictors of abdominal pain experiences and well being ratings are the variables perceived...
stress and neuroticism (possibly because of their shared variance – see above), and also quite importantly perceived severity. It is difficult from these results to ascertain definitely whether or not the results support a Transactional or a latent variable model of stress and illness. The fact that the strongest predictors of the subjective aspects of disease experience are generally perceived stress and neuroticism, plus the fact that these two variables correlate highly, and also because the other variables tend to add relatively little or no additional explained variance, the research may be interpreted as supporting a latent variable model; namely, that a latent variable exists somewhere in the shared variance between neuroticism, perceived stress, and also possibly perceived severity, and that this may be quite close to Watson and Pennebaker's (1989) concept of negative affectivity. More discussion on whether the regression equations support a transactional or a latent variable model of stress and illness can be seen later (see section 4.7.4.).

The role of perceived disease severity in predicting outcome is also important. At the very least it has implications on treatment and care of patients with these illnesses (as discussed later). Furthermore it suggests that perceptions of disease severity are important to consider, and possibly include in the concept of negative affectivity. In other words, negative affectivity may be closely related to neuroticism/perceived stress, but the results seem to suggest that the inclusion of perceived disease severity could also be important. One thing that should be remembered, however, is that the disease outcomes we are talking about here are pain and well being - the more subjective / disease experiences / perceptions of disease. Thus perceptions of one's disease in terms of disease severity are predictive
of perceptions of one's disease in terms of pain and well being. It could be that perceived severity may also be seen as an outcome variable and that in this case we are using one variable based on perceptions of health status to predict another very similar variable – the problems of interpreting this are obvious. However, the implications of this variable for health care professionals still remains, and will be discussed later.

There are two main differences between the equations trying to predict abdominal pain experiences and those trying to predict well being ratings. First, in the equations trying to predict abdominal pain, age, disease length, and internal health locus of control add some predictive power to the overall equations in addition to the already mentioned variables perceived stress/neuroticism, and perceived severity. For the equations trying to predict well-being ratings, these variables (age, disease length, and internal health locus of control) did not add very much predictive power to the equations over and above that already added by perceived stress/neuroticism and perceived severity. Secondly, powerful others health locus of control did appear to be important in predicting disease activity, and this was not noted for the equations trying to predict abdominal pain experiences.

4.7.3. Hierarchical multiple regressions – psychological and clinical variables.

When attempting to predict disease activity, more clinical indices relating to disease activity are extremely useful. The inclusion of clinical indices facilitates comments on the predictive power of the psychological variables over and above that of the clinical
factors. Similar to previous findings the regression equations attempting to predict the more objective factor of number of soft stools over the last week, were not significant. This is interesting because it means that in these disease populations the clinical indices do not significantly predict even the most objective aspect of disease activity. It must be noted, though, that the summed variance explained by the three clinical indices, had the overall equations been significant, was greater than the summed variance explained for the psychological variables. Also similar to previous findings, the regression equation attempting to predict overall disease activity using neuroticism (amongst the other variables) (see section 3.10.3.) did not significantly predict overall disease activity. However, the equivalent regression equation with neuroticism replaced by perceived stress did significantly predict overall disease activity. This may be due, however, to a statistical artefact, as the equation involving perceived stress was only just significant, and the equation involving neuroticism was only just non-significant. Who is to say there is any great difference between the two? What is more interesting, however, are the results for the more subjective aspects of the disease activity index. The percentage variance explained by each of the variables in the equations can be seen in figures 4.7.3.1 and 4.7.3.2. for the equations trying to predict abdominal pain experiences and well being ratings respectively.
From these graphs (figures 4.7.3.1. & 4.7.3.2.) it can be seen that the overall pattern of the variance explained by the psychological factors in the regression equations is very similar to when the clinical indices were not included: perceived stress / neuroticism and perceived severity are important predictors of both abdominal pain...
and well being; age and internal health locus of control are important predictors of abdominal pain; and powerful others health locus of control is an important predictor of well being. Of the clinical indices, haemoglobin was a powerful predictor of both of the more subjective aspects of disease experience – abdominal pain and well being ratings. Haemoglobin has been thought of as being a good clinical indicator of inflammatory bowel disease activity because of its' involvement with nutrient absorption (in this case iron) and there are several recent studies looking at the role iron absorption and haemoglobin have in these diseases (see Schreiber et al, 1996; Fischer, 1996; Dichi & Burini, 1995; Saitoh, et al, 1995; Sandborn, 1997; and Martinez Salmeron, et al, 1996). Thus it is possible to say that, although haemoglobin (a clinical index) is a powerful predictor of disease activity in these disease populations, psychological factors (like perceived stress, neuroticism, perceived severity, internal and powerful others health loci of control) do add significant amounts of explained variance in the outcome of disease experiences. It seems clear from this that it is likely that a combination of clinical and psychological factors orchestrate disease experience. And thus these psychological factors should not be forgotten when trying to help patients live with these diseases. That is, if perceptions of stress and perceptions of severity affect disease experience in a negative sense then maybe some alleviation from negative experiences of disease activity can be achieved by changing these patients' perceptions of their stress and their disease severity.
4.7.4. Latent variable vs. transactional model of stress and illness.

There are two ways of approaching the question as to whether the results from the current cross-sectional study supports a Transactional or a Latent variable model of stress and illness. First, one can look at the correlational evidence and use the links implied by the correlations to infer which model is more likely. Secondly, the results from the regression equations can also provide insight into which model is more likely to be the case.

From the correlational evidence, the current research appears to offer tentative support for the existence of a latent trait with the correlations between the three variables neuroticism, perceived stress and negative emotion coping. With these three variables being closely linked together, then it would seem that the putative Latent variable would be some form of combination of these three – this would be ‘negative affectivity’. Interestingly this tendency to support Watson & Pennebakers’ (1989) idea of ‘negative affectivity’ is more strongly supported in the Ulcerative Colitis patient group; the results for the Crohn’s Disease patients may fit the transactional model of stress and illness better (Lazarus & Folkman, 1987). It may be that different models of stress and illness best fit the data gathered from different disease groups, or alternatively there may be other reasons for differences in the literature as to whether a latent variable or a transactional model of stress and illness is the ‘true’ model (see methodology section below – section 4.8.).
The evidence from the regression equations is also not entirely clear. We have seen that the variables neuroticism, perceived stress, and perceived severity are most predictive of the subjective aspects of disease activity. This may point us to thinking that the Latent variable is supported by these results, but this is unclear. It can just as easily be argued that because of the fact that some of the other variables (namely some loci of control) do also play a role in predicting disease outcome, that a more Transactional approach is closer to the 'truth'. The third possibility of the 'true' model being some form of combination of the two still exists.

In order to identify which model of stress and illness best fits the data from these disease populations, further analysis would have been necessary. It would have been useful to compare the two models by performing some form of structural equation modelling, and analysing the best fit. Unfortunately the scale of the project did not permit this form of analysis. The number of participants was too small and the number of variables too large to allow a statistically meaningful form of this analysis to be carried out. Clearly this provides a springboard for further research. Previous research, albeit in a different disease population, has performed this kind of analysis. Deary et al (1997) found that an integrated model that consisted of elements 'borrowed' from both the Transactional Model and the Latent variable model, was the best fit for the data gathered from their population of diabetes mellitus sufferers. This was such that a latent variable fed into factors like alexithymia, neuroticism, negative emotion coping, somatisation and diabetes severity, but also that a transaction from trait neuroticism through negative emotion coping and somatisation
to diabetes severity was also noted (Deary et al, 1997). This integrated model can be seen below (figure 4.7.4.1.)

![Integrated model of stress and illness reporting](taken from Deary et al, 1997).

Therefore an approach that dogmatically sticks to one or other of the models of stress and illness, may be inappropriate. Clearly this is the problem that actual individuals do not fit into the convenient little black boxes that we propose for them in our conceptual models. Perhaps this should remind us, psychologists trying to measure objectively individuals and their behaviour, to take a more empirical data led approach to theorising about stress and illness.


When attempting to interpret these and similar results, there are several questions of methodology that should be kept in mind, if not answered. Some of these will be discussed here.
One point that should be mentioned here is that of interpretation. At various points I have attempted to draw conclusions, albeit tentatively, from different correlations across the disease populations. It must be mentioned here that differences between the size, or even significance, of differing correlations on the same measures for the different disease groups, were not explicitly analysed. This means that some correlational differences, one group showing a significant correlation the other not, may only be due to them sitting either side of the significance boundary, and may not actually suggest any real tangible difference. Thus the reader is reminded to approach interpretation of these with caution.

Health psychology has an increasing embarrassment of constructs, and measurement of those constructs, to choose from when attempting to answer any specific question. In this particular research, when one looks at any one theoretical construct addressed in the research, one can easily ask why one measurement tool was chosen over another. For example, the stress index chosen – the Perceived Stress Scale – 10 (Cohen et al, 1983), could have easily been something different like the Daily Stress Index (Brantley & Jones, 1989), not to mention other forms of stress measurement – e.g. Life Events; the coping inventory (CHIP – Endler et al, 1992) could just as easily have been the Coping Inventory for Stressful Situations (CISS – Endler & Parker, 1994) or the COPE (Carver et al, 1989). The list goes on. The question that must be asked, however, is to what extent do these separate measures measure separate and independent real entities. This does not just mean which of two stress questionnaires are the better, but rather is one stress questionnaire measuring some of the same real phenomenon as a coping questionnaire. We have
already seen, for example, that perceived stress and neuroticism correlate so highly that it is entirely likely that they may be actually measuring the same underlying phenomenon. Similarly, many of the constructs used daily by health psychologists appear to be related to many others, and in order for theoretical interpretation to be made, clarification of what these measures actually measure independently of one another is needed. It is clear that the stress – illness process is not easily investigated, and the use, and proliferation, of sloppy non-independent constructs can only serve to muddy this water further.

Another issue to consider is that of homogeneity of populations. If individual differences in this underlying central trait closely related to neuroticism do affect many other health-related indices (Marshall et al, 1994), and as in the current research the two disease populations do not differ on neuroticism it cannot be that surprising that the two diseases do not differ on the health-related variables considered (except smoking). However, if the two inflammatory bowel disease populations are considered separately, or indeed together, significant correlations are observed between perceived stress and disease activity; and also very importantly between neuroticism and perceived stress, neuroticism and negative emotional coping and perceived stress and negative emotional coping. These all provide support, albeit tentative, for the existence of a latent variable akin to negative affectivity / somatopsychic distress. Lack of convincing evidence in the literature may be due to mixed populations of stress-responders and non-stress-responders. The inclusion of stress-responders and non-stress responders in the same group being measured may only serve to reduce the size of the effect of
perceived stress on illness measured in that group (Brantley & Jones, 1993).

Assessing individual differences in stress response may address this issue.

Another issue that is mentioned earlier is that of the omnibus effect. In a study with quite a few variables with which to perform correlations the chances of acquiring significant correlations by chance alone are increased - thus the interpretation of some of the smaller correlations in terms of supporting demonstrable real processes or psychological links becomes increasingly difficult.

The majority of research that has been done on the stress – illness link in inflammatory bowel disease, has been shown to have some methodological limitations (as discussed previously). Thus, it would be useful to have research that is not only prospective in nature, and can predict onset of disease exacerbation, but is performed longitudinally to assess the temporal nature of the stress-illness relationship, given that it exists. The next section, therefore, presents a pilot of such a study, which hopefully can address some of these issues.
CHAPTER 5: Pilot – Longitudinal Study.

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5.3. Methods.

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5. Pilot – Longitudinal Study.

5.1. Abstract.

Previous research is critically reviewed, on various methodological grounds, including retrospective sampling and problems of life-event research. The design and procedure of a pilot study is presented, as a means of providing some ideas to future researchers as to how to answer, or at least address, some of these issues. Pilot data are presented from a small sample of eight individuals comprising three men and five women, four Crohn’s disease patients and four ulcerative colitis patients. Participants were assessed at baseline, and again at approximately three months after baseline, on demographics and health behaviours, personality, loci of control, coping styles and disease activity. Prior to follow-up assessment, participants completed 28 consecutive sequential days of diary reporting of perceived stress, mood, and disease activity. The results are assessed and presented descriptively on the whole, due to the small number of participants. Results appeared to support some form of link between perceived stress, mood, and disease activity, in some of the participants – possibly suggesting individual differences. The implications of the results, and further ideas are then discussed, with a view to informing future research.
5.2. Introduction.

As stated in the introductory section of this thesis, life events research into the stress-illness link in Inflammatory Bowel Disease (see section 1.2.3.1.), tends to focus on the kinds of events that cause major life change. These would include events like redundancy, pregnancy, divorce, death of a friend or spouse, etc.. Fortunately in any 'normal' individual's life these events tend to happen with relative infrequency. Therefore, one of the only practical ways to study the effects these events have on a specific disease such as Crohn's disease or ulcerative colitis, which are, in themselves, relatively rare (CD-approximately 6 per 100,000; UC-approximately 10 per 100,000 — Shivanda et al, 1996), is to use retrospective study designs for data collection (Duffy et al, 1991; von Weitersheim et al, 1992; Monk et al, 1970). Such a methodology may be replete with flaws when causal inferences are to be made: -

First, individual variation in the responses to life events questions the use of life events as a measure of stress for small sample sizes and within-subject analysis. A second problem is that with the use of retrospective data collection, patients with these diseases might indeed express greater levels of life events compared to controls, but this might serve to provide them with some explanation of the course of their own illnesses (von Wietersheim et al, 1992). Retrospective studies also often tend to disregard the temporal nature of the illness, the change of the disease activity over an extended period of time. Yet, only knowledge of such changes would enable researchers to examine whether an important increase or decrease in disease activity can be connected to the proposed precipitating stress or stressors. This is especially pertinent given that both Crohn's disease and ulcerative colitis are typified by bouts of exacerbation and remission across time.
Some more methodologically appropriate prospective studies have been carried out using life events measures in order to negate some of the problems mentioned above. Campbell et al (1986) used continuous assessment of stressful life events as predictors, and symptoms of abdominal pain and diarrhoea as outcomes. They found no significant correlation between the occurrence of these two variables. Riley et al (1990) tested 100 patients over a 48-week testing period and found no difference between patients with an acute relapse of ulcerative colitis and those patients in remission, on a modified Paykel Life Event Scale (Paykel, 1974). It must be noted however, that although they measured a sizeable sample on a standardised measure over a long period of time, they only collected data once every 12 weeks (i.e. 4 data points) which might generate some problems drawing inferences from the results (see below for more detail).

Despite various interesting findings, there is, as is common in many areas of research, a problem in drawing comparisons between all these different studies. This is due to a lack of standardised measures of both stress due to life events and measures of disease activity. Differing sampling characteristics, methods of analysis and the lack of appropriate control groups only serve to compound this problem, leading to an equivocal, even contradictory, picture of the stress-illness relationship in these inflammatory bowel diseases.
A further problem with life events studies is that of timing, and more specifically time lapse. Even in longitudinal studies, when the subject is asked to report the life events occurring in the previous month or two, the chances are that the resultant immunologically mediated disease activity might not be recorded accurately. It has been reported that the time lag for the effect of minor life events on the common cold is approximately four days (Evans & Edgerton, 1991), implying that it only takes this length of time for stressors to be displayed physiologically. Thus, to analyse this by means of assessment with time in the order of months may miss the subtle relationship between stress and disease. This error could be corrected by collecting data on a daily basis, and thus due to the infrequency of major life events in normal existence, a measure of daily minor stressors/perceived stress may be more appropriate to this finer temporal analysis. Similarly, in studies prompting the subject to remember stressful life events, some bias in recall is likely. In other words, personological differences may result in differing memory of, or attention to stressful events. For example, subjects high in neuroticism may attend more to stress/life events and thus remember more, they may have superior memories of negative experiences or they could actually experience more stressful events (Adler & Mathews, 1994).

Any evidence supporting the claim of a causal relationship between stressful life events and inflammatory bowel diseases must be treated cautiously. The reason for this can be seen in the problems of comparisons between studies, as well as the various methodological flaws associated with life events studies (see above). The use of daily stress measures along with prospective study designs would avoid these
pitfalls. Because of this, researchers interested in the stress-disorder relation have focused attention on minor stressful events and daily 'hassles' as opposed to the problematic major life events style research paradigms (Brantley et al, 1987). Indeed it has been suggested that measures of daily stress may serve to account for a larger percentage of the variance in prediction of physical symptoms than major life events measures have managed (DeLongis et al, 1982).

To date, however, relatively few studies have been conducted that use daily measures of stress when considering the stress-illness relationship in inflammatory bowel diseases. One study that has used a psychometrically sound instrument to measure daily stress and its relation to Crohn's disease was conducted by Garrett and colleagues (1991). They recorded the perceived impact of minor stressful events using the Daily Stress Inventory (DSI-Brantley & Jones, 1989) along with both the signs and symptoms of the disease activity for 28 consecutive days. They found a direct relationship between daily stress and Crohn's disease activity when the effects of previous major life events were controlled for. This result was not reflected in the findings from the within-subject analyses, which were not consistent, which may reflect the limited sampling period of 28 days, during which substantial change in either disease activity or daily hassles may not have occurred. The inconsistent within-subject correlations also advocate the importance of taking into consideration individual differences in possible mediating or antecedent personological factors of the stress-illness process.
Good prospective or longitudinal studies may be relatively rare, because of various problems associated with performing this kind of research. If the researcher is trying to capture the time just prior to a period of disease flare-up, then it would be necessary for them to do at least one of two things. These are to ensure that the sampling time frame is long enough to capture a disease flare-up episode, or to assess enough individuals so that, by chance, some of them experience a flare-up within the sampling time frame. Ideally, large numbers of individuals should be assessed intensively for a long period of time, to maximise the chances of witnessing as many of these episodes as possible. Obviously there are problems associated with this. First, the time and effort that the researcher/research team would need to give to the problem often makes such research prohibitively expensive, both in terms of financial and human resources (the difficulties of finding funding bodies to fund such research merely exacerbate this problem). Another very significant problem is the demands that such research may make on participants. Given that most individuals do not experience these periods of high disease activity very frequently, and consequently live 'normal' full lives, to require them to be intensively studied for long periods of time may be too much to ask. Also, the continuous measurement may indeed have an effect on measures under scrutiny. For example, in trying to measure daily stress, the process of the measurement may itself be stressful. Another problem is that, although health psychology is generally blessed by unusually compliant and conscientious subjects, their vigilance in maintaining measurement regimes may, and often will, wane. This may mean that individuals' scores may not display any variance after a while as they become habituated to particular responses to measurement. This is a particular problem when measures are repeated daily (so subjects become familiar with the measurement), and when measures are self-report.
Another problem associated with measuring stress and illness in particular is time lag. If a subject's attention to daily measurement is already waning and they find themselves experiencing high levels of stress/stressors, then they may feel that completing measurements at this time is the last thing they wish to do. This is unfortunate as it is precisely at this moment that those data become vitally important, and subjects may not realise this until a few days later when they experience the putative increase in disease/illness activity. However, no research is without its flaws, and the job of the researcher performing this research is to try to balance these factors. They must employ a measure that is recognised by the academic forum, but which is also relatively unintrusive to the subject, and maintains interest in continuing in the study. Similarly the researcher must balance their resources in time, money, etc., by having a sample size large enough and/or a sampling time frame long enough, to permit a discriminative study.

Despite the Garrett et al (1991) study showing significant improvement on retrospective life events study design, by being one of the very few prospective studies to measure daily stress in this disease population, it is not without fault. As has already been mentioned the study does not truly consider the effect that possible individual differences may have on the results, nor does it monitor the relation between stress and Crohn's disease for long enough. Over and above these criticisms, however, the study is flawed in design by its lack of a control group with which to compare the disease group. [Although it is noted that the inclusion of a control group or not depends on the aims of the research – if the focus is individual differences then a control group may neither be necessary or appropriate.] The
possible inclusion of a control group, monitoring for a longer period and appropriate measures of the individual differences in the stress process would have greatly improved this study and made the results far more exciting and interesting.

Stone et al (1993) have written an excellent review article which outlines a lot of the potential benefits and problems of performing research on daily stress, mood and coping. They state that:

"a ubiquitous finding in studies of daily stress and mood, regardless of measures, samples, etc., is that higher levels of stress directly relate to negative mood." (pg 9)

This does not, however, imply any causal direction to this relationship. Is there any time lag between perceived stress/events checked, and an increased negative mood? One clear way of trying to assess this would be to try to predict subsequent mood levels just after a period of acute stress. Greene et al (1996) found just that: on testing 11 inflammatory bowel disease patients over 7 consecutive days every month for a year, they found a positive concurrent relationship between psychosocial stress and disease activity. However, they also noted the apparently contradictory finding that the month succeeding a particularly stressful month patients reported greater well being (this will be discussed in greater detail in a later section).

As this example suggests, there are several methodological/statistical problems associated with this temporal analysis. For example, when measuring stress and its
dependencies repeatedly for many days the problem of autocorrelation (along with others) becomes a pertinent issue - so specialised statistical techniques need to be employed to compensate for these (West and Hepworth, 1991). The day of the week may have an effect on the relationship between stress and mood, and therefore on health outcomes. That is, the types of stressors that are experienced during the working week may be categorically different from those experienced at the weekend. Despite these problems with this research paradigm, same-day links between stress and mood are quite robust. This, however, is not reflected in results trying to assess a lagged effect of stress on mood, where the evidence is far from conclusive (Stone et al, 1993). Stone et al (1993) also note large individual differences in the magnitude of the relationship between stress and negative mood, and that these differences may mask the true effect. That is, there may be ‘stress-responders’: individuals who respond more negatively to stressors than non-responders. In this light, the level of perceived stress may be preferable as a measure than a checklist of stressors experienced. The reason why some individuals may respond more negatively to the same stressor is still under discussion, although a lot of research is being performed in the area of individual differences in personality.

Therefore, despite the improvement of the methodology from retrospective life events data collection to prospective daily stress measurement, the ‘true’ picture of the stress-illness relationship in inflammatory bowel disease still remains unclear. The present study aims to provide a pilot of a design in this field of research that can
improve on some of the limitations mentioned above (although not all because of the limitations of time/effort/resources/etc., mentioned above).

First, the current study's use of standardised measures of generally well accepted constructs, may improve the comparability of studies in this area. Secondly the study also incorporates some reliable and valid constructs from the field of individual differences in psychology. Thus at a conceptual level, the researchers are prepared to accept the possibility that the personological make-up of an individual may effect their responses to stressors/daily hassles or disease experience; and that this may be very different from individual to individual. The interplay of lessons learnt from both psychometric and health sciences, may provide invaluable insight into the stress-illness relationship (see section 1.1.2.2.). Thirdly the study employs a combination of prospective data collection, using self-report diaries, and baseline and follow-up measures that are normally employed in one-off cross-sectional studies.

Therefore the study aims can be itemised as follows: -

- To provide a stepping stone, and hopefully some insight, for further research into this area.

- To demonstrate the usefulness of prospective study design, and the use of personological data.
To report on the relationship between perceived stress and illness experience in a small group of Crohn’s disease and ulcerative colitis patients, and also to comment on the role mood plays in this relationship, if any.

And to disseminate some potential benefits and pitfalls of this type of research design within this area of study.

5.3. Methods.

5.3.1. Participants.

Individuals from lists of inflammatory bowel disease patients attending a gastrointestinal out-patient clinic were invited to participate in the study. Out of 20 patients approached, 10 agreed to take part in the study, two of whom dropped out at a later date. This left eight individuals who completed the whole study. These consisted of five women and three men. Their ages, gender, and Inflammatory bowel disease diagnoses were as follows:

<table>
<thead>
<tr>
<th>Subject Code</th>
<th>Age</th>
<th>Gender</th>
<th>I.B.D. Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>60</td>
<td>Male</td>
<td>Ulcerative Colitis</td>
</tr>
<tr>
<td>7</td>
<td>50</td>
<td>Male</td>
<td>Crohn’s Disease</td>
</tr>
<tr>
<td>3</td>
<td>53</td>
<td>Female</td>
<td>Crohn’s Disease</td>
</tr>
<tr>
<td>10</td>
<td>51</td>
<td>Female</td>
<td>Crohn’s Disease</td>
</tr>
<tr>
<td>19</td>
<td>31</td>
<td>Female</td>
<td>Ulcerative Colitis</td>
</tr>
<tr>
<td>20</td>
<td>38</td>
<td>Female</td>
<td>Crohn’s Disease</td>
</tr>
<tr>
<td>8</td>
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<td>Ulcerative Colitis</td>
</tr>
<tr>
<td>9</td>
<td>28</td>
<td>Female</td>
<td>Ulcerative Colitis</td>
</tr>
</tbody>
</table>
5.3.2. Measures.

Subjects were assessed on a series of measures:

a). Demographic details. (see appendix I)

Patients' sex, age, employment and marital status were assessed.

b). Brief disease history. (see appendix I)

Participants were asked to rate their current perceived disease severity on a scale of 1-10 of increasing severity. They were also asked to recall how long ago they had been diagnosed as having inflammatory bowel disease. Patients' medical records were accessed to extract information on medication levels and treatment regimes, and clinical indices (like white blood cell counts, ESR, and haemoglobin levels).

c). Health behaviours. (see appendix I)

Participants were asked whether or not they thought they had a healthy diet; and if they were a non-smoker, an ex-smoker (if so how long ago did they stop smoking), or a current smoker (if so, how many they smoked per day), as it is reported that smoking is related in various ways to inflammatory bowel disease experience (Tobin et al, 1987) (see section 1.2.2.). They were also instructed to categorise themselves into one of six categories of exercise ranging from never exercising to taking exercise at least once every day.
d). Perceived stress / minor life events  (see appendix 2)

The 10 item Perceived Stress Scale (PSS-10, - Cohen et al, 1983) indexes perceptions of stress experienced over the last month. The scale has been validated in a random stratified sample (n=2387) of healthy 'normal' individuals, and internal consistency was satisfactory (as assessed by Cronbach's alpha - 0.75) (Cohen and Willamson, 1988). A two day test-retest reliability as assessed in college students was 0.85 (Cohen et al., 1983), although longer test-retest correlations are lower, due to the short-term nature of minor stress/stressors. This scale has also been shown to possess reasonable predictive validity: in the same college students' samples it correlated with indices of depressive symptomatology significantly higher than life event measures (0.65 vs. 0.18 respectively).

e). Inflammatory Bowel Disease Activity  (see appendix 6)

To assess the disease activity, an index was used that is solely based on those data in The Crohn's Disease Activity Index (CDAI - Best et al, 1976) which can be recorded directly from the patients (frequency of diarrhoea, abdominal pain, general well being). This index (called DA – for Disease Activity) (von Weitersheim et al, 1992) can be used for patients with Crohn's Disease as well as for patients with Ulcerative Colitis. This index has been found to correlate highly with the full Crohn's Disease Activity Index - r = 0.93 (von Weitersheim et al, 1992), thus indicating that the modified index supplies very similar information.
f). Coping strategies/styles (see appendix 5)

Subjects were asked to complete the Coping with Health, Injuries and Problems (CHIP - Endler et al, 1992, 1993) questionnaire of coping strategies/styles. This multidimensional index, which was developed specifically for coping with health problems, is divided into four factors each with 8 items (Palliative, Instrumental, Distraction, and Negative Emotion). This measure has been validated in various populations including college student, adult, and medical patient groups. In particular relevance to the present study, the medical patient population consisted of - 13.0% described as having severe chronic illnesses (diabetes, cancer, etc.), and 5.9% with gastrointestinal problems. Internal Alpha reliabilities for the three broader groups separately (students/adults/patients) for the four factors were satisfactory, ranging from 0.75 to 0.85. The CHIP also showed construct validity in correlations with other existing coping scales, e.g. with the Coping Inventory for Stressful Situations (CISS - Endler & Parker, 1990a) and the Coping Strategy Indicator (CSI - Amirkhan, 1990) – see Endler et al., 1992 for details.

g). Trait personality. (see appendix 3)

Trait personality was assessed using the NEO-Five Factor Inventory (NEO-FFI - Costa & McCrae, 1992). This 60 item personality inventory indexes five relatively stable and independent factors of trait personality each with 12 items: these are neuroticism; extraversion; openness; agreeableness; and conscientiousness. The reliability of these factors as measured by Cronbach's alpha are good, ranging from
.89 to .95 (Costa & McCrae, 1992). Various factors from this index (in particular the variable neuroticism) have also been shown to have significant predictive validity for stress outcomes. For example, Deary et al (1996) found that the personality trait of neuroticism was significantly related to levels of job stress and occupational burnout (Maslach Burnout Inventory) in a representative sample of 39 psychiatrists from 500 consultant doctors.

h). Locus of control (see appendix 4)

This was assessed using the Multidimensional Health Locus of Control Scale (MHLC) as developed by Wallston et al (1978). This 18 item scale indexes three dimensions of health locus of control: Internality (IHLC); Chance (CHLC); and Powerful Others (PHLC), which may be used separately or combined to identify different subgroups or ‘types’. For example, individuals scoring high on internal and low on powerful others loci of control may be described as a ‘type’. The MHLC has been validated in college students, chronic patients (e.g. Arthritis), healthy adults, and persons engaged in preventive health behaviours, in which the alpha reliabilities range from 0.67 to 0.77 for each of the subscales (Wallston, et al., 1978). The scale also has construct validity when compared and correlated with Levenson’s Multidimensional Locus of Control Scale (Levenson, 1973b), which is also based on three scales measuring beliefs in internality, powerful others, and other/externality.
i). Daily Stress. (see appendix 7)

The same 10 item form of the perceived stress scale (Cohen et al, 1983) mentioned above (see section 5.3.2.d.) was completed by the participants every day. This scale was used in preference to the published daily four item version of this scale for two reasons. First, the reliability of the ten item version is superior to that of the four item version. The internal reliabilities, as assessed by Cronbach's alpha are 0.78 and 0.60 for the PSS-10 and PSS-4 respectively (Cohen et al, 1983). Secondly, it was chosen over the four item version because of the ease of comparison with the baseline and follow-up perceived stress scores that this affords.

j). Daily disease activity index. (see appendix 7)

This measure was taken from a development of the Crohn’s Disease Activity Index (Best et al, 1976), by Garret et al (1991) called The Diary of Crohn’s Disease Symptoms. Subjects reported the number of loose or liquid bowel movements, bloody stools, and the frequency of vomiting which occurred during the previous 24 hour period. Ratings of abdominal pain, joint pain, nausea, and abdominal tenderness were derived from a 5-point scale from 0 (no pain/problem) to 4 (severe pain/problem). Three scores were derived from this instrument: a) Disease Activity Sign Score, i.e., the sum of the item scores for frequency of loose/liquid bowel movements, bloody stools, and vomiting; b) Disease Activity Symptom Score, i.e., the sum of the item scores for abdominal pain, nausea, joint pain, and abdominal tenderness; and c) Disease Activity Global Score, i.e., the sum of the symptom and sign scores.
5.3.3. Procedure.

Subjects were assessed on measures a) through h) (see section 5.3.2.) at the start of the study (time = 0), and again at the end of the study 3 months later (time = 3 months). This provided a three month follow up of these measures for these patients, and also enabled the analysis of the test-retest reliability of some of the more trait like variables (such as trait personality).

For the 28 days preceding the follow-up assessment subjects completed the daily diary consisting of measures i), j) and k) (see section 5.3.2). The timetable of assessments can be seen depicted in the Figure 5.3.2.1. below.
[Note – for 3 out of the eight subjects the follow-up date was actually more like 4, or in one case 5, months after the original assessment. This was due to various factors interrupting the commencement of the diary phase of the study. These included public holidays (Christmas & Hogmanay), illness on my part, and trips abroad by a participant. However, all subjects completed 28 consecutive sequential days of diary.

5.3.4. Statistical analysis.

Due to the small number of participants, statistical analysis of some of the results may be misleading, inaccurate, or invalid. Thus the majority of results reported are descriptive and are presented to be of interest to other researchers who may be considering similar research in the future. It is for this reason that this study is presented as a pilot. Statistical analyses which were facilitated by the large quantity of repeated measures data from the diaries, was performed on a Macintosh® Powerbook 165 personal computer utilising SPSS.X® and Statview® software.
5.4. Results.

5.4.1. Baseline/follow up comparisons.

No significant differences were noted between total sample scores on the variables between baseline assessment and follow-up assessment. This may well be due to the small number of participants in the study, although for the personological variables (neuroticism/extroversion/openness/agreeableness/conscientiousness) no differences were expected as these variables are reported as being relatively stable across time (Costa & McCrae, 1992). Any differences between baseline and follow-up which do exist for the subjects individually can be seen in table 5.4.1.
Table 5.4.1. Subjects' scores on all the assessments made at time = 0 and at time = 3 months (approx.).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Subject</th>
<th>2</th>
<th>3</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>19</th>
<th>20</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perceived Severity</td>
<td>t1/t2</td>
<td>4/3</td>
<td>2/3</td>
<td>4/7</td>
<td>4/3</td>
<td>7/5</td>
<td>5/5</td>
<td>3/5</td>
<td>3/5</td>
</tr>
<tr>
<td>Perceived Stress</td>
<td>t1/t2</td>
<td>12/7</td>
<td>18/2</td>
<td>16/19</td>
<td>16/21</td>
<td>36/23</td>
<td>22/19</td>
<td>24/16</td>
<td>20/25</td>
</tr>
<tr>
<td>N</td>
<td>t1/t2</td>
<td>27/25</td>
<td>22/25</td>
<td>23/22</td>
<td>7/30</td>
<td>28/24</td>
<td>32/36</td>
<td>22/28</td>
<td>31/36</td>
</tr>
<tr>
<td>E</td>
<td>t1/t2</td>
<td>19/21</td>
<td>21/20</td>
<td>29/18</td>
<td>38/18</td>
<td>32/22</td>
<td>23/22</td>
<td>32/30</td>
<td>23/23</td>
</tr>
<tr>
<td>O</td>
<td>t1/t2</td>
<td>19/22</td>
<td>32/31</td>
<td>24/24</td>
<td>28/22</td>
<td>34/24</td>
<td>17/17</td>
<td>24/20</td>
<td>26/29</td>
</tr>
<tr>
<td>A</td>
<td>t1/t2</td>
<td>28/29</td>
<td>37/42</td>
<td>31/37</td>
<td>31/32</td>
<td>26/27</td>
<td>32/33</td>
<td>37/36</td>
<td>35/35</td>
</tr>
<tr>
<td>C</td>
<td>t1/t2</td>
<td>38/37</td>
<td>23/20</td>
<td>32/29</td>
<td>32/31</td>
<td>36/31</td>
<td>39/42</td>
<td>32/33</td>
<td>26/29</td>
</tr>
<tr>
<td>IHLC</td>
<td>t1/t2</td>
<td>23/21</td>
<td>23/27</td>
<td>29/24</td>
<td>24/12</td>
<td>14/33</td>
<td>23/18</td>
<td>28/18</td>
<td>23/24</td>
</tr>
<tr>
<td>PHLC</td>
<td>t1/t2</td>
<td>14/15</td>
<td>18/18</td>
<td>14/20</td>
<td>15/23</td>
<td>9/26</td>
<td>17/19</td>
<td>22/15</td>
<td>22/24</td>
</tr>
<tr>
<td>CHLC</td>
<td>t1/t2</td>
<td>23/20</td>
<td>21/21</td>
<td>15/15</td>
<td>15/12</td>
<td>22/21</td>
<td>10/18</td>
<td>23/23</td>
<td>17/24</td>
</tr>
<tr>
<td>Palliative</td>
<td>t1/t2</td>
<td>21/22</td>
<td>27/32</td>
<td>21/20</td>
<td>24/26</td>
<td>33/24</td>
<td>24/30</td>
<td>24/29</td>
<td>38/25</td>
</tr>
<tr>
<td>Instrumental</td>
<td>t1/t2</td>
<td>19/24</td>
<td>35/39</td>
<td>28/21</td>
<td>35/34</td>
<td>26/29</td>
<td>34/27</td>
<td>39/30</td>
<td>31/29</td>
</tr>
<tr>
<td>Distraction</td>
<td>t1/t2</td>
<td>15/17</td>
<td>24/26</td>
<td>23/17</td>
<td>27/20</td>
<td>34/21</td>
<td>19/21</td>
<td>31/24</td>
<td>20/21</td>
</tr>
<tr>
<td>Negative Emotion</td>
<td>t1/t2</td>
<td>8/9</td>
<td>12/12</td>
<td>10/7</td>
<td>16/9</td>
<td>19/12</td>
<td>16/19</td>
<td>13/11</td>
<td>13/16</td>
</tr>
<tr>
<td>No. Soft Stools</td>
<td>t1/t2</td>
<td>5/2</td>
<td>7/4</td>
<td>10/5</td>
<td>15/20</td>
<td>30/22</td>
<td>15/20</td>
<td>6/4</td>
<td>10/50</td>
</tr>
<tr>
<td>Abdominal Pain</td>
<td>t1/t2</td>
<td>1/1</td>
<td>0/1</td>
<td>2/2</td>
<td>1/2</td>
<td>2/1</td>
<td>2/2</td>
<td>2/1</td>
<td>1/3</td>
</tr>
<tr>
<td>Well Being</td>
<td>t1/t2</td>
<td>1/0</td>
<td>1/1</td>
<td>1/2</td>
<td>2/2</td>
<td>2/1</td>
<td>2/2</td>
<td>1/0</td>
<td>1/5</td>
</tr>
</tbody>
</table>

5.4.2. Daily Diary Results.

The daily diary scores for all subjects on the variables perceived stress, mood, and disease activity can be seen in sections 5.4.2.1.a. through h. (see below). The reader is reminded that for each of these graphs the y-axis scale may differ. This is important to remember if comparisons between the cases are to be attempted. However, for ease of interpretation the reader should note the extent of each scale represented here: the perceived stress scale is measured out of a possible maximum of 40 (the higher the score the greater the level of perceived stress); the mood is a
self rating from 1-10 (1 indicating very poor mood, 10 indicating very good mood); the disease activity scale is a composite of the disease symptoms scale (min = 0, max = 16) and the disease signs scale for which responses may vary considerably (i.e. subjects are asked to indicate the absolute number of loose, liquid or bloody stools and or vomiting) therefore no maximum or minimum of this scale is possible.
5.4.2.1. Descriptive results for individual subjects' daily diary scores.
a). Subject – 2: a 60-year-old male with Ulcerative Colitis.

Overall, for this subject there appeared to be a stronger link between perceived stress scores and mood self ratings, than between perceived stress and disease activity (although some links can be seen between perceived stress and disease activity). This link between perceived stress and mood can be seen by looking at the graph at days 5, 7 & 8, 22, and 26. At day 5 this subject reported a greatly reduced mood and a much higher level of perceived stress. Rises in perceived stress and reduced mood scores can also be seen at days 7 & 8, 22, and 26, although these are not as marked as for day 5. Regarding the link between perceived stress and disease activity, some minor evidence of this can be seen around days 7-9, days 22 & 23, and day 26. On days 7-9, it appears that disease activity increases a couple of days after an increased experience of perceived stress. The same pattern can be seen on days 22 & 23. On day 26, however, there is no time lag to the increased disease activity, and both scores for this and perceived stress increase on the same day. Finally on day 13, a marked increase in disease activity is linked with a marked decrease in reported mood.
b). Subject – 7: a 50-year-old male with Crohn’s Disease.

This subject’s reported perceived stress levels for the 28 days varied greatly – implying the sensitivity of this measure. The other measures, however, did not vary very much at all. Most notable possible linkage between the scales can be seen on day 23, where an increase in perceived stress is accompanied by a slight increase in disease activity score.
This subject's scores were quite interesting. Most notable of these are the results for days 8 through 13. During this period, an increase in perceived stress on days 9 and 10 is accompanied by an increase in disease activity scores on day 10 and more markedly on days 11 and 12 - a couple of days after the increase in perceived stress. Similarly, on days 10 and 11 a decreased mood rating is reported by the subject.

Also, an increase in perceived stress on day 16 is accompanied by an increase in disease activity on day 17, although no decrease in mood is noted for these days.

Concerning that issue, the consistently very high mood ratings of the subject for days 13 to 25, and the lack of variation in these scores despite variation in perceived stress and disease activity, may indeed be genuine, but may also indicate some reporting error. The subject may have become bored with diary completion, may have become habituated to high mood reporting, may have not completed that section of the dairy for those days and filled it in after the event, may have been 'dispositionally optomistic' (see Carver et al, 1989), may have been experiencing a high mood rebound from previous stress (see later), or may even have been that happy for that extended period of time.
d). Subject - 10: a 51-year-old female with Crohn’s Disease.

This subject showed much more variation in their scores across the 28 days than did the previous one. She reported two very high peaks of perceived stress on days 7 and 24. Each of these was associated with decreased mood and increased disease activity on or just after the increase in perceived stress. Similarly, at the end of the diary period on days 26 and 27, increased perceived stress is accompanied by an increasing disease activity score, at which point measurement ends. Overall, this subject appears to exhibit good linkage between extreme perceived stress, mood ratings and disease activity.

This subject has an interesting disease activity episode on day 22. The disease activity score for this day is very high, and is accompanied by a high level of perceived stress, and a decreased mood - all on the same day. Other than this day, which may well be exceptional, this subject's perceived stress scores and disease activity scores appear to be well linked. For example on days 2, 5, 11, and 26, an increase in perceived stress is followed a couple of days afterwards by an increase in disease activity. Also there may be two sorts of relationship occurring here: First, Concurrent (which could be reactive – disease activity increase raises perceived stress); and second delayed (stress inducing increase in disease activity).
Subject 20 reported levels of perceived stress and disease activity that also appeared to be well linked to one another. On day 4 an increase in perceived stress is associated with an increase in disease activity. The steady rise of perceived stress from day 6 to 14 is also associated with a steady rise in disease activity, which tails off at the end of this period. Towards the end of the diary period (days 24 to 28) a rise then decrease in perceived stress is mirrored by a similar rise then decrease in disease activity. Mood ratings appear to be related to perceived stress on day 15 only, where a higher level of perceived stress is associated with a drop in mood rating, and no associated change in disease activity.
g). Subject – 8: a 75-year-old male with Ulcerative Colitis.

Subject 8 reported large variation in perceived stress scores, but this did not appear to be associated with any concomitant changes in mood or disease activity. Links between perceived stress and disease activity for this subject are tentative. However, between days 4 and 7 a release from stress is closely followed by a reduction in disease activity. Also on day 15 a peak in perceived stress is associated with a synchronous trough in disease activity. This is of interest as it is opposite to what would be expected.

There are many interesting points to raise from this subject’s diary scores. First, a decrease in perceived stress on day 8 is associated with an improved mood rating for this day. Secondly, the increasing stress reported following this may well be associated with the increase in disease activity on day 10 and a falling mood during this period. Similarly on day 20 a small increase in perceived stress may be associated with the small increase in disease activity the following day. Thirdly, the peak of perceived stress reported on day 24 may be associated with the increasing disease activity ratings 2-3 days after.
Overall, the results from the daily diaries may go some way to providing evidence for links between the variables perceived stress, disease activity, and mood, although this was not reflected by all the subjects' scores for the full course of their diaries. As mentioned earlier, various reasons for this may exist including high demands on the subjects' time and effort resulting in inaccurate or polemic responding, possible individual differences in levels of somatising experienced stress, or specific stressors having specific effects (see discussion).

Also, no discernible differences/similarities could be seen between the two disease diagnoses, or between male and female participants, although this does not necessarily mean that these do not exist. The between group differences may be obscured by the within-groups individual differences.

5.5. Discussion.

Due to the small number of participants, staunch theoretical standpoints based on the available data are difficult to take. Therefore, the potential implications that data, as presented here, may offer to the field of research, will be discussed briefly, with further discussion and integration of this area in chapter 6.
5.5.1. Baseline – follow-up comparisons.

Although no significant differences were noticed between subjects’ scores at baseline and at follow-up, this does not necessarily support (or refute) any theories based on the stability over time of these measures. The lack of differences may be due to the stability of these measures (as would be hypothesised for the more trait like variables); but it is just as likely that real differences exist between subjects scores at baseline and follow-up but that the number of participants prohibits meaningful statistical analysis of these possible differences. Clearly, however, given previous research stating the importance of antecedent personological variables in acting as potential benchmarks for the assessment of individual differences in the stress-mood-outcome relationship (Adler & Mathews, 1994; Watson & Pennebaker, 1989), the value of including them in future research is emphasised. Similarly, given the role that mediating control cognitions and styles of coping are theorised as playing in health research (Stone et al, 1993; Endler et al, 1992), their inclusion in future research is also emphasised.

It may be, therefore, that stressors affect certain individuals (as assessed by personological variables) to react negatively in terms of mood and illness experience, and that this may be orchestrated by control cognitions and coping strategies – i.e. the Transactional model of stress and illness (Lazarus & Folkman, 1987). If these variables are not assessed, however, the important role that they may play could be missed – it is very difficult to explain a no-result finding in terms of individual differences when these are not assessed.
5.5.2. The stress-illness-mood relationship.

It appears from the daily data, that some form of linking between perceived stress, mood, and disease experience does exist. This is, however, more strongly supported by some individuals’ scores than by others, and not at all by others. Reasons for this may be numerous. First, it is possible that individual differences exist in reactions to stressors. It would be hypothesised from the literature that individuals with higher neuroticism scores, more negative coping styles – like negative emotion focused coping - and external loci of control, are more likely to be ‘stress responders’. Secondly, it is possible that particular categories of stressor are more likely to elicit negative reactions in terms of mood and illness reporting, and that in the sampling time frame, some individuals in the study did not experience these. This is a limitation of using the particular perceived stress scale used, as opposed to an event checklist, as well as usual limitations on prospective longitudinal research. Thirdly, from some subjects’ scores it may be argued that floor and ceiling effects on some of the measures meant that the measures weren’t sensitive enough to pick up the linked variance changes. Fourthly (similar to the first point), different individuals may experience variations in disease activity for different reasons. i.e. disease activity may increase because of antecedent stress (and/or the individual being a stress responder), but disease activity may increase or decrease independently of stress (even for stress responders) due to environmental changes – diet, pathogens, day-to-day changes in health behaviours, etc.. Fifthly, daily changes in disease activity, mood, and stress may be totally independent of each other, and thus, by chance, some individuals exhibit changes in one approximately around the
same time as changes in another. This seems to be unlikely (given previous research), although it is possible. Another interesting point to bring up here can be seen by looking at the results from subject 9’s scores. On days 19 – 22, the subject’s scores on perceived stress increase then decrease as disease activity increase. It is possible then that the subject is somatising her stress (hence the increase in disease activity) and that this relieves her stress (hence the decrease in stress after the disease activity scores have increased). Could this be the ‘purpose’ of the link between stress and illness?

Clearly these are many issues which need to be resolved if a clearer picture of the nature of the stress-illness-mood link is to be obtained. Future researchers should bear some, if not all, of these issues in mind when designing and conducting their future research.

5.5.3. Methodological considerations.

As mentioned earlier, a balance needs to be reached for the researchers’ and the subjects’ (not inexhaustible) time and effort. When trying to design a longitudinal prospective study of this type, researchers need to be conscious of their own limitations, what reasonable demands can be placed on the participants in the study, as well as trying to maximise the quality of the research.

Similarly, the problems of floor and ceiling effects, habituation, and autocorrelation, should be considered fully when planning research which entails repeated daily
measures. Thus reliable and valid measures that combat this effect should be employed / developed.

a). Time issues.

The results on time series analysis are equivocal. For example, although some, albeit few, studies provide evidence for a prolonged, or lagged effect of stress on negative mood, there is also some evidence of a rebound effect on positive mood on the day following stress (Stone et al, 1993). Similar to this, the work performed by Greene, Blanchard, and colleagues (1994), although strongly supporting a link between stress and illness in inflammatory bowel disease, found that it is not a simple matter. They found a marked negative effect of previous month on inflammatory bowel disease symptomatology. This suggests a monthly rebound effect, whereby the month succeeding a stressful month certain subjects, experienced a marked pickup in wellness. This has implications when trying to analyse temporally dynamic data. Having to distinguish between subjects experiencing worsened disease activity due to stress and those who are experiencing a rebound effect, when data is grouped, may go some way to explaining small and variable effect sizes. It may also help explain why some researchers have found what they have called stress-responders – they may just have caught those who are reacting negatively to stress at that particular moment in time. In any cross-section of any disease population, there must be times when there are certain individuals who are on the downward slope (i.e. their stress is causing their illness to worsen) and those that are quite the opposite. The experiences of individuals have also been shown to not be static over time, thus it is entirely possible that at any time (or indeed over a period of time – this problem is
not limited to cross-sectional research) a researcher assessing a representative sample of the population will receive a hotchpotch of stress responses (positive, negative, and null), which may be different from how the same subjects will respond some time down the line. Is it surprising, then, that the literature in this area is equivocal? Clearly some consideration of these problems needs to be taken, when conducting research of this kind.

b). Time series analysis.

In order to attempt to provide solutions to some of these problems it would be necessary to employ a reasonable and reliable time series analyses to facilitate interpretation of daily results (see West & Hepworth, 1991). Ideally some form of analysis that could take into account possible individual differences in trait psychology prior to diary completion, and use this information in attempting to analyse the patterns of putative linked variables should be developed/employed. This would enable researchers to address issues like time-lagged effects of one variable on another, given that results at present are equivocal. Akin to many of the other areas of psychology already explored in this thesis there is almost an embarrassment of techniques that the psychologist can choose from when wanting to employ some form of time series /temporal analysis (Box-Jenkins technique, ARIMA, Granger- causality analysis, etc..). Any study wanting to take the format of this pilot and make it into a full blown study could do worse than employ a Granger-causality time series framework. This would enable the testing of specific causal determinations between the putative causal agents – via structured equation modelling. Therefore causal equations would be established from either the bivariate relationships of the
variables, or from the literature, these would then be combined to form structural
equations to predict the outcome variable, in this case (perceived) disease activity. It
must be noted, however, that time series analysis is a rapidly developing area, both in
terms of the numbers of studies now using it in some form or other, and
consequently the techniques available for use, and there are many existing
developments being made daily that need to be considered before embarking on a
study of this type.

c). Day-of-the-week.

Another issue that should be mentioned is the ‘day-of-the-week effect’ (Stone et al,
1993). We are all aware that as individuals we routinely experience very different
forms of stress and stressors at different times during the week. For example, the
events or situation that cause us stress are likely to be very different at the weekend
as compared to during the normal working week. Inherent in this is the idea that
certain stressors, or categories of stressful situations, may have differential effects in
their influence on mood, health or both. For example, Clark and Watson (1988)
found that positive affect and negative affect were differentially predicted by
categories of events. Social activities were the strongest predictors of positive affect,
but were unrelated to negative affect; work events and irritants were related to
negative affect. It is quite plausible, therefore, that the events which are work or
socially related (in this example) will have different frequencies during different times
of the week. Interestingly, the belief of many in more negative moods on Mondays,
was not supported by a study by Stone et al (1985), where subjects’ prospective data
did not distinguish Mondays as particularly stressful, although the subjects still
believed this to be the case. These day-of-the-week effects may also be very different for certain groups of individuals. For example, employment status, hours worked (e.g. shift/normal/night), style of work, etc., may have large effects on how much the day-of-the-week influences the relationship between stress, mood and illness, and should be considered in this type of research.

d). Measure Choice.

As mentioned earlier, another issue is the type of stress/stressor measure chosen for daily assessment. Stone et al (1993) discuss the difference between measuring perceived stress experienced for that day, and an event checklist of ‘hassles’ (with or without a rating scale of how stressful the events were to the individual). Although a lot of findings have been very similar for measures of daily perceived stress and events experienced, there may be an important difference between the two. Both perceived and event stress relate to negative mood – the greater the stress, the greater the negative mood. However, only stress conceptualised through actual events is related (inversely) to positive mood. This may be due to appraisals of stress being conceptually indistinct from (negative) mood measures for the individual and/or the researcher. Event checklists are less prone to this problem, although they do not provide as much information about the appraisal of stress. With the increase in palm-top computing technology, attempts are being made to record by self report stressful events and appraisals of these events as and when they actually happen (Shiffman et al, 1992), which may serve to add further insight to this issue. It needs to be remembered, however, that there may be substantial bias in who agrees
to take part in a very intensive and demanding study like this, as well as in the compliance rates when subjects have agreed to take part.

Given the temporal dynamism of the relationship between stress, mood, and outcome, which is hinted at by the results from this study, and possible individual differences in this relationship, and the potential methodological pitfalls that these encompass, and the difficulties associated with appropriate measure and methodology choice, researchers should be wary of seeing longitudinal prospective studies as the panacea for all the ills of previous research. In other words, although certain forms of research paradigm can negate methodological limitations from others, they can, and do, generate their own problems, which must also be considered.

5.5.4. Conclusions.

We have seen, then, that it appears that stress and illness may be linked dynamically over time in these disease populations, and also that the strength (and even direction) of this relationship may differ across time, across stressful situation categories, across differing individual personological characteristics, even across different days of the week. Clearly this is a minefield if meaningful interpretation of research results is to be attempted.
A definitive study would have high demands: demands on appropriate robust methodological techniques; demands on quality of data – many patients to be assessed intensively over a long period of time; the resultant demands this has on the participants of the study; demands on analytical tools that can cope with analysing individual differences over time when the very day of the week may have an effect – clearly from the discussion above of methodological issues there are many other variables to also be taken into account in the analysis; and last, but by no means least importantly, the financial / resource demands that carrying out such a research study need and deserve.

Clearly, there are many issues which future researchers should bear in mind when considering research of this kind, which have already been mentioned. This type of research also has wider implications for previous research, on the main study mentioned earlier, and on issues of health psychology in general. These issues will be discussed in the conclusions.
CHAPTER 6: Conclusions.

6.1. Research questions answered?

6.2. Implications for further research.

6.3. Implications for health professional practice.

6.4. Final conclusions.
6. Conclusions.

From the main cross-sectional study, we have seen that psychological factors, such as perceived stress, perceived severity, and neuroticism, can be telling when predicting individuals' experiences of disease activity – be they experiences of disease specific pain, or more general feelings of well being. We have also seen, from the pilot of the longitudinal study, that there is tentative descriptive evidence supporting a temporal link between perceived stress and disease activity in these disease populations. This provides an answer for the main question of a link between perceived stress and disease activity, but can answers be proposed for the other research questions that were set at the beginning of the research?

6.1. Research questions answered?

Research questions.

1. What are the characteristics of the two inflammatory bowel disease diagnostic groups, Crohn's Disease and Ulcerative Colitis, in terms of demographic, behavioural or psychological factors, or experiences of disease activity? What are the similarities and differences between these two disease cohorts for these factors?

We have seen that the two disease cohorts do not differ significantly from each other for the demographic behavioural factors (excepting smoking behaviour), and the psychological factors. In terms of experiences of disease activity, the two groups only differed on experiences of well being. We have also found that these results are not unusual for these disease groups - some researchers believe these two disease diagnostic groups are just different manifestations of a greater inflammatory
bowl disease (Whitehead & Shuster, 1985). For future research this may mean that it is methodologically acceptable to perform analysis on the two diseases together, thus essentially doubling the potential sample size. If analysis was to be performed on inflammatory bowel disease patients as a whole, then it would be necessary to firstly confirm that no differences exist between the diseases. For example in the main study reported here, although generally speaking there were little or no differences between the disease groups (excepting smoking behaviour and well being ratings), there were interesting comparative differences for the correlations between the major variables that might provide some important insights. If analysis of inflammatory bowel disease patients as a whole was performed these might be missed. Similarly it has been discussed previously that differences between individuals within a population (e.g. stress responders vs. stress non-responders – or high neuroticism vs. low neuroticism), might be much more telling than group differences between populations (e.g. male vs. female – Crohn’s disease patients vs. Ulcerative colitis patients). Therefore, although it may be acceptable to put the two disease groups together for the purposes of analysis, care is needed to not miss, or at least be aware of the potential for, important group differences.

2. Similarly are there any gender differences in these disease populations in terms of demographic, behavioural or psychological factors, or experiences of disease activity?

We have seen that several gender differences do exist between the men and women in the overall sample, including employment, exercise, perceived stress, distraction oriented coping, neuroticism, overall disease activity, abdominal pain experiences, and well being experiences. We have also seen that some of these gender
differences are not particularly out of the ordinary, e.g. neuroticism scores are commonly found to differ across sex. Therefore we can already see that certain within subject analyses may be more important that certain between subject analyses. Similarly to research question 1, addressed above, this may mean that individual differences in neuroticism, for example, might be more important in overall disease experience and/or outcome than gender, or disease diagnosis.

3. Are there any interaction effects between disease cohort and gender in terms of demographic, behavioural or psychological factors, or experiences of disease activity?

The answer to this research question was no. The lack of any interaction effects between disease cohort and gender may suggest to further research that the idea of analysing inflammatory bowel disease patients in the four groups of male-Crohn’s Disease, female-Crohn’s Disease, male-Ulcerative Colitis, and female-Ulcerative Colitis, and expecting differences between these groups, may not yield any interesting results.

4. Is there a link between stress and illness in the inflammatory bowel diseases Ulcerative Colitis and Crohn’s disease?

We have seen that perceived stress is an important consideration when analysing the factors that affect disease experiences for both these groups. i.e. there is a link between perceived stress and these disease groups, but also that the establishment of causal direction is still unknown, although the results from the pilot section shows
that increase in perceived stress may precede disease activity increase, and therefore implies this direction of causation.

5. How do the factors that are normally associated with this link (given that it exists) relate to themselves?

Do they support a transactional, a latent variable, a combination of the two, or another, model of the stress and illness relationship?

We have seen that many of the factors associated with the stress-illness link do in fact link to each other also. It has been discussed that this may be a result of something called the omnibus effect (if enough correlations are calculated then some will be statistically significant by chance alone) or, alternatively, may be caused by the non-independence of many of the measures that psychologists have at their disposal, which has also been discussed. Thus, to differentiate between these two problems and ‘real’ links between these factors, is difficult and needs further work. It has also become clearer, from the different aspects of the results, that to use the results to support, or refute, either the Transactional or the latent variable Model of stress and illness is difficult. It has also been discussed that maybe a model that is comprised of a combination of these two models of stress and illness may provide the ‘truest’ picture of the link between stress and illness in inflammatory bowel diseases. Clearly further research that is designed specifically to compare the best models of the stress-illness relationship in sufferers of these diseases, would be useful.

6. What factors (demographic, behavioural, psychological, clinical) are useful in predicting disease activity?

What roles do these factors play in predicting disease outcome?
We have seen that the factors that are useful in predicting disease include perceived stress, neuroticism, perceived disease severity, and to a lesser degree some aspects of locus of control. We have also seen that similar to research question 5, to use these results to provide support for either of the two models of stress and illness, is not unproblematic. Similarly more research is necessary here. Importantly we have seen that psychological factors, most notably perceived stress and neuroticism are useful in predicting disease activity over and above certain clinical indices. Therefore carers for patients with inflammatory bowel diseases, the patients themselves, and clinicians supervising their care, should note that there may be more to experiencing these diseases than the clinical factors alone.

6.2. Implications for further research.

The old adage, further research is necessary, is a cliché, and is one of the ways researchers can propagate further research grants, with which to appease their departments, and be well represented on research audit reports. In this instance, however, this old adage holds true, and is not just a job creation scheme. It can be seen from the responses to the research questions set, and the responses that this research has provided for these, that future research should take these considerations on board and work with them to provide a better bank of knowledge surrounding research into psychological factors in these diseases. In preparing this research project, a need was established for a definitive piece of work that addressed the issue of the stress-illness link in this population. The primary aim of the work being to improve on previous research and not to fall into the pitfalls of this other research. Of course, as in all research, some of the pitfalls were avoided.
but other ones were not. Indeed it could even be claimed that new pitfalls have been found to fall into a-fresh. Thus, although the research has helped to address a few issues, it has equally brought up many more that similarly need to be addressed by further research. For example, the results of the daily assessment of disease activity, perceived stress, and mood, suggest the importance of mood as a factor in the stress-illness relationship. Mood was not assessed in the main cross-sectional study, where it perhaps might have provided extra insight into the nature of how stress and illness are related. Future research should consider the inclusion of this factor. Although previous sections have discussed future research implications in some detail, the importance of the role of personality must be noted (either explicitly in terms of personality measures themselves, most notably neuroticism, or more globally in terms of ideas of negative affectivity – which may include various factors relating to personality).

6.3. Implications for health professional practice.

Clearly the desired end point of research into any particular illness, is to generate suggestions, implementations, treatments, and/or regimes that can hopefully alleviate the conditions of the illness for the sufferers. It is easy for those involved in discovering or expanding on the minutia of a particular area to forget this; we need to be reminded to attempt to ease the patients’ dis-ease. In this respect, the current research has generated several implications for people involved in the care and treatment of patients with inflammatory bowel disease as well as for the patients themselves.
As is mentioned in the introduction to this thesis, twentieth century medicine is progressing and is beginning to accept that what constitutes wellness, or for that fact illness, is a complicated matter, and one that cannot easily be distilled to several causative pathogens. This is not to say that modern science has not helped us live longer more fulfilled lives, but that the shift of modern medicine to the promotion of good health, and the alleviation of chronic disease experience is essential. This is particularly evident when considering diseases or conditions that at present we are unable to cure or prevent (e.g. Crohn’s disease and ulcerative colitis). We all need to work together, considering the individual as a whole, and not just the sum of their signs and symptoms, in order to provide the best possible care / treatment that we can (whether this be biological, psychological, behavioural, a mix of all of these, or whatever).

More specifically, we have seen that certain psychological factors assessed here (most notably neuroticism, perceived stress, and perceived disease severity) do significantly predict disease experience outcomes, which should also be considered. The idea is that if it is possible to alter these putative causes of some of the variance in disease experience, then it is possible to alleviate at least some of the negative experiences of disease activity (like perceptions of pain and well being).

Neuroticism, by definition, is a personality trait, and therefore is defined as being a relatively stable factor across time and is resistant to change. This assertion has been empirically backed up (see Costa & McCrae, 1992; Deary & Mathews, 1993).
and it does appear that this is the case. We shall need to look elsewhere in the process for potential intervention sites.

As opposed to neuroticism, the perceived stress scale (Cohen & Cohen, 1983) was designed as a state measure, that could easily be used to assess changing perceived stress over time. It appears that this may be tapping into a changeable, and therefore potentially alterable, underlying factor. Thus if it was possible to alter an individuals' level of perceived stress, we could potentially change his/her disease experience in a positive way. There are several ways this could be performed:

- Firstly, the attempt could be made to reduce the quantity and extent of actual stressors that the individual experiences. To do this the individual would need to try and remove themselves from potentially stressful situations – clearly this is not always possible, and in today’s society is becoming increasingly difficult.

- Second, given the inability to remove or reduce the stressors experienced by the individual, then attempts could be made to reduce the individuals’ reactions to these stressors. This could be done by embarking on stress management courses, cognitive behavioural therapy, relaxation therapy, cognitive restructuring, etc., - we have already seen some attempts to perform research on such an intervention in inflammatory bowel disease (Milne et al, 1986; Blanchard, 1991 – see section 1.2.3.3.) with mixed results; perhaps further research would clarify whether this was a viable and realistic way to relieve patients’ suffering.
Third, given that previous research has found individuals’ coping styles/strategies to be important variables in the mediation of the stress-illness relation (see section 1.1.2.1.a), then more appropriate coping styles could be taught. This could concentrate on teaching coping styles that are more practical in nature (like task oriented coping) as opposed to emotional based coping styles.

Similarly, if perceived disease severity is a real predictor, and not just measuring the same thing as perceived disease activity, then attempts can be made to increase communication between medical professional and patient, so the patient can acquire a more realistic perception of their disease activity. Thus, hopefully, alleviating some of the patients’ worry about their health, and therefore improving their disease experience.

6.4. Final conclusions.

In final conclusion there are a few points that need to be made. First, the need for future research is reiterated. This is greatly facilitated by departments of gastrointestinal medicine that acknowledge the role that psychological factors play in experiences and appraisals of disease activity. Similarly, research into these areas is also facilitated by the commitment and compliance of the individuals who participate in research of this kind. In this respect, I found the participants in the current study to be very accommodating, and also very pleased and keen to participate in the research to the best of their ability.
Secondly, in order to facilitate meaningful research into the role psychological factors (stress, personality, etc.), may play in health, health psychologists need to improve their measurement tools. We do not need more constructs, we need a consolidation of what the existing constructs mean. We need to know what it is these measures are indeed measuring and hopefully this will inform future research as to how to use fewer constructs more efficiently, to say more.

Finally, and probably most importantly, it needs to be reiterated that psychological factors DO play a role in experience of chronic disease, in this case inflammatory bowel disease. This should be remembered at all times by people who treat/care for/live with people with chronic disease. This again emphasises that a holistic approach to medicine might be one way that we, as health psychologists, but also as individuals entering into a new millennium, can improve the health and wellness of the population as a whole, as we try to deal with diseases/conditions/illnesses that are increasingly involved with behaviour and psychological factors. That is, we need to ensure that investment is continued in the pursuit of knowledge of treating diseases that have more holistic origins. In doing so, valuable insights gained from the examination of the psycho-behavioural side of ill-health, can help us to aid the promotion and propagation of good health.
Appendices.

Appendix 1
(Demographic Details and Health Behaviours Questionnaire)

Appendix 2
(Perceived Stress Scale – PSS-10)

Appendix 3
(NEO Five Factor Inventory – NEO-FFI)

Appendix 4
(Multidimensional Health Locus of Control Scale (form A) – MHLC)

Appendix 5
(Coping with Health, Injuries and Problems – CHIP-5)

Appendix 6
(Disease Activity)

Appendix 7
(Daily Diary)

Appendix 8
(Informed consent/information sheet)

Appendix 9
(Oscar Wilde Quote)
APPENDIX 1

Demographic Details and Health Behaviours Questionnaire.

Sex (please tick): Male _____ Female _____

Age: ___________

Employment Status (please tick):
- employed full time _____
- employed part time _____
- unemployed _____
- retired _____
- student _____
- other _____
(please specify) ______________________________

Marital Status (please tick):
- Married ____ Single _____
- Divorced ____ Widowed _____

Do you take exercise? (please tick):
(this does not include simply walking from a to b, purposeful vigorous exercise is what is meant).
- Never _____
- Once a month _____
- Once a week _____
- More than once a week. _____
- Every day _____

Do you smoke? (please tick)
- Yes _____ (if so, how many per day?) ____
- No _____
- Ex-smoker _____ (how long since stopped?) ____years

Would you say you had a healthy balanced diet? (please tick)
- Yes _____
- No _____

How long ago were you diagnosed as having Ulcerative Colitis or Crohn’s disease?
- _______years/months

On a scale of 1-10 (1 not at all severe, 10 extremely severe)
how would you rate the severity of your inflammatory bowel disease? ________
APPENDIX 2

Perceived Stress Scale.
(PSS-10)

Instructions.
The questions in this scale ask you about your feelings and thoughts during the last month. In each case, you will be asked to indicate how often you felt or thought a certain way. Although some of the questions are similar, there are differences between them and you should treat each one as a separate question. The best approach is to answer each question fairly quickly. That is, don’t try to count up the number of times you felt a particular way, but rather indicate the alternative that seems like a reasonable estimate.
For each question choose from the following alternatives:

0 = never
1 = almost never
2 = sometimes
3 = fairly often
4 = very often

1. In the last month, how often have you been upset because of something that happened unexpectedly?

2. In the last month, how often have you felt that you were unable to control the important things in your life?

3. In the last month, how often have you felt nervous or stressed?

4. In the last month, how often have you felt confident about your ability to handle your personal problems?

5. In the last month, how often have you felt that things were going your way?

6. In the last month, how often have you found that you could not cope with all the things you had to do?

7. In the last month, how often have you been able to control irritations in your life?

8. In the last month, how often have you felt that you were on top of things?

9. In the last month, how often have you been angered because of things that happened that were outside of your control?

10. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?
NEO Five Factor Inventory

Form S

Paul T. Costa, Jr., PhD., and Robert R. McCrae, PhD.

Instructions:

Carefully read all the instructions before beginning. This questionnaire contains 60 statements. Read each statement carefully. For each statement circle the response that best represents your opinion.

Circle SD if you strongly disagree or the statement is definitely false.

Circle D if you disagree or the statement is mostly false.

Circle N if you are neutral on the statement, you cannot decide or the statement is equally true and false.

Circle A if you agree or the statement is mostly true.

Circle SA if you strongly agree or the statement is definitely true.

For example, if you strongly disagree or believe that a statement is definitely false, you would circle SD for that statement.

Fill in only one response for each statement. Respond to all of the statements, making sure that you fill in the correct response. If you need to change an answer, make an ‘X’ through the incorrect response and then fill in the correct response.
<table>
<thead>
<tr>
<th></th>
<th>Strongly Disagree</th>
<th>Disagree</th>
<th>Neutral</th>
<th>Agree</th>
<th>Strongly Agree</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>I am not a worrier.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>2</td>
<td>I like to have a lot of people around me.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>3</td>
<td>I don't like to waste my time daydreaming.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>4</td>
<td>I try to be courteous to everyone I meet.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>5</td>
<td>I keep my belongings clean and neat.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>6</td>
<td>I often feel inferior to others.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>7</td>
<td>I laugh easily.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>8</td>
<td>Once I find the right way to do something, I stick to it.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>9</td>
<td>I often get into arguments with my family and co-workers.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>10</td>
<td>I'm pretty good about pacing myself so as to get things done on time.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>11</td>
<td>When I'm under a great deal of stress, sometimes I feel like I'm going to pieces.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>12</td>
<td>I don't consider myself especially &quot;light-hearted&quot;.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>13</td>
<td>I am intrigued by the patterns I find in art and nature.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>14</td>
<td>Some people think I'm selfish and egotistical.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>15</td>
<td>I am not a very methodological person.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>16</td>
<td>I rarely feel lonely or blue.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>17</td>
<td>I really enjoy talking to people.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>18</td>
<td>I believe letting students hear controversial speakers can only confuse and mislead them.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>19</td>
<td>I would rather cooperate with others than compete with them.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>20</td>
<td>I try to perform all the tasks assigned to me conscientiously.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>21</td>
<td>I often feel tense and jittery.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>22</td>
<td>I like to be where the action is.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>23</td>
<td>Poetry has little or no effect on me.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>24</td>
<td>I tend to be cynical and sceptical of others' intentions.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>25</td>
<td>I have a clear set of goals and work towards them in an orderly fashion.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>26</td>
<td>Sometimes I feel completely worthless.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>27</td>
<td>I usually prefer to do things alone.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>28</td>
<td>I often try new and foreign foods.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>29</td>
<td>I believe that most people will take advantage of you if you let them.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>30</td>
<td>I waste a lot of time before settling down to work.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>31</td>
<td>I rarely feel fearful or anxious.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>32</td>
<td>I often feel as if I'm bursting with energy.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>33</td>
<td>I seldom notice the moods or feelings that different environments produce.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>34</td>
<td>Most people I know like me.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>35</td>
<td>I work hard to accomplish my goals.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>36</td>
<td>I often get angry at the way people treat me.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>37</td>
<td>I am a cheerful high-spirited person.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>38</td>
<td>I believe we should look to our religious authorities for decisions on moral issues.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>39</td>
<td>Some people think of me as cold and calculating.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
</tr>
<tr>
<td>Statement</td>
<td>Strongly Disagree</td>
<td>Disagree</td>
<td>Neutral</td>
<td>Agree</td>
<td>Strongly Agree</td>
</tr>
<tr>
<td>--------------------------------------------------------------------------</td>
<td>-------------------</td>
<td>----------</td>
<td>---------</td>
<td>-------</td>
<td>----------------</td>
</tr>
<tr>
<td>40 When I make a commitment, I can always be counted on to follow through.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
<td>SA</td>
</tr>
<tr>
<td>41 Too often, when things go wrong, I get discouraged and feel like giving up.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
<td>SA</td>
</tr>
<tr>
<td>42 I am not a cheerful optimist.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
<td>SA</td>
</tr>
<tr>
<td>43 Sometimes when I am reading poetry or looking at a work of art, I feel a chill or wave of excitement.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
<td>SA</td>
</tr>
<tr>
<td>44 I am hard-headed and tough-minded in my attitudes.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
<td>SA</td>
</tr>
<tr>
<td>45 Sometimes I'm not as dependable or reliable as I should be.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
<td>SA</td>
</tr>
<tr>
<td>46 I am seldom sad or depressed.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
<td>SA</td>
</tr>
<tr>
<td>47 My life is fast passed.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
<td>SA</td>
</tr>
<tr>
<td>48 I have little or no interest in speculating on the nature of the universe or the human condition.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
<td>SA</td>
</tr>
<tr>
<td>49 I generally try to be thoughtful and considerate.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
<td>SA</td>
</tr>
<tr>
<td>50 I am a productive person who always gets the job done.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
<td>SA</td>
</tr>
<tr>
<td>51 I often feel hopeless and want someone else to solve my problems.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
<td>SA</td>
</tr>
<tr>
<td>52 I am a very active person.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
<td>SA</td>
</tr>
<tr>
<td>53 I have a lot of intellectual curiosity.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
<td>SA</td>
</tr>
<tr>
<td>54 If I don't like people I let them know it.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
<td>SA</td>
</tr>
<tr>
<td>55 I never seem to be able to get organised.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
<td>SA</td>
</tr>
<tr>
<td>56 At times I have been so ashamed I just wanted to hide.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
<td>SA</td>
</tr>
<tr>
<td>57 I would rather go my own way than be a leader of others.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
<td>SA</td>
</tr>
<tr>
<td>58 I often enjoy playing with theories or abstract ideas.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
<td>SA</td>
</tr>
<tr>
<td>59 If necessary I am willing to manipulate people to get what I want.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
<td>SA</td>
</tr>
<tr>
<td>60 I strive for excellence in everything I do.</td>
<td>SD</td>
<td>D</td>
<td>N</td>
<td>A</td>
<td>SA</td>
</tr>
</tbody>
</table>

Have you responded to all of the statements? _____ Yes   _____ No
Have you responded accurately and honestly? _____ Yes   _____ No
**APPENDIX 4**

**MULTIDIMENSIONAL HEALTH LOCUS OF CONTROL SCALE (FORM A).**

This is a questionnaire designed to determine the way in which different people view certain important health-related issues. Each item is a belief statement with which you may agree or disagree. Beside each statement is a scale which ranges from strongly disagree (1) to strongly agree (6). For each item we would like you to circle the number that represents the extent to which you disagree or agree with the statement. The more strongly you agree with a statement, then the higher will be the number you circle. The more strongly you disagree with a statement, then the lower will be the number you circle. Please make sure that you answer every item and that you circle only one number per item. This is a measure of your personal beliefs; obviously there are no right or wrong answers.

Please answer these items carefully, but do not spend too much time on any one item. As much as you can, try to respond to each item independently. When making your choice, do not be influenced by your previous choices. It is important that you respond according to your actual beliefs and not according to how you feel you should believe or how you think we want you to believe.

SD = Strongly disagree  
MD = Moderately disagree  
D = slightly disagree  
A = slightly agree  
MA = Moderately agree  
SA = Strongly agree  

<table>
<thead>
<tr>
<th></th>
<th>SD</th>
<th>MD</th>
<th>D</th>
<th>A</th>
<th>MA</th>
<th>SA</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>2</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>3</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>4</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>5</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>6</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>7</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>8</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>9</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>10</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>11</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>12</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>13</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>14</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>15</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>16</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>17</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>18</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
</tbody>
</table>
### CHIP-5

**Health Reactions Inventory**

Norman S. Endler, Ph.D. F. R. S. C., and James D. A. Parker, Ph.D.

The following are ways of reacting to HEALTH PROBLEMS, such as ILLNESSES, SICKNESSES, and INJURIES. These are typically difficult, stressful, or upsetting situations. We are interested in your typical reactions to illness in general, not just your current problem. Please circle a number from 1 to 5 for each of the following items. Indicate how much you have engaged in these activities when you have encountered health problems. Please be sure to respond to each item.

<table>
<thead>
<tr>
<th></th>
<th>1 = Not at all</th>
<th>3 = Moderately</th>
<th>5 = Very Much</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Think about the good times I’ve had.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Stay in bed.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Find out more information about the illness.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Wonder why it happened to me.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Be with other people.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Lie down when I feel tired.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Seek Medical treatment as soon as possible.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Become angry because it happened to me.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Daydream about pleasant things.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>Get plenty of sleep.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>Concentrate on the goal of getting better.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>Get frustrated.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>Enjoy the attention of friends and family.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>Try to use as little energy as possible.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>Learn more about how my body works.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>Feel anxious about the things I can’t do.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>Make plans for the future.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>Make sure I am warmly dressed or covered.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>Do what my doctors tell me.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>Fantasise about all the things I could do if I was better.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>Listen to music.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>22</td>
<td>Make my surroundings as quite as possible.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>23</td>
<td>Try my best to follow my doctors advice.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>24</td>
<td>Wish that the problem had never happened.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>25</td>
<td>Invite people to visit me.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>26</td>
<td>Be as quite and still as I can.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>27</td>
<td>Be prompt about taking medications.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>28</td>
<td>Feel anxious about being weak and vulnerable.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>29</td>
<td>Surround myself with nice things (e.g. flowers).</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>30</td>
<td>Make sure I am comfortable.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>31</td>
<td>Learn more about the most effective treatments available.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>32</td>
<td>Worry that my health might get worse.</td>
<td>1 2 3 4 5</td>
<td></td>
</tr>
</tbody>
</table>
**APPENDIX 6**

**DISEASE ACTIVITY.**

Please try and answer the following questions by thinking about your health as it has been over the last week.

1. Number of liquid or very soft stools over the last week _____

2. Average abdominal pain rating over the last week
   
   (0=none; 1=mild; 2=moderate; 3=severe) _____

3. General well-being over the last week
   
   (0=generally well; 1=slightly under par; 2=poor; 3=very poor; 5=terrible) _____

4. Please indicate your weight:
   
   in_________lbs

   or_________kgms

5. Please indicate your height:

   in_________cms

   or_________ft & inches
APPENDIX 7

Subject code: 
Date: 
Time: 

### Indicate how often today you have:

<table>
<thead>
<tr>
<th>Question</th>
<th>Scale 0-4</th>
</tr>
</thead>
<tbody>
<tr>
<td>been upset because of something that happened to you unexpectedly?</td>
<td>0 - 4</td>
</tr>
<tr>
<td>felt that you were unable to control the important things in your life?</td>
<td>0 - 4</td>
</tr>
<tr>
<td>felt nervous or stressed?</td>
<td>0 - 4</td>
</tr>
<tr>
<td>felt confident about your ability to handle your personal problems?</td>
<td>0 - 4</td>
</tr>
<tr>
<td>felt that things were going your way?</td>
<td>0 - 4</td>
</tr>
<tr>
<td>found that you could not cope with all the things you had to do?</td>
<td>0 - 4</td>
</tr>
<tr>
<td>been able to control irritations in your life?</td>
<td>0 - 4</td>
</tr>
<tr>
<td>felt that you were on top of things?</td>
<td>0 - 4</td>
</tr>
<tr>
<td>been angered because of things that happened that were outside of your</td>
<td>0 - 4</td>
</tr>
<tr>
<td>control?</td>
<td>0 - 4</td>
</tr>
<tr>
<td>felt difficulties were piling up so high that you could not overcome them</td>
<td>0 - 4</td>
</tr>
</tbody>
</table>

### Please rate the following symptoms on a severity scale of 0 to 4 that you have experienced over the past 24 hours.

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Scale 0-4</th>
</tr>
</thead>
<tbody>
<tr>
<td>abdominal pain</td>
<td></td>
</tr>
<tr>
<td>joint pain</td>
<td></td>
</tr>
<tr>
<td>nausea</td>
<td></td>
</tr>
<tr>
<td>abdominal tenderness</td>
<td></td>
</tr>
</tbody>
</table>

### Please indicate the number of occurrences of the following over the past 24 hours:

<table>
<thead>
<tr>
<th>Occurrence</th>
<th>Number.</th>
</tr>
</thead>
<tbody>
<tr>
<td>loose or liquid bowel movements</td>
<td></td>
</tr>
<tr>
<td>bloody stools</td>
<td></td>
</tr>
<tr>
<td>vomiting</td>
<td></td>
</tr>
</tbody>
</table>

Please indicate any medication you have taken over the past 24 hours:

On a scale of 1 - 10 please indicate your overall mood of the previous 24 hours:

1 = very poor
10 = very good

Please indicate the date of the end of your last period:
APPENDIX 8

Informed consent/information sheet.

Monday, June 03, 1996

Dear Sir/Madam,

We are carrying out a study of social and individual influences upon symptom experience of inflammatory bowel disease entitled “Perceived stress, personality and symptom experience in inflammatory bowel disease patients”. This research study is being conducted by a social scientist from Queen Margaret College in Edinburgh in collaboration with the Western General Hospital in Edinburgh.

We are looking for volunteers to take part in this study, in order to further knowledge of the factors triggering the symptoms of inflammatory bowel disease. If you agree to take part you would be required to complete a few questionnaires sent to you by post. This would take approximately 30 minutes of your time.

It is strongly emphasised that participation in this study would not, in any way, affect your standard care or your care plan, and that the data gained from the questionnaires will be treated with total confidentiality. It is also emphasised that you are under no obligation to participate in this study and that you can withdraw at any time.

However, your participation would be greatly appreciated and may provide us with further insight into caring for and helping sufferers of inflammatory bowel diseases.

Please indicate below whether or not you are prepared to take part in this study, by ticking the relevant box and providing your name. A stamped addressed envelope is enclosed for your reply slip. Due to the nature of the study, individuals with ileostomies or colostomies are not required to participate (as such procedures should lessen if not remove symptoms). The surgical processes of investigative biopsy and bowel section removal are not included in this classification and individuals who have received such surgery are still encouraged to participate. Therefore it would be greatly appreciated if you could tick the appropriate box below to indicate that you have received either a colostomy or an ileostomy.

Thank you for reading this and please respond, whether you wish to take part or not.

Andrew Sadler.
Dept. Of Management and Social Sciences, Queen Margaret College.
Tel: 0131-317-3616.

I am willing to participate in this research
I am not willing to participate in this research
I have received a colostomy or an ileostomy

Please specify:

Name:__________________________
Signature:_______________________
“Education is a valuable thing; but it is worth remembering, from time to time, that nothing that is worth knowing can be taught.”

Oscar Wilde
References.


End of document


References.


References


