

NEUROTICISM AS A POTENTIAL MODERATOR
IN THE EUSTRESS-HEALTH RELATIONSHIP

CENTRE FOR NEWFOUNDLAND STUDIES

**TOTAL OF 10 PAGES ONLY
MAY BE XEROXED**

(Without Author's Permission)

NASHWA IRFAN



**Neuroticism as a potential moderator
in the Eustress-Health relationship**

By

Nashwa Irfan, (Honours) B.Sc.

**A thesis submitted to the School of Graduate
Studies in partial fulfillment of the
requirements for the degree of
Master of Science**

**Department of Psychology
Memorial University of Newfoundland
1996**

St. John's

Newfoundland



National Library
of Canada

Acquisitions and
Bibliographic Services Branch

395 Wellington Street
Ottawa, Ontario
K1A 0N4

Bibliothèque nationale
du Canada

Direction des acquisitions et
des services bibliographiques

395, rue Wellington
Ottawa (Ontario)
K1A 0N4

Your file Votre référence

Our file Notre référence

The author has granted an irrevocable non-exclusive licence allowing the National Library of Canada to reproduce, loan, distribute or sell copies of his/her thesis by any means and in any form or format, making this thesis available to interested persons.

L'auteur a accordé une licence irrévocable et non exclusive permettant à la Bibliothèque nationale du Canada de reproduire, prêter, distribuer ou vendre des copies de sa thèse de quelque manière et sous quelque forme que ce soit pour mettre des exemplaires de cette thèse à la disposition des personnes intéressées.

The author retains ownership of the copyright in his/her thesis. Neither the thesis nor substantial extracts from it may be printed or otherwise reproduced without his/her permission.

L'auteur conserve la propriété du droit d'auteur qui protège sa thèse. Ni la thèse ni des extraits substantiels de celle-ci ne doivent être imprimés ou autrement reproduits sans son autorisation.

ISBN 0-612-13908-5

Canada

Abstract

Eustress was conceptualized as the positive affect resulting from positive events. A moderator model was proposed which postulated that neuroticism moderates the relationship between eustress and health. Due to the possibility of specification error (i.e., data may reflect linear or mediating properties as opposed to interactive or moderating properties), an exploratory mediation model was developed to test for any mediating effects eustress may have in the neuroticism-health relationship. Specifically, the direct effects hypotheses for this mediation model proposed that uplifts lead to eustress, which in turn reduces reported symptoms of poor health. Neuroticism reduces eustress, which in turn leads to symptoms (i.e., eustress mediates the relationship between neuroticism and health). Neuroticism also leads to symptoms via other mechanisms not involving eustress (e.g., cognitive interpretation (Harkins, Price & Braith, 1989), or physiological processes (Friedman & Booth-Kewley, 1987)). Three hundred and twenty-two participants completed measures of positive affect, uplifts, neuroticism, and somatic complaints in the first phase of a two phase prospective study. One hundred and ninety-six participants from the original subject pool completed the positive affect, uplifts and somatic complaints measures two weeks later. The results provide support for conceptualizing eustress as the positive affect arising from positive events. With respect to the moderator model, the results failed to support the hypothesis that neuroticism moderates the relationship between eustress and health. The mediator model was not found to have a good fit to the data. The hypothesis that uplifts lead to eustress which in turn reduces symptoms was supported. However, the hypothesis that neuroticism leads to somatic complaints through reducing eustress (i.e., that eustress mediates the relationship between neuroticism and health) as well as through other mechanisms, was not

supported. Analysis on transformed data showed some support for the hypothesized model. However, since this result was based on transformed data, it should be interpreted with caution. Alternative mediator models fit the data and supported the finding that uplifts lead to eustress resulting in low symptoms. Since mediator models fit the data, specification error could have resulted from only testing a moderator model. In other words, the data may have largely reflected linear (mediator) relationships as opposed to interactive (moderator) relationships. Possible explanations for the present findings and suggestions for future research are discussed.

Acknowledgments

I would like to thank Dr. Ted Hannah for his open-minded attitude, motivation, inspiration, and kind support in supervising this thesis. I would also like to thank Dr. Cathy Button and Dr. Christine Arlett who are members on my supervisory committee. In addition, I would like to thank my colleague and friend, David Korotkov, for his tremendous support. Thank you Dave for all of your help especially regarding the methodology and statistical techniques used.

Dedication

I would like to dedicate this project to my father, mother, and brother. Their tremendous support and help in making this thesis possible will always be remembered.

Thank you.

Introduction

Introductory Remarks

The present study is interested in examining the role of the personality trait neuroticism in the relationship between eustress (positive stress) and health. A detailed discussion focusing on eustress, neuroticism, and health, including the reasoning and evidence for focusing on neuroticism as a moderator in the eustress - health relationship, will be provided later. However, for purposes of clarification, before one discusses the concept of eustress, a discussion of the conceptualization of stress in general must first be provided.

Conceptualization of Stress

There is substantial disagreement over the definition of stress. Some researchers conceptualize stress as a stressor (Holmes & Rahe, 1967), as a cognitive response (Lazarus, 1966), or as a biological response to various stimuli (Selye, 1976). Although this discrepancy concerning the definition of stress may be viewed by some as indicative of instability in the stress field, this absence of consensus more properly reflects the rapid expansion of stress research in many divergent directions (Breznitz & Goldberger, 1982). All three definitions focus on one factor, be it a stimulus (stressor), an appraisal, or a biological response. For instance, stress conceptualized as a stimulus, focuses on the change or adaptation required by an individual in response to a stressor (for example, life events). A definition based on cognitive appraisal concentrates on the type of information about the stressor available to the individual (for example, situational context) and how it is processed. A biological definition focuses on the body's physiological reaction to a stressor.

A brief examination of these three definitions will now be presented.

Stress as a stimulus. Stress conceived as a stimulus has been used to describe situations characterized as new, intense, rapidly changing, sudden or unexpected. However, stressful stimuli can also include stimulus deficit, absence of expected stimulation, highly persistent stimulation, fatigue and boredom (Zegans, 1982a). Holmes and Rahe (1967) viewed stress as a stimulus in their reasoning that life events can be conceptualized as stressful stimuli. They maintained that stress may be conceptualized as discrete, time limited events requiring change or adaptation. In their original work, Holmes and Rahe (1967) scaled life events, for example, marriage, change in residence, etc., in terms of the intensity and length of time necessary to accommodate to a life event regardless of its desirability (Rabkin & Struening, 1976). Their initial measure, called The Schedule of Recent Experience (SRE), contained 43 events and a subject's life stress score was the number of events he or she reported experiencing during a recent interval of time (usually 6-24 months). Holmes and Rahe soon recognized that some of the 43 SRE items, for example, death of spouse, required considerably more change and adaptation than did others, for example, Christmas. In response to this, a subsequent instrument, The Social Readjustment Rating Scale (SRRS) (Holmes & Rahe, 1967), was developed. This scale weighted each event using a ratio scale to estimate the amount of change or readjustment required on the part of the individual experiencing the event. Based on this life events research model, it is possible to make predictions about stress and susceptibility to a wide array of diseases (infectious, neoplastic, autoimmune) by determining the magnitude of critical life changes taking place within a limited span of time (Zegans, 1982b). Researchers have since found a significant relationship between the experience of stress, as assessed by life events, and physical illness (Dohrenwend, Pearlin,

Clayton, Hamburg, Riley, Rose, & Dohrenwend, 1982; Dohrenwend & Dohrenwend, 1981; Jacobs & Charles, 1980).

Source of stress: Cognitive appraisal and coping. Stress can be defined relationally by reference to both the person and the environment (Coyne & Lazarus, 1980). Stress requires a judgement that environmental and/or internal demands exceed the individual's resources for managing them. This judgement and the individual's efforts to manage and shape the stress experience are conceptualized in terms of two interacting processes: appraisal and coping (Lazarus & Folkman, 1982).

Appraisal refers to the evaluative process associated with a situational encounter which provides meaning for the individual. Appraisals can be separated into those that are concerned with the recognition that the individual is in jeopardy (appraisal of what is at stake) and those that are concerned primarily with the evaluation of resources and options available for managing potential or actual harm (appraisal of coping). Appraisal of what is at stake refers to the judgement that an encounter is irrelevant, positive, or stressful to our well-being. Stressful appraisals can be further placed into three categories: appraisals of threat, appraisals of harm-loss, and appraisals of challenge. Appraisals of threat and harm-loss are distinguished primarily by their time perspective, with threat referring to the anticipation of imminent harm and harm-loss referring to the judgement that damage has already occurred. Challenge involves not only the judgement that an encounter contains the potential for harm or the potential for mastery or gain, but also that the outcome can be influenced by the individual. Thus, appraisals of challenge involve an interaction of appraisal of stakes and a sense of positive control.

The term "coping" refers broadly to efforts to manage environmental and internal demands

and conflicts among demands (Lazarus, 1981). Such thoughts and acts are actively involved in the coping process.

Appraisal and coping abilities may illustrate the cognitive processes involved in the stress experience for an individual. However, another important mechanism involved in the experience of stress is incorporated in the physiological reactions to stress.

The Stress Response. Considerable research has been conducted to examine the relationship between stress and illness. One of the major contributors to this line of research, Hans Selye, defined stress as the "body's non-specific response to any demand placed on it, whether pleasant or unpleasant" (Selye, 1976). He maintained that stress is indicated by evidence of adrenal stimulation, shrinkage of lymphatic organs, gastrointestinal ulcers, and loss of body weight with characteristic alterations in the chemical composition of the body. The body's non-specific response to any demand was later found to comprise many other changes, collectively referred to as the general adaptation syndrome (G.A.S.). According to Selye (1976), the G.A.S. incorporates three stages - alarm, resistance, and exhaustion - and sequential progression through these stages results in a gradual deterioration of the body's defense mechanisms and ultimately results in a breakdown of specific physiological processes.

In tissues more directly affected by stress, there develops a local adaptation syndrome (L.A.S.). For example, inflammation occurs where microbes enter the body. Chemical alarm signals are sent out by the directly stressed tissues, from the L.A.S. area to centres of coordination in the nervous system, and hence to the endocrine glands, especially the pituitary and the adrenals. These glands produce adaptive hormones to combat deterioration in the body. The adaptive hormones fall into two categories: (a) the anti-inflammatory or glucocorticoid hormones

(ACTH, cortisone, cortisol), which inhibit excessive defensive reactions, and (b) the pro-inflammatory and/or mineralocorticoid hormones (STH, aldosterone, DOC) which stimulate defensive reactions. The effects of these substances can be modified or conditioned by other hormones (e.g., adrenaline, or thyroid hormones), nervous reactions, diet and heredity (Selye, 1976).

Selye (1976) maintained that derailments of the G.A.S. mechanism produce diseases of adaptation, or stress diseases, for example, high blood pressure, diseases of the heart, diseases of the kidney, eclampsia (periods of coma following convulsions during pregnancy), rheumatoid arthritis, among others. Selye also maintained there are other less severe symptoms or somatic complaints one may experience when subjected to stress. Such somatic complaints include: dryness of the throat and mouth, feelings of weakness or dizziness, predilection to become fatigued, insomnia, sweating, frequent need to urinate, diarrhea, indigestion, queasiness in stomach, vomiting, migraine headaches, pain in lower back or neck, and excessive loss of appetite.

It may seem reasonable to conceptualize stress as an interaction between a biological mechanism and a cognitive mechanism in responding to a stressor, such as a change in life events. Stress may refer to the entire process by which one both cognitively appraises and biologically responds to the stressor. The body may respond in a certain way to a particular cognitive appraisal of a stressor. For example, psychological states such as challenge are associated with hormonal response patterns that are not as physiologically harmful as those associated with threat (Lazarus, Cohen, Folkman, Kanner, & Schaefer, 1980a). Research suggests that threat is associated with elevations in both catecholamines and cortisol levels, whereas challenge is

associated only with elevations in catecholamine levels, with cortisol levels remaining normal or even declining (Frankenhaeuser, 1980).

Distress and Eustress

Selye (1976) maintained that stress is the body's nonspecific response to any demand placed on it, whether it is caused by pleasant or unpleasant conditions. One should, however, differentiate within the general concept of stress between the unpleasant or harmful variety, called 'distress', and the pleasant type called 'eustress' (Selye, 1976). Despite Selye's distinction, distress is still usually referred to by the term 'stress' and is characterized by a negative psychological state. This state reflects a negative discrepancy between an individual's perceived state and his or her desired state, provided that the presence of this discrepancy is considered important by the individual (Edwards & Cooper, 1988). Very little research has been conducted examining eustress. Eustress is characterized by a positive psychological state and is often referred to as 'positive stress' or 'good stress' (Mullis, Youngs, Mullis, & Rathge, 1993). It should be noted that some researchers conceptualize eustress as the individual's experience of encountering events requiring change and adaptation but which, at the same time, are growth producing and welcome, that is, having positive emotional consequences (Greenberg, 1987). However, the dominant view holds that eustress is the positive affect arising from experiences with positive events (Edwards & Cooper, 1988). This is the working definition used for purposes of the present study.

Overview of proposed model

To date, most research has focused on the health consequences resulting from major

negative life events, for example, divorce or death of spouse (Kierolt, Janice, Kennedy, Malkoff, & Fisher, 1988; Williams & Siegel, 1989), or from daily minor negative events termed hassles, for example, minor financial problems (DeLongis, Folkman, & Lazarus, 1988; Landreville & Vezina, 1992; Zarski, West, Gintner, & Carlson, 1987). In this relationship, undesirable negative life events and/or hassles are presumed to give rise to negative affect (i.e., distress). In other words, distress can be conceptualized as the negative affect which results from undesirable negative stressful events. It should be noted that negative life events assessed only according to the change and adaptation required by the individual and independently of the emotions arising from these negative life events, are not necessarily indicative of distress. Distress involves the negative affect resulting from undesirable events (Sarason, Johnson, & Siegel, 1978; Pearlin, 1989). The negative emotional consequence of undesirable events is presumed to give rise to poor health. In other words, chronic or long-term distress is thought to have detrimental effects on health (Williams & Siegel, 1989). Much research maintains that focusing solely on a change of life events score independent of the event's desirability is not a good predictor of future health problems (Depue & Monroe, 1986; Maddi, Bartone, & Puccetti, 1987; Rutter & Sandberg, 1992). Research maintaining that perceived undesirability of an event is a stronger predictor of illness than life change shows that positive life events such as getting married are less physically harmful than negative life events such as being fired (Anderson & Arnoult, 1989, Brown & McGill, 1989).

The relationship between distress and poor health may be moderated by certain variables such as social support (Cohen & Hoberman, 1983; Sarason, Sarason, Potter, & Antoni, 1985), positive events (Cohen & Hoberman, 1983), and locus of control (Denney & Frisch, 1981). For present purposes, it should be noted that a variable, for example, x , is a moderator if the

relationship between an independent and dependent variable is a function of the level of x .

Moderator variables will be discussed in more detail later in the introduction. Figure 1 is an example of a moderator model which portrays the relationship between distress and poor health moderated by social support.

The present study proposes a parallel line of reasoning with respect to major positive life events (e.g., marriage) or daily minor positive events termed uplifts (e.g., winning the office hockey pool). In this relationship, desirable positive life events or uplifts give rise to positive affect, termed eustress. In other words, eustress is conceptualized as the positive affect which results from positive events. Eustress, in turn, is presumed to have beneficial effects on health (Edwards & Cooper, 1988).

As in the case of negative life events, where the relationship between distress and poor health may be moderated by certain variables, the relationship between eustress and good health may also be moderated by certain variables. The present study focuses on the personality trait of neuroticism as a possible moderating variable in the eustress - health relationship. The reasoning and evidence for examining neuroticism as a moderator variable in the present study will be discussed later. The basic model of the present study is shown in Figure 2. This model posits that individuals who experience eustress resulting from positive events, have few somatic complaints or health problems. However, neuroticism may moderate this relationship such that those individuals who objectively experience eustress but are also high on neuroticism will experience more somatic complaints than those individuals who experience eustress and are low on neuroticism.

Before considering the various components of the present study, it should be noted that

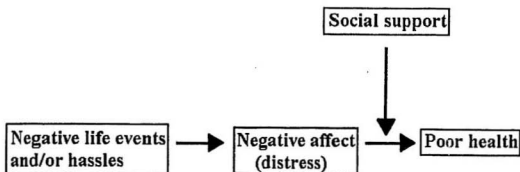


Figure 1. Social support as a moderator in the distress-illness relationship.

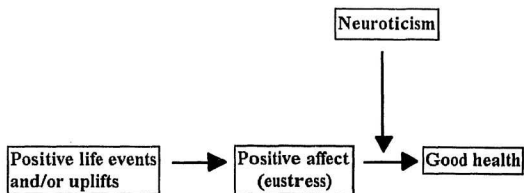


Figure 2. Neuroticism as a moderator in the eustress-health relationship.

since somatic complaints are a major focus of this study, a distinction should be made regarding the types of reports used to assess health status. Subjective health is usually assessed through self-reports of somatic complaints. These self-reports are often associated with actual physical illness or objective health, however are not synonymous with physical illness. The term illness behaviour describes the way people respond to bodily indications which they perceive as abnormal. Illness behaviour involves the manner in which people monitor their bodies, define and interpret their symptoms, take remedial actions and utilize the health-care system (Mechanic, 1983). Examples of illness behaviour include visiting a physician, taking medicine, staying home from work, and complaining of pain or other symptoms. Actual illness is more strongly associated with illness behaviour than self-reports of somatic complaints. Somatic complaints constitute one type of illness behaviour related to actual objective health status. However, somatic complaints do not necessarily reflect objective health. In addition, it is important to note that illness and illness behaviour are not perfectly correlated. For instance, one's illness behaviour may be excessive, as in the case of the hypochondriacal individual, or unusually restrained, as in the case of the stoic. Although health complaints have been empirically linked to objective, concurrent health status (e.g., Linn & Linn, 1980) and subsequent objective health outcomes such as mortality (e.g., Idler, Kasl, & Lemke, 1990), these associations reflect only modest amounts of common variance. Thus, much of the variance in self-report measures of health reflects somatic complaints in the absence of disease (Smith & Williams, 1992). Thus, reference to health and assessment of health in the present study will reflect reports of symptomatology as opposed to objective health status.

The various components of the proposed model will now be discussed in detail.

Relationship between positive events and good health. Research has shown that there is a positive relationship between positive events and good health (Svensson & Theorell, 1983). Miller and Wilcox (1986) administered a hassles scale, an uplifts scale, and psychological and physical health scales to 30 subjects aged 69-93 years in a nursing home. Their results indicated that hassles were negatively related to psychological and physical health, while uplifts were positively related to psychological and physical health. Other studies have shown that the absence of positive events may lead to poor health (Kanner, Kafry, & Pines, 1978). For instance, Evans and Edgerton (1991) had 100 subjects check, at the end of each day, a variety of items dealing with events, mood states, and health. A subsample that had provided several weeks of data and had suffered at least one common cold episode was selected for analysis. Results showed there was a significant decrease in the frequency of desirable events (compared to the number of desirable events normally experienced by these individuals) experienced prior to cold onset. This finding indicates a possible negative relationship between positive events and health problems.

Thus, in general, positive events lead to good health. Conversely, a reduction in positive events may lead to somatic complaints.

An important point to consider is that uplifts may be stronger predictors of health status than positive life events. For comparison purposes, research focusing on hassles as opposed to negative life events as predictors of health will be briefly presented.

Kanner, Coyne, Schaefer, & Lazarus (1981) compared major negative life events with daily hassles in predicting health. They found that hassles were more strongly associated with concurrent and subsequent health than were life events. Major life events had little effect independent of daily hassles, however hassles contributed to symptoms independent of major life

events. In predicting reported symptoms, a substantial relationship remained for hassles even after the effect due to life events had been removed. Moreover, the remaining relationship between hassles and reported symptoms was generally greater than between life events and reported symptoms. Thus, although daily hassles overlap considerably with life events, they also operate quite strongly and independently of life events in predicting symptoms. Other studies have also found that measures of daily hassles are more strongly related to health status than are measures of major life events (DeLongis, Coyne, Dakof, Folkman, & Lazarus, 1982; Monroe, 1983; Weinberger, Hiner, & Tierney, 1987; Zarski, 1984). A possible explanation for these findings is that hassles disrupt the characteristic coping processes required to deal with negative life events. Hassles may function as critical event mediators, that is, events which determine if an independent variable leads to a dependent variable (James & Brett, 1984), in the negative life event - health outcome relationship. They may indicate how a person's daily routine is being affected by life changes and thus be better predictors of health status as opposed to life events. This notion of the mediating role of hassles in the relationship between negative life events and health is generally supported (Kanner et al., 1981; Russell & Cutrona, 1991).

Consistent with the reasoning that daily hassles may be better predictors of health problems as opposed to major negative life events, one can also suggest that uplifts may be better predictors of health as opposed to major positive life events. Little research has been conducted to compare the utility of uplifts versus major positive life events in predicting well-being. However, reports of uplifts are more reliable than reports of positive life events when assessed over similar periods of time and reporting uplifts has less bias associated with them than reporting positive life events. For instance, memory loss is slow for experience with positive life events possibly resulting in

higher frequency scores for reports of desirable life events. There is little evidence, however, for bias or reactivity with regards to reports of experiencing uplifts. Accuracy of self-reports of uplifts have been explored by having peers observe subjects and comparing the two estimates of event frequency. Results showed a moderate correlation between peer and subject frequency ratings ($r=.63$, Reich & Zautra, 1988). It is possible that uplifts also operate as mediators in the positive life event - health relationship perhaps by enhancing the effects of positive life events on health. Based on this reasoning, the present study will focus on uplifts, as opposed to major positive life events, as the predominant precursor of eustress.

Some debate has arisen concerning the question of whether the influence of positive events on well-being is determined primarily by cognitive mechanisms or affective mechanisms.

Vinokur and Caplan (1986) found that positive events are easier to adjust to than negative events. Through a positive cognitive appraisal of positive events, due to their ease of adjustment, positive events may have beneficial effects on well-being. The experience of positive events has also been associated with the perception of having control over the positive event. This, in turn, may lead to greater well-being (Reich & Zautra, 1988). Zautra and Reich (1980) explored the relationship between life events and subjective ratings of well-being. Results showed that positive origin experiences (i.e., experiences which involved personal control) led to reports of greater well-being and less maladjustment than pawn events (i.e., experiences which did not involve personal control) which were either positive or negative in nature. Reich and Zautra (1988) maintain that positive events influence well-being through a mediating mechanism of personal mastery including cognitive control. They reason that individuals feel causally responsible for positive events in their lives and positive events enhance one's sense of control over the events in

one's life. This, in turn, may lead to positive well-being.

Other factors in addition to perceived adjustment and control over positive events may also play a role in the influence of positive events on well-being. For example, Csikszentmihalyi and Figurski (1982) found that a sense of being engaged with an event voluntarily rather than as a requirement was related to its positivity. The voluntary nature of an event may, in turn, lead to good health.

Vinokur and Caplan (1986), however, suggest that the affective response to an event is more reliable and more influential in predicting health than a cognitive mechanism. They maintain that the quality of the affective reaction that accompanies the event may be the most important facet of how the event is experienced and hence the ultimate influence on health. Others also hold that the affect that is generated by positive events regardless of whether clear cognitions are present or not, may be a more accurate indicator of the ultimate influence of the event on health (Zajonc, 1980).

Summary. Positive events are associated with good health. Uplifts may be better predictors of health than positive life events. This study will therefore focus on uplifts instead of positive life events in giving rise to eustress. Much debate has arisen concerning whether positive events lead to good health through cognitive or affective mechanisms. There is evidence for both. However, affective responses to positive events may be more reliable and influential in predicting health.

Relationship between positive events and positive affect. Some research has been conducted to support the notion that positive events are correlated with positive affect. For example, Clark and Watson (1988) studied daily mood ratings and corresponding diary entries to

determine the relationship between common events and two independent mood factors – positive affect and negative affect - in a sample of 18 young adults over a 3-month period. The results indicated an especially robust relationship between positive affect and reported positive social interactions, particularly physically active social events. Others have found similar relations between positive events and positive affect (Brandstatter, 1983; MacPhillamy & Lewinsohn, 1982; Reich & Zautra, 1981; Stone, 1981; Zautra, 1983; Zautra & Reich, 1980; Zautra & Reich, 1983). There seems to be much evidence to support that positive events are related to positive affect.

Relationship between positive affect and good health. Evidence exists showing that positive affect is positively related to good health (Croyle & Uretsky, 1987; Dua & Price 1992) Lubin, Zuckerman, Breyspraak, and Bull (1988) explored the relationship between positive affect and health. They administered the revised Multiple Affect Check List (MAACL-R) to a national probability sample of 1,543 adults. These adults also provided demographic data and self-ratings of health, medication use, and social activities. Results showed that positive affect was related directly to self-ratings of good health.

A negative relation has also been found between positive affect and reports of somatic complaints (Jenkins, Stanton, Klein, Savageau, & Dwight, 1983; Kasl & Cobb, 1982). More specifically, evidence suggests that the absence of positive affect is associated with somatic complaints (Veit & Ware, 1983). Clark and Watson (1988) studied the relationship between reports of physical symptoms and the positive affect arising from daily events. Results showed that low positive affect was correlated with health complaints. Bradburn (1969) found that a lack of positive affect is significantly related to low well-being. This relationship is independent of the

presence of negative affect as a source of low well-being.

Some research has also found a negative relationship between positive mood and pain (Cogan, Cogan, Waltz, & McCue, 1987; Stalling, 1992). For instance, Stalling (1992) conducted an experiment examining the relationship between mood and pain. Mood was experimentally induced and pain was measured by self-reported body aches in 25 body areas. Results indicated that while negative mood had no effect on pain, there was a negative relationship found between positive mood and pain. Positive mood was associated with a reduction in pain ratings.

Based on the evidence to date, it appears that an increase in positive affect leads to low somatic complaints. Conversely, a reduction in positive affect leads to more somatic complaints.

Summary. Research has provided evidence for the following relationships:

1. Positive events are positively related to good health.
2. There is a positive relation between positive events and positive affect
3. There is a positive relation between positive affect and good health
4. A negative relation exists between positive affect and somatic complaints.

Based on this evidence, it is reasonable to suggest that the positive affect from desirable events has a positive impact on health. In other words, eustress leads to good health. Conversely, low eustress leads to somatic complaints.

Processes involved in the influence of eustress on health: The impact of neuroticism

There are two major processes by which eustress may influence health. One process involves the direct effects of eustress on health. Eustress may evoke certain physiological responses, which, in the long run, may serve to improve or protect health. A second process may involve the effect of eustress on coping. Rather than affecting health directly, eustress may

influence health indirectly by facilitating attempts to cope with existing distress, such that the coping process acts as a moderator of the relationship between eustress and health (Lazarus, Kanner, & Folkman, 1980b). There is evidence for both of these processes. The following will first discuss both direct and indirect influences on eustress. Then, a discussion of how and why neuroticism may serve as a potential moderator of the eustress-health relationship will be presented.

With respect to direct effects, Karasek, Russell, and Theorell (1982) describe pathways by which situations involving high demands combined with high control may produce physiological growth and regeneration. The situation of high demand and high control is consistent with Edwards and Cooper's (1988) conceptualization of eustress. In their view, high control implies the ability to meet the demands placed on the individual. If the individual desires to meet these demands and considers meeting them important, then eustress will result. It is suggested that these situations stimulate the production of hormones, such as HDL cholesterol, testosterone, insulin, adrenaline, and growth hormone. When the balance of these anabolic hormones exceeds catabolic hormones (e.g., cortisol), physiological growth may occur. For example, testosterone and growth hormone may actually enhance protein synthesis in the myocardium (i.e., heart muscle), thus contributing to a decrease in the probability of coronary heart disease. While this process is speculative, it nonetheless suggests pathways by which eustress may influence physiological mechanisms which ultimately improve physical health (Karasek et al, 1982).

Eustress may also influence health indirectly by facilitating attempts to cope with existing distress. In general, eustress may facilitate coping by enhancing individual abilities relevant to coping and/or stimulating increased effort directed toward coping. It should be noted that these

effects focus on the reduction of physiological damage associated with existing distress rather than the production of physiological benefit associated with eustress (Edwards & Cooper, 1988). The effects of eustress on coping are discussed by Lazarus et al. (1980b). They identify three mechanisms by which eustress may facilitate coping. First, eustress may serve as a breather from ongoing distress. These breathers or breaks presumably facilitate coping by allowing periods for creative problem-solving. Second, eustress may act as a sustainer of ongoing coping increasing the likelihood that coping efforts will persist. Third, eustress may serve as a restorer, replenishing damaged or depleted resources or developing new resources. For instance, positive experience may bolster damaged self-esteem, which may, in turn, renew coping efforts.

Summary. Eustress may influence health through both direct and indirect processes. With respect to direct processes, eustress may stimulate the production of beneficial hormones. With respect to indirect processes, eustress may facilitate better attempts to cope with existing distress, thereby reducing the negative physiological consequences of distress.

A brief discussion of moderator variables will now be presented followed by a discussion of how and why neuroticism may serve as a moderator in the eustress-health relationship.

The nature and strength of the relationship between distressful life events and illness is influenced by other variables (Schroeder & Costa, 1984), for example, social support (Cohen & Hoberman, 1983). Some people develop chronic disease and psychiatric disorder after exposure to distressful conditions, and others do not. Mere exposure to negative events alone is almost never a sufficient explanation for the onset of illness in ordinary human experience and other factors that influence their impact require consideration. Thus, the question of whether distressful life events commonly precede the onset of a wide variety of physical and psychiatric disorders in

populations lends itself to the consideration of issues such as moderating factors (Nowack, 1990; Williams, 1989).

(i) The Moderator vs. Mediator Variable Distinction. There is a fundamental distinction between moderating and mediating factors in a relationship. With respect to moderation, a variable, z , is a moderator if the relationship between two (or more) variables, for example, x (a predictor or independent variable) and y (a criterion or dependent variable), is a function of the level of z . Z would moderate this relationship if there is a significant x by z interaction in predicting y (James & Brett, 1984). Figure 3 portrays a model of neuroticism moderating the effect of eustress on health. The moderator hypothesis would be supported if the interaction effect (that is, eustress \times neuroticism) significantly predicts health (Baron & Kenny, 1986).

Mediator relations are generally thought of in causal terms. Influences of an antecedent or independent variable are transmitted to a consequence or dependent variable through an intervening mediator (James & Brett, 1984). Figure 4 depicts an example of a potential mediator model, where eustress mediates the influence of neuroticism on health (Baron & Kenny, 1986). In the moderator-predictor relation, both moderators and predictors are at the same level with regard to their role as causal variables antecedent to certain criterion effects. In the mediator-predictor relation, however, the predictor is causally antecedent to the mediator. In other words, moderator variables always function as independent variables whereas mediating events shift roles from effects to causes, depending on the focus of the analysis. Moderator variables specify when certain effects will hold, while mediators indicate how or why such effects occur (Baron & Kenny, 1986). There is more evidence supporting the role of neuroticism as a moderator as opposed to being a mediator (Aldwin, Levenson, Spiro, & Bosse, 1989; Flood & Endler, 1980; King &

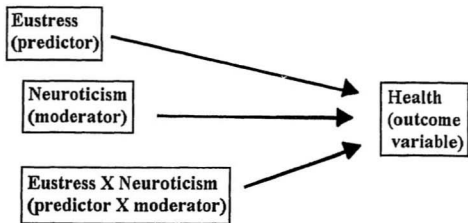


Figure 3. Neuroticism as a moderator in the eustress-health relationship.

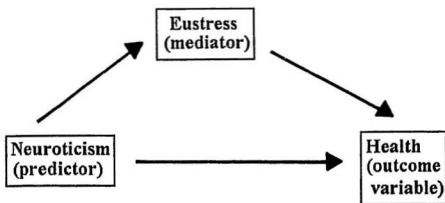


Figure 4. Eustress as a mediator in the neuroticism-health relationship.

Endler, 1990; Phillips & Endler, 1982). Therefore, the present study will focus primarily on a moderator model in which neuroticism is hypothesized to moderate the relationship between eustress and health.

(a) Internal Moderating Variables: Neuroticism. Numerous personal variables may be considered as moderating variables. Such individual factors may include biological and psychological threshold sensitivities, intelligence, verbal skills, morale, psychological defenses, sense of mastery over one's fate, and personality type (Dohrenwend & Dohrenwend, 1969). The effects of most personal variables in moderating distressful conditions are fairly obvious; persons with more skills and assets tend to fare better than individuals with fewer skills and assets. In general, the more competence individuals have demonstrated in the past, the more likely it is that they will cope adaptively with a negative event. The correspondence of personality type to distress reactions and to vulnerability to disease is less clear-cut. Much research, as will be described later, however, provides evidence that the personality trait termed neuroticism defined as the tendency to experience distressing emotions and to possess associated behavioral and cognitive traits such as fearfulness, irritability, low self-esteem, social anxiety, poor inhibition of impulses, and helplessness (Costa & McCrae, 1987) is related to health complaints (Costa & McCrae, 1987; Roll & Theorell, 1987) and is an important moderator variable in the distress - illness relationship (Aldwin et al., 1989; Depue & Monroe, 1986). There is little research showing that other personality dimensions such as extraversion, agreeableness, conscientiousness, and openness to experience (Digman & Inouye, 1986; McCrae, Costa, & Busch, 1986) have strong moderating and/or direct influences on health as compared to neuroticism. Neuroticism is also a broad dimension (Costa & McCrae, 1987) encompassing many facets such as anxiety,

hostility, depression, self-consciousness, impulsivity, and vulnerability (Dolliver, 1987). One may therefore suspect that constructs such as trait anxiety may be just as useful moderators as neuroticism. However, neuroticism provides a more global measure of negative emotions as opposed to other single measures, such as trait anxiety, which are facets of neuroticism. Therefore, it appears more reasonable to use neuroticism as a moderator variable in the eustress - health relationship as opposed to trait anxiety. Individual differences in neuroticism are quite stable and mean levels neither increase nor decrease appreciably with age in adulthood (McCrae & Costa, 1984). It is therefore important that neuroticism be distinguished from episodes of depression or periods of distress-related anxiety.

Neuroticism refers to a chronic condition of irritability and emotionality (Costa & McCrae, 1987). Negative affectivity (NA), a construct characterized by aversive mood states including anger, disgust, guilt, fearfulness, and depression (Watson & Pennebaker, 1989), has been proposed as a term to be used interchangeably with neuroticism (Watson & Clark, 1984). Although NA shares some characteristics with neuroticism, it is not synonymous with it. NA does not include the anxiety and heightened emotionality which is characteristically found in neuroticism (Depue & Monroe, 1986; McLennan & Bates, 1993). In addition, neuroticism is a stable and pervasive trait whereas NA is a temporary, unstable state (Watson & Pennebaker, 1989). Thus, examining neuroticism instead of NA as a moderator in the eustress - health relationship would provide a more stable assessment of one's personality as opposed to assessing a temporary emotion. Neuroticism is a powerful variable, and many other measures of personality used in health research are known to be correlated or are plausibly correlated with neuroticism and reflect its influence (Smith & Williams, 1992). Thus, neuroticism is an important factor in

studies of personality and health. Due to these reasons, in addition to research which will be described shortly, the present study will focus on neuroticism as a potential moderator variable in the eustress - health relationship.

Summary. Neuroticism is a stable pervasive trait which reflects a broad dimension of negative emotions. Past research as will be discussed shortly, provides evidence for neuroticism, compared to other personality dimensions such as extraversion, openness to experience, agreeableness, and conscientiousness, as being related to health complaints as well as being an important moderator in the distress - illness relationship. Thus, the present study focuses on the trait neuroticism as being a moderator in the eustress - health relationship.

The following discussion will theoretically justify the role of neuroticism as a potential moderator in the eustress-health relationship. Moderator research will be discussed followed by the relationship between neuroticism and health and the mechanisms operating in this relationship.

(b) Moderator research. Some research has shown that neuroticism is a significant moderator variable in the relationship between distress and illness. For example, Aldwin et al., (1989) explored evidence of neuroticism moderating the relationship between distress as assessed by life events and hassles and health among a group of elderly men. They found that neuroticism moderated the relationship between distress as assessed by both life events and hassles and health. Individuals scoring higher in neuroticism exhibited higher levels of symptoms under distress than did individuals scoring lower in neuroticism. Thus, neuroticism may determine if individuals will experience illness when subjected to distress.

As mentioned earlier, trait anxiety is not synonymous with neuroticism, however since trait anxiety is a significant component of neuroticism (Dolliver, 1987), for purposes of illustration, the

following brief discussion of anxiety research may provide some support for neuroticism as a moderator variable in the present study. Trait anxiety has been shown to moderate the relationship between specific stressful events and state anxiety. State anxiety is conceptualized as a transitory condition involving unpleasant feelings of fear and apprehension while trait anxiety is conceptualized as a relatively stable personality characteristic indicative of the predisposition to respond with state anxiety under stressful conditions (Spielberger, 1972). Endler (1988) developed a person-by-situation interaction model of anxiety. A major component of this model involved the distinction between state and trait anxiety. Endler argued that trait anxiety is a multidimensional construct composed of a minimum of four facets (social evaluation, physical danger, ambiguity, and daily routines) (Endler, 1988). The differential hypothesis (Flood & Endler, 1980; King & Endler, 1990) of the interaction model of anxiety specifies that differential changes in state anxiety for high and low trait anxious people will occur only when the type of situational threat is congruent with the facet of trait anxiety under consideration. A significant person (high vs low trait anxiety) by situation (stress vs non-stress) interaction for state anxiety is anticipated only when the facet of trait anxiety and situational stress are congruent. For example, an individual exhibiting high ambiguous trait-anxiety will show more state anxiety in an ambiguous stressful situation compared to an individual exhibiting low ambiguous trait anxiety (King & Endler, 1990). Research has provided evidence for this model (Flood & Endler, 1980; Phillips & Endler, 1982). With respect to this model, trait anxiety exacerbates the relationship between specific stressful stimuli and state anxiety.

(ii) Relationship between neuroticism and health. Individuals scoring high on neuroticism report more medical complaints (Costa, 1987; Costa & McCrae, 1980; Larsen & Kasimatis,

1991; Okun & George, 1984; Ormel, 1983). Most research has shown that while neuroticism is related to subjective health indices, it is largely unrelated to objective health status. Costa & McCrae (1987) examined the relationship between personality and organic disease by examining the relationship between neuroticism and objective health indices such as: (a) various manifestations of coronary heart disease (CHD), (b) mortality, and (c) non-life threatening disease (e.g., irritable bowel syndrome (IBS)). Costa and McCrae concluded that neuroticism is related to somatic complaints, but its links to disease have not been proven.

Roll and Theorell (1987) compared patients complaining of chest pain without any obvious organic cause to healthy subjects matched with regard to age and sex. Their results indicated that the patient group had significantly higher scores on neuroticism, vital exhaustion, and critical recent life events. Others have also found positive relations between neuroticism and somatic complaints in the absence of disease (Costa, Fleg, McCrae, & Lakatta, 1982; Valdes, Treserra, Garcia, Pablo, & Flores, 1988).

Summary. Much evidence suggests that neuroticism is positively associated with self-reported somatic complaints, however its links to disease have not been proven.

Research has provided explanations as to how neuroticism negatively influences reports of health. These mechanisms will now be presented.

(iii) Possible mechanisms involved in the influence of neuroticism on health. Numerous mechanisms are involved in the impact of neuroticism on health. They include increased attention to one's physiological functions, cognitive interpretation, poor coping strategies, poor health habits, and physiological mechanisms. These will now be discussed in turn.

(a) Increased attention to one's physiological functions. Research has shown that

increasing attentional focus to one's bodily functions may result in higher symptom reporting.

Fillingim & Fine (1986) conducted a study to determine the effects of internal vs external attentional focus on symptom perception and performance in an exercise setting. In the internal focus condition, subjects were required to run one mile while attending to their own breathing and heart rate. In the external focus condition, subjects ran one mile while listening for a target word heard repeatedly over headphones. Results indicated that participants reported significantly less symptomatology when they were focusing externally than when they were focusing internally.

Research also shows that individuals who experience anxiety or who are high on neuroticism are more attentive toward their biological or physiological functioning. This internal attentional focus may in turn lead to somatic complaints. Pennebaker (1982) maintained that measures of anxiety can be viewed as indicators of attentiveness to symptoms. He found that scores on The Private Self-Consciousness Scale (PSC) (Fenigstein, Scheier, & Buss, 1975), a scale which measures the degree to which subjects report being aware of their thoughts and moods, were significantly correlated with the PILL (Pennebaker, 1982), a self-report inventory of somatic complaints. However, PSC scores were unrelated to reports of health-centre use, aspirin consumption, and class absences. Costa & McCrae (1980) maintained that a possible explanation as to why neuroticism is associated with somatic complaints may be that individuals high on neuroticism are more sensitive or attentive to their bodily states. Costa & McCrae (1987) also argued that people high in neuroticism are more vigilant about bodily changes. They are more apt to misinterpret unusual signs of illness and are more likely to worry about possible diseases. Pennebaker (1982) suggested that the increased attention exhibited by individuals high on neuroticism may result in high somatic complaints through the amplification of bodily concerns.

Affleck, Tennen, Urrows, & Higgins (1992) investigated neuroticism and the pain-mood relation in rheumatoid arthritis. They had subjects with rheumatoid arthritis supply daily reports of their mood and joint pain. A path-analysis suggested that the relation between neuroticism and chronic pain intensity was mediated by the propensity of individuals high on neuroticism to exaggerate their pain. Other researchers have also concluded that neuroticism or anxiety lead to amplification of bodily sensations resulting in somatic complaints (Barsky & Klerman, 1983; Costa & McCrae, 1987; Watson & Pennebaker, 1989).

Summary. Focusing on one's physiological functions leads to higher somatic complaints than focusing externally. Individuals who are high on anxiety or neuroticism focus much attention on their internal biological functioning. This internal focus exhibited by individuals high on neuroticism may lead to an amplification of biological concerns resulting in somatic complaints.

(b) Cognitive interpretation. It has been postulated that the cognitive meaning that individuals associate with pain has a profound effect on pain perception (Kreitler, Carasso, & Kreitler, 1989). Neuroticism is associated with an exaggerated interpretation of pain. For instance, Wade, Dougherty, Hart, Rafii, & Price (1992) examined the relationship between neuroticism and extraversion on the four major stages of pain processing, that of pain sensation intensity, pain unpleasantness, suffering, and pain behaviour, in chronic pain patients. Neither personality variable was related to the first stage of pain processing, pain sensation intensity. However, neuroticism was an important predictor of the other three stages. Wade et al. concluded that the last two stages of pain processing, pain suffering and pain behaviour, presumably involve extensive cognitive appraisal related to the meanings and implications that pain holds for the individuals. Neuroticism, more so than extraversion, was associated with

emotional disturbance, negative pain beliefs, and pain behaviour, and hence may have resulted in an exaggerated perception of pain. Harkins, Price, and Braith (1989) focused on the effects of extraversion and neuroticism on experimental and clinical pain in a group of myofascial pain dysfunction (MPD) patients. Results indicated that patients scoring high on neuroticism gave higher ratings of emotions related to suffering and scored higher on items related to affective disturbance on the Illness Behaviour Questionnaire (IBQ) (Pilowsky & Spence, 1976) as compared to patients scoring low on neuroticism. Harkins et al. concluded that neuroticism does not affect sensory mechanisms of nociceptive processing, but does appear to exert its influence by means of cognitive processes related to the ways in which people constitute the meanings and implications of pain.

Hence, neuroticism appears to influence those stages of pain processing involving the cognitive appraisal of pain. Neuroticism may result in an exaggerated negative cognitive appraisal of pain.

(c) Poor Coping Strategies. Others have speculated that individuals high on neuroticism report more somatic complaints because they employ less effective pain coping strategies compared to individuals low on neuroticism. For instance, Affleck et al. (1992) had seventy-five individuals with rheumatoid arthritis report their pain coping, mood, and joint pain for 75 consecutive days. Pain coping strategies used most often and considered effective included taking direct action to reduce the pain and using relaxation strategies. Strategies which were considered less effective and used least often consisted of expressing emotions about the pain and redefining the pain to make it more bearable. Neuroticism was related to a greater use of emotional expression and less use of relaxation. This association may explain why neurotic individuals

report experiencing more pain.

(d) Poor health habits. Evidence also suggests that individuals high in neuroticism exhibit a variety of poor health habits, including smoking, overeating, failure to exercise, and sleep disturbances. These poor health habits may in turn lead to subclinical problems that appear as somatic complaints (Costa & McCrae, 1987).

(e) Physiological Mechanisms. It is also reasonable to suspect that neuroticism has direct effects on various physiological pathways resulting in somatic complaints. For instance, headaches, colds, backpain, and irritable bowel syndrome (IBS) have long been thought to be associated with poor psychological adjustment. It is possible that physiological pathways can be identified that will account for an association between neuroticism and somatic complaints. Facets of neuroticism such as anger, hostility, depression and anxiety have been associated with elevated levels of corticosteroids (such as cortisol) and catecholamines (such as epinephrine) (Friedman & Booth-Kewley, 1987). Elevations of either corticosteroid or catecholamine levels may result in immuno-suppression and metabolic abnormalities (Goodkin, Antoni, & Blaney, 1986; Krantz, Baum, & Singer, 1983) which, in turn, may result in somatic complaints.

Summary. Based on the research focusing on neuroticism, it appears that when neuroticism acts as a moderator in a relationship, such as the distress - illness relationship, or when neuroticism directly influences health, neuroticism is a detrimental variable in these relationships. For example, with respect to the moderating role of neuroticism in the distress - illness relationship, individuals who experience distress and are low in neuroticism will report less health problems than individuals who are high in neuroticism. Similarly, with respect to neuroticism directly influencing health, individuals low on neuroticism will report less somatic

complaints than individuals high on neuroticism. Mechanisms involved in neuroticism's detrimental impact on health focus primarily on neuroticism as exaggerating internal physiological reactions and cognitive appraisals of pain.

However, little attention has been given to mechanisms involving affect or emotions in the relationship between neuroticism and health. In light of the observation that neuroticism may be a detrimental moderator variable in a relationship, such as the distress - illness relationship, theoretical reasoning and evidence for an additional suggested mechanism of neuroticism's influence on health, that of neuroticism reducing positive affect resulting in somatic complaints, is now presented.

(f) An additional mechanism involved in neuroticism's influence on health: Neuroticism reduces the impact of positive affect on health. Much research has focused on the possible role that cognitive appraisal may have on the influence of neuroticism on health. However, little attention has been given to the role of positive affect in this relationship. It is possible that neuroticism may reduce the positive affect experienced by individuals encountering positive events, and hence may result in somatic complaints. Evidence suggests there is a negative relationship between neuroticism and positive affect. Bouman and Luteijn (1986) studied the relations between the mood related subscale of the Pleasant Events Schedule (PES) (MacPhillamy & Lewinsohn, 1982), depression, and other psychopathology. Subjects completed the PES, the Beck Depression Inventory (Beck, Rush, Shaw, & Emery, 1979), the State-Trait Anxiety Inventory (Spielberger, Gorsuch, & Lushene, 1970), and a test which paralleled the EPI-Neuroticism scale (Eysenck & Eysenck, 1963). Principal components analysis revealed two factors, negative affect and positive affect, where the latter was dominated by PES scores.

Results showed that the PES correlated negatively with depression as well as with anxiety and neuroticism. McFatter (1994) argues that neurotic introverts report exceptionally low positive affect, compared to all other personality types. Others have also found a negative relation between neuroticism and positive affect (McCrae & Costa, 1991).

Based on the evidence that low positive affect may lead to somatic complaints and that there is a negative relation between positive affect and neuroticism, it is reasonable to suggest that neuroticism may reduce positive affect, resulting in somatic complaints.

Summary. Previous research has suggested the following relationships:

1. Positive events are associated with good health
2. Positive events are positively correlated with positive affect
3. Positive affect is correlated with good health and negatively related to somatic complaints
4. Neuroticism is correlated with somatic complaints
5. Neuroticism is negatively correlated with positive affect
6. Finally, when neuroticism acts as a moderator in the distress - illness relationship, it appears to exacerbate the relationship between distress and illness

Based on the evidence provided by past research focusing on neuroticism, eustress and health, the following hypotheses are posited:

Hypotheses

1. Conceptualization of the eustress construct. It is predicted that positive events will

significantly predict positive affect. The positive affect resulting from positive events will then be conceptualized as eustress for the present study.

2. Neuroticism moderates the Eustress-health relationship. Individuals high on both eustress and neuroticism will report more somatic complaints than individuals high on eustress and low on neuroticism. Individuals low on eustress and high on neuroticism will report more somatic complaints than individuals low on eustress and low on neuroticism. This relationship is illustrated in Figure 5.

It should be noted that although the present study focuses on a moderator model, there may be a possibility of specification error (i.e., the data of neuroticism, eustress, and reported symptoms of poor health may not reflect interactive or moderating properties in that neuroticism may not moderate the relationship between eustress and health, but may reflect linear or mediating relationships where eustress may mediate the relationship between neuroticism and health). Thus, as an exploratory assessment, a path analysis will be performed on the neuroticism, eustress, and symptoms of poor health data in order to assess any mediating effects, and direct effects between neuroticism, eustress and health.

3. Exploratory study: This study will assess for mediation and direct effects between neuroticism, eustress, and symptoms of poor health.

- (i) Mediation effects. Eustress mediates the relationship between neuroticism and health, and neuroticism also influences health through other mechanisms not involving eustress. Such mechanisms may include possible physiological processes (Friedman & Booth-Kewley, 1987) or cognitive factors (Harkins, Price, & Braith, 1989)). This

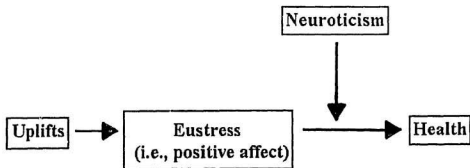


Figure 5. The eustress-health relationship moderated by neuroticism.

relationship is shown in Figure 6.

- (ii) Direct effects. A directional influence exists between uplifts and eustress, neuroticism and eustress, eustress and symptoms, and neuroticism and symptoms
- a) uplifts lead to eustress
 - b) eustress leads to low symptoms
 - c) neuroticism reduces eustress
 - d) low eustress leads to symptoms of poor health
 - e) neuroticism also leads to symptoms via mechanisms not involving eustress

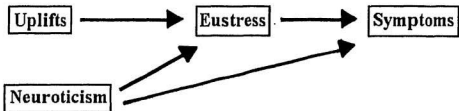


Figure 6. The neuroticism-symptoms relationship mediated by eustress.

Method

Subjects

To obtain a power of .80, (Cohen, 1992) at the $p=.05$ level for an expected medium effect size, approximately 109 subjects were required (Faul & Erdfelder, 1992). However, in order to account for an expected 50% attrition level, 218 subjects were needed. Three hundred and twenty-two undergraduates (98 men and 224 women, Mean age = 20.69 years, SD = 2.16 years) from Memorial University of Newfoundland, voluntarily recruited from psychology courses in personality and developmental psychology, participated as subjects for the first phase of this two phase prospective study. One hundred and ninety-six subjects from the original first phase subject pool then took part in the second phase which was held approximately two weeks later. This represents a return response rate of 61%.

Materials

Various measures were utilized to assess neuroticism, somatic complaints, positive events and positive affect. Measurement of the latter two variables constituted an assessment of eustress. Although it is preferable to administer two measures for each variable in the attempt to maximize the construct validity of the variables of interest, the present study used only one measure for each variable being assessed due to time constraints. However, as will be discussed shortly, all test measures have been found to be both reliable and valid indicators of the proposed theoretical constructs.

Neuroticism. The normal personality dimension of neuroticism was assessed using a 13-item bipolar trait adjective checklist taken from McCrae and Costa, (1985; see Appendix A).

McCrae & Costa selected these items on the basis of the 13 highest factor loadings for neuroticism. Each item was scored on a 9-point scale (where 1 = low on emotionality and 9 = high on emotionality). Total neuroticism scores were obtained by summing the scores on each independent item. McCrae and Costa found that with respect to internal reliability, coefficient alpha was greater than .80. With respect to validity, convergent correlations ranged from .57 to .65 and discriminant correlations were less than .25 (McCrae & Costa, 1985). It should be noted that since this bipolar trait adjective checklist, in addition to being reliable and valid, is comprised of only 13 items, due to time constraints, it was chosen in favour of other also reliable and valid, yet lengthier measures such as the NEO-PI (Costa & McCrae, 1985).

Somatic complaints. Somatic complaints were assessed using a 12-item somatization subscale from the Hopkins Symptom Checklist (HSCL; Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974; see Appendix B). The full HSCL scale consists of five basic dimensions - somatization, obsessive-compulsive, interpersonal sensitivity, depression and anxiety. However, since the present study focused on how neuroticism influences the relationship between eustress and somatic complaints, only the somatization subscale was administered. Each item was scored on a 5-point scale (where 1 = slight or no complaints and 5 = many complaints). Total symptom scores were obtained by summing the scores on each item. The scale has shown to be both valid and reliable (for example, $\alpha = .87$; Derogatis et al., 1974).

Positive events. Positive events were measured with a 53-item uplifts measure (DeLongis, Folkman, & Lazarus, 1988; See Appendix C). This scale is a thoroughly revised version of the uplifts scale used in prior research (i.e., Kanner et al., 1981). In this revised version, in the attempt to avoid a confound between uplifts and health, redundant items and words that

suggested somatic symptoms were eliminated. In order to avoid a confound between assessing uplifts and positive affect, the present study focused on an objective measure of uplifts as opposed to a subjective rating assessment. Thus, the present study was interested in the frequency as opposed to the intensity of the uplifts, where the number of items indicated as being an uplift (i.e., any uplift item rated higher than 0), independent of intensity, that is, independent of the actual value assigned to the item, were summed together to produce a total uplifts score. The scale has been shown to demonstrate good reliability and validity (DeLongis et al., 1988).

Positive affect. To assess positive affect, a 10-item positive affect (PA) scale from the Positive and Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988) was administered (See Appendix D). Each item was scored on a 5 point scale (where 1 = low positive affect and 5 = high positive affect). Total scores were obtained by summing each individual score on the items. The scale has been shown to be both reliable (e.g., coefficient alpha ranges from .86 to .90) and highly valid (convergent correlations range from .89 to .95 and discriminant correlations range from -.02 to -.18; Watson et al., 1988).

Procedure

Test measures were administered to subjects in two phases, spaced two weeks apart.

Phase one. The phase one data collection period took place between February 22, 1995 and March 3, 1995. Participation was solely on a voluntary basis and subjects were told they were free to withdraw from the study at any time. Subjects were given an informed consent form (see Appendix E) and were reassured that all information obtained would remain anonymous and that subject's involvement would in no way influence their course grade. Subjects were also given

an identification code via a code-generator sheet (see Appendix F), in order to match the data obtained in the two phases. It should be noted that the purpose of the code was strictly to enable the researcher to match the data obtained in phase one with the data obtained in phase two, and completely retained full anonymity of all subjects. In phase one, two test orders were randomly administered to the students. Subjects were given one of the following two test orders randomly selected from a pool of $5! = 120$ possible test orders: (a) Hopkins Symptom Checklist (to statistically control for the effects of baseline somatic distress or symptomatology); The positive and negative affect scale; Bipolar trait adjective checklist; information concerning demographics (in order to statistically control for any confounding effects of gender on symptomatology); and The Uplifts scale, and (b) The Positive and Negative Affect Scale; demographic information; The Uplifts scale; Bipolar trait adjective checklist; and Hopkins Symptom Checklist.

Phase two. The phase two data collection period took place between March 10, 1995 and March 17, 1995. Subjects in each class were given the phase two measures exactly two weeks after they completed the phase one measures. Again subjects were informed as to the voluntary nature of the study and reassured of full anonymity. Individuals completed the informed consent form which was identical to the one administered in Phase one, and code generator sheet (see Appendix G). In phase two, two test orders were given to subjects. In this phase the following two orders randomly selected from a pool of $4! = 24$ possible test orders were administered: (a) Hopkins Symptom Checklist; The Positive and Negative Affect Scale; demographic information; and The Uplifts scale, and (b) The Positive and Negative Affect scale; The Uplifts scale; Hopkins Symptom Checklist; and demographic information. Completion of test measures took approximately 15 minutes.

Results

Preliminary Data Screening and Descriptive Statistics

Histogram frequencies of all variables were computed. These revealed that a small number of outliers were present with respect to the Uplifts measure assessed at time2, the positive mood measure (PANAS) at time2, and the symptoms measure (Hopkins symptom checklist) at time2. Previous research has shown that outliers can have a severe impact on the interpretation of results obtained from regression analyses, since they influence the determination of one of several regression lines to be utilized (Tabachnik & Fidell, 1989). One procedure recommended for reducing the impact of outliers, is to alter the deviant score of the variable such that it is either one unit above or below the next extreme score (Tabachnik & Fidell, 1989). Transformed distributions of the variables containing outliers were calculated using this procedure. These transformed distributions subsequently revealed that all points fell within the distribution for uplifts (time 2), mood (time 2) and symptoms (time 2) and no points were detached from their distributions. The means, standard deviations and alpha coefficients for all variables are presented in Table 1.

Because multiple regression was used in the main analyses, the regression assumptions of normality, linearity, and homoscedasticity were assessed. Histogram frequencies revealed substantial skewed distributions for symptoms at time1 ($z = 7.62, p < .01$), and symptoms at time2 ($z = 8.65, p < .01$). In addition, scatterplot analyses revealed violations of linearity and homoscedasticity assumptions for symptoms (time 1) and symptoms (time 2). Tabachnik & Fidell (1989) recommend that transformations should be carried out on non-normal and non-linear distributions since such distributions violate assumptions of regression analysis (Tabachnik &

Table 1

Intercorrelations and descriptive statistics for all variables

	1	2	3	4	5	6	7	8	9	10	11
1. Sex (T1)	*										
2. Sex (T2)	.99	*									
3. Age (T1)	-.02	-.01	*								
4. Age (T2)	-.02	-.01	.99	*							
5. Symptoms (T1)	.18	.17	-.08	-.07	*						
6. Symptoms (T2)	.09	.09	-.12	-.12	.49	*					
7. Uplifts (T1)	.18	.19	-.01	.00	.06	.01	*				
8. Uplifts (T2)	.25	.24	.01	.02	.19	.04	.82	*			
9. Mood (T1)	.00	-.00	-.03	-.04	-.27	-.14	.21	-.04	*		
10. Mood (T2)	.01	.01	.07	.07	-.20	-.32	.26	.13	.54	*	
11. Neuroticism	.11	.10	-.02	.03	.40	.30	-.07	.03	-.39	-.25	*
Means			20.63	20.70	19.28	17.83	32.10	32.10	32.01	32.02	57.14
Standard deviations			2.54	2.16	5.21	4.93	7.95	8.35	6.07	6.56	14.62
alpha (α)					.73	.77	.88	.90	.86	.89	.88

Note. $p < .05$ for correlations = .19; $p < .01$ for correlations = -.39 to .99.

Fidell, 1989). Estimates of statistical significance for non-normal variables are known to be biased, and non-linear relations among variables may also pose serious problems due to a possible under or overestimation of variables (Biddle & Marlin, 1987). However, this recommendation is not universally accepted (Kenny, 1979). In general, some researchers have argued that analyses from transformed variables may be more difficult to interpret (Tabachnik & Fidell, 1989). Thus, initial exploratory, stepwise, hierarchical, and path analyses were performed for both non-transformed data and transformed variables. In all cases results were essentially identical. Hence, only the results for non-transformed data will be presented. Any differences concerning the transformed data will be indicated.

Conceptualization of eustress

It was predicted that positive affect is significantly predicted from uplifts. The positive affect arising from uplifts will be conceptualized as eustress for the present study. To assess the eustress concept, positive mood at both time 1 and time 2 was regressed using stepwise regression on the following variables, sex (time 1), symptoms (time 1), uplifts (time 1) and neuroticism.

Results indicated that uplifts (time 1) significantly predicted both mood (time 1), $p < .05$, and mood (time 2), $p < .01$ (see Table 2).

Thus, for purposes of the present study, conceptualizing eustress as the positive affect arising from positive events appears to be a valid assumption.

Table 2

Main effects in predicting eustress variance

	Time one						
	R cumulative	R ² change	F	Sig. F	b	B	Y intercept
Neuroticism	.40	.16	29.56	<.01	-.16	-.40	41.44
Uplifts (T1)	.42	.02	4.77	.03	.12	.16	37.15
Symptoms (T1)	.45	.02	4.62	.03	-.20	-.17	38.59
	Time two						
	R cumulative	R ² change	F	Sig. F	b	B	Y intercept
Neuroticism	.26	.07	11.14	<.01	-.11	-.26	38.27
Uplifts (T1)	.35	.06	9.95	<.01	.19	.24	31.59

Does neuroticism moderate the eustress-health relationship?

A hierarchical multiple regression analysis was performed to test for the moderating effects of neuroticism in the eustress-health relationship. Variables were entered using a forced entry procedure in an SPSS data-analysis program. Order of entry was determined by the assumed theoretical significance (i.e., the amount of symptomatology variance accounted for by each variable) assigned to each variable in predicting symptomatology at time2. In order to assess any main effects of the variables on symptoms (time 2), the following order of entry was chosen: sex (time1) (since based on previous research (Conger, Lorenz, Elder, Simons, and Ge, 1993; Kaplan, Anderson, and Wingard, 1991; Vebrugge, 1989) gender was expected to account for the largest symptoms (time2) variance); symptoms (time1); eustress (time1); and neuroticism. To assess the moderating effects of neuroticism of eustress on health, the interaction term, eustress \times neuroticism, was entered.

The only significant result found was that symptoms (time 1) significantly predicted symptoms (time2), $p < .01$. Table 3 gives the main and interactive effects in predicting symptoms (time 2) variability.

It could be reasoned that eustress assessed at time 1 was not an accurate reflection of eustress at other times, since subjects assessed at time 1 were possibly experiencing high distress due to midterm exams. Since eustress (time 2) was assessed during a more relaxed or more natural time, it may be a better indicator of eustress. Thus, a second regression analysis was conducted using eustress (time 2). The method and order of entry was the same as that used in the prior analysis. As found in the first analysis, symptoms (time 1) significantly predicted symptoms (time 2), $p < .01$. In addition, eustress (time 2), significantly predicted symptoms at

Table 3

Main and interactive effects in predicting symptoms (time 2) variance

	R cum	Δ R change	F change	sig. F change	b	B	Y-intercept
Sex (T1)	.09	.01	1.47	.23	.91	.09	16.17
Symptoms (T1)	.49	.23	56.43	<.01	.42	.49	9.74
Eustress (T1)	.49	.00	.02	.90	.01	.01	9.50
Neuroticism	.50	.01	3.33	.07	.04	.13	6.88
Eustress (T1) x Neuroticism	.51	.00	.97	.33	.00	.30	12.15

time 2, $p < .01$. Table 4 gives the main and interactive effects in predicting symptoms (time 2) variability.

Summary. In summary, when eustress (time 1) was included, the only significant predictor of symptoms (time 2) were symptoms (time 1). However, when eustress (time 2) was included, a slight improvement in results was obtained since significant predictors of symptoms (time 2) were symptoms (time 1) and eustress (time 2).

Assessing for mediation and direct effects

The data were subjected to a path analysis to assess for any mediational and directional influence among the variables due to the possibility of specification error. Specification error results from an inadequate theoretical framework and may lead to biased estimations. The consequences of possible specification error can be examined by focusing on other theoretical models which relax some of the assumptions of the original theoretical framework. (Gallini, 1983).

Using the procedure outlined in Kerlinger and Pedhazur (1983) a path analysis was performed on the following variables: sex (time 1), symptoms (time 1), eustress (time 1), eustress (time 2), uplifts (time 1), neuroticism, and symptoms (time 2). It should be noted that one of the assumptions of path analysis is that any variable omitted from the model must be unrelated to the predetermined variables (Gallini, 1983). Thus, to avoid a biased interpretation of results, it was necessary to include sex (time 1), eustress (time 1), and symptoms (time 1). A fully recursive just-identified model (i.e., a model encompassing all possible intercorrelations between exogenous (independent) and endogenous (dependent) variables, and one which is assumed to fit

Table 4

Main and interactive effects in predicting symptoms (time 2) variance

	R cum	R^2 change	F change	sig. F change	b	B	Y-intercept
Sex (T1)	.08	.01	1.21	.27	.82	.08	16.32
Symptoms (T1)	.50	.24	58.25	<.01	.43	.50	9.88
Eustress (T2)	.55	.05	13.47	<.01	-.16	-.23	15.67
Neuroticism	.55	.01	1.48	.22	.03	.08	14.41
Eustress (T2) x Neuroticism	.56	.01	1.91	.17	.00	.42	20.96

the observed data perfectly), was necessary (See Fig. 7). This model was used to compare and test the hypothesized model (see Fig. 8). The hypothesized model holds that uplifts give rise to eustress which in turn reduces symptoms. Neuroticism leads to somatic complaints both indirectly through reducing eustress, that is eustress mediates the relationship between neuroticism and health, and through other mechanisms (e.g., physiological factors; Friedman & Booth-Kewley, 1987). In addition to the fully recursive model and hypothesized models, four alternative models based on different theoretical principles were tested as comparison models. Alternative models were included since more than one model may fit the data equally well. Since a particular model cannot be confirmed when there are plausible alternatives which cannot be ruled out by the data, alternative models should also be examined (Cliff, 1983). A brief description of the theoretical principles of each model will now be presented.

Alternative model 1:

Alternative model 1 maintains that neuroticism leads to somatic complaints indirectly through reducing eustress (i.e., eustress mediates the relationship between neuroticism and health) and through other mechanisms (e.g., poor coping strategies (Affleck et al., 1992). Uplifts lead to good health through factors other than eustress (e.g., positive cognitive appraisal; Vinokur & Caplan, 1986).

Alternative model 2:

Alternative model 2 holds that uplifts lead to good health by resulting in eustress. Neuroticism leads to somatic complaints through factors other than reducing eustress.

Alternative model 3:

Alternative model 3 postulates that both uplifts and neuroticism influence health through

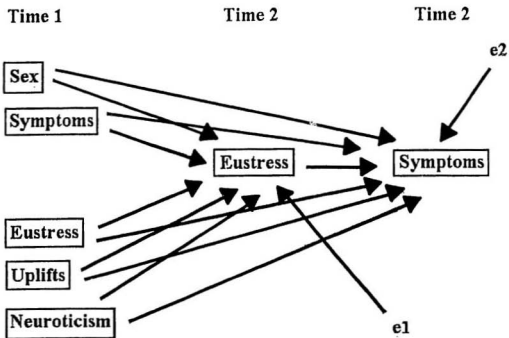


Figure 7. Fully recursive path model.

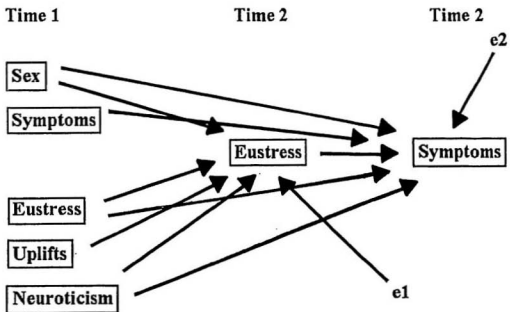


Figure 8. Hypothesized path model.

mechanisms not involving eustress.

Alternative model 4:

Alternative model 4 maintains that uplifts lead to good health indirectly through eustress, and through other factors. Neuroticism leads to somatic complaints indirectly by reducing eustress, and through other mechanisms.

Figures 9, 10, 11, & 12 illustrate the alternative models.

In the attempt to test models, path coefficients (i.e., standardized regression coefficients) were calculated for the recursive, hypothesized, and alternative models. In order to calculate path coefficients, each variable taken to be dependent was regressed on the variables upon which it is assumed to depend. The calculated Betas were the path coefficients for the paths leading from the particular set of independent variables to the dependent variables under consideration (Kerlinger & Pedhazur, 1983). A goodness of fit index, Q, and chi-square, χ^2 , was then calculated for all models. Q indicates the degree of fit between the overidentified model (i.e., a model where one or more path is deleted with respect to the fully-recursive model) being tested and the fully-recursive model. Q ranges from 0-1, where the larger the Q-value (.90 or greater), the better the fit. Chi-square indicates how well the model being tested generally fits the observed data. A non-significant chi-square indicates a good fit. It has also been suggested that a small χ^2/df ratio (ranging between 2-5) is indicative of a good fit to the observed data (Kerlinger & Pedhazur, 1983).

Figures 13, 14, 15, 16, 17, & 18 illustrate the fully-recursive, hypothesized, and alternative models respectively. Table 5 gives Q, chi-square, and the χ^2/df ratio values.

Hypothesized model. With respect to the hypothesized model, although Q was large,

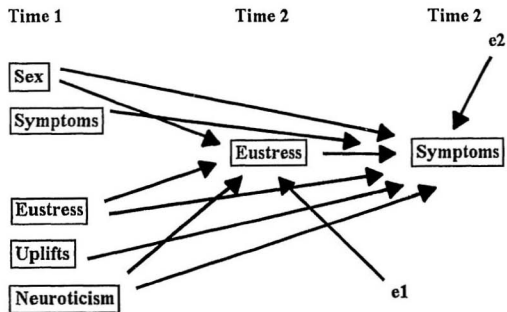


Figure 2. Alternative model 1.

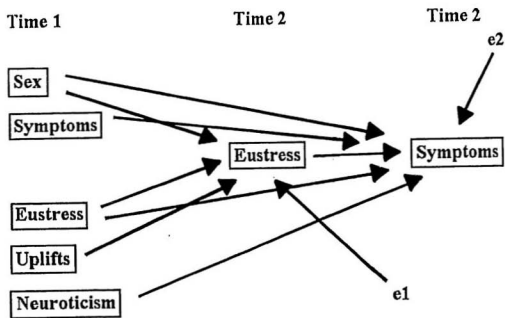


Figure 10. Alternative model 2.

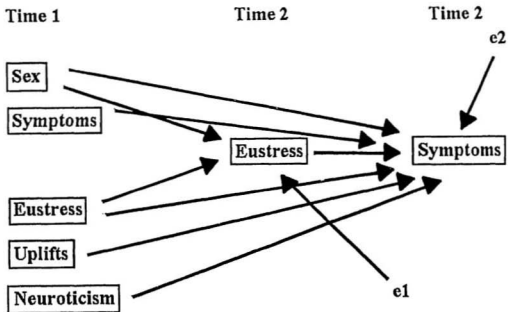


Figure 11. Alternative model 3.

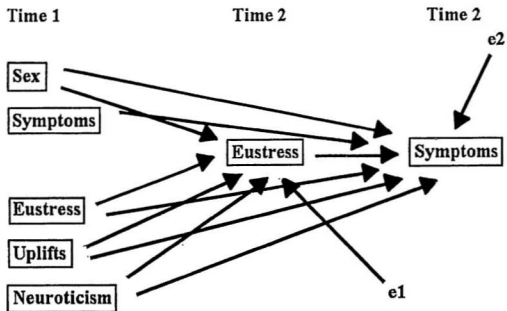


Figure 12. Alternative model 4.

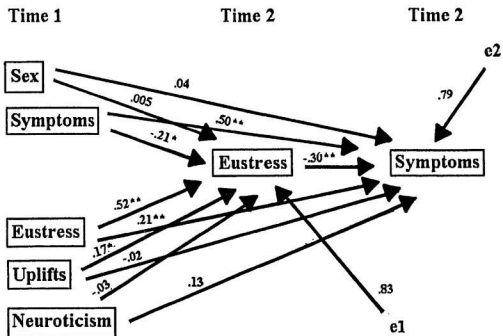


Figure 13. Fully recursive path model.

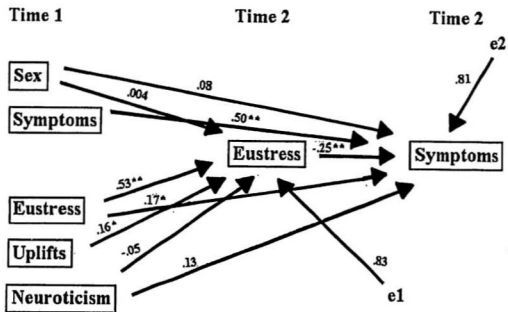


Figure 14. Hypothesized path model.

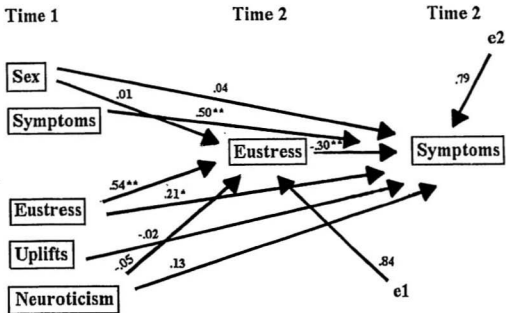


Figure 15. Alternative model 1.

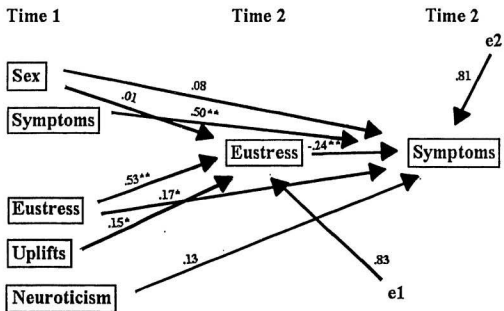


Figure 16. Alternative model 2.

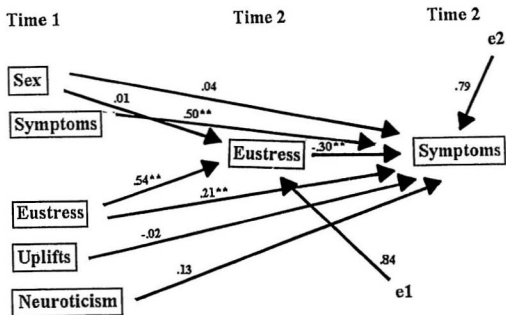


Figure 17. Alternative model 3.

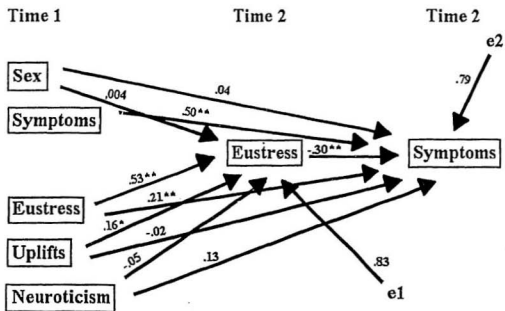


Figure 18. Alternative model 4.

Table 5

Q, chi-square, and chi-square/df values for hypothesized and alternative models

	Q	χ^2	χ^2/df
Hypothesized model	.935	13.04	6.52
Alternative model 1	.956	8.73	4.37
Alternative model 2	.915	17.14	5.71
Alternative model 3	.956	8.68	2.89
Alternative model 4	1	0	0

.935, possibly indicating a good fit between this model and the recursive model, chi-square was large and significant $\chi^2=13.04$, $p<.01$, and the χ^2/df ratio was greater than 5, $\chi^2/df=6.52$, indicating a generally poor fit to the observed data. However, within the hypothesized model, one should note that eustress (time 2) was significantly predicted from uplifts (time 1), $p < .05$, and eustress (time 1), $p < .05$. In addition, symptoms (time 2) were significantly predicted from symptoms (time 1), $p < .01$, eustress (time 1), $p < .05$, and negatively predicted from eustress (time 2), $p < .01$. Neuroticism did not significantly predict eustress (time 2) or symptoms (time 2). Gender did not significantly predict eustress (time 2) or symptoms (time 2).

Alternative model 1. In comparison to the hypothesized model, alternative model 1 displays a set of relationships where the path from uplifts (time 1) to eustress (time 2) is fixed to 0, and a path from uplifts (time 1) to symptoms (time 2) is added. This modification of the hypothesized model appears to increase the fit to the fully recursive model, since Q was large, .956. It also improves fit to the data, since chi-square was small and not significant, $\chi^2=8.73$, $p>.05$, and the chi-square/df ratio was small (4.37 falls between the range of 2 and 5). It can be seen that eustress (time 2) was significantly predicted from prior eustress (time 1), $p < .01$. Symptoms (time 2) were significantly predicted from symptoms (time 1), $p<.01$, eustress (time 1), $p<.01$, and negatively predicted from eustress (time 2), $p<.01$. Neuroticism did not significantly predict eustress (time 2) and symptoms (time 2). Uplifts did not predict symptoms (time 2), and gender did not predict eustress (time 2) and symptoms (time 2).

Alternative model 2. In comparison to the hypothesized model, alternative model 2 involves fixing the path from neuroticism to eustress (time 2) to 0. Although Q was large, .915, chi-square was significant, 17.14, $p<.01$, and the chi-square/df ratio was larger than 5,

χ^2
 $\chi^2/\text{df}=5.71$, indicating a poor fit to the data. Within the model, however, eustress (time 2) was significantly predicted from uplifts (time 1), $p<.05$, and eustress (time 1), $p<.01$. Symptoms (time 2) were significantly predicted from symptoms (time 1), $p<.01$, eustress (time 1), $p<.05$, and negatively predicted from eustress (time 2), $p<.01$.

Alternative model 3. In comparison to the hypothesized model, alternative model 3 involves fixing of the uplifts (time 1) and neuroticism paths to eustress (time 2), to 0 and adding a direct path from uplifts (time 1) to symptoms (time 2). Since $Q=.956$, χ^2 was not significant, $\chi^2=8.68$, $p>.01$, and $\chi^2/\text{df}=2.89$, this model fits the recursive model as well as the observed data. Eustress (time 2) was significantly predicted from eustress (time 1), $p<.01$. Symptoms (time 2) were significantly predicted from symptoms (time 1), $p<.01$, eustress (time 1), $p<.01$, and negatively predicted from eustress (time 2), $p<.01$. Neuroticism, uplifts (time 1), and sex (time 1) did not predict symptoms (time 2). Sex (time 1) did not predict eustress (time 2).

Alternative model 4. In comparison to the hypothesized model, alternative model 4, involves the addition of a path from uplifts (time 1) to symptoms (time 2). This model also significantly fits the fully-recursive model as well as the observed data, since Q was large, $Q=1$, chi-square was not significant, $\chi^2=0$, $p>.05$, and the chi-square/df ratio was small, $\chi^2/\text{df}=0$. Eustress (time 2) was significantly predicted from eustress (time 1), $p<.01$, and uplifts (time 1), $p<.05$. Symptoms (time 2) were significantly predicted from eustress (time 1), $p<.01$, and negatively predicted from eustress (time 2), $p<.01$. Neuroticism did not predict eustress (time 2) and symptoms (time 2). Sex (time 1) did not predict eustress (time 2) and symptoms (time 2). Uplifts (time 1) did not predict symptoms (time 2).

Summary. Path analysis revealed that alternative models 1, 3, and 4 significantly fit the

data, but the hypothesized model and alternative model 2 showed a poor fit to the observed data. However, eustress (time 2) was significantly predicted from uplifts (time 1) and symptoms (time 2) were negatively predicted from eustress (time 2). This supports the hypothesis that uplifts lead to eustress, which in turn reduces symptoms of poor health. Path analysis performed on transformed data, revealed that all models including the hypothesized model significantly fit the data, providing some support for the hypothesized model. However, since this result was obtained from transformed data, it should be interpreted with caution. Since mediator models fit the data, specification error could have resulted from testing only a moderator model. The data appear to reflect linear or mediational patterns to a larger extent than interactional or moderator relationships.

Comparison of Overidentified Path Models

The preceding analysis revealed that more than one model fit the fully recursive model as well as the observed data. However, statistical comparisons of overidentified models (i.e., models maintaining differing theoretical perspectives) also involve a comparison of competing theories and may result in the identification of one model fitting the observed data statistically better than the other models. To obtain a possible 'best fitting' overidentified model, Kerlinger & Pedhazur, (1983), suggest comparing the model with the largest number of estimated parameters or paths with alternative models exhibiting a smaller number of parameters. A goodness of fit index between these two types of models, U , is then computed. U , like Q , can range from 0-1, where the larger the U value, the better the fit between the two models. A chi-square is then computed to determine any significant differences between the model with the most paths and the alternative

model. The smaller the chi-square the better the fit. One can also compute the χ^2/df ratio where a range from 2-5 is indicative of a good fit (Kerlinger & Pedhazur, 1983). If no significant difference is obtained between these two types of models, then the more parsimonious model, that is, the model accounting for the maximum amount of data with the minimum number of theoretical hypotheses or paths, is chosen. However, if a significant difference is found between the model with the most paths and the alternative model, one should favour the model with the most parameters (Valentine, 1992).

With respect to the present study, comparisons between alternative model 4 (i.e., the model with the largest number of parameters) and alternative model 1 and alternative model 3 were conducted. U, chi-square, and χ^2/df values are given in Table 6. Results indicated that U was small, chi-square was large and significant, $p > .05$, and χ^2/df ratios were greater than 5. Thus, alternative model 4 was accepted in favour of alternative models 1 and 3 as being the best fitting model.

Summary. Comparisons were made between alternative model 4 (the model exhibiting the largest number of parameters) and alternative models 1 and 3. Comparisons revealed there were significant differences found between alternative model 4 and the other two models. Hence, alternative model 4 was chosen in favour of alternative models 1 and 3.

Eliminating the possibility of spurious relations

As an exploratory assessment, to assess the possibility that non significant paths in alternative model 4 may be spuriously causing the model to be the best fitting model, (as compared to alternative models 1 and 3) fit indices were computed for these models, where all

Table 6

Overidentifying comparisons between alternative model 4 and alternative models 1 and 3

	U	χ^2	$\frac{\chi^2}{df}$
Alternative model 1	.022	738.15	738.15
Alternative model 3	.022	734.37	367.19

models were theory trimmed. The theory-trimming procedure employs the deletion of non-significant paths from models in an attempt to improve their fit to the data (Kerlinger & Pedhazur, 1983; MacCullum, Roznowski, & Necowitz, 1992). If a theory trimmed model of alternative model 4 was again found to have the best fit over trimmed alternative models 1 and 3 then it is reasonable to assume that spurious relations were not responsible for the original non theory-trimmed alternative model 4 having the best fit.

Results showed that for a theory-trimmed model of alternative model 4 (see Fig. 19) $Q = .979$, $\chi^2 = 4.13$, $p > .01$, and $\chi^2/df = 4.13$. Theory-trimmed models of alternative models 1 and 3 were theoretically identical to each other. In these models, in comparison to the theory-trimmed model of alternative model 4, the path from uplifts (time 1) to eustress (time 2) is fixed to 0. The Q value for trimmed alternative models 1 and 3 is $.958$, $\chi^2 = 12.51$, $p < .01$, and $\chi^2/df = 6.21$. Thus, the trimmed alternative model 4, fit the data significantly better than trimmed alternative models 1 and 3. Hence, the possibility of spurious relations resulting in a better fit of the original non theory-trimmed alternative model 4 over the non-trimmed alternative models 1 and 3 is not very probable.

Summary. The theory-trimmed model of alternative model 4 was found to fit the data significantly better than trimmed alternative models 1 and 3. It is therefore not likely that spurious relations resulted in the original alternative model 4 fitting the data significantly better than the original alternative models 1 and 3.

Threats to internal validity

Statistical tests were also performed to assess possible effects of subject attrition and

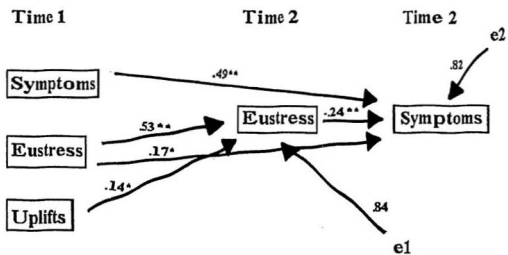


Figure 19. Final theory-trimmed model of alternative model 4.

subject selection (Campbell & Stanley, 1966). With respect to subject attrition, since not all subjects completed measures for both phases it is possible that individuals who completed both phases differed with respect to eustress, neuroticism and somatic complaints compared to subjects who completed only one phase. T-tests were performed between subjects who completed measures for both phases and subjects who completed measures for only one phase. There was no significant difference in the means found between these two groups with respect to all variables (see Table 7); for eustress, $t(df = 122) = -.65, p > .05$; for uplifts, $t(df = 123) = -1.35, p > .05$; for symptoms, $t(df = 121) = .51, p > .05$.

With respect to subject selection, since subjects volunteered from three different classes, it is reasonable to speculate that differences in the variables of interest may be attributed to class membership. A 3(number of different psychology classes) \times 7(number of variables) anova was performed on the data. Comparisons between the means for all three classes with respect to all variables showed no significant differences (see Table 8); for eustress (time 1) $F(2, 311) = .205, p > .05$; for eustress (time 2) $F(2, 191) = .105, p > .05$; for symptoms (time 1), $F(2, 314) = .893, p > .05$; for symptoms (time 2), $F(2, 193) = 1.46, p > .05$; for neuroticism, $F(2, 312) = 1.43, p > .05$; for uplifts (time 1), $F(2, 264) = .109, p > .05$; for uplifts (time 2), $F(2, 159) = .188, p > .05$.

Thus results cannot be attributed to subject attrition or subject selection.

Summary. Analyses were performed to determine whether the results could be attributed to subject attrition and subject selection. With respect to subject attrition, results indicated there were no differences between subjects who completed both phases compared to subjects who completed only one phase with respect to all variables. With respect to subject selection, comparisons between the three classes revealed no significant differences with respect to all

Table 7

Means of eustress, uplifts, and symptoms comparing subjects who completed one phase with subjects who completed both phases

	Eustress	Uplifts	Symptoms
One phase	31.62	31.09	19.44
Both phases	32.10	32.45	19.09

Table 8

Means comparing the three classes for eustress, symptoms, neuroticism, and uplifts (both phases)

EustressT1 EustressT2 SymptomsT1 SymptomsT2 Neuroticism UpliftsT1 UpliftsT2

Class 1	30.04	32.00	18.00	16.06	56.27	31.61	30.79
Class 2	32.15	31.91	19.41	18.12	57.94	32.07	32.23
Class 3	32.37	32.50	19.34	17.32	54.53	32.53	32.20

variables. This eliminates the possibility of results being attributed to subject attrition or subject selection.

Discussion

The present study examined the role of the personality trait neuroticism in moderating the relationship between eustress and health. Eustress was conceptualized as the positive affect resulting from positive events and has been shown to be related to good health. It was reasoned that neuroticism may reduce the eustress experienced by individuals and hence may lead to various health problems or somatic complaints. In other words, individuals high on both eustress and neuroticism were expected to report more somatic complaints than individuals high on eustress but low on neuroticism. Individuals low on eustress and high on neuroticism were expected to report more somatic complaints than individuals low on eustress and low on neuroticism.

Conceptualization of eustress

The assumption that eustress can be conceptualized as the positive affect resulting from positive events was supported. Uplifts (time 1) were shown to predict both positive affect at time 1 and time 2.

Neuroticism as a moderator in the Eustress - health relationship

Using hierarchical multiple regression it was found that the best predictors of somatic complaints, were previous complaints, and eustress. Thus, since the eustress x neuroticism interaction in predicting symptoms (time 2) was not significant, the hypothesis arguing that neuroticism moderates the relationship between eustress and health was not supported.

Assessing mediation and direct effects

Due to a possible specification error, path analyses were carried out on the data to determine any mediator effects in addition to a more specific directional influence between the variables of interest (i.e., neuroticism, eustress, and symptoms). The hypothesized model maintained that uplifts give rise to eustress, which in turn reduces symptoms of poor health. Neuroticism reduces eustress, resulting in somatic complaints. In other words, eustress mediates the relationship between neuroticism and health. Neuroticism may also lead to somatic complaints through other factors, for example, cognitive processes (Harkins, Price, & Braith, 1989) and physiological mechanisms (Friedman & Booth-Kewely, 1987). Path analyses did not provide evidence for the hypothesized model as being one of the models significantly fitting the data. For the hypothesized model, however, it was found that uplifts influence health through their impact on eustress. This finding supports the notion that eustress is associated with good health, or that positive events give rise to eustress which leads to good health. As mentioned in the Introduction, the processes involved in the impact of eustress on health may be both direct (physiological mechanisms) or indirect (coping processes). Since the relationship between eustress and symptoms was negative, it may be reasoned that a reduction in eustress may lead to somatic complaints or poor health. With respect to the mediating role of eustress in the relationship between neuroticism and health, it appears that neuroticism had a statistically non significant influence in reducing eustress. Thus, the hypothesis that neuroticism reduces eustress resulting in symptoms of poor health, was not supported. Also, since a direct path from neuroticism to symptoms was not significant, the hypothesis that neuroticism may lead to somatic complaints through factors other than reducing eustress, for example, increased attentiveness to

one's physiological functions (Costa & McCrae, 1980; Costa & McCrae, 1987; Pennebaker, 1982), exacerbating the cognitive meaning of symptoms (Affleck et al., 1992; Costa & McCrae, 1987; Harkins et al., 1989; Pennebaker, 1989; Wade et al., 1992), poor coping strategies (Affleck et al., 1992), poor health habits (Costa & McCrae, 1987) and physiological pathways (Friedman & Booth-Kewely, 1987) was not supported. It should be noted that transformed data revealed that the hypothesized model did fit the data providing some support for this model. However, since this result was obtained from transformed data, it should be interpreted with caution.

Alternative Models. In addition to the hypothesized model, a number of theoretically alternative models were also tested.

In the original analysis, prior to a theory-trimming analysis, alternative model 4 was found to have the best fit to the observed data. This model maintains that uplifts lead to good health indirectly through eustress and through other factors (such as cognitive mechanisms, Vinokur & Caplan, 1986). Neuroticism leads to reports of somatic complaints indirectly by reducing eustress and through other mechanisms (for example physiological pathways, Friedman & Booth-Kewley, 1987). The only significant results obtained from this model, however, are that uplifts lead to eustress which in turn leads to low symptoms of poor health. As an exploratory assessment, comparisons of theory-trimmed models (i.e., where all non-significant paths were deleted in all models) were performed to assess the possibility that non significant paths in model 4 may be spuriously causing the model to be the best fitting model. However, the possibility of spurious relations resulting in a better fit of the original non theory-trimmed alternative model over other non-trimmed alternative models did not appear to be very likely.

It should be noted that since mediator models fit the observed data, it appears that a model

focusing on interactive effects (i.e., a moderator model) does not adequately describe the observed data. A linear relationship or mediator model appears to be a more accurate description of the observed data. Thus, it was concluded that specification error could have resulted from testing only a moderator model.

Possible explanations of major findings

The present study did not support the finding of past research (for example, Bouman & Luteijn, 1986) which suggested that neuroticism is negatively related to positive affect arising from positive events (i.e., eustress). A possible explanation for the present findings with regards to the relationship between eustress and neuroticism, may be due to methodological problems. Previous research (Bouman & Luteijn, 1986) administered two measures of neuroticism such as the State-Trait Anxiety Inventory (Spielberger et al., 1970), and a parallel test of the EPI - Neuroticism scale (Eysenck & Eysenck, 1963). It is possible that administration of only a single measure (bipolar-trait adjective checklist) in the present study was not sufficient to assess the neuroticism construct. The construct validity of assessing neuroticism may have been maximized by administering more than one measure in addition to the bipolar trait adjective checklist. Also, past researchers who have found a negative relationship between neuroticism and positive affect (McCrae & Costa, 1991) used the NEO-PI (Costa & McCrae, 1985). The NEO-PI explicitly measures facets of neuroticism such as depression, anxiety, hostility, self-consciousness, impulsivity, and vulnerability as compared to other measures such as the bipolar-trait adjective checklist. Hence, administration of the NEO-PI may have been more likely to be measuring the neuroticism construct. Also, since eustress was conceptualized as the positive affect resulting

from positive events, perhaps a more comprehensive measure of positive affect could have enabled a negative relationship between eustress and neuroticism to be detected.

Another point worth mentioning is that since the present study was interested in the frequency as opposed to the intensity of uplifts, the uplifts measure could have also required subjects to rate how often each event had occurred in the past week. This may have also resulted in a stronger relationship between eustress and neuroticism.

In addition, previous research supporting a positive relationship between neuroticism and somatic complaints (for example, Okun & George, 1984) was not supported by the present study. A possible explanation may be that previous research used a method of measuring health, where subjects were asked to rate their health as 'excellent', 'good', 'fair', or 'poor' (Okun, & George, 1984). Since subjective health status is largely related to neuroticism (Costa & McCrae, 1987), an item on overall self-health rating requiring subjects to rate their health as 'excellent', 'good', 'fair', or 'poor', could have been added to the somatic complaints measure. Also, with reference to the present study, subjects may have been exhibiting a bias with respect to completing the symptoms checklist. Medical symptoms such as 'faintness or dizziness', 'pains in heart or chest', 'trouble getting breath', 'hot or cold spells' may be socially viewed as being more serious than other symptoms such as 'headaches', and subjects may have been reluctant to admit to experiencing these slightly more serious problems. In assessing symptoms, it may have been wise to include more items postulated to arise from neuroticism (for example, symptoms related to immuno-suppression, Friedman & Booth-Kewely, 1987) such as colds, flu, or sore throat, however which are not socially viewed as 'major symptoms'. Admitting to such minor medical complaints would probably not cause the subject to be socially conscious.

In addition, previous research (Ormel, 1983), assessed reports of somatic complaints by studying the frequency, duration, and intensity of minor health problems (e.g., headaches) as opposed to only assessing the intensity of health problems (both major and minor) in the present study. The additional information concerning symptom frequency and duration assessed in past research may have enhanced detection of a relationship between neuroticism and reports of somatic complaints.

Another explanation for failing to find a significant relationship between neuroticism and somatic complaints in the present study may be that neuroticism was measured during a time period where students were also involved with mid-term examinations. Thus, students who would not normally score moderate or high on neuroticism (during more normal or relaxed times) may have rated themselves as being more worried, nervous, high-strung, or emotional as assessed by the bipolar trait adjective checklist. Thus, the relationship between neuroticism and symptoms may not have been validly assessed due to subjects possible inaccurate rating of neuroticism during an emotional time. Previous research did not assess neuroticism at a generally emotional time (Okun & George, 1984; Ormel, 1983).

Summary

The hypothesis that eustress can be conceptualized as the positive affect resulting from positive events was supported. Neuroticism was not found to moderate the eustress-health relationship. However, eustress significantly predicted symptoms. A hypothesized mediator or linear model was not found to have a good fit to the data. The direct effects hypotheses suggesting that uplifts lead to eustress, which in turn reduces symptoms of poor health was

supported. The direct effects hypothesis maintaining that neuroticism reduces the impact of eustress resulting in somatic complaints was not supported. In addition, the hypothesis that neuroticism may lead to somatic complaints through factors other than reducing eustress was not supported. Transformed data showed some support for the hypothesized model. However, since this finding is based on transformed data, it should be interpreted with caution. Since path analytic models fit the data, specification error could have resulted from testing only a moderator model since the data may reflect linear or mediational relationships to a greater degree than interactive or moderator properties. Possible reasons as to why symptoms did not result from neuroticism reducing eustress or through other mechanisms, may include inappropriate assessment of the neuroticism construct due to methodological problems and time factors, and subject bias with regards to rating symptoms.

Future research

Future researchers may wish to investigate any mediating factors involved in the impact of eustress on health. For instance, it would be interesting to test whether eustress leads to any physiological changes, (for example, stimulation of the production of HDL cholesterol, testosterone, insulin, adrenaline, and growth hormone) which may ultimately lead to improved health (Karasek et al., 1982). Figure 20 depicts a model examining such possible mechanisms.

One may also wish to investigate how eustress affects health through influencing coping processes directed toward existing distress (Lazarus et al., 1980b). Figure 21 illustrates a model examining such a relationship.

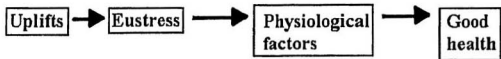


Figure 20. Physiological mediator model .

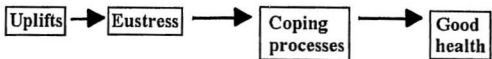


Figure 21. Coping process mediator model.

References

- Aldwin, C.M., Levenson, M.R., Spiro, A., & Bosse, R. (1989). Does emotionality predict stress? Findings from the normative aging study. Journal of Personality and Social Psychology, 56(4), 618-624
- Affleck, G., Tennen, H., Urrows, S., & Higgins, P. (1992). Neuroticism and the pain-mood relation in rheumatoid arthritis: Insights from a prospective daily study. Journal of Consulting and Clinical Psychology, 60(1), 119-126.
- Anderson, C.A., & Arnoult, L.H. (1989). An examination of perceived control, humour, irrational beliefs, and positive stress as moderators of the relation between negative stress and health. Basic and Applied Social Psychology, 10, 101-117.
- Baron, R.M. & Kenny, D.A. (1986). The moderator-mediator variable distinction in Social Psychological research: Conceptual, strategic, and statistical considerations. Journal of Personality and Social Psychology, 51(6), 1173-1182.
- Barsky, A.J., & Klerman, G.L. (1983). Overview: Hypochondriasis, bodily complaints, and somatic styles. American Journal of Psychiatry, 140, 273-283.
- Beck, A.T., Rush, A.J., Shaw, B.F., & Emery, G. (1979). Cognitive therapy of depression. New York: Wiley.
- Biddle, B.J., & Marlin, M.M. (1987). Causality, confirmation, credulity, and structural equation modelling. Child Development, 58, 4-17.
- Bouman, T.K. & Luteijn, F. (1986). Relations between the Pleasant Events Schedule, Depression, and other aspects of Psychopathology. Journal of Abnormal Psychology, 95(4), 373-377.
- Bradburn, N. (1969). The structure of well-being. Aldine, Chicago.
- Brandstatter, H. (1983). Emotional responses to other personas in everyday life situations. Journal of Personality and Social Psychology, 45, 871-883.
- Breznitz, S. & Goldberger, L. (1982). Stress research at a crossroads. In Goldberger, L. & Breznitz, S. (Eds.) Handbook of stress. Theoretical and Clinical aspects p.3-6. New York: Macmillan Publishing Co.
- Brown, J.D. & McGill, K.L. (1989). The cost of good fortune. When positive life events produce negative health consequences. Journal of Personality and Social Psychology, 57, 1103-1110.

- Campbell, D.T., & Stanley, J.C. (1966). Experimental and quasi-experimental designs for research. Boston: Houghton Mifflin Co. pp.5-16.
- Clark, L.A. & Watson, D. (1988). Mood and the mundane: Relations between daily life events and self-reported mood. Journal of Personality and Social Psychology, 54(2), 296-308.
- Cliff, N. (1983). Some cautions concerning the application of causal modeling methods. Multivariate Behavioral Research, 18, 115-126.
- Cogan, R., Cogan, D., Waltz, W., & McCue, M. (1987). Effects of laughter and relaxation on discomfort thresholds. Journal of Behavioral Medicine, 10(2), 139-143.
- Cohen, J. (1992). A power primer. Psychological Bulletin, 112(1), 155-159.
- Cohen, S. & Hoberman, H.M. (1983). Positive events and social supports as buffers of life change stress. Journal of Applied Social Psychology, 13(2), 99-125.
- Conger, R.D., Lorenz, F.O., Elda, G.H.Jr., Simons, R.L., & Ge, X. (1993). Husband and wife differences in response to undesirable life events. Journal of Health and Social Behaviour, 34, 71-88.
- Costa, P.T.Jr., Fleg, J.L., McCrae, R.R., & Lakatta, E.G. (1982). Neuroticism, coronary artery disease, and chest pain complaints: Cross sectional and longitudinal studies. Experimental Aging Research, 8, 37-44.
- Costa, P.T.Jr., & McCrae, R.R. (1980). Somatic complaints in males as a function of age and neuroticism: A longitudinal analysis. Journal of Behavioral Medicine, 3(3), 245-257.
- Costa, P.T. (1987). Influence on the normal personality dimension of Neuroticism on chest pain symptoms and coronary artery disease. American Journal Cardiology, 60, 20J-26J.
- Costa, P.T., Jr., & McCrae, R.R. (1985). The NEO Personality Inventory manual. Odessa, FL: Psychological Assessment Resources.
- Costa, P.T., Jr., & McCrae, R.R. (1987). Neuroticism, somatic complaints, and disease. Is the bark worse than the bite? Journal of Personality, 55(2), 299-316.
- Coyne, L.C., & Lazarus, R.S. (1980). Cognitive style, stress perception, and coping. In I.L. Kutash, & L.B. Schlesinger (Eds.), Handbook of stress and anxiety: Contemporary knowledge, theory, and treatment. San Francisco: Jossey-Bass.
- Croyle, R.T., & Uretsky, M.B. (1987). Effects of mood on self-appraisal of health status. Health Psychology, 6(3), 239-253.

- Csikszentmihalyi, M., & Figurski, T.J. (1982). Positive experiences are associated with voluntarism. Journal of Personality, 50, 15-28.
- DeLongis, A., Folkman, S., & Lazarus, R.S. (1988). The impact of daily stress on health and mood: Psychological and social resources as mediators. Journal of Personality and Social Psychology, 54(3), 486-495.
- DeLongis, A., Coyne, J.C., Dakof, G., Folkman, S., & Lazarus, R.S. (1982). Relationship of daily hassles, uplifts, and major life events to health status. Health Psychology, 1, 119-136.
- Denney, D.R., & Frisch, M.B. (1981). The role of neuroticism in relation to life stress and illness. Journal of Psychosomatic Research, 25(4), 303-307.
- Derogotis, L.R., Lipman, R.S., Rickels, K., Uhlenhuth, E.H., & Covi, L. (1974). The Hopkins Symptom Checklist (HSCL): A self-report symptom inventory. Behavioral Science, 19, 1-15.
- Depue, R. A., & Monroe, S.M. (1986). Conceptualization and measurement of human disorder in life stress research: The problems of chronic disturbance. Psychological Bulletin, 99(1), 36-51.
- Digman, J.M., & Inouye, J. (1986). Further specification of the five robust factors of personality. Journal of Personality and Social Psychology, 50, 116-123.
- Dohrenwend, B.P., & Dohrenwend, B.S. (1969). Social status and psychological disorder. New York: Wiley-Interscience.
- Dohrenwend, B.P., & Dohrenwend, B.S. (1981). Stressful life events and their contexts. New York: Prodist.
- Dohrenwend, B.P., Pearlin, L., Clayton, P., Hamburg, B., Riley, M., Rose, R.M., & Dohrenwend, B. (1982). Report on stress and life events. In G.R. Elliot & C. Eisdorfer (Eds.), Stress and human health. Analysis and implications of research. New York: Springer.
- Dolliver, R.H. (1987). Testing the test. A review of the NEO Personality Inventory. Journal of Counseling and Development, 65, 107-108.
- Dua, J. & Price, I. (192). Psychometric analysis of the subscales of the Thought and Real-Life Experiences Scale. Behaviour Change, 9(2), 104-111.
- Edwards, J.R., & Cooper, C.L. (1988). The impacts of positive psychological states on physical health: A review and theoretical framework. Social Science and Medicine, 27(12), 1447-1459.

- Endler, N.S. (1988). Hassles, health and happiness. In M.P. Janisse (Eds.), Individual differences, stress, and health psychology. New York: Springer
- Evans, P.D., & Edgerton, N. (1991). Life-events and mood as predictors of the common cold. British Journal of Medical Psychology, 64(1), 35-44.
- Eysenck, H.J., & Eysenck, S.B.G. (1963). Eysenck Personality Inventory, Form A and B. San Diego, CA: Educational and Industrial Testing Service.
- Faul, F., & Erdfelder, E. (1992). GPOWER: A priori, post-hoc, and compromise power analyses for MS-DOS (Computer program). Bonn, FRG: Bonn University, Dep. of Psychology.
- Fenigstein, A., Scheier, M.F., & Buss, A. (1975). Public and private self-consciousness: Assessment and theory. Journal of Consulting and Clinical Psychology, 43, 522-527.
- Fillingim, R.B., & Fine, M.A. (1986). The effects of internal versus external information processing on symptom perception in an exercise setting. Health Psychology, 5(2), 115-123.
- Flood, M., & Endler, N.S. (1980). The interaction model of anxiety: An empirical test in an athletic competition situation. Journal of Research in Personality, 14, 329-339.
- Frankenhaeuser, M. (1980). Psychobiological aspects of life stress. In S. Levine & H. Ursin (Eds.), Coping and health. New York: Plenum.
- Friedman, H.S., & Booth-Kewley, S. (1987). The 'Disease-prone personality'. American Psychologist, 42(6), 539-555.
- Gallini, J. (1983). Misspecifications that can result in path analysis structures. Applied Psychological Measurement, 7(2), 125-137.
- Goodkin, K., Antoni, M.H., & Blaney, P.H. (1986). Stress and hopelessness in the promotion of cervical intraepithelial neoplasia to invasive squamous cell carcinoma of the cervix. Journal of Psychosomatic Research, 30, 67-76.
- Greenberg, J.S. (1987). Comprehensive Stress Management. Second Edition. Dubuque, Iowa: Wm. C. Brown Publishers.
- Harkins, S.W., Price, D.D., & Braith, J. (1989). Effects of extraversion and neuroticism on experimental pain, clinical pain, and illness behavior. Pain, 36, 209-218.
- Holmes, T.H., & Rahe, R.H. (1967). The Social Readjustment Rating Scale. Journal of Psychosomatic Research, 11, 213-218.

- Idler, E.L., Kasl, S.V., & Lemke, J.H. (1990). Self-evaluated health and mortality among the elderly in New Haven, Connecticut, and Iowa and Washington counties, Iowa. American Journal of Epidemiology, 131, 91-103.
- Jacobs, T.J., & Charles, E. (1980). Life events and the occurrence of cancer in children. Psychosomatic Medicine, 42, 11-24.
- James, L.R., & Brett, J.M. (1984). Mediators, moderators, and tests for mediation. Journal of Applied Psychology, 69(2), 307-321.
- Jenkins, C.D., Stanton, B., Klein, M.D., Savageau, J.A., & Dwight, E.H. (1983). Correlate of angina pectoris among men awaiting coronary by-pass surgery. Psychosomatic Medicine, 45, 141-153.
- Kanner, A.D., Coyne, J.C., Schaefer, C., & Lazarus, R.S. (1981). Comparison of two modes of stress measurement: Daily hassles and uplifts versus major life events. Journal of Behavioral Medicine, 4(1), 1-39.
- Kanner, A.D., Kafry, D., & Pines, A. (1978). Conspicuous in its absence: The lack of positive conditions as a source of stress. Journal of Human Stress, 4(4), 33-39.
- Kaplan, R.M., Anderson, J.P., & Wingard, D.L. (1991). Gender differences in health-related quality of life. Health Psychology, 10(2), 86-93.
- Karasek, R.A., Russell, R.S., & Theorell, T. (1982). Physiology of stress and regeneration in job-related cardiovascular illness. Journal of Human Stress, 8, 29-42.
- Kasl, S.V., & Cobb, S. (1982). Variability of stress effects among men experiencing job loss. In Handbook of stress: Theoretical and Clinical aspects (Goldberger, L., & Breznitz, S. (Eds.), 445-461. New York: Free Press.
- Kenny, D. (1979). Correlation and Causality 45-73. John Wiley and sons: New York.
- Kerlinger, F.N., & Pedhazur, E.J. (1983). Multiple Regression in behavioral research. 577-635. New York: Holt, Rinehart, & Winston.
- Kiecolt, G., Janice, K., Kennedy, S., Malkoff, S., & Fisher, L. (1988). Marital discord and immunity in males. Psychosomatic Medicine, 50(3), 213-229.
- King, P.R., & Endler, N.S. (1990). The interaction model of anxiety: A critical appraisal of current research methods. Personality and Individual Differences, 11(3), 233-237.

- Krantz, D.S., Baum, A., & Singer, J.E. (Eds.). (1983). Handbook of psychology and health: Vol. 3. Cardiovascular disorders and behaviour. Hillsdale, N.J.: Erlbaum.
- Kreitler, S., Carasso, R., & Kreitler, H. (1989). Cognitive styles and personality traits as predictors of response to therapy in pain patients. Personality and Individual Differences, 10(3), 313-322.
- Landreville, P., & Vezina, J. (1992). A comparison between daily hassles and major life events as correlates of well-being in older adults. Canadian Journal on Aging, 11(2), 137-149.
- Larsen, R.J., & Kasimatis, M. (1991). Day-to-day physical symptoms: Individual differences in the occurrence, duration, and emotional concomitants of minor daily illnesses. Special Issues: Personality and daily experience. Journal of Personality, 59(3), 387-423.
- Lazarus, R.S. (1966). Psychological stress and the coping process. New York: Wiley, 1974.
- Lazarus, R.S., Cohen, J.B., Folkman, S., Kanner, A., & Schaefer, C. (1980a). Psychological stress and adaptation: Some unresolved issues. In H. Selye (Ed.), Selye's guide to stress research, vol. I. New York: Van Nostrand.
- Lazarus, R.S., Kanner, A.D., & Folkman, S. (1980b). Emotions: A cognitive phenomenological analysis. In Plutchik, R., & Kellerman, H. (Eds.), Emotion: Theory, research, and experience. New York: Academic Press.
- Lazarus, R.S. (1981). The stress and coping paradigm. In C. Eisdorfer, D. Cohen, A. Kleinman, & P. Maxim (Ed.), Theoretical bases for psychopathology. New York: Spectrum.
- Lazarus, R.S., & Folkman, S. (1982). Coping and adaptation. In W.D. Gentry (Ed.), The handbook of behavioral medicine. New York: Guilford.
- Linn, B.S., & Linn, M.W. (1980). Objective and self-assessed health in the old and very old. Social Science and Medicine, 14, 311-315.
- Lubin, B., Zuckerman, M., Breytspreek, L.M., & Bull, N.C. (1988). Affects, demographic variables and health. Journal of Clinical Psychology, 44(2), 131-141.
- MacPhillamy, D.J., & Lewinsohn, P.M. (1982). The Pleasant Events Schedule: Studies on reliability, validity, and scale intercorrelations. Journal of Consulting and Clinical Psychology, 50, 363-380.
- MacCullum, R.C., Roznowski, M., & Necowitz, L.B. (1992). Model modifications in covariance structure analysis: The problem of capitalization on chance. Psychological Bulletin, 111(3), 490-504.

- Maddi, S.R., Bartone, P.T., & Puccetti, M.C. (1987). Stressful events are indeed a factor in physical illness: Reply to Schroeder and Costa (1984). Journal of Personality and Social Psychology, 52(4), 833-843.
- McCrae, R.R., & Costa, P.T., Jr. (1984). Emerging issuers, enduring dispositions: Personality in adulthood. Boston: Little Brown.
- McCrae, R.R., & Costa, P.T., Jr. (1985). Updating Norman's "Adequate Taxonomy": Intelligence and Personality dimensions in natural language and in questionnaires. Journal of Personality and Social Psychology, 49(3), 710-721.
- McCrae, R.R., Costa, P.T., Jr., & Busch, C.M. (1986). Evaluating comprehensiveness in personality systems: The California Q-set and the five factor model. Journal of Personality, 54, 430-446.
- McCrae, R.R., & Costa, P.T. Jr. (1991). Adding liebe und arbeit: The full five-factor model and well-being. Personality and Social Psychology Bulletin, 17(2), 227-232.
- McFatter, R.M. (1994). Interactions in predicting mood from extraversion and neuroticism. Journal of Personality and Social Psychology, 66(3), 570-578.
- McLennan, J., & Bates, G.W. (1993). Vulnerability to psychological distress: Empirical and conceptual distinctions between measures of neuroticism and negative affect. Psychological Reports, 73, 1315-1323.
- Mechanic, D. (1983). The experience and expression of distress: The study of illness behaviour and medical utilization. In Mechanic, D. (Ed.) Handbook of health, health care, and the health professions. New York: Free Press.
- Miller, M. J. & Wilcox, C.T. (1986). Measuring perceived hassles and uplifts among the elderly. Journal of Human Behaviour and Learning, 3(1), 38-46.
- Monroe, S.M. (1983). Social support and disorder: Toward an untangling of cause and effect. American Journal of Community Psychology, 11, 81-97.
- Mullis, R.L., Youngs, G.A., Jr., Mullis, A.K., & Rathge, R.W. (1993). Adolescent stress: Issues of measurement. Adolescence, 28(110), 267-279.
- Nowack, K.M. (1990). Initial development of an inventory to assess stress and health risk. American Journal of Health Promotion, 4(3), 173-180.

- Okun, M.A., & George, L.K. (1984). Physician- and self-ratings of health, neuroticism and subjective well-being among men and women. Personality and Individual Differences, 5(5), 533-539.
- Ormel, J. (1983). Neuroticism and well-being inventories: measuring traits or states? Psychological Medicine, 13, 165-176.
- Pearlin, L. I. (1989). The sociological study of stress. Journal of Health and Social Behavior, 30, 241-256.
- Pennebaker, J.W. (1982). The psychology of physical symptoms. New York: Springer-Verlag.
- Phillips, J.B., & Endler, N.S. (1982). Academic examinations and anxiety: The interaction model empirically tested. Journal of Research in Personality, 16, 303-318.
- Pilowsky, I., & Spence, N.D. (1976). Illness behavior syndromes associated with intractable pain. Pain, 2, 61-71.
- Rabkin, J.G., & Struening, D.L. (1976). Life events, stress and illness. Science, 194, 1013-1020.
- Reich, J.W. & Zautra, A. (1981). Life events and personal causation: Some relationships with distress and satisfaction. Journal of Personality and Social Psychology, 41, 1002-1012.
- Reich, J.W. & Zautra, A.J. (1988). Direct and stress-moderating effects of positive life experiences. In Cohen, L.H. (Ed), Life events and psychological functioning: Theoretical and methodological issues. Newbury Park, CA: Sage Publications.
- Roll, M., & Theorell, T. (1987). Acute chest pain without obvious organic cause before age 40 - Personality and recent life events. Journal of Psychosomatic Research, 31(2), 215-221.
- Russell, D.W., & Cutrona, C. E. (1991). Social support, stress, and depressive symptoms among the elderly: Test of a process model. Psychology and Aging, 6(2), 190-201.
- Rutter, M., & Sandberg, S. (1992). Psychosocial stressors.: Concepts, causes and effects. European Child and Adolescent Psychiatry, 1(1), 3-13.
- Sarason, I.G., Johnson, J.H., & Siegel, J.M. (1978). Assessing the impact of life changes: Development of the Life Experiences Survey. Journal of Consulting and Clinical Psychology, 46(5), 932-946.
- Sarason, I.G., Sarason, B.R., Potter, E.H., & Antoni, M.H. (1985). Live events, social support, and illness. Psychosomatic Medicine, 17(2), 156-163.

- Schroeder, D.H., & Costa, P.T., Jr. (1984). Influence of life event stress on physical illness: Substantive effects or methodological flaws? Journal of Personality and Social Psychology, 46, 853-863.
- Selye, H. (1976). The stress of Life. New York: McGraw Hill.
- Smith, T.W., & Williams, P.G. (1992). Personality and health: Advantages and limitations of the five-factor model. Journal of Personality, 60(2), 395-423.
- Spielberger, C.D., Gorsuch, R.L., & Lushene, R.L. (1970). STAI manual for the State-Trait Anxiety Inventory. Palo Alto: Consulting Psychologists Press.
- Spielberger, C.D. (1972). Conceptual and methodological issues in anxiety research. In Spielberger, C.D. (Eds.), Anxiety: Current trends in theory and research, Vol. 2. New York: Academic Press.
- Stalling, R.B. (1992). Mood and Pain: The influence of positive and negative affect on reported body aches. Journal of Social Behaviour and Personality, 7(2), 323-334.
- Stone, A.A. (1981). The association between perceptions of daily experiences and self- and spouse-rated mood. Journal of research in personality, 15, 510-522.
- Svensson, J., & Theorell, T. (1983). Life events and elevated blood pressure in young men. Journal of Psychosomatic Research, 27, 445-456.
- Tabachnik, B.G., & Fidell, L.S. (1989). Using Multivariate Statistics. Harper and Row: New York.
- Valdes, M., Treserra, J., Garcia, L., Pablo, J., & Flores, T. (1988). Psychogenic pain and psychological variables: A psychometric study. Psychotherapy and Psychosomatics, 50, 15-21.
- Valentine, E.R. (1992). Theories and explanations. In Conceptual issues in Psychology (Second Edition) Rutledge: New York.
- Veit, C.T., and Ware, J.E.Jr. (1983). The structures of psychological distress and well-being in general populations. Consulting and Clinical Psychology, 51(5), 730-742.
- Verbrugge, L.M. (1989). The twain meet: Empirical explanations of sex differences in health and mortality. Journal of Health and Social Behavior, 30, 282-304.

- Vinokur, A., & Caplan, R.D. (1986). Cognitive and affective components of life events: Their relations and effects on well-being. American Journal of Community Psychology, 14(4), 351-370.
- Wade, J.B., Dougherty, L.M., Hart, R.P., Rafii, A., & Price, D.D. (1992). A canonical correlation analysis of the influence of neuroticism and extraversion on chronic pain, suffering, and pain behaviour. Pain, 51, 67-73.
- Watson, D., & Clark, L.A. (1984). Negative affectivity: The disposition to experience aversive emotional states. Psychological Bulletin, 96, 465-490.
- Watson, D., Clark, L.A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. Journal of Personality and Social Psychology, 54(6), 1063-1070.
- Watson, D., & Pennebaker, J.W. (1989). Health complaints, stress and distress: Exploring the central role of negative affectivity. Psychological Review, 96(2), 234-254.
- Weinberger, M., Hiner, S.L., & Tierney, W.M. (1987). In support of hassles as a measure of stress in predicting health outcomes. Journal of Behavioral Medicine, 10, 19-32.
- Williams, J. S., & Siegel, J.P. (1989). Marital disruption and physical illness: The impact of divorce and spouse death on illness. Journal of Traumatic Stress, 2(4), 555-562.
- Zarski, J.J. (1984). Hassles and health: A replication. Health Psychology, 3, 243-251.
- Zarski, J.J., West, J.D., Gintner, G.G., & Carlson, J. (1987). The stress-illness paradigm: Relationship to social interest and coping. Journal of Mental Health Counselling, 9(4), 227-235.
- Zajonc, R.B. (1980). Feeling and thinking: Preferences need no inferences. American Psychologist, 35, 151-175.
- Zautra, A.J. (1983). Social resources and the quality of life. American Journal of Community Psychology, 11(3), 275-289.
- Zautra, A., & Reich, J.W. (1980). Positive life events and reports of well-being: Some useful distinctions. American Journal of Community Psychology, 8, 657-669.
- Zautra, A.J., & Reich, J.W. (1983). Life events and perceptions of life quality: Developments in a two-factor approach. Journal of Community Psychology, 11, 121-132.

- Zegans, L.S. (1982a). Stress and the development of somatic disorders. In Goldberger, L., & Breznitz, S. (Eds.) Handbook of stress. Theoretical and Clinical aspects. p.137. New York: MacMillan Publishing Co.
- Zegans, L.S. (1982b). Stress and the development of somatic disorders. In Goldberger, L., & Breznitz, S. (Eds.) Handbook of stress. Theoretical and Clinical aspects. p. 138. New York: MacMillan Publishing Co.

Appendix A

Bipolar Trait Adjective Checklist

Listed below are 13 dimensions of trait adjectives. For each dimension, please circle the number most closely resembling how you would generally describe yourself. Work quickly by choosing the response which is the first to come to mind. Complete all 13 questions. Thank you.

1 calm	2	3	4	5	6	7	8	9 worrying
1 at ease	2	3	4	5	6	7	8	9 nervous
1 relaxed	2	3	4	5	6	7	8	9 high-strung
1 unemotional	2	3	4	5	6	7	8	9 emotional
1 even-tempered	2	3	4	5	6	7	8	9 temperamental
1 secure	2	3	4	5	6	7	8	9 insecure
1 self-satisfied	2	3	4	5	6	7	8	9 self-pitying
1 patient	2	3	4	5	6	7	8	9 impatient
1 not envious	2	3	4	5	6	7	8	9 envious
1 comfortable	2	3	4	5	6	7	8	9 self-conscious
1 not impulse- ridden	2	3	4	5	6	7	8	9 impulse-ridden
1 hardy	2	3	4	5	6	7	8	9 vulnerable
1 objective	2	3	4	5	6	7	8	9 subjective

Appendix B

The Hopkins Symptom Checklist

This scale consists of 12 health problems or somatic complaints. Read each item and then mark the appropriate answer in the space next to that word. Indicate to what extent you have experienced the symptom(s) during the past week including today. Use the following scale to record your answers.

1	2	3	4	5
very slightly or not at all	a little	moderately	quite a bit	extremely
_____	Headaches		_____	Trouble getting your breath
_____	Faintness or dizziness		_____	Hot or cold spells
_____	Pains in the heart or chest		_____	Numbness or tingling in parts of your body
_____	Feeling low in energy or slowed down		_____	A lump in your throat
_____	Pains in the lower part of your back		_____	Weakness in parts of your body
_____	Soreness of your muscles		_____	Heavy feelings in your arms or legs

Appendix C

The Uplifts Scale

Uplifts are events that make you feel good; they can make you joyful, glad, or satisfied. This questionnaire lists things that can be uplifts in day-to-day life. Please indicate how much of an uplift each item was for you in the past week including today by circling the appropriate number.

0 = None or not applicable

1 = Somewhat

2 = Quite a bit

3 = A great deal

1. Your child(ren)	0	1	2	3
2. Your parents or parents-in-law	0	1	2	3
3. Other relative(s)	0	1	2	3
4. Your spouse	0	1	2	3
5. Time spent with family	0	1	2	3
6. Health or well-being of a family member	0	1	2	3
7. Sex	0	1	2	3
8. Intimacy	0	1	2	3
9. Family-related obligations	0	1	2	3
10. Your friend(s)	0	1	2	3
11. Fellow workers	0	1	2	3
12. Clients, customers, patients, etc.	0	1	2	3
13. Your supervisor or employer	0	1	2	3
14. The nature of your work	0	1	2	3
15. Your work load	0	1	2	3
16. Your job security	0	1	2	3
17. Meeting deadlines or goals on the job	0	1	2	3
18. Enough money for necessities (e.g., food, clothing, housing, health care, taxes, insurance)	0	1	2	3
19. Enough money for education	0	1	2	3
20. Enough money for emergencies	0	1	2	3
21. Enough money for extras (e.g., entertainment, recreation, vacations)	0	1	2	3
22. Financial care for someone who doesn't live with you	0	1	2	3
23. Investments	0	1	2	3
24. Your smoking	0	1	2	3
25. Your drinking	0	1	2	3
26. Mood-altering drugs	0	1	2	3
27. Your physical appearance	0	1	2	3
28. Contraception	0	1	2	3
29. Exercise(s)	0	1	2	3
30. Your medical care	0	1	2	3
31. Your health	0	1	2	3
32. Your physical abilities	0	1	2	3
33. The weather	0	1	2	3
34. News events	0	1	2	3
35. Your environment (quality of air, noise level, greenery)	0	1	2	3
36. Political or social issues	0	1	2	3
37. Your neighbourhood	0	1	2	3
38. Conserving (gas, electricity, water, gasoline, etc.)	0	1	2	3
39. Pets	0	1	2	3
40. Cooking	0	1	2	3
41. Housework	0	1	2	3
42. Home repairs	0	1	2	3
43. Yardwork	0	1	2	3
44. Car maintenance	0	1	2	3
45. Taking care of paperwork (e.g., paying bills, filling out forms)	0	1	2	3
46. Home entertainment (e.g., TV, music, reading)	0	1	2	3
47. Amount of free time	0	1	2	3
48. Recreation and entertainment outside the home (e.g., movies, sports, eating out, walking)	0	1	2	3
49. Eating (at home)	0	1	2	3
50. Church or community organizations	0	1	2	3
51. Legal matters	0	1	2	3
52. Being organized	0	1	2	3
53. Social commitments or organizations	0	1	2	3

Appendix D

The PANAS (PA scale)

This scale consists of a number of words that describe different feelings and emotions. Read each item and then mark the appropriate answer in the space next to that word. Indicate to what extent you have felt this way during the past week including today. Use the following scale to record your answers.

1	2	3	4	5
very slightly or not at all	a little	moderately	quite a bit	extremely
_____	attentive			_____ inspired
_____	interested			_____ proud
_____	alert			_____ determined
_____	excited			_____ strong
_____	enthusiastic			_____ active

Appendix E
INFORMED CONSENT FORM

I, _____, the undersigned agree to my participation in the research study described.

(Signature of Participant)

(Date)

To be signed by the Investigator:

To the best of my ability I have fully explained to the subject the nature of this research study. I have invited questions and provided answers. I believe that the subject fully understands the implications and voluntary nature of the study.

(Signature of Investigator)

(Date)

(Telephone Number)

If you have any questions concerning this research study, feel free to contact Nashwa Irfan, at 739-6034.

Thank you.

Appendix F

CODE GENERATOR SHEET (PHASE ONE)

Thank-you for participating in this research study. You are requested to complete the following four questionnaires as quietly and accurately as possible. Please read all instructions carefully and do not leave out any questions.

In the attempt to match your responses obtained at this time with the responses you are requested to give at a future time, you will need to create a code based on the following information:

- 1) What are the last two digits of your MUN ID number? ____
- 2) What are the two digits representing the ~~month~~ of your birthday? ____
- 3) What are the two digits representing the **date** of your birthday? ____

This information will make up your code. All participation is voluntary. For participants, I would like to reassure you that anonymity of all data obtained from the questionnaires is guaranteed and that participation in this study will not influence your course grade. Please do not remove this sheet. Thank-you.

Nashwa Irfan

Appendix G

CODE GENERATOR SHEET (PHASE TWO)

Thank you for participating in the final phase of this research project. Your participation in Phase one was greatly appreciated. You are requested to complete the following three questionnaires as quietly and as accurately as possible. Please read all instructions carefully and do not leave out any questions.

In order to match the responses obtained in phase two with those obtained in phase one, please generate a code by answering the following questions:

- 1) What are the last two digits of your MUN ID number? ____
- 2) What are the two digits representing the **month** of your birthday? ____
- 3) What are the two digits representing the **date** of your birthday? ____

This information will make up your code. All participation is voluntary. Again, I would like to reassure you that anonymity of all data obtained from the questionnaires is guaranteed. Participation in this study will not influence your course grade. Please do not detach this sheet. Thank-you.

Nashwa Irfan



