2002

INDIVIDUAL DIFFERENCES IN SECRETORY IMMUNOGLOBULIN A (S-IgA) REACTIVITY TO ACUTE STRESS

WETHERELL, MARK ANTHONY

INDIVIDUAL DIFFERENCES IN SECRETORY IMMUNOGLOBULIN A (S-IgA) REACTIVITY TO ACUTE STRESS

by

MARK ANTHONY WETHERELL

A thesis submitted to the University of Plymouth
in partial fulfilment for the degree of

DOCTOR OF PHILOSOPHY

Department of Psychology
Faculty of Human Science

January 2002

"There are many circumstances, some of which are of common occurrence in human medicine, where the physical, chemical, physiological, and probably psychological factors which affect the host, play far more decisive parts in the causation of disease than does the presence of this or that micro-organism"

Rene Dubos, 1902-1982

MARK ANTHONY WETHERELL

INDIVIDUAL DIFFERENCES IN SECRETORY IMMUNOGLOBULIN A (S-IgA) REACTIVITY TO ACUTE STRESS

Abstract

Secretory immunoglobulin-A (S-IgA) is an antibody found on all surfaces of the common mucosa and serves as a first line of defence against pathogens. S-IgA is the predominant antibody in human secretions and unlike many other immune parameters, can be measured non-invasively in saliva. In addition to being an efficient indicator of health status, S-IgA levels are sensitive to variations in subjective and objective levels of stress, both of which are also influenced by state and trait factors. Stress is known to play an important role in susceptibility to infections of the common mucosa, and as such, the role of S-IgA as a potential moderating variable between stress and health is of increasing clinical importance. This thesis assessed the roles of retrospectively reported health status (minor health complaints) and state and trait factors upon levels of S-IgA following acute stress (S-IgA reactivity). Stress was manipulated using a multi-tasking performance battery, which unlike other laboratory based stressors is analogous to a variety of working environments.

In a series of studies (3), S-IgA reactivity was observed following the stressor on one occasion, two occasions (24 hours apart) and following repeated stress on one occasion (cumulative acute stress). Volunteers classified as in poor health using a specifically designed health questionnaire, demonstrated consistently reduced S-IgA reactivity when compared to volunteers classified as being in good health. Furthermore, the discrepancy in S-IgA reactivity between good and poor health volunteers was most evident following cumulative acute stress. That is, poor health volunteers demonstrated progressive reductions in S-IgA reactivity as the accumulation of stress became greater. Volunteers in poor health were also characterised by negative state and trait characteristics, which in addition, were also independently associated with reduced S-IgA reactivity to acute stress.

The findings indicate that negative state and trait characteristics are associated with reduced S-IgA reactivity to acute stress, levels of which influence post-stress susceptibility to illness. Further, deleterious effects of acute stress are most apparent in poor health volunteers following cumulative acute stress analogous with the stressors encountered in a variety of working environments.

Table of Contents

PART THREE: CONCLUSIONS

List of Figures

List of Tables

Acknowledgements

Mum and Dad

Eternal gratitude to Mum & Dad. You told me the best way I could repay my thanks, and here it is, I've done it. Thank you.

Kirsteen & Luke – Thanks for keeping me going.

Kirsteen, what can I say, but thanks for everything, but most of all, for just being you. Luke, thanks for giving me fantastic work avoidance and stress relief, the therapeutic power of a good bundle should never be underestimated!

My Friends

Andy and Sid, thanks for your continual help, advice, support but most of all, for being great friends - I couldn't have found any better. Thanks to Liz for being there and with Andy Lee providing the venue for my alternative music career. Also thanks to Svenn for keeping a constant supply of caffeine and CDs during those endless final weeks. To the Friday Footy boys – thanks for helping me to realise that I could always give it all up and go professional! I'd also like to thank Swindon Town Football Club for making the stressful times even more so.

My Supervisors

Thanks to my supervisors Michael Hyland and Jack Harris for bridging the gap between psychology and immunology. While giving me the freedom to explore my own ideas, you have always given me support when I needed it. Above all, thanks for being people that I can look up to and for teaching me that there is more to the PhD experience than a thesis.

Thanks also to Annette Wrathmell for my ELISA education, Matt Bristow for our conversations about 'spit and stuff', Mike Gleeson and his team for S-IgA analyses, and 'Aunties' Gilly and Lisa for smoothing corners and cutting red tape.

Author's Declaration

At no time during the registration for the degree of Doctor of Philosophy has the author been registered for any other University award.

This study was financed with the aid of a studentship from the Faculty of Human Sciences.

Relevant scientific seminars and conferences were regularly attended at which work was often presented; external institutions were visited for consultation purposes and several papers are being prepared for publication.

Conferences Presentations

Invited Participation

Wetherell, M.A., Hyland, M.E., & Harris, J.E., The Interaction between minor health complaints and personality characteristics in relation to the S-IgA response to Acute Stress. The proceedings of the British Psychological Society, February, 2001.

Wetherell, M.A., Hyland, M.E., & Harris, J.E (2001) Health Status as a moderator of S-IgA reactivity to cumulative acute stress. *The Proceedings of the British psychological Society, February, 2001.*

Wetherell, M.A., Hyland, M.E., & Harris, J.E (2001) Cumulative effects of acute stress on secretory Immunoglobulin-A. *The Proceedings of the British psychological Society, February, 2001.*

Wetherell, M.A., Hyland, M.E., & Harris, J.E (2001) Individual differences in S-IgA reactivity to acute stress: The effects of health status and personality. *The Proceedings of the British Psychological Society, February, 2001.*

Winner of CDR Sponsors Prize at Psychobiology Annual Scientific Meeting, 2000

Wetherell, M.A., Hyland, M.E., & Harris, J.E. (2001) Immune Responses to Cognitive Workload: The effects of multi-task performance on S-IgA. *The Proceedings of the British Psychological Society, February, 2001.*

Wetherell, M.A. & Hyland, M.E., & Harris, J.E. Minor Health Complaints (MHCs) Salivary Immunoglobulin-A (S-IgA) and Perceived and Actual Cognitive Performance. *The Proceedings of the British Psychological Society*, September, *2001.*

Wetherell, M.A. & Hyland, M.E. Is Too Much Information a Bad Thing? The effects of cover letter length on questionnaire response rates. *The Proceedings of the British Psychological Society*, September, *2001.*

Conferences / Workshops Attended:

Psychobiology Section of the British Psychological Society, Annual Scientific Meeting (September, 1998, 1999, 2000, 2001)

British Psychological Society Annual Conference (March, 1998, 1999, 2000, 2001)

British Psychological Society, Division of Health Psychology / European Health Psychology Society Annual Conference, September, 2001.

British Psychological Society, Media and press Training Workshops, 1998.

External Contacts

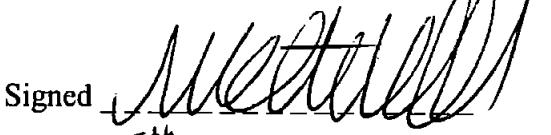
Psychobiology Section of the British Psychological Society

Professor Mike Gleeson, University of Birmingham

Dr. Angela Clow, University of Westminster

Dr. Matt Bristow, Anglia Polytechnic University

Dr. Kavita Vedhara, University of Bristol

Signed 

Date 5th June 2002

1. Introduction

The links between psychological factors and health status are well-established in the scientific as well as anecdotal literature - we are all aware that 'stress can make you ill'. However, it is also apparent that even under the same stressful conditions, not everybody gets ill, and further, while some people seem to thrive on stress, others find it extremely unpleasant. This suggests that the way people cope with stress, either actively, or at a sub-conscious level, can mediate the effects of stress upon the immune-system and therefore influence subsequent vulnerability to illness.

There is now an increasing body of evidence suggesting that IgA plays an important role as a mediator between psychological factors and health status. The literature regarding IgA and psychological factors will be explored extensively in Chapter Two. This introduction will therefore briefly discuss the background to this research, general research questions and the structure of the thesis.

This thesis therefore explores the effects of a previously unused stressor upon IgA reactivity. It is generally acknowledged that acute stress induces a temporary up-regulation of IgA. The majority of acute stress research has involved the use of lab-based stressors. These stressors have provided important information regarding the precise effects of acute stress upon IgA reactivity, however, they are often lacking in external validity. The current stressor (Synwork) is a multi-tasking battery, designed to effectively mimic any working environment where an individual is required to attend and respond to several stimuli simultaneously. In a series of three studies the effects of the Synwork battery were assessed in relation to IgA reactivity. The stress and temporal intensity of the current stressor are analogous with previously used lab-based stressors and as such, patterns of IgA reactivity observed in the current research are expected to be analogous with previous findings. However, as previously stated the Synwork

battery is a multi-tasking environment and as such, it is reasonable to assume that IgA reactivity observed in response to the stressor is more analogous with that observed following everyday working stressors. It is therefore suggested that the findings of the current research can be extrapolated to a range of working environments with greater confidence than previous findings.

It is generally accepted that there is a negative relationship between levels of IgA and ill-health. That is, greater incidence of ill-health is associated with lower levels of IgA. Furthermore, periods of stress are generally associated with an increased risk of post-stress infection. This thesis attempted to explore the role of retrospectively reported health status in relation to changing levels of IgA in response to acute stress (IgA reactivity). The relationships between health and stress are complex, it is suggested that stress can lead to ill-health, however, ill-health itself can also cause stress. This thesis seeks to explore whether those people who experience greater frequencies of ill-health, and moreover, different kinds of ill-health, maintain their poor health status as a result of poor IgA reactivity to acute stress.

As previously discussed, when faced with stress, not all people become ill. This thesis therefore attempts to explore individual differences in response to the manipulated stressor. That is, to explore which factors are associated with positive IgA reactivity to acute stress. As such, personality characteristics, state mood and perceptions of workload following the stressor were assessed in an attempt to identify those individuals that seemed better equipped to deal with stress, and thus those individuals that are likely to be less prone to post-stress infection.

It should be noted that this research is of an exploratory nature and the series of studies should be viewed as a developmental process. That is, findings from each of the studies

provided the rationale for assessment in subsequent studies, and further each study provided further knowledge to the area as a whole. Whilst each study is preceded by a set of specific aims and objectives, the thesis has several general objectives. Firstly, to explore IgA reactivity following a novel but potentially advantageous stressor. Secondly, to explore the role of retrospective health status in relation to IgA reactivity. Finally, to explore other psychological factors that could potentially mediate between health status and the effects of the stressor.

The thesis is divided into three parts. Part one (Chapters 2, 3 & 4) provides background information to the current research project, and provides a review of relevant literature regarding IgA and health status, stress and other psychological factors. Part one also contains information regarding the development and testing of a health questionnaire for use in the experimental studies, and discusses the methodologies used in the thesis. Part two (Chapters 5, 6 & 7) comprises three experimental studies, which should be viewed as progressive in nature. That is, each study builds upon the foundations of its predecessor. Finally, part three (Chapter 8) comprises the final conclusions from the thesis. The findings from the thesis as a whole are drawn together and discussed in light of previous work,. Further, recommendations for further research, and the wider implications of the research findings are discussed.

Part One

Background Information

Part one comprises background information to the thesis. Chapter two comprises a review of S-IgA literature, Chapter three contains information regarding the development of the Minor Health Complaints Questionnaire (MHCQ), and Chapter four discusses the methodologies used in the thesis.

2. Literature Review

2.0 Chapter Overview

This chapter comprises a review of the literature relevant to the current thesis. The sources used are not exhaustive, however, they are considered as the most appropriate in setting a context for the current body of work. A basic introduction to the immune system will be provided, with a specific focus upon S-IgA. The relevance of S-IgA will then be discussed in relation to its' use as a valid indicator of health status, observed changes in response to stress, and individual differences in state and trait characteristics. The literature is discussed in relation to the aims and objectives of the thesis (the specific aims and hypothesis are presented in Chapter 1), and criticisms of the literature are discussed as a rationale for the series of studies and conclusions presented.

2.1 The Immune System: An Overview

Extensive reviews of the immune system are available in a large number of fundamental immunology texts (e.g., Kuby, 1997). The current body of work is concerned only with one specific aspect of the immune system, and as such, only information pertaining to this facet of the immune system will be discussed.

The immune system is a complex network of organs and tissues generating cells that protect the body from potentially harmful foreign substances, e.g., pathogens, or infectious agents, such as viruses, bacteria, fungi, and other parasites. The immune system allows for the recognition and subsequent destruction of such agents. The immune system is made up of two main kinds of immune protection. Firstly, cell-mediated immunity is carried out by cell-destroying or cytoxic cells. That is, following recognition, these cells directly or indirectly kill target cells, e.g., bacteria, tumour or transplanted cells. The second type of immunity, and moreover, of more relevance to

the factors being assessed in this thesis, is humoral immunity. Humoral immunity comprises the secretion from lymphoid cells of protein molecules (antibodies) to all bodily fluids, these antibodies are capable of binding to specific foreign molecules (antigens) and either neutralising them or facilitating their destruction and removal by other immune cells (e.g., cell-mediated) or pharmacological agents. A fully efficient response requires the involvement of both cellular and humoral responses.

Antibodies are formed from lymphocytes (the cells responsible for the recognition of antigens). There are two types of lymphocytes, both of which are primarily produced in the bone marrow. T-cells originate in the bone marrow but then migrate to the thymus where full maturation occurs. There are two major types of T-cell; T-cytoxic (Tc), and T-helper (Th) cells. Th cells take one of two forms (Th1 and Th2), characterised by the immune response they elicit. In basic terms, Th1 immunity activates cell-mediated responses, whereas, Th2 immunity activates humoral immunity. Following recognition of an antigen, Th cells secrete lymphokines (e.g., cytokines) which are responsible for the activation of the second type of lymphocytes, B-cells. B-cells develop fully in the bone marrow and when mature express a unique antigen-binding receptor known as an immunoglobulin (a type of antibody). Immunoglobulins are graphically presented a Y-shaped structure, made up of two heavy and two light chains made up of polypeptides, sequences of which determine the specificity of the molecule. . The two arms of the "Y-structure" provide the site for binding with a specific antigen.

There are five major immunoglobulin classes (IgM, IgG, IgD, IgE & IgA), which vary in their specificity and therefore their role in immune defence. The specific purpose of IgD is unknown, however it is thought to aid activation of B-cells following recognition of an antigen. IgM and IgG are the predominant immunoglobulin classes present in the blood and IgE is responsible for eliciting immune response to allergens and parasites.

Finally, IgA (the focus of this thesis) is found on all surfaces of all mucosae and acts as a first line of defence on the upper-respiratory, urino-genital and gastro-intestinal tracts by preventing antigens attaching to epithelial surfaces.

While IgA is found in serum, it is the predominant antibody in human secretions (e.g., saliva, tears and breast milk). IgA in such secretions is referred to as secretory IgA (S-IgA), molecules of which are structurally different to those found in serum. That is, S-IgA contains a secretory component or piece which is thought to protect the IgA molecule from enzymatic breakdown in the mucosa. The production and subsequent secretion of S-IgA is presented in Figure 2.1.

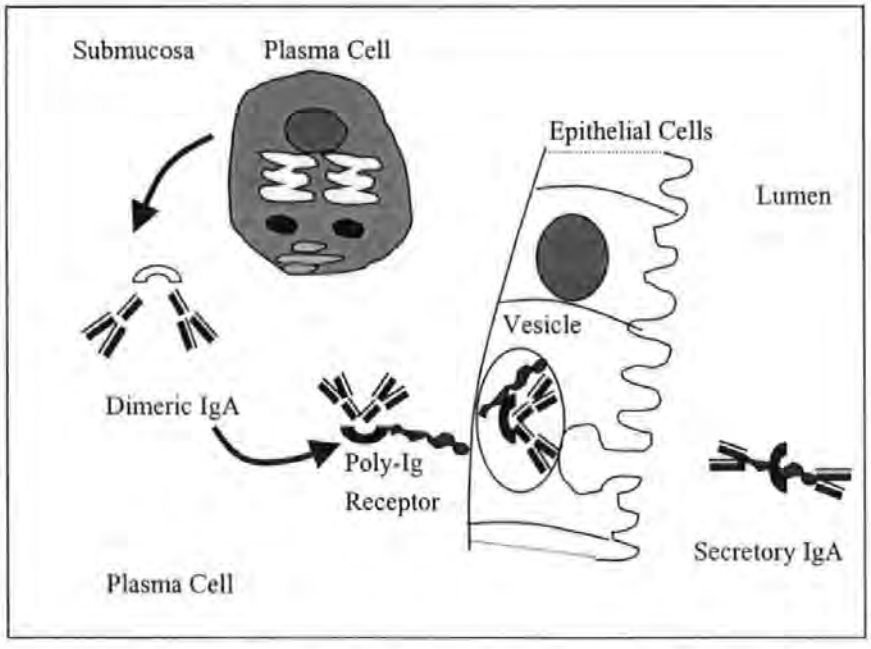


Figure 2.1 Production of and Release of S-IgA onto the Mucosal Surface (adapted from Evans, Hucklebridge & Clow, 2000).

Plasma cells produce dimeric IgA which migrates through the submucosa towards the mucosal epithelial cells. Dimeric IgA then binds with a polymeric immunoglobulin receptor (Poly-Ig) on the proximal surface of the epithelial surface. The resulting complex (dimeric IgA + poly-Ig receptor) is then endocytosed in a membrane vesicle and transported to the lumen facing surface of the epithelial cell. Through a process of enzymatic cleavage the IgA complex is secreted into the mucosa where as the

combination of dimeric IgA and the secretory component from the poly-IgA receptor becomes S-IgA.

Most other immune parameters; serum and tissue immunoglobulins, lymphocytes, interleukins etc., are difficult to monitor non-invasively. Mucosal secretions and saliva in particular, therefore provide the most tangible body fluids that may be easily sampled. Further, the immunological purpose of S-IgA provides evidence that S-IgA is an important clinical indicator of health status. The following sections therefore discuss the role of S-IgA in susceptibility to illness, and attempts to explain why S-IgA is one of the measures of choice in psychoneuroimmunological research.

2.2 S-IgA & Health

The known role of S-IgA in immunological defence suggests that levels of S-IgA must be related to health status. S-IgA provides a first line of defence protecting the upper-respiratory, gastro-intestinal and urino-genital tracts, and as such, it should follow that frequencies of illness manifesting in these tracts should be inversely related to levels of S-IgA. That is, an absence or depression of S-IgA should result in increased susceptibility to such illness, whereas an abundance of S-IgA should help to maintain the integrity of these tracts and thus decrease vulnerability to pathogens gaining entry through these portals. As such, many studies have assessed the relationship between levels of S-IgA and frequencies of illnesses.

Tomasi (1976), provided the first empirical evidence of a negative association between S-IgA and ill-health. Tomasi (1976) suggested that individuals who selectively lack S-IgA have a high association with various diseases, resulting in recurrent infections of the upper respiratory tract and increased frequencies of allergic disorders such as eczema and asthma. Later studies have tended to focus upon the relationship between

levels of S-IgA and frequencies of upper-respiratory tract infections (URTIs), e.g., coughs, colds, sore throats and influenza. The studies involving this proposed relationship are well documented, as such, this review will discuss data and findings arising from a meta-analysis conducted by Jemmott and McClelland (1989). The discussion of this meta-analysis, is in no way belittling the individual studies that have contributed to this research area. However, when discussing the studies individually an inconsistent pattern emerges which in part can be attributed to variation in methodologies (e.g., use of healthy and infected volunteers) and changes in their specific research foci. It is only when the data is taken as a whole, and analysed using consistently appropriate methods that a consistent pattern emerges. The meta-analysis demonstrated overwhelming evidence supporting the view that S-IgA concentrations are indeed related to incidences of URTIs ($p < 0.000025$).

Despite this apparent evidence suggesting that lower levels of S-IgA are indeed associated with increased incidence of illness, Jemmott and McClelland (1989) warn of the danger of misinterpretation. That is, although lower S-IgA seems to be associated with increased illness incidence, actual infection depends upon a host of other factors. That is, in normal circumstances an individual with lower than average levels of S-IgA might not become infected with a URTI. Actual contraction of illness is of course not only determined by an individuals susceptibility to the illness, but moreover, whether that individual is actually exposed to a pathogen. Further, the virulence of the pathogen and the immune capacity of the individual at the time of exposure, i.e., the immune system is a robust system and deficits in one department can be counterbalanced by over-activity in another. As such, Jemmott & McClelland (1989) suggest that lower S-IgA is most appropriately viewed as a risk factor. That is an individual with lower S-IgA is more vulnerable to infection, however, whether illness actually manifests is influenced by a host of other factors. This is especially pertinent with regards to the

experimental designs utilised in studies of S-IgA and infectious illness. That is, volunteers are assessed during a fixed period of time ranging from weeks to months. Although one might reasonably expect incidences of illness in normal individuals during such time periods, this is not always the case. However, knowledge of the immunological role of S-IgA taken with the meta-analytical findings of Jemmot and McClelland (1989) suggest that, in normal healthy individuals, lower S-IgA levels increase the risk of subsequent illness.

More recently, Gleeson (2000) has described reduced S-IgA levels and subsequent increased susceptibility to URTIs in athletes following intensive training regimes. Although the sample population utilised in this study does not further knowledge with regards to illness in normal healthy volunteers, the findings provide further support that lower S-IgA levels are indeed associated with increased frequencies of illness. That is regardless of the fact that levels of S-IgA have been somewhat artificially lowered (i.e., it is the exercise not natural variation that is responsible for the lowering of S-IgA), volunteers with the lowest levels of S-IgA experienced the greatest frequencies of URTIs.

Although S-IgA is ubiquitous to all mucosae, the majority of research concerning the relationship between S-IgA and illness have focused upon frequencies of URTIs. This is not surprising when it is noted that infections of the upper-respiratory tract comprise illnesses that are experienced by the majority of the population on a regular basis, (e.g., coughs and colds) and that are the most symptomatic. However, the focus upon URTIs is to the detriment of research concerning other minor health complaints that manifest in the urino-genital and gastro-intestinal, where S-IgA is also known to play an influential protective role. There is also an absence of research concerning diffuse symptoms that may be indicative of general illness. Also, previous research (especially those studies

utilising a manipulated stressor of some kind) has focused upon susceptibility to illness after an event. The current body of work therefore views the relationship between S-IgA and health as a cyclical process. That is, there is strong evidence to suggest that lower S-IgA is a risk factor for illness, however, what can be said about the levels of S-IgA in individuals who are most susceptible to illness? From a pure immunological perspective it could be argued that such individuals would have greater levels of S-IgA as a result of frequent infection triggering the production and proliferation of S-IgA. However, it is likely that in such individuals the need for S-IgA (in response to infection) will outweigh production and secretion. Hence individuals who experience greater frequencies of minor health complaints will demonstrate a deficit in S-IgA, this deficit probably being responsible for their increased susceptibility.

However, it should be noted that levels of S-IgA are not purely a function of illness (and *vice-versa*). S-IgA levels and indeed frequencies of illness are also influenced by a host of other psychosocial factors, hence S-IgA is of key interest in psychoneuroimmunological studies. This thesis argues that it is the inter-relationships between these psychosocial factors, S-IgA and illness that is of the greatest importance. The following sections will therefore discuss the relationships between S-IgA and psychosocial factors (i.e., stress, state and trait factors).

2.3 Stress and S-IgA

First inspection of the literature regarding S-IgA and stress is likely to reveal a mixed pattern of results. Moreover, reviews of the literature (e.g., Van Rood, Bogaards, Goulmy & Van Houwelingen, 1993, Valdimarssdottir & Stone, 1997) argue for a lack of consistency among research findings. However, some of the observed inconsistencies can be attributed to confusions concerning the definition of stress. That is, the word stress is often used without an adequate explanation of what the stress is. This is an especially pertinent issue with regards to S-IgA and stress research, where a variety of stressors have been utilised. As such, for the purposes of the current research, it is essential to discuss this confusion, and clarify the stress and stressors used in the previous and current research.

Attempts to classify the stress or stressor used have also resulted in confusion with regards to findings concerning stress and S-IgA. This is most apparent where the stressor is described as either *acute* or *chronic*. Although such descriptors should alleviate the confusion, however, the terms acute and chronic tend to vary between researchers. For example, within the field of acute stress research, examination stress (i.e., monitoring S-IgA over a two week examination period) would be described as chronic. However, researchers of chronic stress would have a different perspective on what warrants a chronic stressor. A prime example of chronic stress for these researchers, would be the stress experienced by an individual caring for a disabled partner (i.e., long term, continual - insidious chronic stress). The major underlying factor in acute and chronic stress is time. This is especially important with regards to responses to stress because a variety of time dependent chemicals are secreted at differing times during the stress response. The time duration of the stressor will therefore influence the type and intensity of release of these chemicals and will influence immune reactivity accordingly.

For the purposes of this research, the term acute stress refers to a short and temporary stressor. As such, the term chronic stress will be used to describe stressor that are longer in duration (i.e., days, weeks and months, rather than minutes). Such stressors (e.g., care-giving) are undoubtedly chronic in nature, and moreover often demonstrate similar patterns of S-IgA reactivity as the short term chronic stress studies that will be discussed later. However, it could be argued that chronic stress research is a different area of research altogether, which although undoubtedly driven by the same mechanisms, is influenced by a variety of other mechanisms. As such, this review of the literature will deal primarily with acute stress studies, although studies of chronic stress will be discussed in light of important methodological issues.

In the earliest studies of acute stress, the term acute referred to changes over the course of days. Although in the context of the current research, the term acute refers to minutes rather than days, it is important to discuss these early studies in order to familiarise the reader, but moreover, to exemplify the apparent discrepancies in the literature.

The earliest study concerning the effects of acute stress upon S-IgA was conducted by McClelland, Floor, Davidson and Saron (1980). Acute stress was administered using perceptual learning tasks, which the authors reported to be a mild stressor.
Levels of S-IgA obtained on the day of the stressor was subsequently compared with samples obtained the previous day where no stressor task was administered. S-IgA concentrations on day two S-IgA (the day of the stressor) were significantly lower than those samples on day one. However, as with many seminal research findings, this study has several inadequacies, and as such, the findings do not necessarily lead to the conclusion that acute stress leads to a reduction in S-IgA. The most important issue is the choice of stressor. That is, although the authors report the tasks to be mildly

stressful, there is no objective evidence to support this claim. Secondly, no information was provided concerning the timing of the samples. That is, contemporary research (e.g., Hucklebridge, Clow & Evans, 1998), provides clear evidence of a diurnal cycle in S-IgA. Given the absence of information concerning time of day of samples, it is impossible to attribute changes in S-IgA to the stressor alone. Further, given the transient nature of S-IgA change, a host of other factors, psychosocial and otherwise, could have influenced the reduction in S-IgA from day one to day two.

Despite the apparent inadequacies of this pioneering study, similar findings were observed in subsequent studies, where the term acute refers to days and weeks rather than minutes. However, despite the longer time period, in these studies the term acute is analogous with contemporary studies in that the utilised stressor was acute in nature. That is, several studies utilised student examination periods as a stressor. Although the S-IgA sampling was taken over longer periods, sampling during the acute stress periods, can be considered to be following (or during) a period of what is now referred to as acute stress (examinations). The choice of stressor in these studies is also worthy of further discussion. That is, exams are inevitable for students, who form the majority of samples in much psychological research. Further, with regards to external validity (a concept that will be discussed in more detail later), examinations are naturally occurring in that the stressor is an expected part of the volunteers activities, As such, changes in S-IgA (or any other measure) during these periods has direct relevance in the real world. It is therefore not surprising that exams became a popular stressor in this research area, and have provided important information regarding both the acute effects of examination stress, and the more chronic effects of exam periods compared with low-stress periods.

The first study of this kind was conducted by Jemmott, Borysenko, Borysenko, McClelland, Chapman and Meyer (1983). The authors compared S-IgA in dental students during periods of low and high stress (exam or test periods). The high stress periods can therefore be considered to be following periods of acute stress, although the total period of sampling spanned months. Findings demonstrated lower S-IgA during the high stress periods, when compared with the other sampling points. Further, self-report stress measures taken during all sampling periods provided further evidence to support the notion that S-IgA levels are lower during periods of high stress.

A similar paradigm was adopted by Mouton, Fillion, Tawadros & Rejean (1989). The authors assessed S-IgA over a two year period comprising two low-stress periods, and two high-stress periods (examination periods). A significant reduction in S-IgA was observed between the final exam period (high-stress) and the summer vacation period (low-stress). As with Jemmott *et al.*, (1983) the degree of stress was also supported by self-report stress measures. Examination stress was again utilised by Jemmot and Magloire (1988), however, in this case the sampling periods are more in line with the view of acute stress adopted in the current research. Saliva samples were obtained at three points; a first baseline measure (5 days before examination), a second stress measure (on the day of the examination), and a final low-stress measure (2 weeks after the examination). Levels of S-IgA were significantly lower on the day of the exam when compared to baseline and low-stress sampling points.

More contemporary studies of examination stress have also demonstrated a reduction in S-IgA following examination periods. These contemporary studies will be briefly discussed in order to demonstrate the consistency in the findings, but moreover to exemplify the importance of timing of samples in acute stress research, which will be discussed in more depth later on. In a series of studies Deinzer and colleagues assessed

S-IgA change before and after examination stress. In the first study (Deinzer & Schuller, 1998), S-IgA was sampled 25 days prior to assessments, and then before and after both a written and an oral examination. S-IgA was significantly lowered during both exams and post-stress on both occasions when compared with the baseline measurement. Further, this suppression remained until six days post-stress, after which, S-IgA levels slowly began to rise. In a second study (Deinzer, Kleineidam, Stiller-Winkler, Idel & Bachg, 2000), the study was replicated but extended in order to observe the point at which post-stress S-IgA recovery occurred. Similar suppression was observed, however, in a two week period post-stress no significant S-IgA recovery was observed.

The earlier studies of examination stress, which at the time were described as inducing acute stress, demonstrated a reduction in S-IgA. However, using examination stress McClelland, Ross & Patel (1985) demonstrated an increase in S-IgA. Volunteers provided three saliva samples; a first immediately following an exam, a second 1¾ hours after the exam, and a finally volunteers were asked to return to provide a third sample at a time when "they were feeling relaxed". Although this final measure was taken post-stress, the subjective reports of "feeling relaxed" allowed for the use of this sample as a pseudo baseline measure. Compared with the baseline, higher S-IgA was observed in both stress measures, with the greatest increase apparent immediately following the stressor. No data is provided regarding time of sampling and as such, observed reduction could be attributed in part to diurnal variation. However, this study provided the first challenge to the notion that acute stress is associated with a reduction in S-IgA. More importantly, this finding demonstrated the importance of sampling time in relation to the stressor. That is, unlike other studies of this kind, samples were taken *immediately* following the stressor. The evidence regarding examination stress in the longer term consistently suggests suppression of S-IgA. However, sampling S-IgA over

a shorter time period (McClelland *et al., 1985*) is more in line with the notion of acute stress in more contemporary studies, as well as the research presented in this thesis.

Further support for the notion that acute stress actually mobilises or increases S-IgA was provided by Evans, Bristow, Hucklebridge, Clow and Walters (1993) albeit indirectly. Further, without directly assessing the effects of a stressor *per se*, the findings supported both longer term findings analogous with Mouton *et al.,* (1989) and Jemmott *et al.,* (1988) as well as the immediate effects akin to those observed by McClelland *et al.,* (1985). Evans *et al.*, (1993) assessed frequencies of desirable events and S-IgA over a two week period. As predicted, and in line with previous literature, net desirable events were associated with higher S-IgA. That is, higher S-IgA was associated with increased frequency of desirable events. However, within-sample analyses revealed a contrary and, at first, paradoxical pattern of results. That is, contrary to hypotheses higher S-IgA was observed on days of higher negative mood, and further on days of greater than average numbers of undesirable events.

In this study, between and within sample analyses have therefore allowed for the assessment of both the immediate and longer term effects with regards to changes in S-IgA. Further, although the study did not assess the effect of an imposed stressor, there is not a great conceptual leap from daily hassles to stressors. As such, this study has several important implications, not least stressing the importance of assessing not only gross, but also individual response to stimuli. Firstly, in line with previous research, Evans *et al., (1993)* demonstrated that greater frequencies of desirable events (longer term) are associated with higher S-IgA, and therefore it follows that undesirable events are associated with lower S-IgA. Secondly, and of great theoretical importance, in the short term, increased daily hassles / negative mood are associated with higher S-IgA. Although using previous literature as a benchmark the findings of Evans *et al., (1993)*

seem somewhat paradoxical, there are strong parallels between their findings and those observed by McClelland *et al., (1985)* immediately following an exam. Similarly, the authors suggest the similarity of findings with that of McClelland & Kirshnit (1988) who observed increases in S-IgA following negative affect inducing manipulations, and further, with the findings of Stone, Cox, Valdimarsdottir, Jandorff and Neale (1987) who also observed high S-IgA on days of high negative mood. That is, in the immediate short-term, manipulations to negative mood either through hassles or manipulated stressors are associated with a rise in S-IgA. The authors further suggest that the current findings in light of previous research suggest the potential importance of the timing of S-IgA measures. That is, the increases and decreases observed at differing time periods may represent a complex pattern of S-IgA reactivity to mood altering situations such as stress.

Although I have stated that it does not take a huge conceptual leap to apply the findings of Evans *et al.*, (1993) to manipulated stressors, at this point, the findings involving S-IgA and acute stress were still mixed. Increased daily hassles are associated with higher immediate S-IgA, however, the collation of daily hassles / mood data relies on self-report. The assertions and links to other findings however, were pointing to a contrary pattern of reactivity than that predicted by earlier examination studies. However, in order to assess whether acute stress per se also led to increased mobilisation of S-IgA, acute stress, not daily hassles needed to be implemented. As such, two studies made this conceptual leap, and directly assessed the assertion that stress (not just daily hassles) increased S-IgA levels. Further, these studies employed more rigorous procedures than previously observed, e.g., multiple baselines and immediate post-stress sampling which allowed for the direct assessment of the effects of acute stress upon S-IgA reactivity.

In the context of the current thesis, the external validity of the stressor utilised in this research area remains an important factor. Extrapolation to the real world is also addressed in the first of the two studies by virtue of the utilised stressor. Evans, Bristow, Hucklebridge, Clow & Pang (1994) used an assessed student presentation as a means of stressing volunteers. Saliva samples were collected on four occasions on a neutral (non-stress) day during the week prior to the assessment. Further samples were taken prior to the assessment (at the same times of day as the neutral samples) and immediately post-stress. S-IgA levels were higher immediately post-stress, although not significantly so when compared with baseline. The absence of significance can in part be attributed to the small sample size ($n = 7$), and as such, the study undoubtedly lacked the power to detect significant changes even if an effect were present. Although this finding lends tentative support to the notion that actual acute stress mobilises S-IgA, the lack of significance, despite the undoubted power issues, was an obvious problem. However, the second study from this group provided even greater support for this assertion.

Bristow, Hucklebridge, Clow and Evans (1997), also utilised students taking part in assessed presentations. This study further assessed the immediate effects of the stressor by sampling S-IgA over a 4 hour time-span. The four samples were obtained; upon arrival, 30 minutes prior to their assessed presentation, immediately after assessment, and finally 30 minutes after the presentation. Significant changes in S-IgA were observed in the study, with the highest S-IgA levels observed immediately following the assessment. A subsequent decline (although not getting back to baseline levels) were observed 30 minutes post-stress. A similar pattern was observed with regards to the volunteers self-reported arousal, suggesting that the hypothesised sampling periods were indeed indicative of low and high stress situations. This design allowed for the short term changes in S-IgA in response to a stressor to be assessed. The significant

increase in S-IgA immediately post-stress therefore provided statistical support for the notion that acute stress increases S-IgA.

A similar methodology was applied by Spangler (1997). Using an oral examination of students, Spangler (1997) sampled S-IgA 15 minutes prior to the assessment, and five minutes and 15 minutes post-assessment. Both post-stress measures demonstrated significantly higher S-IgA when compared to baseline (15 minutes pre-stress), however, the greatest increase was observed immediately (5 minutes) post-stress. S-IgA reactivity was also greater in those volunteers classified as high in ego-control (other factors that can mediate S-IgA reactivity will be discussed in more detail later).

Thus far a more consistent picture is emerging. While earlier studies suggested an immuno-suppressive role of acute stress, contemporary studies (Deinzer *et al.*, 1998, 2000) using the same examination stress paradigm have demonstrated that this suppression follows in a period of days following a stressor, when compared with baseline measures. Subsequent studies have assessed acute stress in a smaller time-span, giving strong support for the notion that in the short term stress leads to a temporary mobilisation or increase in S-IgA. It is these acute stress studies which are more akin with the research presented in this thesis. As such, the potential flaws in these studies must now be addressed.

I have stressed the importance of extrapolation to the real world in the studies utilising assessed presentation or exams, i.e., they are assessing a real phenomenon, which while not applicable to everyone, is real and essential for those taking part. Other studies have therefore assessed S-IgA in real world setting other than examination stressors and also demonstrated that short-term stress can lead to increases in S-IgA. Firstly, Kugler, Reitjes, Tews, and Schedlowski (1996) utilised an innovative naturalistic study. Kugler

et al., (1996) assessed S-IgA change in professional football coaches during their team's matches in relation to subjective ratings of excitement. S-IgA increases were observed during the match, with a peak during the half-time break. Subsequent samples demonstrated that S-IgA levels returned to baseline levels approximately one hour after the match. Further, there was a high degree of association between measures of perceived excitement and increases in S-IgA providing evidence for a link between arousal and S-IgA activation. Another naturalistic study was conducted by Zeier, Brauchli, and Joller-Jemelka (1996). They assessed changes in S-IgA in air-traffic controllers, an occupation characterised by high stress levels. Zeier *et al.*, (1996) sampled S-IgA before and after periods of radar monitoring and observed significant post-stress increases in S-IgA.

Although these naturalistic studies have great external validity, and furthermore provide more evidence for the notion that acute stress elicits increases in S-IgA, such stressors do not occur in isolation. That is, observed fluctuations in S-IgA may not occur simply as a result of the stressor. There is no doubt that such phenomena are stressful. For example, Kugler *et al.*, (1996) observed association between perceived levels of excitement and S-IgA increases, and as Bristow *et al.*, (1997) suggest their oral presentation involved the common anxiogenic experience of public speaking with the added stress of outcomes being directly related to degree class. Furthermore, perceived stress levels were near maximum during the stressor period. However, researchers were merely taking a snapshot of immune reactivity during a period in the volunteers' lives. As such, these studies are analogous with observational studies (with the obvious exception of collecting biological data). That is, the volunteers were participating in their normal lives, part of which was an assessed presentation, a work shift, or other stressor which they had undoubtedly been expecting. This expectancy could therefore lead to arousal before the assessed event.

Although the Bristow *et al.*, (1997) study assessed S-IgA changes over a small time-span, expectancy and subsequent relief could be important factors in the observed results. It is plausible that in expectancy of the event, immune suppression had occurred in the days or even weeks leading up to the event. This being the case, post-stress increases could be attributed to a rebound effect, i.e., S-IgA levels returning to normal following the stressor. Related to this is the influence of relief that the stressor is now over. That is, relaxation has been demonstrated to increase S-IgA (*c.f.*, Van Rood *et al*, 1993). It is acknowledged that post-stress measures are not directly akin to relaxation, however, given that volunteers had been building up to this event for some time, the post-stress increases could be attributed to relief that the stressor is now over.

Several studies have further utilised the assessed presentation / exam design. Whilst suggesting an increasing role of anticipation, the findings of these studies indicate that the role of relief or euphoria upon finishing the task are unlikely. Firstly, a study (Bosch, Brand, Ligtenburg, Bermond, Hoogstraten & Nieuw-Amerongen, 1998) sampled S-IgA in dental students 30 minutes prior to an expected exam, and then two and six weeks post-exam. They observed significantly elevated S-IgA during the pre-exam sample when compared with both prospective baseline samples. Similarly, Huwe, Hennig, & Nettir (1998), sampled S-IgA immediately before and after a 30 minute examination, and then again four weeks post-exam. Analogous with the findings of Evans *et al.*, (1993) Bristow *et al.*, (1997) and Spangler, (1997) the authors observed significant increases in S-IgA immediately post-exam when compared with the four week low-stress sample. However, as with Bosch *et al.*, (1998) S-IgA immediately before the examination were also significantly greater than low-stress levels.

These studies therefore demonstrate an influential effect of anticipation upon S-IgA prior to manipulated stress. Such anticipation is always likely to contaminate research

findings when stressors such as assessed examinations or presentations are used. While such increases in S-IgA have been demonstrated, the anticipation of such tasks makes it very difficult to assess how much of the observed increase can be attributed to the stressor itself. In order to establish the extent to which the stressor is responsible for S-IgA increases, a number of laboratory stressors have been used.

Before discussing these laboratory stressors, it is appropriate to discuss the ethics of administering stress to volunteers. As well as the great external validity, the other advantage of academic style stressors is that they are not imposed by the researcher – the researcher is merely taking an advantage of an event that would have occurred anyway. One alternative is, therefore, laboratory based experimentation. Ethical constraints limit the amount of stress that can be administered in the lab, and to these ends, it is unlikely that lab based stressors are as stressful as academic style stressors. However, given that much of the stress in academic style stressors could be attributed to anticipation, lab based stressors will have no or very little anticipatory stress. Volunteers are obviously aware that they are going to take part in an experiment, however, unless the volunteer is particularly anxious, it is unlikely that volunteers will build up a huge anticipation prior to taking part in a psychology experiment. Further, given that many of the volunteers in such research are psychology undergraduates they have a vague idea of what is expected in experimentation.

One of the first lab based studies of acute stress and S-IgA (Carroll, Ring, Shrimpton, Evans, Willemson & Hucklebridge, 1996), assessed S-IgA reactivity to a computer game. The session comprised of six minutes rest period, 30 minutes of the computer game (a level style shootem-up), followed by a 20 minute recovery period. S-IgA was sampled four times during the study; once during the rest period (4 minutes), twice during the stressor (6 and 24 minutes), and finally 18 minutes into the recovery period.

The reader will note the relatively short time span of the entire sampling period when compared to previous studies of acute stress. S-IgA was greater during both task samples when compared with both the rest and recovery samples. However, this increase was significantly greater at the second stressor sample (24 minutes).

This study provided preliminary evidence for the notion that the S-IgA increases hypothesised to follow acute stress, are also evident following laboratory based tasks. Such a finding has the added benefit of a reduction in the contamination by anticipation, which is likely to have influenced the earlier studies That is, in lab based studies there is no reason to assume that the volunteers were stressed prior to experimentation. Further evidence for the beneficial use of lab based stressors was provided by Willemson, Ring, Carroll, Evans, Clow and Hucklebridge (1998). The authors used two stressors, one psychological (Paced Auditory Serial Arithmetic Task – PASAT) and one physical (cold pressor task), tested four weeks apart. S-IgA was sampled at rest (following a 15 minute rest period following entry to the lab), immediately following each of the stressors, and then again between three and four minutes into a post-stress rest period. Both stressors elicited increases in S-IgA, that is significant increases in S-IgA from rest to post-task. The authors suggest that S-IgA change is indeed sensitive to acute lab based stress, and moreover, seems to respond to diverse (psychological and physical) stimuli in a similar fashion. It is further suggested that these findings demonstrate the feasibility of eliciting S-IgA using lab based stressors, i.e., where an increases in control and reduction in anticipatory stress is expected.

A similar paradigm was utilised by Ring, Carroll, Willemson, Cooke, Ferraro and Drayson (1999). Knowledge that acute lab based stressor could elicit increases in S-IgA allowed for greater assessment of the potential mechanism driving the S-IgA response. Ring *et al.*, (1999) also used two stressors known to differentially exert

sympathetic and parasympathetic nervous activation. That is, the PASAT was used to elicit sympathetic activation, whereas a paced breathing task was utilised to elicit parasympathetic activation. As with the work of Willemson *et al., (1998)* S-IgA was sampled immediately prior to and following the stressor tasks. A significant increase in S-IgA concentration (but not secretion rate – this concept will be discussed in more detail with regards to study 2) was observed following the PASAT, however, very little difference in S-IgA levels were observed in response to the paced breathing task. This finding supports the work of Willemson *et al.*, (1998) and again replicates the finding that acute laboratory stress induces increases in S-IgA. Further, the discrepancy in findings between the PASAT (sympathetic) and the paced breathing task (parasympathetic) suggest that in the short term, increases in S-IgA are mediated by sympathetic nervous stimulation. That is, as expected, the PASAT elicited an increase in alpha and beta-adrenergic activity (as assessed through cardiovascular measurements), indicating stimulation of the sympathetic branch of the nervous system.

A further replication of this paradigm was attempted (Winzer, Ring, Carroll, Willemson, Drayson & Kendall, 1999), however, the paradigm was expanded in order to further assess the potential mechanism mediating S-IgA activation in response to acute stress. The authors counterbalanced the presentation of the PASAT and the cold pressor task (one week apart), however, both tasks were followed by the exercise task. In addition, the counterbalanced administration of either 40 mg of propranolol (a non-specific beta-adrenergic blocker) or placebo was given at both sessions. As with Ring *et al.*, (1999) the PASAT significantly increased S-IgA concentration (but not secretion rate), however, there was no significant change in S-IgA in response to the cold pressor task. Further, with regard to mediating mechanism, propranolol had no effect on response to either the PASAT or cold pressor task. However, in contrast, the beta-blocker volunteers demonstrated a greater increase in S-IgA following the exercise task,

when compared to placebo. This indicates, that while, sympathetic activation may still be a potential mechanism for S-IgA mediation, activation of the beta-adrenergic system is not the cause of S-IgA increases. The underlying mechanisms of S-IgA activation will be discussed in more detail in light of the work presented in this thesis.

The acute lab based tasks have demonstrated that tasks such as mental arithmetic also elicit increases in S-IgA. However, although the findings of Willemson *et al.*, (1998) has not been replicated with regards to S-IgA secretion rates (S-IgA concentration and saliva flow rates are discussed in more detail in light of studies 1 and 2 - *c.f.*, chapter 7), the trends in the studies involving acute lab based stress are all consistent with an increase in S-IgA following acute stress. Further, the lack of significance with regard to S-IgA secretion following these tasks may not be entirely attributable to differential effects of the stressor upon saliva volume. At a basic methodological level, it should be noted that the studies of Ring *et al.*, (1999) and Winzer *et al.*, (1999) both had fewer than 20 volunteers, and as such, the lack of significance could be an issue of power rather than saliva volume. The consistency of the trends also supports the notion that acute lab based stress does increase S-IgA.

Further evidence for this notion is provided by another study by Willemson *et al.*, (Willemson, Ring, McKeever & Carroll, 2000). Using a slightly larger sample (n = 27), the authors again used mental arithmetic (PASAT) as a stressor. This study also assessed the effects of task difficulty and task order upon S-IgA activation. The findings regarding this addition to the paradigm will be discussed in light of the work presented in this thesis. Regardless of other manipulations, the authors observed significant increases in both S-IgA concentration and secretion rate following the stressor. It should also be remembered that this study was essentially the same as their previous work, with the exception of a larger sample size.

Using mental arithmetic as a stressor, S-IgA increases have also been observed by Ohira, Watanabe, Kobayashi & Makiko (1999), with a larger sample size (n = 38). Volunteers were required to perform a mathematical addition task, where speed and accuracy were emphasised. Although in itself this was stressful, in addition, volunteers were exposed to random administrations of noise (1 second at 100db). As with the work of Willemson *et al.,* (2000), significant increases in S-IgA from baseline to stress were observed. Owing to the combination of stressors it is impossible to attribute the S-IgA increases to either the mental arithmetic or the noise. However, the findings once again demonstrate that S-IgA increases can be elicited by acute lab based stressors.

The significance of the post-stress increases in S-IgA observed by Willemson *et al.*, (2000) and Ohira *et al.*, (1999) therefore provide greater support for the notion that acute lab based tasks elicit increases in S-IgA, and further, that earlier discrepancies in findings can be attributed, in part, to a lack of power. That is, the consistency of the trends, and the subsequent significance of this study indicate that the effect was evident, it was simply not detected given the small sample sizes.

Other acute lab based manipulations have also been used in attempt to elicit changes in S-IgA. That is, the advantages of lab based studies (i.e., reductions in expectancy of the stressor) were maintained, however, other stressor formats were applied in order to evaluate the robust nature of the S-IgA response to acute stressors. While assessing the effects of lab based manipulations upon S-IgA, these studies also assess the impact of trait and state characteristics in relation to S-IgA reactivity. These factors will be explored in more detail in Section 2.4, however, these studies will initially be discussed in relation to the effects of lab based manipulation upon S-IgA reactivity. Firstly, Harrison, Carroll, Burns, Corkill, Harrison, Ring and Drayson (2000) assessed the effects of film presentations varying in affective content upon S-IgA reactivity.

Previous research of this kind has demonstrated S-IgA increases following exposure to humorous stimuli, however, the authors suggest that previous work was potentially confounded by other general features of the presented affective stimuli. As such, Harrison *et al.*, (2000) assessed S-IgA changes from rest following exposure to three films of varying affective content (classified through prior subjective ratings). The film presentations therefore comprised; one humorous film, one exciting / stressful film, and one film of didactic content. All three film presentation elicited increases in S-IgA, however, there was very little variation in post-stimuli S-IgA with regard to affective content. Although the authors were attempting to explore affect induced changes in S-IgA, the post-stress increases in S-IgA add further support to the notion that acute lab-based stimulation can elicit increases in S-IgA.

Further, in an attempt to assess the effects of affective manipulation upon S-IgA reactivity, Hucklebridge, Lambert, Clow, Warburton, Evans and Sherwood (2000) conducted two experiments. The authors wished to assess whether a manipulated increase in hedonic tone would elicit S-IgA down-regulation analogous with that observed following chronic stress, and further, whether induced positive mood would elicit S-IgA up-regulation. In the first experiment (Hucklebridge *et al.*, 2000) S-IgA was sampled prior to volunteers being required to recall and write about a life situation for a period of 10 minutes that had either elicited feelings of happiness or great guilt. Post-stress S-IgA was then obtained immediately following the 10 minute session, and then 30 minutes post-stress following a period of neutral activity. Both recall conditions elicited significant increases in S-IgA both immediately post-stress and 30 minutes post-stress. Further, although there was no significant difference in S-IgA between the happy and guilty recallers, there was a trend for volunteers recalling happy situations to demonstrate greater up-regulation of S-IgA. The second experiment also induced states of happiness and sadness, however, whilst affect in the first experiment

was induced by subjective experience, the second experiment used music with known validity with regard to affect change. Volunteers provided a baseline saliva sample immediately prior to and following listening to either a sad or happy piece of music for a period of 30 minutes. As with experiment one, both manipulations elicited significant increases in S-IgA, however, there was no significant difference in reactivity between the happy and sad conditions.

The studies of Harrison *et al.*, (2000) and Hucklebridge *et al.*, (2000) support evidence suggesting that lab based manipulations moderate S-IgA reactivity. Further, the findings regarding S-IgA and stress now appear more consistent. That is, while chronic stress has a down-regulatory effect, in the short-term, stress, or more accurately acute stress, elicits a temporary increase in S-IgA. The differences in S-IgA reactivity between chronic and acute stress can bee attributed to the action of the HPA axis (c.f., Evans, Hucklebridge & Clow, 2000). Chronic stress leads to stimulation of the HPA axis and subsequent immune-suppression brought about by the release of corticosteroids. However, in the case of acute stressors, the time period is too short for immune reactions to be mediated by the HPA axis. However, when dealing with stressors, it is not just the time period of stress, but moreover, individual perceptions of stress which will mediate the driving mechanism. Stress, and perceptions of stress can come in many forms, and the recent studies by Harrison *et al.*, (2000) and Hucklebridge *et al.*, (2000) indicate that the stressor can in fact take the form of a manipulation of mood. These studies have not demonstrated clear differences with regard to mood manipulation and S-IgA, moreover, they seem to suggest that it is the manipulation more than the mood that elicits changes (i.e., a generic acute stress effect). However, they do provide evidence that affective states can moderate reactivity to acute stress. The next section will therefore explore the roles of traits, states and individual differences in the perceptions of stress in relation to S-IgA levels.

2.4 S-IgA and the Roles of Trait and State

The studies of stress and S-IgA clearly indicate that stressful experiences elicit changes in S-IgA. Further, in several of the studies there is a cross-over between assessment of stress and affective states. For example, Evans *et al.*, (1993) demonstrated that daily hassles (analogous with affective state) moderates S-IgA, and further, Hucklebridge *et al.*, (2000) provided preliminary evidence for changes in S-IgA in response to manipulations of affective state. Although these studies have been discussed in light of changes in S-IgA in response to stress, their findings indicate that state and trait characteristics can also moderate S-IgA reactivity. Furthermore, the majority of research concerning state and trait factors involve the investigation of the mediating role of these factors. That is, given the general association between stress and illness, it was assumed that in all individuals, stress led to illness, however, this was not always the case. Individual differences were therefore seen as influential in moderating the association between stress and ill-health. That is, everybody gets stressed, but not all people become ill. Anecdotal evidence suggests that some people thrive on stress, and further even seek stress in their everyday lives. Such evidence suggests the existence of specific factors that actually mediate the effects of stress, i.e., some factors may increase perceptions of stress, whilst others may serve to reduce the effects of stress, and therefore reduce susceptibility to stress-related illness. Moreover, many studies have suggested the role of S-IgA as key to this moderating process between stress and illness.

Given the knowledge that some individuals cope better with stress, i.e., experience less stress-related illness, what characteristics do they possess which serve to protect them against stress, and further, what effect, if any, do these factors have upon their S-IgA levels? Martin and Dobbin (1988) assessed the potential role of sense of humour as a buffer to stressful experiences. Specifically they assessed whether a sense of humour moderates the effects of daily hassles upon S-IgA concentrations. The study provided

some support for the stress-buffering effect of sense of humour, however, they observed no association between sense of humour and S-IgA. Humour was further investigated by Dillon, Minchoff and Baker (1985), however, this study assessed the moderating role of sense of humour upon S-IgA in response to humorous stimuli (a humorous video). The authors observed an increase in S-IgA concentration immediately following presentation of the humorous stimuli. However, somewhat paradoxically, they observed an inverse relationship between S-IgA reactivity to the stimulus and sense of humour. That is, those with low sense of humour scores demonstrated the greatest post-stimulus increases in S-IgA. To add to the confusion, Dillon *et al.*, (1985) also reported positive relationships between baseline S-IgA levels and sense of humour (i.e., higher S-IgA in those with the highest sense of humour scores). Similar results were observed by Lefcourt, Davidson-Katz & Keuneman (1990). They observed increases in S-IgA following three humorous films, however, no relationships were observed between baseline S-IgA and sense of humour.

The results regarding sense of humour and S-IgA levels are therefore mixed. However, it should be noted that some of the inconsistency could be attributed to differing methodologies, ie., differences in both the tools used to assess sense of humour and potential differences in the humorous stimuli, or moreover, perceptions of the stimuli. Preliminary evidence has been provided that sense of humour is associated with higher S-IgA. However, inconsistency in the findings, regardless of potential differences in methodology, do not provide evidence that humour has a moderating effect on S-IgA, and moreover, that "laughter is the best medicine".

Other mediating factors with potential immune enhancement have also been assessed. Green, Green and Santoro (1987) and Green and Green (1988) assessed the effects of relaxation upon S-IgA reactivity. The authors assessed the effects of specific types of

active relaxation (e.g., guided visualisation and massage) and demonstrated increases in S-IgA in relaxation but not control conditions. Further, Janoski and Kugler (1987) assessed the effects of active relaxation (progressive relaxation and focused breathing) and imagery (imagining positive immune function). They observed increases in S-IgA following both the relaxation and visual imagery sessions when compared with control conditions. Visual imagery was also utilised by Rider and Welden (1990) in an attempt to assess whether S-IgA increases are more salient following specific visualisation (music and image focus on biological mechanisms) or non-direct imagery (music and non-specific imagery). They observed S-IgA increases following both the specific and non-direct imagery interventions, compared with the control condition (no treatment), but no real differences between the imagery types.

Music has also been identified as moderating positive immune-enhancement. McCraty, Atkinson, Rein and Watkins (1996) assessed the effects of different kinds of music (rock, new-age and designer) upon S-IgA reactivity. In this case, the designer music, a piece called 'Heart Zones', was specifically designed to facilitate mental and emotional states , and to these ends is analogous with the previous work utilising active relaxation and imagery. The authors observed increases in S-IgA following periods of listening to the designer music, but not the other music genres. Further, the greatest increases in S-IgA were observed in those volunteers who listened to the designer music whilst practising self-induced appreciation (a form of positive imagery).

These studies suggest that specific factors are associated with immune-enhancement. However, it should be noted that such interventions (specifically imagery, massage etc.) must be administered over extended periods of time (e.g., regular session over periods of weeks) before immune-enhancement is observed. As such, these studies provide evidence that certain lifestyles, rather than states or traits *per se*, provide immune-

enhancement. This is an important concept in relation to the data and model forwarded in this thesis. That is, these studies suggest that relaxed, stress-free lifestyles can increase levels of S-IgA over a longer period of time. Periods free from stress, spent engaged in relaxing activity can therefore be seen as periods of time where the immune-system can be replenished. That is, chronic stress has an immuno-suppressive effect, whereas, similar period of time spent engaged in relaxing activities result in immune-enhancement. The concept of immune replenishment is key to the findings presented in this thesis, and will therefore be discussed in more detail later.

The majority of research regarding moderating characteristics and S-IgA have focused upon negative states and traits. That is, those factors that moderate the association between negative characteristics and ill-health. In a series of studies, McClelland and colleagues assessed the role of inhibited power motivation in relation to S-IgA levels. Individuals high in inhibited power motivation are described as being hard-driving and assertive, however, they demonstrate an inability to express aggression. Such individuals are characterised by ill-health and increased susceptibility to disease, and as such, provide an excellent sample population for the assessment of S-IgA as a mediating mechanism between these characteristics and ill-health. First McClelland *et al.*, (1980) assessed the role of S-IgA in the relationship between power motivation, stressful life events and URTIs. Volunteers classified as being high in the need for power, high in inhibition of aggression and with greater frequencies of power stress, reported greater frequencies of URTIs, but more importantly lower S-IgA, than all other volunteers. However, following a mildly stressful task, S-IgA in these volunteers did not differ from the rest of the sample.

This study provides evidence that negative trait characteristics (need for power, power stress and inhibition) are related to increased frequencies of URTIs, perhaps brought

about by increased vulnerability due to a lowering of S-IgA. However, these same traits appeared to have no moderating effect upon S-IgA reactivity to a manipulated stressor. The potential moderating effect of these factors was investigated further (Jemmott, Borysenko, Borysenko, McClelland, Chapman & Meyer, 1983), using the previously discussed examination paradigm. Jemmott, *et al.*, (1983) assessed S-IgA during three examination (stressful) periods, and two low-stress points. Volunteers classified as high in the need for power and high in active inhibition demonstrated progressive reductions in S-IgA through to the second low-stress sampling point. Whilst all volunteers demonstrated reductions in S-IgA during examination periods, volunteers low in need for power and low in inhibition demonstrated greater S-IgA recovery during the low-stress sampling periods. Further, volunteers classified as high in need for affiliation with peers and low in inhibition demonstrated higher S-IgA during all sampling points. This study therefore provides evidence of a moderating effect of specific traits upon S-IgA reactivity to stress. Using a similar paradigm, McClelland, Ross and Patel (1985) provided further evidence for moderating effect of power motivation upon S-IgA reactivity to stress. That is, in a smaller time scale, the authors observed greater suppression of S-IgA following the examination in volunteers whose need for power was greater than their need for affiliation.

In contrast, using the examination paradigm, Jemmott and Magloire (1988) identified a factor that demonstrates a positive mediating role between stress and S-IgA. It is suggested that social support is related to positive health outcomes either through a buffering mechanism (buffers at times of stress) or through a more direct route (i.e., continual buffering regardless of stress). The authors observed lower S-IgA during examination periods, however, volunteers who reported having greater social support demonstrated higher S-IgA during all sampling periods. Further, the authors also assessed perceived social support in relation to actual need for social support, this

distinction provided further support for the notion that individual perceptions are often as or even more important that more objective measurements.

Coons, Montello and Perez (1995) also assessed trait characteristics in relation to S-IgA reactivity to examination stress. However, in a variation of the examination paradigm, Coons *et al.*, (1995) assessed musicians before and after a piano examination in relation to factors relating to confidence and denial. Pianists classified as high in confidence and low in denial demonstrated the greatest post-stress elevations in S-IgA. However, in contrast, pianists classified as high in denial demonstrated depressed post-stress S-IgA levels. More importantly, within the context of the current body of work, is how the authors interpret their findings. That is, Coons *et al.*, (1995) suggest that those volunteers who were high in denial demonstrated post-stress reductions in S-IgA as their S-IgA was already at a near maximal level of secretion. As a consequence, in response to the stressor, S-IgA levels could only go down. In contrast, those volunteers who demonstrated post-stress increases in S-IgA (volunteers classified as high in confidence), have lower S-IgA levels prior to the examination and thus had a greater capacity to respond to stress. The authors describe their findings in terms of the Yerkes-Dodson inverted U performance gradient. That is, those who demonstrated post-stress increases, were lower down the arousal curve, and thus could demonstrate positive reactivity. However, those who demonstrated post-stress reductions were already near the peak of the arousal curve, and subsequently could only demonstrate a decline in S-IgA.

The authors explanation of their results implies some sort of finite supply of S-IgA, which regardless of increased stress load cannot be exceeded, perhaps through a negative feedback mechanism that prevents total depletion of the supply. This phenomena has also been demonstrated by Ohira, *et al.*, (1999). These authors

classified volunteers in terms of the classic Type A / Type B personality types, and assessed S-IgA reactivity in response to mental arithmetic and loud noise. Type A volunteers demonstrated higher pre-stress S-IgA levels when compared with Type B volunteers. However, in response to stress, Type A volunteers demonstrated very little S-IgA reactivity, whereas Type Bs demonstrated significant increases in S-IgA. Ohira *et al.*, (1999) suggest that the lack of reactivity in Type A volunteers represents a poorer immune reaction to stress, and could therefore be responsible for the well researched increased susceptibility to illness in such volunteers. However, within the context of the current thesis, the findings of Ohira *et al.*, (1999) are analogous with those of Coons *et al.*, (1995). That is, Type A volunteers are unable to demonstrate positive reactivity to the stressor, as they are already secreting S-IgA at a consistently high level. In terms of the inverted-U model, Type As are already near asymptote, and as such, can only demonstrate a decline in S-IgA. In contrast, Type Bs, are lower down the arousal curve, and therefore have a greater capacity to respond to stress. Clear evidence for such a concept is provided by Ohira *et al.*, (1999). That is, Type A behaviour is characterised by urgency, hostility and an excessive drive to succeed, and to some extent analogous with the high power-motivated volunteers who demonstrated lower S-IgA in the McClelland *et al.*, (1980) study. Such behaviour patterns can also be characterised by continual and prolonged attendance to stimuli. Such attendance is in the short-term arousing and is likely to elevate S-IgA accordingly, moreover, the Type A volunteers were characterised by higher pre-stress S-IgA levels. However, in terms of a finite supply of S-IgA, individuals are unable to maintain such high levels of immune-enhancement, and there will come a point, where reductions will be observed. This phenomena has been demonstrated by both Coons *et al.*, (1995) and Ohira *et al.*, (1999) and will be discussed in depth in light of the findings presented in this thesis.

The studies assessing trait and state characteristics demonstrate that specific factors can influence S-IgA levels, but moreover, can moderate S-IgA reactivity to acute stress. In

the main, negative states and traits demonstrate clearer and more consistent effects upon S-IgA levels and reactivity when compared to positive characteristics. Many of these negative states and traits are characterised by increased perceptions of stress and over-attendance to environmental stimuli. In light of the current body of work, these concepts will be further explored. It will be argued that state and trait factors, as well as individuals' perception of work demands and stress are of vital importance when evaluating immune-responses to stress. That is, such factors will influence everyday levels of S-IgA, and indeed other immune parameters, levels of which will subsequently effect an individuals' capacity to respond to additional stressors. As a final outcome, S-IgA reactivity, moderated by other factors, will influence subsequent susceptibility to post-stress antigenic attack.

2.5 Summary and Conclusions

Several issues have been addressed in this literature review. Firstly, with regards to health status, the bulk of research has focused upon URTIs. As a result the current research will attempt to assess a wider variety of minor health complaints, which are all (to varying degrees) moderated by S-IgA levels. Further, the relationship between ill-health and S-IgA levels can be viewed as cyclical. That is, lower S-IgA is associated with increased frequencies of health complaints. However, do frequencies of health complaints influence subsequent levels of S-IgA, i.e., when the need for S-IgA exceeds the supply. The supply of S-IgA is also related to the other issues raised in the literature review. That is, consistent evidence has been provided that levels of S-IgA can be temporarily increased following an acute stressor. This post-stress increase could be viewed as essential in ensuring that an individual is not at greater risk from infection following the stressor. If illness prone volunteers demonstrate an S-IgA deficit, as a result of a demand in excess of production, they will demonstrate reduced S-IgA reactivity to stress. It is this reduced reactivity that is suggested as responsible for

maintaining a cycle of ill-health, i.e., poor immune response to stress leads to greater risk of illness, incidence of which will moderate S-IgA levels.

With regards to the stress research, a range of stressors have been utilised. Naturalistic stressor obviously provide the greatest external validity. However, when attempting to assess S-IgA changes in response to the stressor *per se*, naturalistic stress studies are complicated by the effects of expectancy and other factors that in everyday life cannot be teased apart from the effects of the stressor. When assessing the effects of the stressor, laboratory studies provide a more controlled environment, where conclusions regarding changes in S-IgA following the stressor can be suggested with greater confidence. However, what laboratory stressors gain in control, they lose in external validity. The current stressor is a multi-tasking battery analogous with any working environment where an individual is required to attend and respond to several stimuli simultaneously. This stressor obviously has greater external validity, but in addition could provide greater knowledge regarding individual differences in S-IgA reactivity to stress. That is, the perceptions of stress brought about by the stressor task will vary between individuals. These perceptions of stress will therefore be assessed in relation to S-IgA reactivity.

Finally, perceptions of stress, and therefore subsequent S-IgA reactivity, are also likely to be influenced by state and trait characteristics. The literature regarding these factors suggest that negative states and traits are associated with lower S-IgA in the long-term. moreover, such negative characteristics are also associated with increased frequencies of ill health. The current series of studies will therefore assess all of these factors as moderating S-IgA response to stress, and subsequent risk of illness.

3. The Minor Health Complaints Questionnaire

3.0 Overview

An analysis of available tools for the assessment and classification of minor health complaints (MHCs) was conducted. Although there are a variety of tools available for the assessment of disease specific and generalised symptoms, no tool could be efficiently used to classify different types of minor health complaints *per se*. The Minor Health Complaints Questionnaire (MHCQ) was therefore developed in order to assess frequencies of minor health complaints (MHCs) in the general population. Further, the MHCQ allowed for the classification of MHCs in relation to their symptomotologies. That is, unlike other tools, the MHCQ is designed to be multi-dimensional and allow for the assessment of distinct clusters of MHCs simultaneously. These classification would then be used as a measure of health status in subsequent studies concerning the effects of acute stress on sIgA reactivity. MHCs were originally classified using principal components factor analysis with varimax rotation on data from a postal survey on the local population (n = 942). Nine distinct cluster of MHCs were identified, and a final cluster of total ill-health was derived through the combination of all MHC items within the MHCQ. The internal consistency of each of the clusters was derived through the use of Cronbach's Alpha. Mean data for each MHC cluster were then derived as a method of further classification. That is, individuals could be identified as either low (good health) or high frequency (poor health) for each of the clusters.

The mean age of the classification sample was 55 years of age, and therefore considerably older than the expected mean age of participants in subsequent studies. In order to ensure that the classification and mean cluster scores could be appropriately applied to subsequent samples, MHCQ data from studies two and three (discussed in Chapters 5 and 6 respectively) were assessed in relation to the identified clusters. That is, mean cluster scores, and internal consistency of clusters were derived and compared

with those from the first study. The mean age from the second sample was considerably lower (mean age = 30), and similar to that of all experimental studies.

The relationship between negative affectivity (NA) and health status are also discussed. Frequencies of health complaints in relation to reporting of psychological complaints (that contribute to the concept of NA) in the data, are discussed in relation to models of NA and health. The influence of NA on the validation of the derived clusters are also discussed.

In summary, this chapter contains details of the classification of MHCs into 10 MHC clusters. These clusters showed reasonably high internal consistency within the derived sample, and a subsequent younger sample. Further, mean scores for each MHC cluster are presented for both an older and a younger sample population.

3.1 Introduction

3.1.1 The Measurement of Minor Health Complaints

There are relatively few questionnaires / scales that are appropriate for the measurement of minor health complaints (MHCs) in a normal population. Many questionnaires are disease specific whilst other measurement tools focus upon more general concepts related to health status, for example, scales of; psychological well-being, mental states, social support and quality of life. Although some of the more specific questionnaires contain items concerning minor health complaints they are usually specific to the main disease state under analysis. For example, the EORTC Quality of Life Questionnaire contains several items concerning minor health complaints, however, the EORTC is used to assess quality of life in cancer patients and as such the items concern symptoms associated with cancer treatments (e.g., nausea, headache and loss of appetite).

Similarly, subsections of some questionnaires concentrate more on minor health complaints, however, these are best used in association with the other component parts of questionnaire.

The following discusses various scales which assess MHCs in some way and their efficiency in eliciting data regarding the classification of MHCs.

The Cornell Medical Index (Brodman, Erdman, Lorge & Wolff, 1949), or CMI was developed for use by physicians in order that they can quickly collect data regarding the medical history of their patients. The index is completed by the patients themselves, but the data can be used directly by physicians. This is achieved through the simple style of language used in the index, which can be easily understood by patients, but easily translated into medical terminology (e.g., *Does you heart often race like mad?* - is interpreted by physicians as degree of tachycardia.). The CMI contains 195 questions with *yes* or *no* response categories and is estimated to take between 10 and 30 minutes for completion. The index is divided into 18 sections comprising, *physical problems* (e.g., respiratory systems, digestive tract and sensory systems), personal habits / frequency of illness (e.g., fatigability), and moods and feelings (e.g., inadequacy, depression and anxiety). Responses are then scored in order that respondents can be classified in relation to the severity of disorder, i.e., more than 25 positive (Yes) responses indicate the presence of a serious disorder, and a medically significant emotional disturbance is considered present with scores over 30. A localised medical problem is diagnosed if positive replies are clustered within one or two sections, but if the responses are scattered throughout the index, a more diffuse medical problem is evident. More than two or three positive responses within the *moods and feelings* section indicate some sort of psychological disturbance.

As with many measurement tools, the CMI can be criticised in terms of structure and content. For example, it is acknowledged that the accuracy of interpretation of symptoms within the index is dependent upon medical knowledge, and as such the CMI is only an effective tool when used within medical settings. Further, accurate frequencies of symptoms are hard to establish within the CMI. Frequency of illness within the CMI is established through questions such as; Do you suffer badly from frequent severe headaches? As such, it is impossible to establish time periods for the reported illness. Further, the structure of the questions can be deemed ambiguous owing to the variations in the interpretation of the word "frequent", i.e., some respondents may consider frequent to mean several times a week, whereas others may give responses applied to a year.

Although the CMI can be an effective tool in the medical diagnosis of specific disease states, its use as a tool for the classification of minor health complaints is limited. For example, the aforementioned headache item is very much in the minority as far as minor health complaints are concerned. That is, the majority of items are related to symptoms (which can be subsequently interpreted by a physician as indicative of particular disease states), and as such, its use as a tool for the classification of minor health complaints is inappropriate .

The Hopkins Symptom Checklist (Derogatis, Lipman, Rickels, Uhlenhute & Covi, 1974) rates physical symptoms that subjects have experienced in the last week (e.g., trouble getting your breath, faintness, dizziness) on a five-point scale ranging from; *not at all (1)* to *extremely (5).* Although the checklist does focus upon health symptoms it assesses general symptoms associated with minor health complaints, not the frequency or severity of the minor health complaints themselves. The assessment of frequency in the Hopkins checklist is potentially useful, as the tool could be applied for varying time

periods, perhaps during times of differing stress (e.g., examination periods), and symptoms can be related to fluctuations in other environmental stimuli. However, the tool focuses upon general symptoms, which although could be subsequently classified, are not necessarily indicative of any specific health complaints or disease states.

The Quality of Well Being Scale (Bush & Kaplan, 1973), or QWB, summarises a person's current symptoms and associated disability as a single score (this score is also adjusted for social undesirability and quality-adjusted life years). The QWB is easily applied to individuals and populations and can be used to assess quality of life in any disease state. The QWB assessment commences with a structured interview used to record symptoms experienced in the previous 8 days and the respondent's level of functioning. The QWB also records the presence of symptoms or problem complexes (CPXs) that refer to the previous day (e.g., Cough, wheezing, or shortness of breath, with or without fever, chills, or aching all over / Headaches, or dizziness, or ringing in the ears, or spells of feeling hot, nervous or shaky).

Although the QWB contains questions regarding minor health complaints, it assesses symptoms rather than actual minor health complaints. The CPXs are also very wordy and each item contains many symptoms which could be associated with a variety of minor health complaints. The data is also collected through a combination of structured interview and questionnaire and must be administered by trained interviewers. The data collection process and the complex scoring of the scale make the QWB a very time consuming method of data collection. Although it has undoubted uses within clinical settings, there is not enough emphasis on specific minor health complaints, their frequencies and associated symptoms to warrant its' use as a measure or classification tool of minor health complaints *per se*.

The Somatic Anxiety Questionnaire (Schwartz, Davidson & Goleman, 1978), or SAQ, is a sub-scale of the Cognitive and Somatic Anxiety Questionnaire. Subjects are required to evaluate the extent to which they feel various symptoms (e.g., rapid heartbeat) when they feel anxious. This questionnaire does not contain any specific minor health complaints items, but focuses upon general symptoms which are associated with anxiety. As such, the SAQ is not a suitable tool for the measurement of minor health complaints *per se*, although its' use as an assessor of changes in anxiety related symptoms following a stressor would be useful.

The most efficient tool for the assessment of MHCs is the Pennybaker Inventory of Limbic Languidness (Pennybaker, Burnam, Schaeffer & Harper, 1977). The Pennybaker Inventory (PILL) records the frequency of occurrence of a large number of common physical symptoms. The scale allows researchers to see what types of specific symptoms are being experienced by an individual and how often they occur. The PILL has also been effectively used as a measure of illness perception (e.g., to assess whether people's perceptions of their heart rate correlate with their heart related symptoms).

The PILL contains 54 items made up of a variety of specific complaints (e.g., headache / constipation) and more general symptoms (e.g., cold hands or feet even in hot weather / numbness or tingling in any part of the body). Respondents are asked to rate each item on a 5-point scale ranging *from Have never or almost never experienced the symptom* (1) to *More than once every week* (5). Originally the PILL was scored by summing the total scores for each of the 54 items, however, the authors now suggest a simpler scoring method is adequate. The simpler method involves the scoring of only those items where the respondent scores 3 or higher (*Every month or so / Ever week or so / More than once every week*).

The PILL is by far the best existing measure of minor health complaints. It is intended to measure the frequency of occurrence of minor health complaints and achieves this through simple assessment and scoring techniques. The scale encompasses a wide variety of symptoms ranging from general, that is, multi-causal (e.g., hands tremble or shake), to more specific, that is symptoms that respondents would associate with specific illness (e.g., constipation / asthma or wheezing) However, all symptoms / complaints are measured over the same time period, regardless of their likelihood of occurrence. Although the PILL contains many health complaint items, the range of health complaints is relatively limited. That is, although differing items (e.g., wheeze and constipation) are included, in order to fully assess the frequencies of health complaints and classify them by symptomotology, a wider range is needed.

3.1.2 The Minor Health Complaints Questionnaire (MHCQ)

In response to the lack of adequate tools for the measurement of minor health complaints, a new tool has been developed with the specific purpose of minor health complaint measurement. It should be noted that it was necessary to develop the MHCQ instead of using existing measurement tools for a variety of reasons. Other than the PILL, other assessment tools were lacking in response variation (e.g., offered a yes / no response), had too few or too many items, or items that were either too specific to a disease state, or conversely offered only items concerning general symptoms. Although the PILL offers a wide range of complaints it is still not entirely appropriate for the current research. That is, it is intended that the MHCQ include several items which are absent from the PILL, in particular, hypothesised indicators of ill-health. Further, items included in the MHCQ will be categorised in terms of likelihood of occurrence. That is, for more frequently occurring complaints (e.g., headaches), respondents will be asked how often they have experienced the complaint in the last month, whereas for more infrequent complaints (in a healthy individual), e.g., colds and flu, respondents will be

asked about frequency within the last year. The new questionnaire will be used primarily as a classification tool in subsequent studies involving S-IgA reactivity to acute stress. To these ends, a tool which assessed a variety of differing MHCs was needed. That is, it was important to assess S-IgA reactivity in relation to a variety of different complaints.

The Minor Health Complaints Questionnaire (MHCQ) is based upon an earlier questionnaire devised to assess minor health complaints in relation to lifestyle and diet (Hyland & Sodergren, 1998). In addition, the MHCQ comprises more somatic complaints (i.e., eczema, sneeze, blocked or runny nose, sore throats, cystitis and itchy eyes), more psychological complaints (day dreams and clumsy), and several hypothesised indicators of ill-health (thirsty, hunger, frequency of night-time urinations and feeling either too hot or too cold). The MHCQ has been constructed under the premise that there is a general factor of ill health, and as a result all MHC items on the MHCQ can be summed to produce a total MHC score. Further, owing to the wide variety of minor health complaints included in the MHCQ, distinct clusters of MHCs can also be summed to produce MHC cluster scores. These clusters are based upon a theoretical rationale suggesting that, whilst all MHCs are related and as such should contribute to a total MHCs score, specific MHCs are related through either a similarity in their symptomotology or their site of infection. Respondents of the MHCQ will therefore produce several MHC scores – one score will represent their total ill health and several others will represent their health related to clusters of specific complaints.

Another potential advantage of the MHCQ is it's inclusion of psychological complaints, including anxiety and depression (two dominating facets in Negative Affectivity). There is a known positive relationship between negative affectivity and ill-health, as will be discussed in more detail in the following section. Although responses in self-

report health questionnaires can be somewhat biased by the influence of Negative Affect, the inclusion of this psychological component will allow for its' influence to be assessed both in combination with and independently of the reporting of somatic complaints.

3.1.3 The Relationship Between Negative Affectivity (NA) and Ill-health

3.1.3.1 Ill-health and Symptom Reporting

Medical diagnosis relies heavily upon the symptomotologies as reported by the patients themselves. Although objective measures of health are used, the preliminary method of diagnosis is that of patient self report. That is, it is the individual that experiences the symptoms, and as a result chooses whether or not to seek medical help. The initial diagnosis of a physician is therefore symptom-centred and based purely upon the information passed on by the patient. That is, the physician must rely upon the patients reports of the intensity, duration and location of the pain or sensation before any sort of classification of the complaint can be made. Similarly, the use of questionnaires in the measurement of health status are also, by their very nature, subjective. The MHCQ (or any other health questionnaire) will be completed by volunteers, and as such, the possible influence of negative affectivity upon responses to the MHCQ must be assessed.

However, it has been proposed that the adoption of a more person-centred approach could lead to more appropriate diagnoses. That is, the self-report method of symptom reporting is, by its very nature, a subjective process. As such, the perception of individuals symptoms are a combination of the somatic symptoms themselves, personality characteristics and the transient feelings of the patient at the time that the symptoms are presented. Further, the style of feedback that the patient received from the practitioner could further influence the subjectivity of the reported symptoms, e.g.,

an empathetic response may encourage the over-emphasis of symptoms if the patient attributes the presentation of symptoms to a sympathetic response

The relationship between patients perceptions and their actual health can therefore be described in terms of their subjective and objective health status. This distinction was further developed by Coe (1978), who distinguished between *illness*, a psychosocial condition derived from an individuals perceptions of their symptoms, and *disease*, a biomedical condition based upon the objective health status of the individual.

The relationships between symptom perception, illness and symptom reporting are therefore very complex, but can be simplified in terms of three models detailing the relationship between subjective illness and objective disease.

Several models have been suggested to explain the relationships between NA and reporting of ill-health. The *Naive Realism* model assumes an positive relationship between symptom reports and objective health status. That is, symptom reports are a reliable indicator of their health status and as such should be taken at face value. Further, this method is frequently employed in the social sciences, where self ratings schemes and symptom checklists are often used as measures of objective health status and severity of illness. The use of this model can also be supported by the significance of correlations between patient reports and those made by the physician (Linn & Linn, 1980) based upon the same symptoms. However, although significant, the strength of these associations is only modest.

A second model, *the psychiatric-categorical model*, is based upon the concept of hypochodriasis, and assumes that certain groups of individuals are preoccupied with their own health, resulting in increased visits to health professionals. For these people,

self-reporting of symptoms is not consistent with any objective measure of health status, that is, they have a consistent unfounded belief in their own ill health which is unrelated to their objective health status. As such, people are categorised as either physically or mentally ill. That is, those people that actually have a physical illness, and those that are suffering from hypochodriasis. Although this model does provide an adequate descriptor of many individuals it cannot be applied to all. That is, physical and mental illness are not mutually exclusive, and as such, it is inappropriate to assume that a person classified as hypochodrial has no objective ill health. Further, hypochondriacs are classified not through a validated measurement procedure, but usually through an informal evaluation by the physician which suggests that the patient has a tendency to report unfounded symptoms.

The final model suggests that the relationship between complaints and objective ill health varies between individuals. That is, there is no simple distinction between under reporters and hypochondriacs, but instead, a continuum of reporting behaviour. As such, symptom reports must be evaluated not in terms of the symptoms themselves, but in terms of personality characteristics of the individual. This continuum, or *dimension of somatic concern* suggests that all health assessments reliant upon self report may be biased by the personality characteristics of the individual. This predisposition to reporting could originate from childhood, i.e., sick children being rewarded with treats, or could be invoked in later life through financial disability benefits. In general, the further along the continuum towards hypochondria, the higher the levels of emotional distress. At the extreme end of the scale, a hypochondriac may experience many negative emotions. As such, there is a very strong relationship between negative emotions and ill health.

3.1.3.2 Personality and Ill-health.

Each of the three models of ill-health and symptom reporting would result in a strong correlation between negative emotions and ill health. Further, specific negative emotions have been identified as those most strongly associated with ill health. That is, for one reason or another, those people who report, or actually suffer from an increase in ill health also demonstrate higher levels of negative emotions than in normal individuals. The relationship between psychological factors and physical complaints is well established, however satisfactory explanations for these relationships and the direction of causality have yet to be concluded.

In general these relationships have been tested, giving rise to hypothesised relationships between psychological factors such as; chronic stressors (Pearlin, Lieberman, Menaghan & Muulan, 1981), minor daily stressors or hassles (Delongis, Coyne, Dakof, Folkman & Lazarus, 1982) and general health state. The relationship has been extensively explored in relation to the personality trait of *neuroticism*. Costa and McCrae (1987) describe neuroticism as " a broad dimension of individual differences in the tendency to experience negative, distressing emotions and to possess associated behavioral [sic.] and cognitive traits". Individuals with high levels of neuroticism are therefore generally; fearful and irritable with low self esteem, social anxiety and have a poor inhibition of impulses. The authors cite a body of evidence suggesting an association between neuroticism and poor physical health. For example, it has been demonstrated that individuals with high levels of neuroticism are more likely to report medical complaints of all kinds (McCrae, Bartone & Costa, 1976 and Costa & McCrae, 1980).

However, it is suggested that the interpretation of such associations could be ambiguous. In order to resolve this ambiguity, Costa and McCrae (1987) conducted an

eight year longitudinal study on a sample of 347 generally healthy females, using the Cornell Medical Index (CMI). The authors report a highly significant correlation between physical and psychiatric health. Further, subsequent analyses allowed the authors to conclude that the observed associations were not a function of other confounding variables, i.e., the correlations were not due to the presence of a small but highly neurotic group of individuals within the sample, neither can any significance be attributed to social desirability. That is, although it could be assumed that individuals may find it socially desirable to indicate high levels of neuroticism, these reported levels were also reflected in the reports of the respondent's friends and family members.

Although the evidence presented so far, offers strong support for the notion that neuroticism is typically associated with somatic complaints, other factors could in part, be responsible for the strength of association. For example, the majority of research in the current focus, relies upon self-report health measures, as such, responses are undoubtedly influenced by both the pervasive (trait) and current (state) mood of the respondent. Although there is some evidence of an association between the health assessments of patients and those of health professionals (LaRue, Bank, Jarvik & Hetland, 1979; Linn & Linn, 1979), the strength of the association is generally no more than moderate. Costa and McCrae (1985) suggest that to assume that measures of somatic complaints reliably reflect patient's objective level of health is naive on behalf of both medics and psychologists. That is, it is naive to assume that reports of somatic complaints accurately reflect objective organic complaints, and further from a psychological perspective, it is naive to ignore that any self report is undoubtedly influenced by the personality of the individual.

Regardless of objective health, responses to health related items are likely to be more negative if the respondent is or has been in a negative mood, or possesses characteristics

indicative of a stable level of negative mood. In contrast, positive mood states are more likely to be reflected through more positive responses to the same health items. Further, Watson and Pennybaker (1989) suggest that many of the hypothesised correlations between psychological factors and health complaints, which are derived from self report measures, may be spuriously inflated as a result of the underlying influence of neuroticism. Therefore, owing to the pervasive influence of neuroticism on responses to health research, objective and subjective measures of health cannot be accurately equated.

Beyond the role of neuroticism, Watson and Pennybaker (1989) suggest that emotional experience is influenced by two broad, bi-polar dimensions - negative affect (NA) and positive affect PA (Tellegen, 1982, cited in Watson & Pennybaker, 1989), both of which can be measured in terms of trait and state mood changes. Although negative affectivity does contain an element of neuroticism, it is a more general dimension containing a range of negative mood states such as; guilt, fear, anger disgust, scorn, anxiety and depression. Conversely, positive affectivity reflects positive mood states such as; excitement, energy, optimism and enthusiasm.

In a previous review Watson and Clark (1984), suggest that individuals with high NA are more likely to experience severe levels of distress and dissatisfaction in all situations regardless of the amount of life stress they are exposed to. Further, their research indicates that high NA individuals can be identified by specific negative characteristics. For example, high NA individuals are more likely to view themselves, others and the world in general, in a negative fashion. As such they will dwell on their own failings, and will rarely see the positive side to their actions or the actions of others. They tend to be very introspective, with a low self opinion, and a bleak outlook on life in general. In contrast, low NA individuals tend to be more satisfied with their lives, are generally

content and secure. In contrast, high PA individuals tend to be more extroverted with high levels of energy. They tend to have a very high level of activity, which is reflected in their maintenance of a happy and interesting life.

As such, NA and PA can both have an underlying influence on self report questionnaires in a similar process to that of neuroticism. In response to the possible interference of NA and PA in the measurement of health complaints, Watson and Pennybaker (1989) conducted a large scale project, incorporating both previous and new data, in which they assessed correlations between a variety of health complaints scales (including several tools discussed earlier in this chapter) and levels of trait NA and PA.

Their review demonstrates that health complaint scales, across all samples are positively correlated with NA, with r-values generally in the region of .35. Further, the strength of these associations is almost as strong as the associations between the health measures themselves. That is, despite the diversity of many of the symptom measures used, their remains a consistently strong association between NA and symptom reporting. Further, PA, in the majority of cases was totally independent of the same measure.

3.1.3.3 Suggested Links Between NA and Ill Health

There is therefore, undoubtedly a strong relationship between NA and ill health, however, the nature of this relationship is not as clear as the strength of associations. There are three main explanations for this relationship between NA and ill health. The first suggests that a predisposition to negative emotionality can be causally linked to the development of ill health. That is, many negative emotional traits contributing to high NA states (anger, hostility, anxiety and depression), have been causally implicated in the development of a variety of health complaints ranging from asthma to coronary

heart disease. In its most general form this explanation can be described as the *Psychosomatic Hypothesis*.

The Psychosomatic Hypothesis

In order to discuss the ability of this model to fully explain the association, the relationship between NA and coronary heart disease (CHD) will be discussed. As with other somatic complaints, NA is highly correlated with many of the physical symptoms of CHD (i.e. angina pectoris). Further, angina pectoris is associated with CHD, but is there a direct causal link between NA and CHD? In order to assess this, the association between NA and objective measures / predictors of CHD must be assessed. Hypertension (high blood pressure), is a well documented risk factor in the onset of CHD, however, research concerning the relationship between measures of NA and objective measurements of hyper tension are mixed. Several studies have attempted to compare NA levels in both normal and hypertensive individuals. However, results indicate either no differences (Cochrane, 1969, 1973; Costa, McCrae, Andres & Tobin, 1980; Robinson, 1969), or lower NA scores for hypertensive individuals (Watson & Pennybaker, 1986).

Further, Watson and Pennybaker (1986), attempted to assess the relationships between PA and NA and more objective measures related to CHD. Measures of NA showed little or no correlation with blood pressure, and in some instances demonstrated a significant negative correlation. Further, measures of PA were generally unrelated to measures of blood pressure. The authors therefore conclude that measures of trait mood are unrelated to objective measures of blood pressure.

Trait mood measures were also assessed in relation to serum levels, in particular, cholesterol and uric acid concentrations. High cholesterol is a well documented risk

factor for the development of CHD, however, concentrations showed little or no correlation with measures of either NA or PA. Further, uric acid, as well as being a strong predictor of CHD, is also highly responsive to temporary fluctuations in stress levels (Kasl, 1968). Despite being a long term predictor of CHD, and a reliable measure of transient stress, uric acid showed little or no correlation with measure of PA. Further, significant negative correlations were observed between NA and uric acid concentrations. That is, as with the blood pressure findings, results concerning serum levels indicate that individuals with high NA have better cardiac health than those with high PA.

These findings indicate that although NA is correlated with complaints indicative of CHD, it is unrelated to objective measures of cardiac health. The most objective measure of CHD is incidence of the disease itself. However, assessments between NA and actual CHD are problematic owing to their longitudinal and prospective methodologies. Several studies of this kind have been conducted and have generally shown that NA levels are unable to predict later onset of CHD (Costa *et al.*, 1982). However, later meta-analyses (Booth-Kewley & Friedman, 1987) suggests that NA may be positively correlated with actual incidences of CHD.

The psychosomatic hypothesis precludes that high NA individuals are more likely to develop ill health by virtue of their level of trait mood, however, the data presented in relation to objective measures of CHD show little or no correlation to measures of NA. In fact, the data demonstrate that individuals high in PA seem to be higher CHD risks than those with high NA. Although only one disease type has been reviewed, further research has shown little or no relationship between NA and other objective measures of health. That is, NA is unrelated to cancer morbidity (Keehn, Golberg, & Beebe, 1974), immunocompetence (Kiecolt-Glaser, Ricker, George, Messick, Speicher, Garner &

Glaser (1984), and overall mortality rates (Keehn *et al.*, 1974). Further, trait mood is not significantly associated with absenteeism, hospitalisation, health visits or health compromising behaviours (Watson & Pennybaker, 1984). That is, high NA individuals seem to report symptoms relating to a wide variety of health complaints, however, objective measures of these same complaints do not justify the incidence of symptom reporting in these individuals.

Despite the absence of evidence regarding NA as a cause of ill-health, the relationship with minor health complaints could be more direct. That is, no studies have assessed objective measures of minor health complaints in relation to NA. Further, any simple measures of objective health (e.g., visits to a GP), are unlikely to show any relationships, since many individuals will not present such basic symptoms to a doctor. Other evidence (Stone, Cox, Valdimarsdottir, Jandorf & Neale, 1987), suggests that levels of S-IgA change in relation to daily fluctuations of both NA and PA.

The Disability Hypothesis

A second explanation of the relationship between NA and ill health suggests that high frequencies of health complaints can lead to increases in NA. That is, an accumulation of health problems can alter the personality of the individual. Ill health can undoubtedly lead to a reduction in self esteem and increased feelings of dissatisfaction. *The Disability Hypothesis* therefore suggests that an increase in ill health can lead individuals to develop high levels of NA. That is, high NA, is another negative consequence of ill health.

Using the disability hypothesis, Watson and Pennybaker (1984), suggest that if trait mood scores are a reflection of an accumulation in pain and discomfort as a result of ill

health, both NA and PA scores should be correlated with symptom scores. That is, as symptom reporting increases NA scores should increase and PA scores should decrease (i.e., ill health should lead to high NA and conversely good health should lead to high PA). Although their findings demonstrate significant and consistent relationships between ill health and NA, there were no consistent negative relations between ill health and PA. This evidence cannot therefore provide support for the notion that ill health leads to high NA, as symptom reporting cannot be dissociated from the likelihood of a high state NA at the time of illness.

If ill health does directly lead to high NA, then it could be assumed that there would be a strong relationship between the severity of ill health and increases in NA scores. However, hospitalised patients do not demonstrate consistently higher NA scores than healthy individuals across an array of health complaints. Further, many individuals with MHCs produce higher NA scores than individuals suffering from severe ill health. However, this could be due to differences in the perception of such illnesses. That is, a severe illness is likely to have been diagnosed and treatment prescribed, conversely, minor health complaints may be more transient in nature and have no real diagnosis. As such, by their very nature, minor health complaints are generally viewed as less debilitating and sufferers will strive to continue with their normal lifestyles. If their illness impinge on their normal quality of life, this may lead to high NA. With regards to more severe illness, such illness will undoubtedly degrade quality of life, however, it is likely that such individuals will have made radical lifestyle changes in response to their diagnosis.

Although ill health can undoubtedly lead to increased feelings of dissatisfaction, the evidence is not strong enough to support the disability hypothesis as an all-encompassing explanation of the relationship between ill health and high NA. Further,

if the disability hypothesis were accepted , it would be very difficult to confirm the direction of causality. That is, regardless of objective health measures or prospective studies, it would be impossible to confirm whether the psychological leads to the physical or vice-versa.

Alternative Explanations

Both the psychosomatic and disability hypotheses seek to explain why individuals with high NA have more health complaints than those with low NA. In order to do this both models assume that health problems are actually correlated and that individuals with high NA are physically different from those individuals with low NA. That is, although they acknowledge the influence of NA, it is assumed that relationships between health complaints occur independently of NA. The evidence presented does not seem to support either the psychosomatic or disability models as sole linear explanations for the relationship between ill health and NA.

In response to apparent inadequacies in the psychosomatic and disability hypotheses Watson and Pennybaker (1989), suggest a third explanation termed *the Symptom Perception Hypothesis*. In contrast to previous explanations the symptom perception hypothesis assumes no physical differences between high and low NA individuals. Moreover, it suggest that high NA individuals are more likely to perceive, respond to, and complain about body sensations, particularly those of a negative nature.

Watson and Pennybaker (1984) firstly point out that whilst NA is highly correlated with health complaints, it is unrelated to health *per se*. Subjective health complaint measures, are by their very nature, subjective. As such, it is possible that the magnitude of correlation between NA and ill health, is in part, caused by the subjective nature of the health scales used. However, this does not necessarily suggest a lack of validity in

the measures used. Many of the self report measures that have been used in previous research, correlate highly with more objective measures such as physicians ratings and health related visits (Pennybaker, 1982). With regard to self report measures, the authors suggest that such scales not only account for variance within health responses, but also variance that is more subjective and psychological in origin. It is this second source of variance, which the authors suggest as the primary cause of association between NA and ill health. That is, those individuals who are high in NA, are more likely to respond in a negative fashion to self report scales. When the scale is measuring health, this is reflected in an increase or over exaggeration of symptoms.

It is further suggested that this likelihood to respond is not to be confused with a state of hypochodriasis, that is, correlations can be explained in relation to a minority group of hypochondriac individuals. Hypochondriacs would not only complain via self report health measures, but would be more likely to engage in more objective behaviours such as visiting the GP, or taking more self prescribed medicine. This is clearly not the case as NA is neither correlated with health visits or increased incidences of over the counter medicines such as aspirin (Watson & Pennybaker, 1984). However, the symptom perception hypothesis suggests more than simple over exaggeration as the primary cause of the correlation. The model suggests that the personality traits of high NA individuals make them overly vigilant with regard to their health and the world in general. Despite rejecting differences in the biological make up of high and low NA individuals, Watson and Pennybaker (1984), draw on the postulated existence of the Behavioural Inhibition System (BIS).

Gray (1985), suggests that the BIS, is located in the septo-hippocampal system, and serves to compare expected stimuli, with those which are actually processed. If the observed and expected stimuli match, then the BIS takes no action. However, if the

observed stimuli is not recognised, the BIS will begin to control the actions of the individual. The BIS attends particularly to those stimuli identified as most important to the individual, (i.e., novel stimuli which require the most checking with existing stimuli patterns). Gray (1985) suggests that high NA individuals have an overactive BIS, and as such are constantly attending to stimuli. This process of checking leads to an increase in the individuals anxiety, and as such the individual will have higher NA. High NA individuals are therefore constantly scanning their environment for new and potentially harmful stimuli, and as a result have increased levels of NA.

Watson and Pennybaker suggest that the BIS can contribute to the symptom perception hypothesis in two ways. Firstly, as a result of an overactive BIS, high NA individuals are more likely to be aware of normal body sensations, or aches and pains, which may be otherwise dismissed by normal individuals. Secondly, the continual process of checking increases levels of anxiety to the extent that, regardless of the actual severity of the stimuli, high NA individuals will perceive the stimuli as harmful, or in the case of health sensations, painful or pathological.

In their extensive review of literature concerning NA, and with particular reference to the validity of the psychosomatic and disability hypotheses, Watson and Pennybaker conclude that there is no biological difference *per se*, between high and low NA individuals. Moreover, the personality traits which contribute to NA, are themselves responsible for the correlation between NA and ill health. That is, the combination of an introspective lifestyle, with high negative emotions, hostility, anxiety and depression, make these individuals attenuate more strange body sensations. Further, the pervasive nature of these personality traits leads to a faulty interpretation of the symptoms that they process, and as a result, they sub-consciously exaggerate both the frequency and severity of symptoms. As such the existence of NA serves as a general nuisance factor

in health research, preventing true association between facets of NA and ill health to be founded.

3.1.4 Conclusions

There is undoubtedly a strong and pervasive relationship between NA and ill-health, and to a lesser extent a relationship between PA and reports of good health. However, as the literature suggests, at best, this relationship can be viewed as cyclical. That is, regardless of the proposed mechanism, the causal direction of the relationship is hard to establish. Although the symptom perception hypothesis attempt to address the cyclical nature of the relationship, it is important to note that the three explanations are not mutually exclusive. Individual differences may therefore provide a clearer notion of direction of causality. That is, in some individuals NA may lead to ill-health, whereas in others, NA may occur as a result of ill-health. Further, ill-health may cause NA, but NA then leads to an over-exaggeration of symptoms.

Regardless of direction of causality, the known relationships serve to warn of the influence of NA in subjective measurements of health status. With regards to the current research, S-IgA is know to be influenced by ill-health (increases in response to antigen) and mood. As such, it is essential that the reader is aware of the possible influence of NA, firstly upon health status, and secondly upon levels of S-IgA. Although it would be futile to attempt to disentangle the NA – Ill-health link further in the current research, it should be noted that the MHCQ comprises psychological items which contribute to the dimension of NA. As such, the influence of NA upon health status and potential effects upon S-IgA can be observed and accounted for.

3.2 Method

3.2.1 Content and Distribution

Potential respondents were selected from the local telephone directory using a simple algorithm (produced in Excel) which randomly selected a page from the directory and an entry from each page. British Telecom suggest that 98% of British households own a telephone, and although many people exclude themselves from inclusion in local directories, the present source of respondents was favoured over the use of the electoral role, which excludes large homogenous groups of potential respondents. It was assumed that most of the entries in the phone book would be male. Therefore, in order that similar numbers of males and females were included in the distribution, two questionnaires were sent to each selected entry along with instructions that the questionnaires should be completed by the oldest male and oldest female in the household. In total 2,500 questionnaires were distributed (1,250 randomly selected respondents), in hand written envelopes. Each envelope contained a covering letter, two questionnaires and a freepost return envelope. The questionnaires were completely anonymous and identifiable by either an orange or green mark on the first page. These marks were used as identifiers for a sub-study concerning the effects of covering letter length on questionnaire return rates (See Authors Declaration).

Each questionnaire contained eight pages. Page one contained a title (Health in Plymouth), information regarding the source of the questionnaire, a brief description of the study, and simple instructions regarding the completion and appropriate return of the questionnaires. The remaining pages consisted of items of a general nature, physical health, and questions concerning medication, family income and education. The response categories varied depending on the likely frequencies of response. Items concerning psychological health were also included, contained within which were items specifically concerned with anxiety and depression. Responses to these items were used

as a measure of NA in subsequent analyses. A copy of the MHCQ is presented in Appendix A.

The questionnaires were distributed in staggered batches (approximately 200 questionnaires per batch) over a period of approximately two months. Subsequently, questionnaires were returned over a similar period of time, with any returns received after this time being excluded from analysis. Upon return, response details were coded and recorded.

Reliability of Derived Clusters: Younger and Older Samples

As would be expected with a health survey of this kind, the mean age of the sample was much higher than would be expected for the subsequent experimental studies (see section 4.1). To ensure that the classification of MHCs could be applied to other age-groups, i.e., that the contributory MHCs to each cluster are not specific to a particular age group, the data from two of the subsequent experimental studies (N = 109) are also presented (sample two). MHCs were classified into the clusters derived from sample one, mean scores and internal consistency were calculated for each cluster. As such, the results section contains mean and internal consistency data for an older population (sample 1), and a younger population (sample 2).

3.2.2 Treatment of Results

Demography

Frequencies for items concerning demographic information (age, sex, income, education) were calculated and presented both in raw and percentage formats.

Factor Analysis

Principal axes factoring with varimax rotation was applied to the data in order to classify MHCs in relation to their symptomotologies. That is, despite the fact that all health complaints are related by virtue of a general factor of ill-health, in order to identify distinct clusters of related complaints an orthogonal rotation (Varimax) was applied to the data. When selecting factors with eigen-values greater than 1, a 9-factor solution was derived.

Internal Consistency of Identified Clusters

Cronbach's Alpha (α) was applied to all identified clusters as a measure of internal consistency for both sample one and sample two.

Calculation of MHC cluster scores

Scores were calculated for each respondent for each MHC within each of the 9 identified clusters for both sample one and sample two (i.e., scores corresponding to response categories were summed to produce a score for each classification). As scoring scales differed between items, it is inappropriate to make comparisons between clusters, however, the main use of the clusters was for subsequent comparisons between individuals within their cluster scores.

The Influence of Negative Affectivity (NA)

To demonstrate the influence of NA upon symptom reporting in the current research, levels of NA were assessed for each of the identified MHC clusters, in low and high frequency individuals. That is, NA was assessed following the classification of the sample by virtue of their frequencies of complaints for each cluster. Scores of NA were derived by producing average scores (for each individual) on the items of *Anxious* and *Depressed* (two major contributors to NA).

3.3 Results

3.3.1 Demography

Table 3.1 presents the return figures for the MHCQ. Although the total return rate was reasonably high (45.12%), the number of completed questionnaires (excluding incomplete or spoilt returns) was slightly lower (37.68%). There were approximately equal numbers of males (47.3%) and females (53.7%), in the sample (as presented in Table 3.2). However, with regards to age, by far the greatest response was received from respondents aged over 60 years (39.6%). Further, it is noted that responses increased in accordance with age category (presented in Table 3.3).

	Returned	***Completed***	***Uncompleted***
Raw	1128	942	186
%	45.12	37.68	7.44

Table 3.1 MHCQ Response Rates

Sex	***Male***	***Female***
Total	449	493
%	47.7	52.3

Table 3.2 MHCQ Response rates by sex

Age	***<20***	***21 - 30***	***31 - 40***	***41 - 50***	***51 - 60***	***Above 60***
Total	5	77	129	155	203	373
%	0.5	8.2	13.7	16.5	21.5	39.6

Table 3.3 MHCQ Response rates by age

3.3.2 Classification of MHCs by Symptom Clusters

Principal components analysis was applied to population one data. There is strong evidence for a general factor of ill-health, and indeed, the internal consistency of the total ill-health cluster is high ($\alpha = .80$). However, for the purposes of identifying clusters of distinct minor health complaints, a varimax rotation was applied. Nine factors were extracted comprising those factors with eigen-values greater than one (sum of squared loadings = 1.05, total variance = 53.75%). As such, Table 3.4 illustrates the

derived 9-factor solution. Only those items with a factor loading greater than ± .3 were selected (Child, 1970) as contributory items to each of the factors (shown in bold).

MHCQ Item	1	2	3	4	5	6	7	8	9
Heart-burn	**0.59**	-0.01	-0.12	0.15	0.10	0.13	-0.10	0.17	0.10
Anxiety	**0.57**	0.09	**0.47**	0.06	-0.10	0.00	0.09	0.10	0.04
Depressed	**0.57**	0.19	**0.48**	0.04	-0.08	0.05	0.00	0.00	-0.03
Difficulty sleeping	**0.43**	**0.42**	0.03	0.13	0.09	-0.02	0.19	0.11	-0.04
Tired for no reason	**0.43**	**0.53**	0.05	0.14	0.16	0.16	0.13	0.11	0.02
Headaches	**0.43**	0.28	0.12	0.26	0.18	0.09	0.10	-0.22	-0.07
Constipation	**0.37**	0.06	-0.08	-0.02	0.07	**0.30**	**0.40**	0.09	0.15
Eczema	**0.36**	0.02	0.15	-0.03	**0.31**	0.00	-0.17	-0.20	0.26
Itchy eyes	**0.32**	0.12	0.00	0.00	**0.54**	0.08	0.08	0.08	0.06
Thrush	0.25	0.13	0.09	0.02	0.01	-0.06	**0.63**	-0.30	-0.17
Number of night-time urinations	0.20	0.03	-0.01	-0.05	0.15	0.05	0.16	**0.66**	-0.24
Thirsty for no reason	0.20	**0.60**	0.02	0.10	0.06	0.28	0.04	0.02	-0.06
Mouth ulcers	0.15	-0.04	0.02	**0.50**	0.03	0.19	0.12	0.01	0.11
Athletes foot	0.13	0.03	-0.10	0.13	-0.06	0.05	-0.08	-0.04	**0.67**
Explosive diarrhoea	0.10	0.05	0.10	0.04	0.03	**0.82**	-0.03	0.06	0.02
Sore throats	0.09	0.16	0.00	**0.81**	0.06	0.07	0.03	-0.13	-0.07
Day dreams	0.06	0.24	**0.49**	0.01	-0.03	0.03	-0.05	0.10	0.15
Wheeze	0.06	0.07	0.15	**0.33**	**0.30**	-0.10	-0.05	0.23	0.23
Clumsy	0.06	-0.02	**0.75**	0.01	0.23	0.01	0.03	-0.02	-0.04
Watery diarrhoea	0.05	0.15	0.09	0.13	0.08	**0.81**	0.00	0.02	0.03
Feeling too hot or too cold	0.03	**0.60**	0.09	0.00	0.08	-0.04	0.26	0.09	0.04
Rate of urination	0.02	0.10	0.13	-0.07	0.06	0.06	-0.13	**0.79**	0.07
Colds & flu	0.02	0.09	0.01	**0.78**	-0.01	-0.03	0.00	-0.03	0.07
Accident prone	0.00	0.11	**0.69**	0.03	0.13	0.18	0.15	0.07	0.02
Sneezing without a cold	0.00	0.12	0.06	-0.03	**0.71**	-0.02	0.04	0.07	-0.02
Fungal infections of groin or scalp	-0.04	0.02	0.14	-0.01	0.14	0.03	0.16	-0.04	**0.72**
Blocked or runny nose without a cold	-0.04	0.04	0.15	0.16	**0.70**	0.12	-0.02	0.04	0.03
Hungry even after a meal	-0.04	**0.65**	0.10	0.07	0.10	0.03	-0.23	-0.08	0.05
Cystitis	-0.16	0.01	0.13	0.14	0.03	-0.02	**0.71**	0.10	0.11

Table 3.4 Rotated Factor Solution

Descriptive labels were then given to each factor based upon the theoretical relationships between the comprising items, including a total ill-health cluster derived from all MHCQ items. (Cluster labels and comprising minor health complaints can be seen in Table 3.5). Although the assignation of labels to MHC clusters is essentially subjective, the reader is reminded that contributory MHCs were selected only if their factor scores were greater than ± .3. Once these MHCs were identified, the assignation of labels was based upon either similarity in symptomotology or site of infection.

Cluster Label	*Comprising Items*
Total Ill-health	All Complaints
Generalised Stress-related Complaints	Heartburn / Anxiety / Depression / Difficulty sleeping / Tired for no reason / Headaches / Constipation / Eczema / Itchy eyes
Indicators of Ill-health	Difficulty sleeping / Tired for no reason / Thirsty for no reason / Too hot or too cold* / Hungry even after a meal / Day dreams
Psychological Complaints	Anxiety / Depression / Day-dreams / Clumsy / Accident prone
Immune Challenge Complaints	Mouth ulcers / Sore-throats / Wheeze / Colds & flu
Atopic Complaints	Eczema / Itchy eyes / Wheeze / Sneezing without a cold / Blocked or runny nose without a cold
Gastric Complaints	Constipation / Watery diarrhoea / Explosive diarrhoea
Urinary-tract Complaints	Number of night-time urinations * / Urination flow
Microflora Imbalance	Constipation . Thrush / Cystitis
Fungal Complaints	Fungal infections of groin or scalp / Athletes foot

* Although these items were identified as being contributory to the MHC clusters of Indicators of Ill-health, Urinary-tract complaints and Total Ill-health, owing to time and space restriction, these items were omitted from subsequent administrations of the MHCQ. As such, subsequent data regarding means and internal consistencies of the clusters do not include these items. However, it should be noted that in the absence of these items, contributory items to each cluster were unchanged.

Table 3.5 Cluster Labels and Contributory MHCs

3.3.3 Internal Consistency of Identified MHC Clusters

As discussed in the treatment of results section, the MHC clusters are derived from data collected from a large postal survey. The mean age of the sample (55 years) is considerably larger than would be expected from the undergraduate samples used in subsequent experimental studies. As such, data from subsequent studies were combined and classified using the derived MHC clusters. The internal consistency of derived MHC clusters were then assessed (Cronbach's α) in both the younger and older samples to ensure that the MHC clusters could be appropriately applied to other samples. Table 3.6 presents Cronbach's α data from the both sample one and sample two.

The alpha coefficient of some of the identified clusters, in particular, urinary-tract, Microflora and fungal complaints, falls well below the figure usually considered to be adequate (see e.g., Cronbach, 1951). However, the number of contributory MHCs to

these particular clusters is low, leading to a reduced Alpha coefficient. Further, it was decided to include these clusters as they were logically consistent. That is, it is suggested that the contributory MHCs can be objectively identified through their similarity in symptomotology. Despite differences in the mean age between the two samples, the differences in Alpha coefficients for each cluster between the two samples was small, and as such, it is assumed that the identified clusters are an adequate basis for between subjects comparisons of health status.

Cluster	N of Cases		N of Items	Cronbach's α	
	One	Two		One	Two
Total Ill-health	923	100	29	.80	.83
Stress-related	932	109	9	.71	.70
Indicators	929	109	6	.65	.64
Psychological	940	109	5	.68	.68
Immune Challenge	935	109	4	.56	.61
Atopic	928	109	5	.52	.59
Gastric	934	109	3	.56	.69
Urinary-tract	937	109	2	.45	.10
Microflora	930	100	3	.31	.39
Fungal	937	109	2	.26	.47

Table 3.6: Internal Consistency of MHC Clusters in Samples One and Two

3.3.4 Comparison of Cluster Scores in Population One and Population Two

As previously stated, the mean age of individuals forming the basis for the MHCs classifications, is considerably older than would be expected in the participants in subsequent experimental studies. Table 3.7 presents the mean scores and standard deviations for MHC clusters in both an older (population 1) and younger (population 2) sample. The differences in mean scores between the samples are not dramatic, however, the younger population demonstrate higher mean scores for all MHC clusters. Standard deviation are large (in comparison to the means), however, this is to be expected in surveys of health status, where, frequencies of health complaints are varied.

	Population 1		Population 2	
	Mean	**S.D**	**Mean**	**S.D**
Total Ill-health	21.94	9.63	26.64	10.68
Stress-related	7.52	5.55	10.48	5.26
Indicators	3.37	3.30	6.10	3.64
Psychological	1.79	1.96	3.64	2.50
Immune Challenge	5.20	1.64	6.72	3.20
Atopic	3.41	2.40	4.20	2.98
Gastric	1.30	1.94	1.59	2.18
Urinary-tract	1.14	1.16	0.61	0.79
Microflora	2.13	1.35	1.91	1.57
Fungal	2.18	0.70	1.36	1.05

Table 3.7: Comparison of Cluster Means

3.3.5 The Influence of Negative Affectivity (NA)

Using the MHC cluster means as a method of classification (mean splits), individuals were classified as either low frequency (good health) or high frequency (poor health). Table 3.8 presents the mean NA scores (anxiety and depression) for good and poor health individuals on each MHC cluster. With the exception of fungal complaints, individuals classified as in poor health, demonstrated significantly higher NA scores ($p < 0.001$) in all MHC clusters.

MHC Cluster	***Category***	***Mean (SD)***	***DF***	***t***
Total Ill-health	Low	. 34 (.45)	686.35	-15.25*
	High	.96 (.70)		
Stress related Complaints	Low	.32 (.43)	651.92	-16.85*
	High	.97 (.68)		
Indicators of Ill-health	Low	.42 (.51)	683.25	-10.57*
	High	.86 (.70)		
Psychological Complaints	Low	.15 (.23)	561.78	-34.86*
	High	1.13 (.55)		
Immune Challenge Complaints	Low	.54 (.60)	586.06	-4.2*
	High	.73 (.69)		
Atopic Complaints	Low	.51 (.58)	688.78	-6.05*
	High	.76 (.69)		
Gastric Complaints	Low	.52 (.59)	531.48	-6.02*
	High	.79 (.69)		
Urinary-tract Complaints	Low	.55 (.61)	453.82	-4.03*
	High	.74 (.68)		
Micro-flora Imbalance	Low	.52 (.60)	381.34	-6.51*
	High	.84 (.68)		
Fungal Complaints	Low	.59 (.63)	938	-1.40
	High	.69 (.67)		

* $p < 0.001$

Table 3.8 Mean NA scores in Individuals in Good and Poor Health

3.4 Discussion

3.4.1 Demography

Despite the possible problem of the majority of telephone book entries being male, there were approximately equal numbers of returns from both males and females in the sample. Several of the female respondents indicated that they were widowed, but were still entered under their husband's name in the phone book. A slightly higher female life expectancy may therefore contribute to the higher female response rates in the sample. Further, by far the greatest return rate was received from the *above 60* age group. In fact, response rates grew progressively greater as age increased. This could be due to a greater number of entries from older individuals in local telephone directories, or may reflect a greater willingness to respond from older individuals. Further, the greater response rates could be simply attributed to time constraints. That is, older individuals are more likely to be retired and therefore have more time in which to respond, than younger individuals who are probably more active in terms of both career and social commitments.

It was acknowledged that the average age of the sample was considerably larger than would be expected in subsequent experimental studies (using undergraduate volunteers). This discrepancy could therefore invalidate the reliability of the derived MHC clusters, i.e., the observed associations between MHCs within clusters, regardless of theoretical rationale. However, as will be discussed in more detail later, comparisons between the mean MHC cluster scores and internal consistency of the clusters in both younger and older samples, suggest the valid use of the derived MHC clusters as a method of classification in the current research.

3.4.2 Classification of MHCs and Assigning of Cluster Labels

Nine MHC clusters were derived from a rotated principal components analysis. These nine factors accounted for over half of the variance. Although the solution leaves a lot of unaccounted variance, this is to be expected in data regarding health status. That is, health status has large inter-individual variation, and as such, any solution is likely to leave a lot of error variance. Before inspecting the salient MHCs within each cluster and assigning labels, it is assumed that the extraction of nine factors indicates that for one reason or another, the MHCQ can be sub-divided into nine distinct clusters. It should be noted that several items appear in more than one cluster. The contribution of MHCs to more than one factor is deemed as valid considering the general nature of health complaints and the multi-causal nature of many of the symptoms, e.g., the MHCs of eczema and itchy eyes both manifest following generalised stress, however, the symptoms are also dominant in atopic individuals

The association between contributory items in each cluster could be viewed from a either a symptomotology or frequency perspective. That is, contributory items within each cluster could be associated through similarity in symptomotology, i.e., individuals who experience X MHC are also likely to experience MHCs Y and Z. Alternatively, the clusters may have been defined by their frequency of occurrence, i.e., all items that occur in similar frequencies over a specific time period may have been clustered together. Further, the derivation of clusters may have been based upon a combination of both symptomotology and frequency of occurrence, i.e., associated MHCs may have similar symptomotologies (or site of infection) and occur in similar frequencies over a specified period of time.

When selecting contributory items to each cluster, only those MHCs with a factor loading of $\pm$ .3, were deemed as salient to that factor. Although the selection of

contributory items can be viewed as a subjective process, the employment of this technique ensures that contributory items are primarily identified from a statistical perspective, not using subjective constructs. Following the selection of contributory items, for the purposes of description, associations between items within each cluster was sought.

Upon inspection of comprising items, the former view (associations based upon similarity in symptomotologies) seems to be responsible for the derivation of MHC clusters. For the majority of MHC clusters, the labels are very encompassing of the comprising items. For example, all psychological (or non-somatic) complaints loaded within the same cluster and were therefore classified as *Psychological* complaints . It should also be noted that the most salient items within this cluster were anxiety and depression, and as such, this cluster comprises facets of negative affectivity. Similarly, on the basis of similarity of symptoms, encompassing explanations can be given to the clusters of *fungal*, *gastric* and *urinary-tract* complaints. However, some of the derived cluster descriptions need more detailed explanation and justification for the possible associations between comprising MHCs.

The Indicators of Ill-health cluster is based upon the work of Hyland & Sodergren (1997). All contributory MHCs have been previously identified as efficient predictors of general ill-health, either through hypothalamic disturbance or in the cases of "difficulty sleeping" and "tired", may reflect the action of inflammatory cytokines. That is, the initial response to antigen that encourages the conservation of energy through fatigue and general malaise. All previously identified indicators of general ill-health demonstrated salient loadings within the same factor.

The *immune challenge* cluster contains several items relating to upper-respiratory tract infections (colds and flu, sore throats and wheeze). In addition, the cluster comprises the item of mouth ulcers, incidence of which is indicative of levels of oral immunity. As discussed in chapter one, all of these items are implicated in the action of S-IgA. That is, S-IgA acts upon the upper-respiratory tract, and is the most dominant antibody in human secretions. The items comprising this cluster can therefore be associated through basic challenge to the immune system (either primarily, or as a secondary symptom in the case of wheeze), in particular, infections of the mucosa.

The most salient items within the cluster of *microflora imbalance* are "thrush" and "cystitis". There was a definite female bias to these items (i.e., more females giving positive response than males), and as such, it would have been appropriate to assign the label of gynaecological complaints. However, the MHC of "constipation" is also a salient item within the cluster. All three contributory MHCs can be related to the systems theory of dysfunctional gut syndrome (DGS), where such symptoms can arise through the imbalance of competing microflora, e.g., Candida albicans (Hyland & Sodergren, 1998). As such, the contributory MHCs within this cluster are all attributed to some sort of imbalance in the microflora of the gut and urino-gynaecological tracts.

Although MHC clusters were derived objectively, apriori predictions based upon theoretical rationales were made. Based upon knowledge concerning upper respiratory tract infections (URTIs), it was assumed that complaints that act upon this area would be associated (i.e., colds, sore throats, wheeze, sneeze, itchy eyes and blocked or runny nose). As has already been discussed, the complaints of colds, sore throats, wheeze (and mouth ulcers) demonstrated salient loadings within one cluster (immune challenge complaints). Despite predictions of these complaints being associated with other complaints which act upon the upper respiratory tract, the complaints of; wheeze,

sneeze, and blocked or runny nose, demonstrated salient loadings in another distinct cluster of complaints. That is, these complaints clustered with the complaints of eczema and itchy eyes. Although several of these complaints act upon the upper respiratory tract, others have very different sites of action. However, all of the complaints within this cluster can be categorised as atopic. Magnam and Vervloet (2000) suggest that the expression of atopy can vary during life, but atopic individuals generally experience dermatological complaints (e.g., eczema), rhinitis (e.g., blocked or runny nose, sneeze and itchy eyes), and asthma (e.g., the symptom of wheeze). The description of this cluster as *Atopy* therefore seems to adequately encompass all of the comprising complaints.

Upon inspection the cluster of generalised stress-related complaints the contributory MHCs within this cluster seem varied. However, it is suggested that the comprising MHCs all manifest following stress. Anecdotal evidence suggests that many people experience these symptoms at times of stress, however more experimental evidence provides support for the notion that these symptoms can all occur following or during stress.

The greatest anecdotal evidence can be provided for the psychological complaints of anxiety and depression. That is, many people have personal experience of experiencing anxiety and depression during or following times of stress. More specific evidence is also by provided by Wheatley (1997), who reports high levels of anxiety and depression in users of a stress clinic. Wheatley (1997) further suggests that the direction of causality between anxiety / depression and stress is cyclical. That is, stress leads to increased feelings of anxiety and depression, levels of which can subsequently lead to increased susceptibility to stressful situation. Similarly, in a sample of medical

practitioners and senior management in the health services, individuals reporting more job-related stress demonstrated higher levels of anxiety and depression (Caplan, 1994).

The gut related symptoms of "heart burn" and "constipation" are both symptoms of irritable bowel syndrome (IBS). Although the possible causes of IBS are varied, and under much debate, one common cause (or more appropriately risk factor) is stress. For example, symptoms of IBS, including heart burn and constipation are more severe on days of high distress (Kellner, 1994), or days high in stress and daily hassles (Dancey, Taghavi & Fox, 1998). Although this research is specific to IBS, many people experience gut problems during times of stress.

Similarly, the sleep related complaints of "difficulty in sleeping" and "tired" can both be related to stress. For example, sleep disturbance (e.g., difficulty sleeping and subsequent tiredness) was greater in individuals who reported greater distress at work (Loewenthal, Eysenck, Harris, Lubitsh, & Gorton, 2000). Further, increased incidence and severity of "headaches" are reported in those individuals who report higher levels of anxiety and depression and general distress (Holroyd, Stensland, Lipchik,, Hill, O'Donnell & Cordingley, 2000).

Alabadies, Kent. & Gawkrodger (1994) have assessed the effects of stress upon dermatological disorders. They suggest that the onset and exacerbation of disorders such as "eczema" are highly associated with stressful situations. Further, individuals who experience and subsequently report complaints such as eczema and rhinitis symptoms such as "itchy or dry eyes" are generally experience more stress, however it is acknowledged that both the experience and reporting of these symptoms could be related to the experiences of stress (Michel, 1994).

Finally, all complaints (from all clusters) were combined to give a *Total Ill-health* score. This cluster demonstrated high internal consistency for both the younger and older samples, and is therefore assumed to be a valid measure of total ill-health. As previously stated, the cluster contains MHC from all of the derived clusters, including the identified indicators of ill-health. The justification for a total ill-health cluster is based upon the notion that there is a general factor of ill-health. That is, all ill-health complaints are related in some way. From a negative affectivity perspective, this cluster could be explained by the symptom perception hypothesis. That is, in some individuals, an overactive behavioural inhibition system (BIS) leads to the over-perception and therefore reporting of symptoms. However, although levels of negative affectivity were higher in those individuals who reported more ill-health complaints, this was not the case for all MHC clusters. This concept will be discussed in more detail in a subsequent section of this chapter.

Another justification would be the association of all ill-health symptoms through the common mucosa. That is, the common mucosa is ubiquitous to all major tracts of the body, and as such, provides a first line of defence to any antigens entering the mucosa. S-IgA plays a major role in mucosal defence. further, the major purpose of the MHCQ is for subsequent comparisons between health severity with regards to S-IgA reactivity. It is therefore appropriate to use the common mucosa as indicative of the association between all MHCs contributing to the cluster of total-ill health.

This section has therefore detailed justifications for the labels applied to the derived MHC clusters. However, as discussed earlier, regardless of theoretical association, nine distinct clusters of MHCs were derived from a principal components analysis, all demonstrating moderate to high internal consistency. Although labels and an adequate rationale have been applied here, it is acknowledged that other factors could be

responsible for the observed associations. Following this line of enquiry, it would be acceptable to apply objective labels to the clusters (e.g., cluster 1, cluster 2 etc.), and make no attempt to rationalise the associations between contributory MHCs. However, previous literature has provided a solid justification for the labels and associations between comprising MHCs. Moreover, given that the MHCQ was developed in an attempt to classify ill-health (in some way), it seems appropriate to apply labels and explanations for the observed associations, based upon theoretical and experimental evidence.

3.4.3 Internal Consistency of MHC Clusters

As previously discussed, the derived MHC clusters were based upon data from a sample with an average age greater than would be expected in subsequent experimental studies. As such, it was important to firstly assess whether the derived clusters were internally consistent, and secondly, whether they were reliably stable across age categories. That is, the derived clusters could be age specific, and the associations between comprising MHCs within clusters could be peculiar to an older sample. It was known that subsequent experimental studies would use undergraduate as a sample population, and therefore the average age in the latter studies would be considerably younger. In order to assess the appropriateness of applying the derived clusters to a younger sample, mean cluster scores (the chosen method for subsequent classification with regards to health status) were compared for the older and a younger sample.

It would be appropriate to analyse the younger sample data using the same methods used to derive the original clusters. However, the sample size of the younger sample is considerably smaller, and as such, analysis through principal components (or any other factor analytical method) would lead to erroneous conclusions. The internal consistency of clusters in both the older and younger samples were therefore assessed using

Cronbach's Alpha (α). There are various rules of thumb that can be applied to coefficients of internal consistency (e.g., Cronbach, 1951). However, coefficients greater than .7 are generally regarded as sufficient grounds for generalisation. However, the accepted value is also dependent upon the range of correlations ordinarily observed in the data. For example, data regarding health often gives rise to fairly small correlations. As such, a value of .7 could be viewed as too stringent. That said, several high Alpha coefficients were observed in the original data. In particular, the clusters of *Total Ill-health* and *Generalised Stress-related Complaints* demonstrated good internal consistency, further the clusters of *Indicators of Ill-health*, *Psychological Complaints*, *Immune Challenge Complaints, Atopy* and *Gastric Complaints* all demonstrate moderate to high Alpha coefficients. This pattern is also apparent in the application of the clusters to the younger sample. Again, *Total Ill-health* and *Generalised Stress-related Complaints* demonstrated high Alpha coefficients, while, with the exception of Urinary-tract Complaints, all other clusters demonstrated moderate to high Alpha coefficients.

The potential impact of using "unreliable" measures is acknowledged. That is, if a measure / cluster is deemed to be statistically unreliable subsequent uses of the cluster as a means of comparison or association could be insensitive. For example, mean splits will be created using the derived clusters, and will be subsequently used to classify individuals with regard to their health status. If differences between classified groups are relatively small, these differences may not be demonstrated as a result of the lack of reliability. It is therefore noted that in the initial stages of this research, the findings will not be generalised. Further, as the clusters are largely exploratory, it may in this context be important to include measures which although may be unreliable may also be of theoretical and practical importance. The potential reliability (or absence of) will be

discussed in more detail following applications of the clusters in subsequent experimental studies.

3.4.4 Cross-Sample Comparisons of Cluster Means

Given the reasonably high Alpha Coefficients for both the younger and older samples, and the exploratory nature of the current research, the derived factors are deemed as valid clusters of distinct health complaints. As previously discussed, the intention is to classify individuals (based upon their cluster score) for each of the derived MHC clusters. Ideally, several categories for each cluster would be defined (e.g., low, moderate and high), however, given the intended sample sizes for subsequent studies, it is accepted that increases in the numbers of categories will drastically reduce the number of individuals that can be allocated to each category. Consequently, it was decided that individuals could be classified into one of two categories. That is, by using mean data, individuals will be classified as either good health (low frequency) or poor health (high frequency) for each of the MHC clusters. It is acknowledged that the use of mean splits will decrease distinction in those individuals who's scores fall very close to the mean, however, such similarity in scores would occur in a classification scheme with more categories, or indeed any other measurement scale.

As previously discussed, the MHC clusters were derived from an older sample. As such, it was appropriate to compare mean scores for each cluster in both the young and older samples. It was noted that the younger sample demonstrated slightly higher means for the majority of MHC clusters (with the exceptions *of Urinary-tract, Microflora,* and *Fungal Complaints*). The higher incidence of ill-health in the young may seem somewhat surprising given knowledge of deterioration of the immune system in later life. However, it is to be remembered that the younger sample consisted mainly of undergraduates. The lifestyle of such individuals can account for the higher

incidence of MHCs. That is, undergraduates, in the main, live in communal settings in close proximity to one another, increasing the pooling, transfer and mutation of antigens, especially in complaints such as colds and flu. It should also be noted that undergraduates are not particularly renowned for living a healthy lifestyle. As such, factors such as poor diets, in combination with proximity and contact with others, are likely to increase incidences of many health complaints.

As subsequent studies will utilise undergraduate volunteers (and indeed this younger population comprised data from two of the subsequent studies), it was appropriate to classify individuals in subsequent studies using the mean MHC cluster data derived from the younger sample. Although it is expected that the mean data in subsequent studies will be similar to that discussed here, the similarity of the means will be assessed in each study to ensure that classifications based upon this sample can be appropriately applied to subsequent samples.

3.4.5 The Influence of Negative Affectivity

The influence of NA on the MHC clusters cannot be directly assessed. However, the current findings add further to the notion that negative affectivity is associated with ill-health. With the exception of fungal complaints, individuals classified as being in poor health demonstrated higher levels of NA. The lack of difference between good and poor health individuals in the cluster of fungal complaints may reflect the influence of desirability of symptom reporting. That is, it may be the case that the relationship between NA and ill-health is most evident in more socially desirable symptoms. However, it is acknowledged that if this were a major influence, then the lack of difference would also be apparent for other socially undesirable symptoms (e.g., MHCs within the gut or microflora clusters).

The current research makes no further attempt to assess the direction of causality between NA and ill-health, but makes the reader aware of its existence and potential influence on research of this kind. As such, it should be noted that the derived cluster of *Psychological Complaints* contains many facets of NA. The cluster therefore allows the role of NA to be assessed in subsequent studies.

3.4.6 Conclusions and Recommendations

The MHCQ was developed in an attempt to classify MHC (in some way) for the purpose of categorising individuals with regards to their health status. Nine distinct clusters of MHCs were derived, further, a cluster of total ill-health complaints was developed. Following the observation that the original sample was considerably older than would be expected in subsequent experimental studies, the derived clusters were applied to a sub-sample (taken from two subsequent studies). Both the younger and older sample demonstrated moderate to high internal consistency for each of the MHC clusters. Given the internal consistency, and moreover the exploratory nature of the current research, a decision was made to utilise all of the 10 MHC clusters in subsequent studies. That is, regardless of the internal consistency coefficients, all of the derived clusters can be justified from a theoretical perspective.

Individuals are to be categorised as either good health (low frequency) or poor health (high frequency) for each of the MHC clusters. As such, mean data for each cluster was calculated. Given the age of the derivative sample , mean data were also calculated for a younger, and therefore more applicable sample. In the majority of MHC clusters, mean scores were marginally higher in the younger sample. Given that these data were similar to that which would be expected in subsequent studies, the mean data from the younger sample will be used as the method of categorisation in all subsequent studies.

However, the mean cluster scores for each subsequent sample will be compared with the pre-defined means to ensure that similarity.

Finally, the influence of negative affectivity upon the reporting of ill-health complaints was assessed. In the majority of MHC clusters, negative affectivity was higher in those individuals categorised as in poor health. These data support the notion that ill-health is associated with negative affectivity, however, the current research makes no attempt to unravel the direction of causality. Moreover, the influence of negative affectivity will be continually assessed through the use of the psychological complaints cluster (which comprises facets of NA). As such, individuals can be categorised in terms of their somatic health, and psychological health, including negative affect.

4. Methods

4.0 Methods Overview

This chapter details the methods used throughout the current research. Specifically, the chapter comprises general information concerning the materials, equipment and procedures used in the following experimental studies. However, it should be noted that whilst this chapter explains the general methodologies, overviews and specific information peculiar to individual studies will be detailed within subsequent chapters.

4.1 Materials

4.1.1 Questionnaire methods

4.1.1.1 The Minor Health Complaints Questionnaire (MHCQ)

The MHCQ has been developed to produce a total ill health score and nine MHC clusters related through similarity in symptomotology. (the breakdown of MHCs into MHC clusters can be seen in Chapter 3).

Scoring

Total ill health and MHC cluster scores are obtained through summing response scores of all MHCs within each MHC cluster. Individuals are subsequently classified as either in good health (low frequency of MHCs within cluster) or in poor health (high frequency of MHCs within cluster). The categorisation of individuals is based upon mean splits derived from a sample population (see Chapter 3). Although it is expected that cluster means in subsequent studies will be similar to those derived in chapter two, the within sample cluster means will be compared with the pre-defined means in each study to ensure there is no major discrepancy. If a discrepancy is observed, a decision will be made concerning the use of either the pre-determined means or the within sample means. this decisions will be based upon the degree of discrepancy and the

sample size. That is to ensure that categorisation using the predefined means allow for appropriate sample sizes within each category.

Although there are differing numbers of complaints in each cluster it is unnecessary to transform scores into a universal scoring format since comparisons are only made between identified individuals / groups in relation to particular MHC clusters.

4.1.1.2 Positive and Negative Affect Schedule (PANAS)

The PANAS (Watson, Clark & Tellegen, 1988), consists of two 10-items mood scales measuring Positive Affect (PA) and Negative Affect (NA): two highly distinctive dimensions. PA reflects the extent to which a person feels alert, active and enthusiastic; high PA is a state of high energy, full concentration and pleasurable engagement, whereas low PA is characterised by sadness and lethargy.

High NA is characterised by subjective distress and unpleasurable engagement that subsumes a variety of aversive mood states including; anger, contempt, disgust, guilt, fear and nervousness. In contrast, low NA is characterised by calmness and serenity. A copy of the PANAS can be seen in Appendix B

Scoring

The PANAS is scored by summing response scores for each domain. That is, all negative emotions are summed to produce the NA score, and all positive items are summed to produce the PA score.

4.1.1.3 NEO Five-Factor Inventory (McCrae & Costa, 1987)

The NEO Five-Factor Inventory (NEO-FFI) is a 60 item version of the 240 item NEO Personality Inventory – Revised (NEO-PI-I). The questionnaire takes approximately 15

minutes to complete and is suitable for any individual aged over 17 years old. The NEO-FFI produces global information regarding the five personality domains of; Neuroticism (N), Extraversion (E), Openness (O), Agreeableness (A), and Conscientiousness (C). As such, respondents to the NEO-FFI produce scores for each of the five personality domains. Personality characteristics typical of the five domains are detailed below. A list of NEO-FFI items and their contributory categories can be seen in Appendix B

Neuroticism

The core of the neuroticism domain can be described as a general tendency to experience negative affects such as; fear, sadness, embarrassment, anger, guilt and disgust. However, neuroticism includes more than just susceptibility to psychological distress. Individuals who are high in neuroticism are also more prone to irrational ideas, are less capable of controlling their impulses, especially when spurred by negative emotion, and typically cope more poorly during times of stress. In contrast, individuals who score low on neuroticism are more emotionally stable. Such individuals are typically calm, even tempered and relaxed. They are more capable of controlling their instincts regardless of the provoking emotion and are less distressed by stressful situations or experiences.

Extraversion

Individuals who score high on extraversion are typically sociable, assertive and active. They tend to like exciting and stimulating experiences and often strive to place themselves in situations that would provoke such emotion. In contrast, individuals who score low on extraversion are typically more withdrawn and less sociable. They are generally pessimistic and would try to avoid the very situations that high extraverts strive for.

Openness

The domain of openness can be described as a general openness to new experience. Individuals high in openness tend to be intellectually curios and as such have a preference for variety in all areas of their life. Owing to their intellectual curiosity, open individuals are often more sensitive to aesthetics and have a very active imagination supported by independence of judgement. Individuals low in openness are typically guarded against new experience. They tend to be more content with their current situation and have very little ambition to encounter new experiences.

Agreeableness

The agreeable person is fundamentally altruistic. Individuals high in agreeableness tend to see the good in situations and as such are typically very friendly and sociable. In contrast, individuals who are low in agreeableness tend to be narcissistic and very conceited. As such, low agreeable individuals are perceived as anti-social and demonstrate symptoms akin to those associated with paranoid personality disorders.

Conscientiousness

The conscientious individual is highly motivated, strong-willed and determined. Although these appear to be positive traits, in highly conscientious individuals these traits can lead to fastidious and compulsive behaviour. Highly conscientious individuals can often become extremely obsessed by the most menial tasks whilst striving for perfectionism in their work. In contrast, low conscientious individuals are typically hedonistic and are far less concerned with the detail of tasks or the quality of work produced.

Scoring

Each domain within the NEO-FFI comprises 12 items, each scored on a Likert style response scale ranging from 0 to 4 (with reverse scoring for negative items). Within each domain, scores from each of the 12 items are summed to produce a raw score for each domain, with male and female respondents weighted differently. Each raw score is then converted to a global t-score which can be classified into either one of five or three categories. The classifications scheme can be seen in Table 4.1.

5-Category	Very Low	Low	Average	High	Very High
3-Category	LOW		Average	High	
t-score	< 35	36 – 45	46 – 55	56 - 65	> 66

Table 4.1. Classification Scheme for the NEO-FFI

4.1.1.4 NASA-TLX Perceived Workload Questionnaire

The Task Load Index, or NASA-TLX (Hart & Staveland, 1988) is a measure of perceived workload and can be used to self rate perceptions of workload related to any task. The questionnaire comprises visual analogue scales for the workload facets of; *mental demand*, *physical demand*, *temporal demand*, *performance*, *effort* and *frustration*. Respondents are required to mark on each facet scale at the point which most reflect their performance. To aid respondents, each workload facet is demonstrated through use of example. For example, the workload facet of effort is explained as "how hard did you have to work, mentally and physically, to achieve your level of performance". The points marked on each facet scale provide respondent's raw scores for each workload facet. A copy of the NASA-TLX can be seen in Appendix B

Scoring

The point at which the respondent marks the line of each workload facet provided the raw score. The scale also incorporates a weighting or importance scale, in order that

both the respondent's facet demand for the task and their over-all perception of that facet are taken into account. Respondents are required to indicate which facet is most important to them through 15 facet pairings (i.e., every facet is paired with every other facet and in each case respondents are required to select which of the two facets is most important to them). The combination of the raw scores and the weighting scheme produce adjusted scores for each of the perceived workload facets.

The adjusted score is produced using the equation in Figure 4.1.

Adjusted Score = I + C
I = A / B x C

Where: I = Importance of item
A = Number of times each facet is chosen from the 15 pairings
B = Total number of facet pairings (15)
C = Raw facet response (mm: up to maximum of 100)

Figure 4.1. Calculation of adjusted workload scores in NASA-TLX

4.1.2 Stressor tasks

In the past a variety of stressor tasks have been used to induce immune suppression and activity. Previous use of stressors include; humorous, exciting and didactic film presentations (e.g., Harrison, *et al.*, 1999), mental arithmetic tasks (e.g., Willemson, *et al.*, 1999), positive and negative mood manipulation (e.g., Hucklebridge, *et al.*, 2000.) and academic examinations (e.g., Bosch, *et al.*, 1998), *c.f.* Chapter 2).

Whilst it is acknowledged that previous research has manipulated tasks that in one way or another would be encountered in everyday life, none of the previously used stressor tasks adequately simulate working environments. The current research will utilise a computer based performance task which simulates any task which requires multi-tasking.

4.1.2.1 Synwork Test Battery

Synwork is a synthetic work environment for the PC (Elsmore, 1990)and was created in response to the need for a laboratory based performance testing situation intermediate between tests typical of performance assessment batteries and full-blown simulators. Synwork is therefore designed to assess multi-task performance, as well as performance on the individual components of the battery and can be easily run for a standard desk-top computer.

The battery involves attendance to four tasks running simultaneously. Firstly, the upper left of the screen contains a memory task. A string of letters (2-8) appear for a pre-set amount of time. Participants are asked to memorise the letters to their best of their ability. At fixed intervals during the session different target letters appear in a small box below the original letter string. Participants have to click the mouse on either *yes* or *no* depending on whether they think the target letter belongs to or does not belong to the original letter string. If participants are unable to remember the original letter string, they can retrieve it at no cost (other than time). Participants receive 10 points for a correct response, and lose 10 points for an incorrect or missed response.

Secondly, the upper right of the screen contains a mental arithmetic task. Participants are required to add together either two or three 3-digit numbers. The problems are self paced with a new problem being presented upon the completion of each subsequent problems. Participants are awarded 10 point for a correct answer and have 10 points deducted for an incorrect answer.

The bottom left of the screen contains a visual tracking task. The task involves a cursor moving to one of the extreme ends of a horizontal line. Participants are required to reset the cursor before it reaches either end of the scale. The number of points awarded are

dependent upon the cursor distance from the centre of the scale. That is, the longer the period of time before the reset, the more points are awarded (up to a maximum of 10 points for a reset at the extreme ends). However, if the cursor reaches and remains at one of the extreme ends, the participant loses 10 point every 0.5 seconds.

Finally, an auditory monitoring task is presented in the bottom right of the screen. Throughout the session a tone is sounded at a fixed time period (base-level tone). Further, a different tone is sounded every time another task is missed or answered incorrectly. Participants are instructed to ignore all of these tones but to report a higher pitched target tone which replaces the base-level tone (probability of target tone = 1:20). Participants are awarded 10 point for correct identification of the target tone, and are deducted 10 points for a missed tone or an incorrect report.

In all session participants are instructed to get as higher score as possible. In order to achieve their score they are further instructed that they should adopt whatever strategy they perceive as most effective in obtaining as high a score as possible. Their total Synwork score is displayed in the middle of the screen allowing participants to keep a constant check on their current level of performance.

Although the Synwork battery is designed as a measure of multi-task performance, the nature of the battery itself, i.e., the combination of tasks simulating a working environment, can be stressful for some individuals and arousing for others. As such, the Synwork battery is not being used as a performance measure, but as a stressor task. The use of the battery also has external validity and increases potential extrapolation of findings, since the battery is a simulation of working environments encountered in every day life.

4.2 Procedures

4.2.1 Experimental Briefs and Instructions

In all studies, volunteers were told that the experiments assessed the relationships between health status (and personality and mood in studies 2, 3 and 4), cognitive performance and immunity. The saliva sampling procedures were explained and a justification for the measurement of S-IgA was detailed (i.e., that S-IgA is one index of general immunity). In addition, it was explained that the saliva sample would be used to assay S-IgA only, and that all data and saliva samples were anonymous (data and saliva samples could only be identified by code and not name). In all studies, volunteers were given a demonstration of the Synwork battery and detailed instruction which they could refer to at any time. Volunteers were also made aware of their right to withdraw either themselves, or their data at any time.

Volunteers were allowed to assume that the emphasis was on their cognitive performance. Further, in studies where volunteers were exposed to the stressor on more than one occasion, they were instructed to attempt to better their previous performance. However, following the experiments, volunteers were informed as to the precise nature of the studies. That is, the links between S-IgA and health, S-IgA and personality / mood, and S-IgA reactivity to acute stress were detailed. Volunteers were then informed that very little emphasis was placed upon their actual performance in the tasks. Moreover, the tasks were used as an acute stressor, in attempt to elicit differences in S-IgA reactivity dependent upon previous health status and personality / mood. Volunteers were then given the opportunity to ask any questions about the research.

4.2.2 Saliva Collection

All collection of saliva was unstimulated. That is, volunteers were not given anything to stimulate saliva production (*c.f.*, Navazesh, 1993) All Volunteers were nil by mouth (other than water) for one hour prior to the experimental session. In all studies saliva samples were taken immediately before and after exposure to the stressor.

Study One

Volunteers were asked to empty their mouth of saliva. They were then asked to dribble into a pre-weighed sterilised vial up to a specified level (1ml).

Study Two and Three

Volunteers were asked to empty their mouth of saliva. They were then asked to collect saliva in the bottom of the mouth (without moving the jaw or stimulating saliva production in any way) for a period of two minutes. Following the collection period, volunteers were asked to empty the collected saliva into a pre-weighed sterilised vial.

4.2.3 S-IgA Assay Procedure

The same assay procedure is utilised in all S-IgA analyses throughout the current research. All analyses were conducted blind by an independent body (Professor Mike Gleeson, University of Birmingham), who had no knowledge of any experimental manipulations that had occurred during S-IgA sampling. The S-IgA assay procedure is detailed below.

Thawed saliva samples were centrifuged at 5,000 rpm for 5 minutes at room temperature prior to analysis. The concentrations of IgA in saliva (s-IgA) were determined by sandwich-type enzyme linked immunoassay (ELISA). All saliva samples were divided into 100µl aliquots and assayed at a dilution of 1 in 1,000.

Samples were then added to each plate as the first layer. The primary antibody of anti-human IgA (Sigma 1-8760) which was used at a dilution of 1 in 800. Samples were analysed in quadruplicate against a range of standards (Human IgA, Sigma 1-2636), 0 – 400 ng / ml. A reference sample was incorporated into each microtitration plate, and all samples from each participant were analysed on a single plate. The final layer consisted of a peroxidase conjugated anti-human IgA (Sigma A-4165) and the substrate ABTS. Absorbencies were measured at 405 nm.

Part Two

Experimental Studies

Part Two comprises three experimental studies. Chapter five presents StudyOone (one stress exposure), Chapter six presents Study Two (two stress exposures), and Chapter seven presents Study Three (cumulative acute stress exposure).

5. Study One

5.0 Chapter Overview

This study assessed the effects of the Synwork battery as a stressor upon concentrations of S-IgA in healthy volunteers in relation to previous episodes of minor health complaints. Previous research has demonstrated S-IgA increases following a variety of naturalistic and laboratory based stressors. The current study assessed the effects of a stressor (Synwork battery), which has not been used in this context before. The Synwork battery is a multi-tasking battery which simulates many working environments. That is, any environment that involves an individual attending to several tasks simultaneously.

Previous research has also assessed S-IgA concentrations in relation to current health status, and also as a factor in susceptibility to upper respiratory tract infections. Episodes of health complaints over a six month period have also been negatively associated with S-IgA levels (Evans, Hucklebridge & Clow, 1993). However, the current study assessed retrospective episodes of minor health complaints during a year long period prior to experimentation, in relation to both resting levels of S-IgA as well as with regard to the S-IgA response to the stressor.

Significant increases in S-IgA concentrations were observed immediately following the stressor. This finding is compatible with previous research assessing S-IgA reactivity to acute stress. However, in the sample as a whole, there were no association between S-IgA concentrations and ill-health. Further exploration of the data revealed that not all volunteers demonstrated post-stress up-regulation of S-IgA. The sample could therefore be divided into Increasers (those demonstrating post-stress up-regulation), and Decreasers (those demonstrating post-stress down-regulation). The S-IgA concentrations were assessed separately for these two groups in relation to their

previous health status. As in the sample as a whole, S-IgA concentrations in Increasers demonstrated little or no association with health status. However, S-IgA concentrations were negatively associated with health status in individuals demonstrating down-regulation (Decreasers). That is, in the Decreaser sub-sample, individuals with the lowest S-IgA experienced the greatest number of retrospective health complaints. Similarly, differing patterns of relationships between S-IgA and perceived workload demands were observed between Increasers and Decreasers. Little or no relationships were observed in Increasers. However, within the Decreasers group, there was a trend for volunteers with lower S-IgA to report greater workload demands, and significantly greater temporal demand following the stressor task.

Although the identification of the Increasers and Decreasers is a novel finding and further, of great theoretical interest, secondary analyses classified individuals in relation to their health status. That is, the MHCQ was devised to classify individuals as being in either good or poor health. As such, it would be more appropriate to consistently apply this method of classification throughout the research. Further, this method of classification would allow for the assessment of S-IgA reactivity to stress, not just pre and post-stress levels. Although the distinction between Increasers and Decreasers would be lost, it would still be possible to observe the underlying trend of reduced S-IgA / reactivity being associated with poorer health. Moreover, this trend should be more readily observed in the sample as a whole, not just in those demonstrating down-regulation.

This method of classification revealed a similar pattern to that observed in the preliminary analyses. That is, volunteers classified as in poor health demonstrated reduced S-IgA reactivity to acute stress, for all of the identified MHC clusters. Further, there was trend for volunteers in poor health to report greater perceived workload

demands following the stressor. It is suggested that those volunteers with poorer retrospective health status have a reduced immuno-capacity / reserve. Subsequently these individuals demonstrate reduced S-IgA reactivity to acute stress. However, it is further acknowledged the relationships between health status, perceived workload demands and S-IgA reactivity to acute stress are complex. A preliminary model has therefore been developed and is presented in an attempt to explain these complex interactions.

5.1 Introduction

5.1.1 Acute Stressor Tasks

As detailed in chapter two, a variety of naturalistic and laboratory methods have been utilised to induce acute and chronic stress. The current research utilised a new stressor task (Synwork). Synwork was developed as a multi-tasking cognitive performance battery, in order to assess performance following or during a range of pharmacological or environmental stimuli. As such, the task induces either arousal or acute stress (depending upon the individual), and should therefore elicit similar immune reactivity to that observed following other acute stressors.

5.1.2 Minor Health Complaints

The possible mediating role of previous episodes and frequencies of minor health complaints upon S-IgA reactivity to acute stress were assessed. Minor health complaints were classified using the MHCQ (see Chapter 3)

5.1.3 Perceived Workload

The NASA-TLX (*c.f.* Chapter 4) was used to assess the perceived workload of the stressor task. It has been suggested (see Chapter 2) that the S-IgA increases observed following acute stress are attributed to arousal. Although this can be viewed as a semantic issue (i.e., what is stress and what is arousal?), as a result of individual differences, the same task can be perceived as either stressful or arousing depending upon the individual, and how they cope with stressful situations. As such, the perceived workload demands of the task were assessed. Although no definitive distinction can be made between whether an individual perceives a task as arousing or stressful, the NASA-TLX can be used as a guide. That is, a preliminary assumption is made that individuals who find the task stressful will report greater workload demands. In contrast, if the task is perceived as arousing, rather than stressful, lower workload

demands will be reported. More credibility can be given to this assumption if those individuals who perceive fewer workload demands demonstrate greater S-IgA reactivity, i.e., if up-regulation of S-IgA is attributed to arousal (and not stress), those individuals who demonstrate the greatest S-IgA reactivity should also report the lowest workload demands. If this phenomena is observed, it can be assumed that the NASA-TLX can be used as a method of classifying a task as either stressful or arousing, although only in conjunction with other measures (i.e., S-IgA reactivity).

5.1.4 Aims and Hypotheses

Aims

1. To assess the effects of a novel acute stressor upon S-IgA reactivity.
2. To assess the effects of previous episodes of minor health complaints upon S-IgA responses to the Synwork battery.
3. To assess individual differences in the perceived workload of the stressor task, i.e., the NASA-TLX will indicate the degree of perceived stress elicited by the stressor task – some individuals will find the task more demanding than others.

Hypotheses

1. That S-IgA concentrations will increase following five minutes on the Synwork battery.
2. That the magnitude of S-IgA reactivity will be moderated by the retrospective health status of the individual. That is, individuals classified as being in poor health (for any MHC cluster) will demonstrate reduced S-IgA reactivity when compared with individuals classified as in good health.
3. That reduced S-IgA reactivity will be observed in those volunteers who perceive the stressor task as more demanding, whereas, volunteers who perceive the task to be less demanding will demonstrate greater S-IgA reactivity.

5.1.5 Summary

The purposes of this study were three-fold. Firstly, the S-IgA concentrations and S-IgA reactivity were assessed in response to a novel stressor. Secondly, the possible moderating effects of retrospective health status upon S-IgA reactivity to acute stress were assessed. It was suggested that the stressor task would elicit similar S-IgA up-regulation analogous with that observed following previously used stressor tasks. However, the magnitude of this reactivity would be influenced by previous health status. That is, individuals who had experienced greater frequencies of MHCs (those classified as in poor health on individual MHC clusters) would demonstrate reduced S-IgA reactivity to acute stress. Finally, the perceptions of stress elicited by the stressor task were assessed in relation to S-IgA reactivity. It was assumed that greater S-IgA reactivity would be demonstrated by those individuals who perceived the task as arousing rather than stressful.

5.2 Methods

5.2.1 Sample

Sixty participants were obtained from a self-selecting sample of stage one psychology undergraduates from the University of Plymouth. Participants were asked to sign up for the study and were then randomly allocated to one of six experimental sessions, with each session containing 10 participants being tested simultaneously. All experimentation was conducted in the month of November, 1999.

5.2.2 Materials

5.2.2.1 Questionnaire Methods

Minor health complaints were assessed and classified using the MHCQ, and perceived workload assessed using the NASA-TLX Perceived Workload Questionnaire (Full details can be seen in Chapters 3 and 4 respectively).

5.2.2.2 Stressor Task

The Synwork multi-tasking battery was used as the stressor task in the current study (Full details can be seen in Chapter 4)

5.2.3 Experimental Protocol

All six experimental sessions took place between 1000 and 1500 hours – these times having been previously identified as demonstrating the least diurnal variation in S-IgA (Hucklebridge, Clow & Evans, 1998). All participants were nil by mouth for a minimum of one hour prior to experimentation, but were allowed to drink water *ad libitum*.

Volunteers (10 in each session) were informed what the experiment entailed. Although participants were not informed of specific aims of the study, they were informed that they would be required to complete a minor health complaints questionnaire, perform some tasks on the computer, complete a questionnaire regarding their perception of the tasks, and provide two saliva samples. Participants were further informed that the saliva samples would be used to measure one immune parameter (S-IgA), and that the samples would not be used for any other purpose. Participants were then given instructions on how to complete both questionnaires (MHCQ and NASA-TLX), and given a brief demonstration of the Synwork battery.

All participants then began the MHCQ. Upon completion of the MHCQ participants were asked to provide their first saliva sample. Participants were then instructed to dribble into the vial in front of them up to the marked level (marked level = approximately 1 ml). All participants then commenced the Synwork task and were instructed to try and get a high a score as possible. Immediately following five minutes on the battery (session programmed to automatically finish after five minute),

participants were instructed to dribble into the second vial up to the pre-marked level. Participants were then instructed to complete the NASA-TLX perceived workload questionnaire in relation to the Synwork session they had just completed.

Upon completion of the NASA-TLX participants were then given a full debriefing. Participants were informed that the study was assessing changes in S-IgA following the cognitive stress induced by the Synwork battery in relation to their previous episodes of minor health complaints. The experimenter was then available to answer any other questions that the participants had in relation to the study.

5.2.4 Treatment of Results

5.2.4.1 Classifications of Health Status

Volunteers were classified as being in either good or poor health with regards to frequencies of health complaints comprising the previously identified MHC clusters. (See Chapter 3 for full explanation of classification process). Frequency distributions of scores for each of the MHC clusters can be seen in Appendix C.

5.2.4.2 Statistics

Relationships between raw S-IgA measures, S-IgA reactivity, and other variables (e.g., MHC cluster scores and perceived workload demands) were assessed using Spearman's Rho. Within-subject changes in S-IgA reactivity were assessed using Student's t-tests for related samples, whilst, differences, primarily in S-IgA reactivity in relation to classification on other variables (e.g., health status) were assessed using t-tests for unrelated samples.

5.3 Results

The result section is divided into six parts. Part 5.3.1 comprises demographic data of the sample. Next, part 5.3.2 details S-IgA reactivity to the stressor, part 5.3.3 details the relationships between S-IgA reactivity and ill-health clusters and part 5.3.4 details S-IgA reactivity and perceived workload.. Further inspection of the data revealed that a sub-sample demonstrated down-regulation of S-IgA following the stressor. The differences between these identified individuals and the rest of the sample are assessed in part 5.3.5. Finally, part 5.3.6 utilises the classifications of health status as discussed in chapter three. That is, the S-IgA reactivity and perceived workload reports of individuals classified as in either good or poor health on each of the identified MHC clusters will be compared.

5.3.1 Sample Demographics

The sample was taken from a psychology undergraduate population. Table 5.1 demonstrates that the majority of the sample were female (85%). Further, Table 5.2 presents classification of the sample by age category. The majority of the sample were aged under 20 years, with over 90% aged under the age of 30.

	Number	Percent
Male	9	15.0
Female	51	85.0
Total	60	100.0

Table 5.1 Sex of Volunteers

	Number	Percent
< 20	38	63.3
20 - 30	17	28.3
31 - 40	3	5.0
41 - 50	1	1.7
51 - 60	1	1.7
Total	60	100.0

Table 5.2 Age of Volunteers

5.3.2 S-IgA Reactivity to Acute Stress

Post-stress concentrations of S-IgA (mean = 160.91μg/ml) were significantly higher than pre-stress concentrations (mean = 130.79μg/ml). That is, significant reactivity ($t_{(59)}$ 6.23, $p < 0.001$) was observed following five minutes of acute stress. The mean data for pre and post-stress concentrations, and S-IgA reactivity to the stressor are presented in Table 5.3, and graphically in Figure 5.1. Distributions of S-IgA data are presented in Appendix C.

	N	Minimum	Maximum	Mean	Std. Deviation
Pre-stress	60	30.00	272.50	130.7917	56.3276
Post-stress	60	55.00	286.50	160.9083	55.3564
S-IgA Reactivity	60	-48.00	106.00	30.1167	37.0328

Table 5.3 Mean S-IgA concentrations and S-IgA Reactivity (in μg/ml)

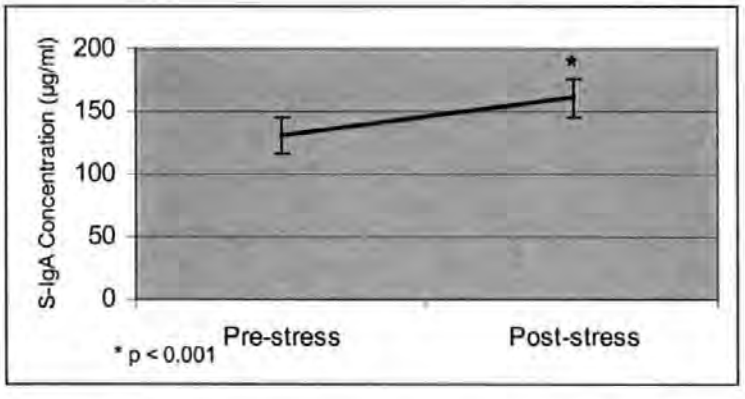


Figure 5.1 S-IgA Reactivity to Acute Stress (and S.E.M)

5.3.3 S-IgA Reactivity and MHC Cluster Scores

Relationships between S-IgA reactivity, that is, pre post-stress differences, and scores for each of the identified MHC clusters were assessed. (Table 5.4). No significant relationships were observed between S-IgA reactivity and scores on any of the MHC clusters, however, in the main observed relationships are negative, albeit small. That is,

individuals with the greatest S-IgA reactivity reported the fewest frequencies of health complaints within each MHC cluster, but not significantly so. Distributions of MHC cluster scores are presented in Appendix C.

		TOTAL	STRESS	INDICATE	PSYCH	IMMUNE
S-IgA Reactivity	Correlation Coefficient	-.095	-.008	.008	.001	-.147
	Sig. (2-tailed)	.469	.952	.952	.997	.264
	N	60	60	60	60	60

		ATOPY	GASTRIC	URINARY	FLORA	FUNGUS
S-IgA Reactivity	Correlation Coefficient	-.158	-.137	.157	-.027	.079
	Sig. (2-tailed)	.228	.296	.232	.839	.546
	N	60	60	60	60	60

Table 5.4 Relationships (Spearman's Rho) Between S-IgA Reactivity and MHC Cluster Scores

5.3.4 S-IgA Reactivity and Perceived Workload

The relationships between S-IgA reactivity and facets of perceived workload (as assessed using the NASA-TLX) were assessed using Spearman's Rho. The observed relationships are presented in Table 5.5. No significant relationships were observed, however, the relationship between S-IgA reactivity and temporal demand approaches significance ($r_{(60)}$ -.23, $p = 0.08$). That is, individuals with the greatest S-IgA reactivity reported the stressor to be less temporally demanding than did individuals with reduced S-IgA reactivity, but not significantly so.

		Mental Demand	Physical Demand	Temporal Demand	Effort	Performance	Frustration
S-IgA Reactivity	Correlation Coefficient	.153	-.115	-.196	.057	.044	.141
	Sig. (2-tailed)	.244	.383	.134	.667	.741	.282
	N	60	60	60	60	60	60

Table 5.5 Relationships (Spearman's Rho) Between S-IgA Reactivity and Perceived Workload

5.3.5 Preliminary Analyses

Exploration of the data revealed that a small sub-set of the sample (n = 14) demonstrated down-regulation of S-IgA following the stressor. Although this sub-sample is small, given the highly significant trend for post-stress up-regulation, it was important to assess whether these individuals differed from the majority of the sample in any way. The following section therefore details analyses that compare differences between the two identified groups (Increasers, who demonstrated up-regulation, and Decreasers, who demonstrated down-regulation), in relation to health status, and perceived workload.

5.3.5.1 Identification of Increasers and Decreasers

Descriptive statistics for pre- and post-stress S-IgA concentration for Increasers and Decreasers are presented in Table 5.6. It should be noted that the mean pre-stress S-IgA concentrations for Decreasers is greater (although not significantly greater) than that of the Increasers. Although it could be argued that the observed up and down-regulation reflects a regression to the mean, it should be borne in mind that these data represent individual responses to the same stressor, as such, there is no reason to assume that the direction of reactivity is simply a statistical artefact. Further, the standard deviations for both pre and post-stress concentrations in both groups are similar, and therefore represent similar variation within the mean response, regardless of direction of reactivity. This issue will be discussed further in relation to health status, and other potential moderators of this mechanism later.

	N	Minimum	Maximum	Mean	Std. Deviation
Pre-stress	46	30.00	272.50	125.9783	57.0444
Post-stress	46	75.50	286.50	171.5326	51.3288

	N	Minimum	Maximum	Mean	Std. Deviation
Pre-stress	14	79.50	244.00	146.6071	52.7655
Post-stress	14	55.00	230.00	126.0000	55.4589

Table 5.6 Descriptive Data for Increasers and Decreasers (µg/ml)

5.3.5.2 Increasers and Decreasers, and Health Status

Thus far in the results, S-IgA reactivity has been the focal point for analyses. However, when comparing relationships between S-IgA and other variables, between Increasers and Decreasers, it is inappropriate to use S-IgA reactivity. That is, it is the direction of S-IgA reactivity which has provided the basis for classification. In statistical terms, S-IgA reactivity has been used as the independent variable. It would therefore be inappropriate to use this classification as a dependent variable. As such, it is already known that S-IgA reactivity, and subsequent analyses using S-IgA reactivity, will differ between these identified groups. It is not known however, whether relationships between pre and post-stress concentrations and other variables, differ between the groups.

Table 5.7 presents the observed relationships between pre and post-stress S-IgA concentrations and health status for Increasers (n = 46). With the exception of the urinary-tract cluster, all relationships with pre and post-stress S-IgA concentrations were positive but small. Significant relationships were observed between pre and post-stress S-IgA and scores for the urinary-tract cluster ($r_{(46)}$ -.30, $p < 0.05$ for both). That is, individuals with

high pre and post-stress S-IgA concentrations had fewer frequencies of urinary-tract complaints.

With the exception of post-stress S-IgA concentrations and microflora complaints, all relationships were negative with the Decreaser group (see Table 5.8). Although no significant relationships were observed, more salient relationships (regardless of direction)were observed, than for Increasers. Specifically, salient negative relationships were observed with the facets of; total ill-health, psychological and gastric complaints. Moreover, the relationships between pre and post-stress S-IgA concentrations and the cluster of immune challenge complaints, were approaching significance ($r_{(14)}$ -.50, p = 0.07, $r^{(14)}$ - .49, p = 0.08, respectively). That is, individuals with the highest pre and post S-IgA concentrations had experienced the fewest immune challenge complaints.

		TOTAL	STRESS	INDICATE	PSYCH	IMMUNE
Pre-stress	Spearman	.132	.100	.055	.190	.094
	Sig. (2-tailed)	.380	.509	.718	.205	.533
	N	46	46	46	46	46
Post-stress	Spearman	.105	.082	.127	.161	.066
	Sig. (2-tailed)	.488	.590	.400	.286	.665
	N	46	46	46	46	46

		ATOPY	GASTRIC	URINARY	FLORA	FUNGUS
Pre-stress	Spearman	.104	.097	-.299	.051	.043
	Sig. (2-tailed)	.491	.522	.043	.737	.776
	N	46	46	46	46	46
Post-stress	Spearman	.053	.022	-.303	-.015	.123
	Sig. (2-tailed)	.725	.882	.041	.919	.417
	N	46	46	46	46	46

Table 5.7 Relationships (Spearman's Rho) Between Pre and Post-stress S-IgA concentrations and Health Status in Increasers

		TOTAL	STRESS	INDICATE	PSYCH	IMMUNE
Pre-stress	Spearman	-.384	-.060	-.133	-.303	-.498
	Sig. (2-tailed)	.176	.839	.650	.292	.070
	N	14	14	14	14	14
Post-stress	Spearman	-.434	-.111	-.215	-.294	-.487
	Sig. (2-tailed)	.121	.706	.460	.307	.077
	N	14	14	14	14	14

		ATOPY	GASTRIC	URINARY	FLORA	FUNGUS
Pre-stress	Spearman	-.041	-.409	-.065	-.100	
	Sig. (2-tailed)	.889	.146	.826	.734	
	N	14	14	14	14	14
Post-stress	Spearman	-.053	-.267	-.151	.049	
	Sig. (2-tailed)	.858	.357	.606	.868	
	N	14	14	14	14	14

Spearman's Rho coefficients not produced for cluster of fungal complaints as no variation in cluster scores

Table 5.8 Relationships (Spearman's Rho) Between Pre and Post-stress S-IgA concentrations and Health Status in Decreasers

5.3.5.3 Increasers and Decreasers, and Perceived Workload

Relationships between pre and post S-IgA concentrations and facets of perceived workload were assessed in both Increasers and Decreasers. Table 5.9 presents the Spearman's Rho coefficients for the Increasers. No significant relationships were observed, however, with the exception of pre-post S-IgA and perceived performance, all relationships were either near zero or positive. A significant relationship was observed between pre-stress S-IgA and temporal demand (r (26) .29, p = 0.05). That is, Increasers with higher pre-post S-IgA concentrations demonstrated a propensity to report higher levels of temporal demand following the stressor task.

Salient negative relationships were observed between pre and post stress S-IgA concentrations and facets of perceived workload in Decreasers (Table 5.10). Specifically, salient negative relationships were observed between pre- and post-stress S-IgA and mental demand. Moreover, significant negative relationships were observed between both pre and

post-stress levels and temporal demand ($r_{(14)}$ -.63, $p < 0.05$, $r_{(14)}$ -.53, $p < 0.05$, respectively), and pre-stress S-IgA and effort ($r_{(14)}$ -.62, $p < 0.05$). This relationship loses significance post-stress, but maintains salience. That is, Decreasers with lower pre-stress S-IgA demonstrated a propensity to perceive greater temporal demand and effort following the task. Further, Decreasers with lower post-stress S-IgA perceived greater temporal demand, and demonstrated a trend to report that greater effort was required by the tasks.

		Mental Demand	Physical Demand	Temporal Demand	Effort	Performance	Frustration
Pre-stress	Spearman	.172	.027	.288	.155	-.165	.063
	Sig. (2-tailed)	.253	.859	.052	.305	.273	.676
	N	46	46	46	46	46	46
Post-stress	Spearman	.234	-.095	.199	.158	-.070	.081
	Sig. (2-tailed)	.118	.529	.184	.295	.646	.591
	N	46	46	46	46	46	46

Table 5.9 Relationships (Spearman's Rho) Between Pre and Post Stress S-IgA Concentrations and Facets of Perceived Workload in Increasers

		Mental Demand	Physical Demand	Temporal Demand	Effort	Performance	Frustration
Pre-stress	Spearman	-.349	.277	-.629	-.618	.146	.209
	Sig. (2-tailed)	.221	.337	.016	.019	.619	.474
	N	14	14	14	14	14	14
Post-stress	Spearman	-.367	.158	-.532	-.473	.084	.020
	Sig. (2-tailed)	.197	.589	.050	.088	.775	.946
	N	14	14	14	14	14	14

Table 5.10 Relationships (Spearman's Rho) Between Pre and Post Stress S-IgA Concentrations and Facets of Perceived Workload in Decreasers

5.3.5.4 Summary and Discussion

Associations between S-IgA and health and S-IgA and perceived workload revealed differing patterns of relationships between individuals identified as Increasers and Decreasers. In the main Increasers demonstrated small, but positive relationships between pre and post S-IgA concentrations and MHC clusters, and facets of perceived workload. In contrast, Decreasers demonstrated negative and stronger relationships. Specifically, significant relationships were observed between S-IgA concentrations and the perceived workload facets of temporal demand and effort, and near significant relationships between S-IgA and the MHC cluster of immune challenge complaints. These differing patterns of relationships indicate that different associations are manifested between health and perceived workload in those individuals who demonstrated post-stress down-regulation of S-IgA. That is, within this sample, individuals with high S-IgA experience fewer health complaints and demonstrate a propensity to find the stressor task less demanding. In contrast, individuals in this same group with low S-IgA experience greater frequencies of complaints and demonstrate a propensity to find the task more demanding.

Despite these differences between Increasers and Decreasers, the analyses conducted in this section do not account for the previously identified classifications of health status. That is, the MHCQ was developed to allow for the classification of individuals through the frequency of occurring complaints, for a variety of MHC clusters. The purpose of this classification method is to ensure consistency throughout the current research, and in future research projects. As such, health status will now be the focus of classifications for subsequent analyses. Although the use of this method will result in the loss of the distinction between Increasers and Decreasers, the observed relationships should still be apparent. That is, if Decreasers (those who demonstrate post-stress down-regulation of S-

IgA) who have low S-IgA concentrations experience higher frequencies of health complaints, then individuals classified as in poor health (higher frequencies of complaints) should have lower S-IgA, or reduced reactivity to the stressor. The health status method of classification should also allow for the observation of these differences in the whole sample, not just those who demonstrate down-regulation (i.e., those who have experienced greater frequencies of health complaints should demonstrate reduced S-IgA reactivity to the stressor.

5.3.6 Secondary Analyses

As previously discussed, the following section used the MHCQ clusters as a method of classifying individuals. As such, this section assesses S-IgA reactivity between those individuals in good and poor health on each of the MHC clusters. Similarly, using the same classification method, differences in perceived workload were also assessed in these same individuals.

5.3.6.1 Health Status and S-IgA Reactivity to Acute Stress

Using the pre-determined means, volunteers were classified as being in either good (low frequencies of complaints) or poor (high frequencies of complaints) retrospective health with regard to total ill-health and nine MHC clusters. S-IgA reactivity to the stressor was then compared between the two groups. No significant differences were observed (at $p < 0.05$), however, consistent trends in reactivity between the two groups were apparent. Mean data for pre- and post-stress S-IgA by health status are presented in Appendix C.

Total Ill-health

Volunteers with higher frequencies of total ill-health complaints (poor health) demonstrated reduced S-IgA reactivity to the stressor (n = 34, mean = 24.40 μg/ml, SEM = 6.67), than did volunteers classified as in good total health (n = 26, mean = 37.60 μg/ml, SEM = 6.62), but not significantly so. The difference in reactivity between volunteers with good and poor total ill-health is presented in Figure 5.2.

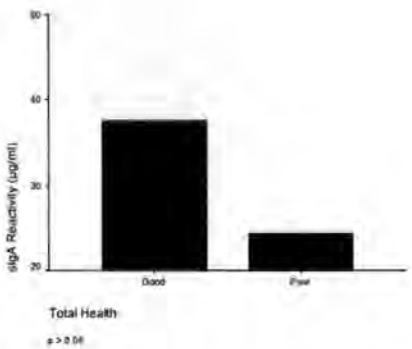


Figure 5.2 S-IgA Reactivity in Volunteers with Good and Poor Total Ill-health

Generalised Stress-related Complaints

Figure 5.3 presents the S-IgA reactivity to acute stress for volunteers classified as in either good or poor health with regards to generalised stress-related complaints. There was a non-significant trend for volunteers classified as in poor health (n = 31) with regards to stress-related complaints to demonstrate reduced S-IgA reactivity (mean = 27.08 μg/ml, SEM = 6.33) when compared with volunteers classified as in good health (n = 29, mean = 33.36 μg/ml, SEM = 7.28).

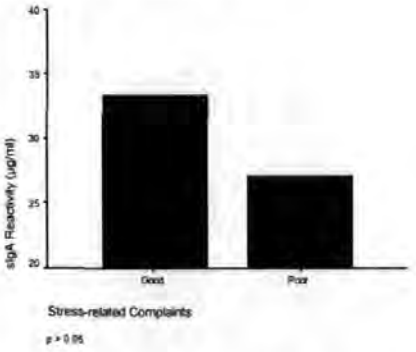


Figure 5.3 S-IgA Reactivity in Volunteers with Good and Poor Stress-related Complaints

Indicators of Ill-health

Volunteers classified as in poor health with regards to indicators of ill-health demonstrated reduced S-IgA reactivity to the stressor (n = 40, mean = 29.25 µg/ml, SEM = 6.17) when compared to those volunteers classified as in good health (n = 20, mean = 31.85 µg/ml, SEM = 7.51), but not significantly so. The differences in S-IgA reactivity between volunteers classified as in either good or poor health are presented in Figure 5.4.

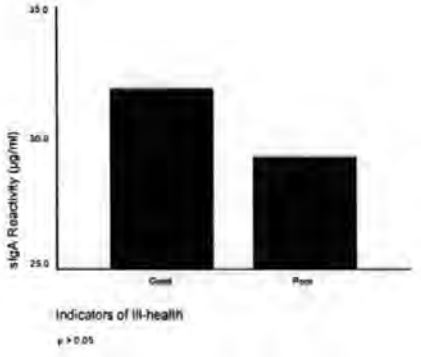


Figure 5.4 S-IgA Reactivity in Volunteers with Good and Poor Indicators of Ill-health

Psychological Complaints

Figure 5.5 presents data for the differences in S-IgA reactivity to acute stress between volunteers classified as either in good or poor health with regards to frequencies of

psychological complaints. There was a trend for volunteers classified as in poor health (n = 20) with regards to psychological complaints to demonstrate reduced S-IgA reactivity to acute stress (mean = 29.50 μg/ml, SEM = 5.77) when compared with those classified as in good health (n = 40, mean = 31.35 μg/ml, SEM = 8.72).

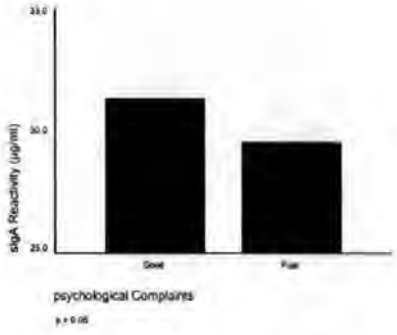


Figure 5.5 S-IgA Reactivity in Volunteers with Good and Poor Psychological Complaints

Immune Challenge Complaints

Volunteers classified as in poor health (n = 20) with regards to frequencies of immune challenge complaints demonstrated reduced S-IgA reactivity to acute stress (mean = 27.88 μg/ml, SEM = 6.11) when compared with those classified as in good health (n = 40, mean = 34.26 μg/ml, SEM = 7.72), but not significantly so. The differences in S-IgA reactivity between volunteers classified as in either good or poor health are presented in Figure 5.6.

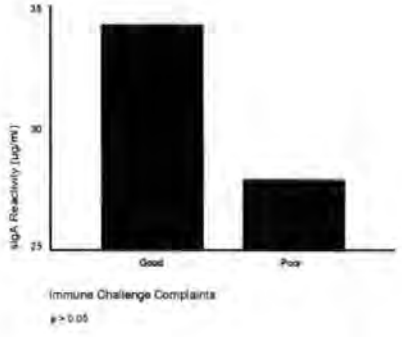


Figure 5.6 S-IgA Reactivity in Volunteers with Good and Poor Immune Challenge Complaints

Atopic Complaints

Figure 5.7 presents the differences in S-IgA reactivity to acute stress between volunteers classified as in either good or poor health with regards to frequencies of atopic complaints. There was a trend for volunteers classified as in poor health (n = 20) with regards to frequencies of atopic complaints to demonstrate greater S-IgA reactivity to acute stress (mean = 32.80 μg/ml, SEM = 8.07) than did those classified as in good health (n = 40, mean = 28.78, SEM = 5.99).

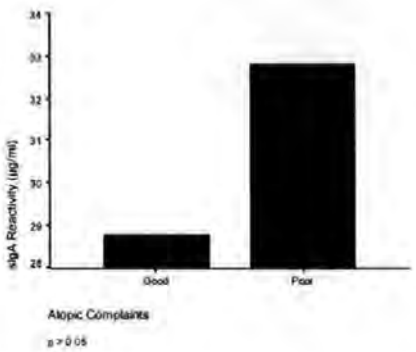


Figure 5.7 S-IgA Reactivity in Volunteers with Good and Poor Atopic Complaints

Gastric Complaints

Volunteers classified as in poor health (n = 20) with regards to frequencies of gastric complaints demonstrated reduced S-IgA reactivity to acute stress (mean = 21.5 μg/ml, SEM = 7.18) when compared with those classified as in good health (n = 40, mean = 34.53 μg/ml, SEM = 6.15), but not significantly so. The differences in S-IgA reactivity to acute stress between volunteers classified as in either good or poor health are presented in Figure 5.8.

Figure 5.8 S-IgA Reactivity in Volunteers with Good and Poor Gastric Complaints

Urinary-tract Complaints

Figure 5.9 presents the differences in S-IgA reactivity to acute stress between volunteers classified as in either good or poor health with regards to frequencies of urinary-tract complaints. There was a trend for volunteers classified as in poor health (n = 21) with regard to frequencies of urinary-tract complaints to demonstrate greater S-IgA reactivity to acute stress (mean = 37.35 µg/ml, SEM = 8.06) than did those classified as in good health (n = 39, mean = 26.22 µg/ml, SEM = 5.92).

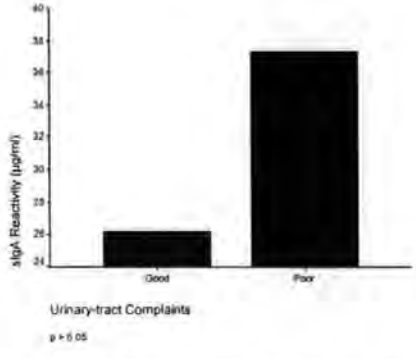


Figure 5.9 S-IgA Reactivity in Volunteers with Good and Poor Urinary-tract Complaints

Microflora Complaints

Volunteers classified as in poor health (n = 53) with regards to frequencies of microflora complaints demonstrated greater S-IgA reactivity to acute stress (mean = 30.79 µg/ml,

SEM 5.01) than did those classified as in good health (n = 7, mean = 25.00 µg/ml, SEM = 16.52), but not significantly so. The differences in S-IgA reactivity to acute stress between volunteers classified as in either good or poor health are presented in Figure 5.10. It should be noted that female bias in this sample are likely to be responsible for the large discrepancy in sample numbers between those in poor and those in good health. That is, the microflora cluster of ill-health contains the MHCs of cystitis and thrush, high frequencies of which are usually reported by females, not males.

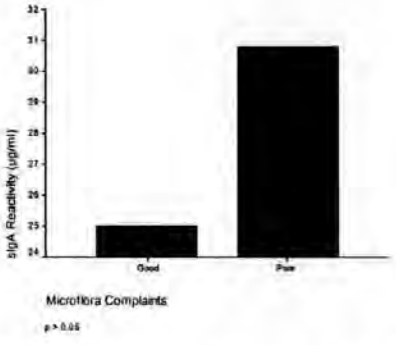


Figure 5.10 S-IgA Reactivity in Volunteers with Good and Poor Microflora Complaints

Fungal Complaints

All volunteers within the sample reported scored either zero or one for the cluster of fungal complaints. As such, volunteers could not be classified as either in good or poor health with regards to frequencies of fungal complaints.

5.3.6.2 Health Status and Perceived Workload

Facets of perceived workload (as assessed using the NASA-TLX) immediately following the stressor were compared in volunteers classified as in either good or poor health on each of the MHC clusters. Tables of means for each health cluster are presented in the Study Appendix C.

Total Ill-health

There was a trend for volunteers classified as in poor health with regards to total ill-health (n = 26) to report more mental demand and effort. Further, poor health volunteers reported significantly greater frustration ($t_{(58)}$ –3.55, $p < 0.001$) following the tasks, when compared with those classified as in good health (n = 34). In contrast, good health volunteers reported marginally greater perceived performance than did poor health volunteers.

Generalised Stress Complaints

There was trend for volunteers classified as in poor health (n = 31) with regards to frequencies of generalised stress complaints to report greater mental, physical demand and effort. Further, poor health volunteers reported significantly greater frustration ($t_{(58)}$ –2.95, $p < 0.01$) following the stressor than did volunteers classified as in good health (n = 29). In contrast, there was a trend for good health volunteers to report greater temporal demand, and marginally greater perceived performance following the stressor than poor health volunteers.

Indicators of Ill-health

There was trend for volunteers classified as in poor health with regards to ill-health indicators (n = 40) to report greater mental demand. Further, poor health volunteers reported significantly greater frustration ($t_{(58)}$ –1.99), $p = 0.05$) following the stressor than did volunteers classified as in good health (n = 20). Conversely, there was a trend for volunteers classified as in good health to report greater temporal demand and perceived performance.

Psychological Complaints

No significant differences were observed for reports of perceived workload between volunteers classified as in either good or poor health with regards to frequencies of psychological complaints. However, there was a trend for volunteers classified as in poor health (n= 40) to report greater mental demand, effort and frustration, than those classified as in good health (n= 20).

Immune Challenge Complaints

There was a trend for volunteers classified as in poor health with regards to immune challenge complaints (n = 39) to report greater temporal demand and perceived performance. Further, poor health volunteers reported significantly greater mental demand ($t_{(58)} -1.98$, $p = 0.05$) and effort ($t_{(58)} -2.97$, $p < 0.01$) than did those classified as in good health (n = 21). In contrast, there was a trend for volunteers classified as in good health to report greater physical demand following the stressor.

Atopic Complaints

There was a trend for volunteers classified as in poor health (n = 20) with regards to frequencies of atopic complaints to report greater mental and temporal demand and effort than those classified as in good health. Further, volunteers in poor health reported significantly greater frustration (t (58) -2.40, $p < 0.05$) following the stressor than did those in good health. In contrast, there was a trend for volunteers in good health to report greater physical demand and perceived performance.

Gastric Complaints

No significant differences were observed between good and poor health volunteers with regards to frequencies of gastric complaints on any perceived workload facet. However, there was a trend for volunteers classified as in poor health (n = 20) to report greater mental and temporal demand, effort and frustration following the stressor than volunteers in good health (n = 40).

Urinary tract complaints

There was a trend for volunteers classified as in poor health (n = 21) with regards to frequencies of urinary-tract complaints, to report greater mental and temporal demand, and effort. Further, volunteers in poor health reported significantly greater frustration ($t_{(58)}$ – 1.96, p = 0.05) following the stressor than those in good health. Conversely, there was a trend for volunteers in good health (n = 39) to report greater perceived performance.

Microflora Complaints

No significant differences were observed between volunteers in good and poor health with regards to microflora complaints on any perceived workload facet. However, there was a trend for volunteers classified as in poor health (n = 53) to report greater mental demand and effort following the stressor than volunteers in good health (n = 40).

Fungal Complaints

Following classification, all volunteers in the current sample were classified as being in good health with regards to frequencies of fungal complaints. As such, no analyses between groups on facets of perceived workload could be conducted.

5.4 Discussion

5.4.1 Demographics of Sample

As would be expected from a sample of undergraduate students, the sample had a mean age of 24 years of age. Further, 91.6% of the sample were aged below 30 years of age. There was also a large difference in the numbers of males and females within the sample. There was also a discrepancy between the age of the current sample and the age of the sample used to derive the MHC clusters. As such, volunteers were classified using standardised means derived from a combination of several experimental studies with a mean age analogous to that used at present. However, it should also be noted that theses standardised means did not differ greatly from the within-sample means in this study. It was therefore considered appropriate to apply the younger sample standardised means to the sample in the current study.

5.4.2 S-IgA Reactivity to Acute Stress

In support of previous findings (*c.f.* Chapter 2), significant increases in S-IgA concentrations were observed following the stressor task. Previous work (e.g., Willemson, 2000) indicate that these increases could be attributed to stimulation of the autonomic nervous system and subsequent activation of the transepithelial secretory mechanism resulting in S-IgA release into saliva. Physiologically this increases reflects the body's response to a perceived threat. This up-regulation of the immune system, namely an increase in S-IgA concentration ensures that the body is not more susceptible to either biological challenge that may accompany the stressor, or to increased susceptibility during and immediately after exposure to the stressor, i.e., during the time period where the body is identifying and preparing appropriate defences to the stressor. The S-IgA released following the stressor is not specific to a particular antigen, moreover, it comprises S-IgA produced following previous antigenic exposure. The observed S-IgA therefore represents S-IgA reactivity in the common mucosa, which,

although not specific has the potential to protect against pathogens and antigens previously encountered. Further, owing to the use of Synwork as a simulation of working environments, the present findings are also analogous with observed increases in S-IgA following acute naturalistic challenges (*c.f.* Chapter two). It can therefore be assumed that the alternative use of a cognitive performance battery can be applied to physiological stress research. That is, based upon the concept that cognitive performance tasks can elicit stress in some individuals and arousal in others, the Synwork battery was adapted for use as a stressor task. The observed results are analogous with previous results assessing the effects of various stressors upon S-IgA reactivity. However, as previously discussed (Chapter 4), the Synwork battery was designed as a compromise between full-blown simulators and basic tasks and is therefore the performance measure of choice in research where simulation of a working environment is required. The external validity of the Synwork battery can also be applied to the current findings. That is, like other stressor, the current stressor elicited up-regulation of S-IgA. However, the Synwork battery has greater external validity than previously used stressors, and as such, the observed S-IgA reactivity may be analogous with a variety of tasks in everyday life.

5.4.3 S-IgA Reactivity and Health Status

Potential relationships were observed between S-IgA reactivity and frequencies of previous health complaints. It was hypothesised that S-IgA reactivity to the stressor would be reduced in those individuals with poorer retrospective health. Mean S-IgA reactivity to the stressor was assessed in relation to the scores for each of the previously identified MHC clusters. A mixed pattern of relationships was observed, however, none of the relationships were statistically significant, moreover, using the r^2 values, none of the relationships could be viewed as salient in any one particular direction.

Previous research concerning health status and S-IgA has assessed symptoms following the stressor. In the main, increases in symptoms are observed following down-regulation of S-IgA, however, it should be noted that these relationships are most salient in vulnerable groups (e.g., over-trained athletes), or following a chronic stressor (e.g., long periods of examination stress). The current study assessed the potential mediating role of retrospective health upon S-IgA reactivity. That is, do people who have generally poorer health have a propensity to demonstrate reduced S-IgA reactivity to stress? It was therefore assumed that those individuals with poorer health (i.e., those individuals with greater scores for each of the MHC clusters) would demonstrate reduced S-IgA reactivity. However, the absence of negative (or any salient) relationships between S-IgA reactivity or any of the identified MHC clusters suggests that retrospective health has no mediating effects on S-IgA reactivity to acute stress.

5.4.4 S-IgA Reactivity and Perceived Workload

It was hypothesised that perceptions of workload would be negatively associated to S-IgA reactivity. That is, perceived workload scores reflect how demanding volunteers found the stressor task. It was assumed that those individuals who reported the greatest workload scores would demonstrate reduced S-IgA reactivity to the stressor. That is, the greatest S-IgA reactivity would be observed in those volunteers who found the task arousing but not stressful. In contrast, volunteers who reported high workload demands from the stressor would demonstrate reduced reactivity, as they perceived the task to be more stressful than arousing.

There was no consistency in the direction of observed relationships between S-IgA reactivity and facets of perceived workload. That is, the facets of; mental, physical and temporal demand, effort and frustration all represent how demanding the task is. If an individual found the task generally demanding (and therefore stressful), the

relationships between S-IgA reactivity and the aforementioned facets would all be negative. That is, the more demanding the perceptions of the task, the lower the S-IgA reactivity. In contrast, the facet of performance is more positive, i.e., if individuals found the task less demanding they would achieve a greater score and therefore report greater perceptions of performance. As such, there should be a bi-polar relationship between the facet of performance and the remaining facets. This was not the case in the current data where a mixed pattern of relationships between S-IgA reactivity and perceived workload was observed. However, the relationship between S-IgA reactivity and the facet of temporal demand did conform to hypothesis. That is, although not statistically significant, the observed relationship was considerably more salient and suggested a trend for increased perceptions of temporal demand in those individuals with the lowest S-IgA reactivity.

Although this relationship conforms to the hypothesised direction of association, in isolation it is of very little statistical or theoretical interest. That is, if the hypothesised relationships were to be fully supported, the relationship would be observed in all other facets of perceived workload.

5.4.5 Increasers and Decreasers

A previously unreported phenomena was identified in the preliminary analyses. That is, despite an overall increase in S-IgA concentration immediately following acute stress, approximately one quarter of the sample population demonstrated post-stress down-regulation of S-IgA. Given the overall significant post-stress increase in S-IgA, attempts were made to identify factors which may differentiate between these individuals (Decreasers) and the majority of the sample (Increasers).

Before the differences in reactivity were explored in relation to possible influential factors, it was acknowledged that those volunteers who demonstrated S-IgA down-regulation had higher pre-stress S-IgA concentrations than did those who demonstrated up-regulation. As discussed in the results section, it could be argued that the observed up and down-regulation reflects a regression to the mean. Regression to the mean is a statistical artefact most appropriately applied to a test re-test design. Individuals who demonstrate extreme scores at the test session, and who may be selected on the basis of these scores, usually produce scores more analogous to the population mean at the re-test session.

In this instance, Increasers demonstrate lower pre-test S-IgA concentrations and demonstrate subsequent up-regulation. Conversely, Decreasers demonstrate higher pre-stress S-IgA and subsequent down-regulation following the stressor. Strong evidence for a regression to the mean would be provided if further phenomena were present in the data, i.e., if the standard deviations for the post-stress data were considerably smaller than those observed in the pre-test data. That is, extreme scores could be responsible for shifting the mean either higher (Decreasers) or lower (Increasers). If regression to the mean were apparent, the variation of the post-stress measure would be considerably lower, as more volunteers produce scores closer to the mean. Pre and post stress standard deviations for both the Increasers and the Decreasers are analogous to those produced in the sample as a whole. Further, while there is a reduction in variation from pre-stress to post-stress in Increasers, this reduction is very small. In contrast, a very small increase in variance from pre-to post-stress in Decreasers was apparent. This suggests that, whilst the distinction between Increasers and Decreasers is by its very nature, based upon extreme measures, the variation around these measures is fairly stable. As such, it can be argued that factors other than a regression to the mean are

responsible for the observed up and down-regulation of S-IgA post-stress. These factors are discussed in more detail in the following sections.

5.4.5.1 Increasers and Decreasers, and Health Status

Differences between Increasers and Decreasers regarding health status (MHC cluster scores) were explored. Contrary to prior hypothesis there were little or no relationships between S-IgA reactivity and frequencies of minor health complaints in the sample as a whole. However, preliminary analyses revealed mixed results regarding the previous health status of Increasers and Decreasers. The original hypothesis suggested that S-IgA reactivity would be reduced in those volunteers with the greatest frequencies of health complaints. However, the magnitude, and moreover the direction of post-stress S-IgA reactivity was used to classify volunteers as either Increasers or Decreasers. As such, it was inappropriate to use S-IgA reactivity as a dependent variable in subsequent analyses. Relationships between pre- and post-stress S-IgA concentrations and MHC scores were therefore assessed individually for the sub-samples of Increasers and Decreasers. A modified derivative hypothesis could then be assessed, that is, that S-IgA would be lower in individuals with greater frequencies of health complaints.

Associations between pre and post S-IgA concentrations and previous episodes of minor health complaints were then explored individually for both Increasers and Decreasers. In general there were very small positive relationships between MHC cluster scores and pre and post S-IgA concentrations in Increasers. In contrast, stronger negative correlations were observed between MHC cluster scores and pre and post S-IgA concentrations in Decreasers. These relationships are mostly clearly illustrated in the cluster of immune challenge complaints. The relationship demonstrates that within the Decreasers group higher S-IgA concentrations are associated with a reduced frequency of immune challenge complaints. That is, those individuals who have the highest S-IgA

concentrations (pre and post) have reported the fewest immune challenge complaints. In contrast, those individuals who demonstrated the lowest S-IgA concentrations reported the greatest frequencies of immune challenge complaints. This pattern of association was also evident in several other MHC clusters, in particular, those of total ill-health, psychological and gastric complaints.

It is therefore apparent that Increasers and Decreasers have different patterns of data. Firstly, as their descriptors indicate, the two groups demonstrate different S-IgA stress responses. Secondly, Decreasers demonstrate a distinct pattern of association between S-IgA concentrations (pre and post) and immune challenge scores, that support original hypotheses. That is, those Decreasers who demonstrate the lowest S-IgA are those individuals who have reported the most immune challenge complaints. It could be argued that the distinction between Increasers and Decreasers with regard to health status could be more apparent. That is, if health status moderates S-IgA concentrations / reactivity, and can distinguish between Increasers and Decreasers, the two groups should demonstrate contrasting patterns of association (i.e., Increasers = positive relationships, Decreasers = negative relationships). However, such a discrepancy would be contrary to the original hypothesis. That is, it was hypothesised that in general, greater frequencies of health complaints would be associated with lower S-IgA reactivity / concentrations. Positive relationships between S-IgA and health status would therefore contradict this prediction.

It should also be noted that previously observed relationships between S-IgA and prospective health status following a stressor are most salient in vulnerable groups (e.g., over-trained individuals, IgA deficient individuals, or in individuals following a chronic stressor). It is therefore suggested that the Decreasers group are analogous with the

vulnerable samples employed in previous research. That is, it is only in this identified group, where the proposed negative relationship between S-IgA and health is apparent.

5.4.5.2 Increasers and Decreasers, and Perceived Workload

As with health status, associations were also assessed between S-IgA concentrations and perceived workload facets for both Increasers and Decreasers individually. As with the analyses concerning health status, the distinction between the groups is based upon direction of reactivity. As such, the analyses were conducted between pre and post-stress S-IgA levels and facets of perceived workload for Increasers and Decreasers individually.

It was hypothesised that some volunteers would perceive the task to be arousing, and would subsequently report lower levels of perceived demand. These individuals would demonstrate higher S-IgA reactivity. In contrast, some volunteers would find the task more stressful and would report higher levels of perceived workload and demonstrate lower levels of S-IgA accordingly. Due to the inappropriateness of using S-IgA reactivity as a dependent variable, a derivative of the original hypothesis was applied to these analyses. That is, it was suggested that volunteers who perceived the task to be more stressful (greater reports of perceived workload) would demonstrate lower pre and post-stress S-IgA concentrations.

There were no significant relationships observed in the Increasers, although with the exception of perceived performance all associations were small and positive in direction. That is, in Increasers, higher reports of mental demand were associated with higher pre and post S-IgA concentration. In contrast, significant negative relationships were observed in Decreasers between S-IgA concentrations and the facets of temporal demand and effort. That is, those Decreasers with the highest S-IgA reported the least

temporal demand and perceived effort, and in contrast, those with the lowest S-IgA reported the greatest workload demands.

As with the relationships observed with Increasers and Decreasers with regards to health status, differing patterns of relationships were observed with regards to perceived workload. Again, little or no relationships were observed in Increasers, however, in support of the derivative hypothesis, significant negative relationships were observed between S-IgA concentrations and facets of perceived workload. That is, Decreasers with lower S-IgA reported the greatest perceived workload following the stressor.

5.4.5.3 Summary and Discussion

It is argued that the observed differences in direction of S-IgA reactivity are not a statistical artefact, but are in fact moderated by other factors. Although the hypothesised relationships were not apparent in the sample as a whole, negative associations between health status and S-IgA were observed in the identified group of Decreasers. That is, despite demonstrating higher pre-stress S-IgA than the majority of the sample, Decreasers with higher S-IgA reported fewer health complaints.

Similarly, different patterns of association were also observed between Increasers and Decreasers with regard to S-IgA and perceived workload. That is, whilst there was little or no relationship observed in Increasers, Decreasers demonstrated significant negative association on facets of temporal demand and effort. These negative association reveal that within the Decreasers, individuals who perceived the greatest effort and temporal demand had the lowest pre and post-stress S-IgA concentrations.

Given the observed elevation is S-IgA pre-stress, it is plausible to suggest the role of an S-IgA reserve as a mechanism responsible for post-stress reductions. That is, owing to previous infection, Decreasers have higher levels of S-IgA. Following acute stress, healthy, or previously healthy individuals would subsequently demonstrate immune activation, and therefore an increase in S-IgA to counter-act the impact of the stressor. However, in Decreasers, their S-IgA is already at a higher level, and as such, they have a diminished reserve, and a modified immune response to acute stress. The proposed existence of this model will be discussed in more detail both later in this chapter, and with regards to data in subsequent studies.

These analyses revealed a previously unidentified phenomena, and through analyses regarding health status and perceived workload, it is argued that specific factors may, in part moderate the differences in S-IgA. However, as discussed in the results section, these result do not utilise the MHCQ classification method. Although the use of this method will result in the loss of distinction between Increasers and Decreasers, similar patterns should still be evident. That is, the original hypotheses can be evaluated, i.e., that S-IgA reactivity will be reduced in volunteers with greater frequencies of ill-health. Further, the same MHC classification can be applied to perceived workload, to assess whether their ill-health can moderate perceptions of stress. Moreover, the combination of these factors as moderators in S-IgA reactivity to acute stress can be appropriately evaluated.

5.4.6 Classification of MHCs and S-IgA reactivity

In support of the original hypothesis regarding health status and S-IgA reactivity, there was a trend for volunteers classified as being in poor health to demonstrate reduced S-IgA reactivity to acute stress when compared to those in good health. This trend was apparent for the clusters of; total ill-health, stress-related, indicators of ill-health,

psychological, immune challenge and gastric complaints. Moreover, although no significant differences were observed, these differences were most pronounced for the clusters of total ill-health, immune challenge and gastric complaints.

The previously discussed model of S-IgA reserve or capacity could also account for these differences in S-IgA reactivity. That is, those volunteers in poor health have a reduced S-IgA capacity, and as such, demonstrate reduced S-IgA reactivity to acute stress. In contrast, volunteers in good health have an enhanced S-IgA capacity, and as such, demonstrate normal S-IgA reactivity to acute stress. When discussed in relation to previous data concerning health status following a stressor, a cyclical pattern of S-IgA reactivity and health status could be apparent. That is, individuals in good health have a good S-IgA reserve and demonstrate post-stress up-regulation. This up-regulation is important to ensure that the individual is not more susceptible to infections following the stressor. As such, these individuals stay in good health. A different process would be observed in individuals in poor health. Such individuals have a reduced S-IgA capacity and subsequently demonstrate reduced post-stress S-IgA reactivity. This diminished reactivity increases susceptibility to post-stress infections. As such, these individuals stay in poor health. This process is cyclical, as health status moderates reactivity to a stressor (be it laboratory based, or any stressor encountered in every day life) and the magnitude or direction of this reactivity will subsequently moderate their health status.

Although this is only a preliminary model, the current data, in conjunction with previous data regarding prospective health status support the concept. However, no suggestions as to passive underlying mechanisms driving this process are suggested at this point, as it would be premature and speculative in the absence of any other biological data. Further, it is difficult to suggest a causal mechanism, as given the

cyclical nature of the process there is no appropriate point to enter the cycle. In basic terms it could be that those individuals with cyclical poor health, have a reduced immuno-capacity as their resources are in the main, allocated to defence against infection. Subsequently, such individuals are unable to allocate the required resources to the stress response.

However, this is an extremely simplistic method of explanation, which is uncorroborated by knowledge of the immune response to antigens. That is, an antigenic challenge creates an immune response, in this case S-IgA. In addition, memory cells are also produced to aid a rapid response following future challenge by a specific antigen (Kuby, 1997). Subsequently, following challenge, S-IgA, as a consequence of an enhanced half-life, persists in the common mucosa. It is this S-IgA that is ordinarily observed following acute stress, i.e., a "wash-out" (Carpenter, *et al.*, 1998), of previously synthesised specific IgA.

This process is obviously counter to the existence of an S-IgA-reserve, however, as previously discussed, the current model is only in its preliminary stages, and it is acknowledged that other factors may also moderate the process. It should also be remembered that S-IgA responses to antigen describe responses to antigen only, and unlike the S-IgA-capacity model, do not encompass information regarding stressor reactivity. It is therefore plausible, that the health status of the individual can moderate the allocation of immune resources following a stressor. This is obviously not a conscious process and would also be influenced by other factors such as personality, mood and coping styles of the individual. However, regardless of the mechanism, the current data indicate a trend for individuals in poor health to demonstrate reduced S-IgA reactivity to acute stress, which may be explained through the use of an S-IgA-capacity model.

Three MHC clusters did not follow this direction of reactivity. That is, volunteers classified as in poor health with regards to frequencies of atopy, urinary tract and microflora infections demonstrated greater S-IgA reactivity than did those classified in good health. This obviously complicates the model further. Atopy is an IgE driven response, that is, the comprising items are all allergic-type responses, which would elicit an IgE response (*c.f.* Chapter 2). IgE activation is mediated by Th2 responses. Shifts to Th2 activation, from the more standard Th1 / IgG response, promote IgE synthesis, the production of histamine (stimulated by IgE), a host of other pharmacological mediators, and the attraction of eosinophils. This response is therefore dominant in atopic individuals (Evans, Hucklebridge & Clow, 2000) and is also responsible for the enhanced production of S-IgA . Although the Th1 – Th2 is not strictly bi-polar, it can be viewed as a balance. Using the S-IgA capacity model, it could be hypothesised that atopic individuals would therefore have a greater reserve of IgA available at times of acute stress.

However, acute stress elicits a shift to Th1 and subsequent activation of mucosal activation. This notion leads onto potential individual differences in perceptions of acute stress. That is, in the main, acute stress activates Th1, whereas more chronic stress elicits a shift to Th2 (Evans, *et al.*, 2000). Atopic individuals may therefore interpret the stressor as more stressful, or indeed, may interpret many daily stressors as being more stressful than arousing. As such, these individuals are dominated by Th2 determined responses. However, the initial interpretation of the stressor elicits Th1 stimulation and subsequent mucosal activity (S-IgA), but to a lesser extent than in normal individuals, as the balance is tipped in favour of Th2. This concept, although complex, may explain the increased S-IgA reactivity in volunteers classified as in poor health with regards to atopic complaints. However, the notion of perceptions of stress will be addressed further in the next section and subsequent chapters.

More intriguing is the reverse reactivity in clusters of urinary tract and microflora complaints. S-IgA dominates in the urino-genital and gastric tracts. Further, the comprising complaints in these clusters also take primary action in one or both of these two tracts. As such, it would be expected that individuals classified as in poor health with regards to frequencies of these complaints would demonstrate reduced S-IgA reactivity in the same way as demonstrated in poor health individuals on clusters of total ill-health *etc.* The reversed pattern of reactivity in these individuals could also be attributed to the interpretation of the stressor. That is, individuals classified as in poor health for these clusters may be predisposed to interpret stressors as more stressful. As with the atopic individuals, the stressor elicits Th1 activation, however, in the main, such individuals are dominated by Th2, resulting in the continual production of IgA which replenishes their IgA store. This notion could also be levied at their response in everyday life. As such, the interpretation of the stressor is likely to be influenced by personality as well as their transient mood at the time of the stressor. Perceptions of stress will be discussed in the following section, and other contributory factors will be introduced in subsequent chapters.

5.4.7 Classification of MHCs and Perceived Workload

It was hypothesised that reduced S-IgA reactivity would be observed in volunteers who reported the greatest perceived workload demands following the stressor. In all MHC clusters there was tendency for volunteers classified as in poor health to perceive the stressor as more demanding. In particular, volunteers in poor health reported the stressor to be more frustrating than did volunteers in good health. Although using the MHC clusters as a method of classification does not allow for the direct assessment of S-IgA reactivity and perceived workload, the method can be applied to both S-IgA and perceived workload independently. For example, X individual is classified as in poor

health with regards to frequencies of total ill-health complaints, they demonstrate reduced S-IgA reactivity and perceive the stressor to be more frustrating.

There is therefore a general trend for individuals in poor health to perceive greater demand from the stressor. This concept has been demonstrated previously (Wetherell, 2000) with regards to perceived workload whilst infected with a common cold. That is, infected volunteers reported significantly greater workload demands (on all facets) when compared to both their own healthy session and healthy controls. Anecdotal evidence suggests that at times of illness, an individual may not perform as well on a task and as a result they may feel that the task is in someway more demanding. However, the current data suggest that perceived workload, and in particular perceived frustration is greater in those individuals with poor retrospective health. Further, for one reason or another, these same individuals demonstrate reduced S-IgA reactivity (with the exception of atopy, urinary-tract and microflora complaints).

Perceived workload demands can therefore be added to the proposed model of immuno-capacity as a potential moderating factor. However, it is acknowledged that the reduced S-IgA reactivity could be a consequence of; previous health status, perceptions of workload or a combination of both factors. Further, as with the proposed cyclical nature of health status and S-IgA reactivity, the influence of perceived workload could be more complex. That is, the relationships between the factors are not linear, moreover, they reflect a complex network of interaction, where the gross effect is more important than the constituent relationships, e.g., poor health may predispose greater workload demands, or vice-versa, similarly, reduced S-IgA reactivity may occur as a result of health status, perceptions of the task or a combination of factors. Given the likelihood of these complex relationships, other, as yet unidentified factors may also influence the network.

In the previous section, perceived workload demands were suggested as moderating a shift towards either Th1 or Th2 driven responses, and possibly responsible for the observed increases in S-IgA reactivity in volunteers in poor health for clusters of atopy, urinary-tract and microflora complaints. It was suggested that those individuals who perceived the task as more stressful may have experienced a subsequent shift towards Th2 immunity and subsequent increased production of S-IgA. There were no notable differences in the perceived workload demands of individuals in these clusters compared with the remaining MHC clusters. As such, no support can be provided for the notion that volunteers classified as in poor health for the clusters of atopy, urinary-tract and microflora complaints experience greater stress and therefore demonstrate a shift towards Th2 immunity. However, it is likely that this shift is not a temporary one, and as such, reflects a general bias in these individuals. Such a bias could therefore occur as a result of their general perceptions of stress. This process will be explored in subsequent chapters.

5.4.8 Summary, Conclusions and Recommendations

As predicted, the current stressor elicited a significant mean increase in S-IgA concentrations. Further, the current stressor has high external validity, that is, it is a simulation of any environment involving attendance and response to more than one simultaneous stimulus. The observed increase may therefore be analogous with a wider variety of stressor encountered in everyday life. Contrary to the original hypotheses, no differences in S-IgA reactivity were observed with regards to previous episodes of minor health complaints or perceived workload demands.

However, the current stressor has elicited a previously unidentified phenomena – that of down-regulation following an acute stressor. When assessed independently of the majority of the sample, those demonstrating down-regulation provided support for

derivatives of the original hypotheses. That is, within this sub-sample, there was a trend for volunteers classified as in good health to demonstrate higher S-IgA, conversely, volunteers in poor health demonstrated lower S-IgA before and after the stressor. Further, although this method of classification did not allow for direct assessment of S-IgA and perceived workload, there was a trend for individuals in poor health (with lower S-IgA) to report greater perceived workload demands following the task.

The current data have given rise to a provisional model concerning S-IgA capacity. That is, a model encompassing the relationships between health status, perceived workload and S-IgA reactivity. It is acknowledged that the model is very basic, however, it has been developed in an attempt to interpret and combine data from the current and previous literature. Further, the model does explain the reduced S-IgA observed in those individuals with poorer health, and suggests the cyclical nature of S-IgA reactivity and health status. Figure 5.11 presents the model of S-IgA capacity, which can be used to explain the current data, and helps to serve as a graphical explanation of the findings of the current study.

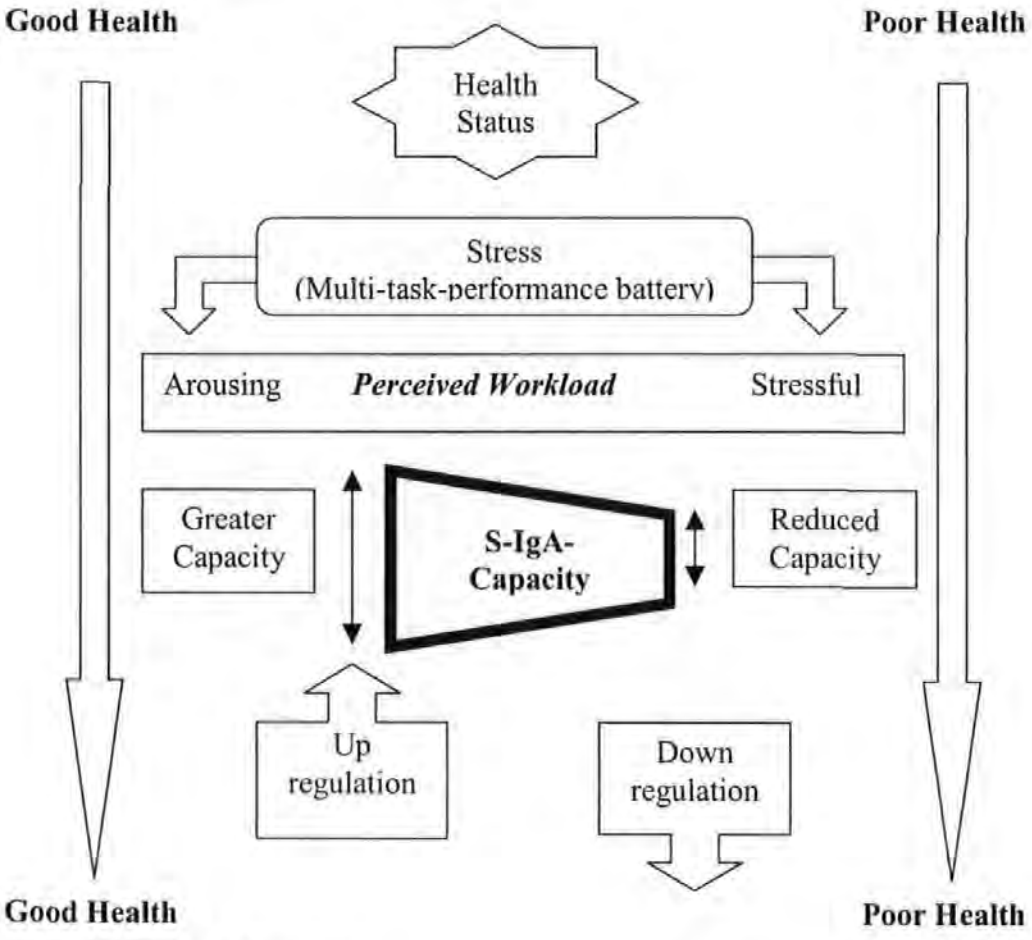


Figure 5.11 S-IgA Capacity

In summary, it is suggested that the use of the Synwork battery as a stressor is sensitive enough to identify individual differences in S-IgA. That is, the demands of the task are perceived differently by different individuals, and as such, different patterns of relationships between health, perceptions of workload and S-IgA reactivity can be observed. Given this sensitivity and its external validity the current stressor will be utilised in subsequent studies.

The data regarding perceived workload demands suggest that other factors could moderate health status and S-IgA reactivity, but moreover, the relationships between the two. As such, future studies will assess other potential moderating factors, in particular, mood and personality in relation to health status, perceived workload and S-IgA reactivity to acute stress.

6. Study Two

6.0 Chapter Overview

This chapter reports the findings from a study designed to investigate the effects of the stress induced by the Synwork battery in relation to previous episodes of minor health complaints and personality characteristics on two separate sessions. The study had several main aims.

Reinforcement of Study One

S-IgA responses to acute stress were measured over two consecutive occasions to assess whether individuals demonstrate similar patterns of immune reactivity to the same stressor on different occasions. Further, as in study one, immune reactivity was assessed in relation to previous episodes of minor health complaints and perceived workload.

Saliva Flow Rates

The effects of the stressor upon saliva flow rates were assessed in order that observed S-IgA reactivity could be attributed to the manipulated stressor, not changes in saliva secretion *per se*. The findings concerning saliva flow rates influenced the role of S-IgA in subsequent analyses. That is, the stressor elicited increases in saliva volume. Increases in S-IgA concentration could therefore be attributed to the stressor not an artefact of differential dilution. However, it was acknowledged that both pre and post-stress S-IgA measurements could be influenced by saliva flow. As such, S-IgA secretion rates were adopted as the preferred measure of S-IgA in all subsequent analyses. Analyses regarding health status and perceived workload in relation to S-IgA secretion rates were therefore assessed.

Personality and Mood

Immune reactivity was measured in relation to personality and mood characteristics to assess whether certain personality / mood types demonstrate different patterns of immune reactivity. Similarly, health status and perceptions of workload were assessed in relation to personality and mood to assess whether certain mood / personality types predispose individuals to experience specific symptoms or perceive the stressor in different ways.

Familiarity to the Stressor

The influence of familiarity to the stressor (Willemson *et al*, 2000) was assessed across the two sessions. That is, Willemson *et al.*, would suggest that less immune reactivity is demonstrated to a familiar stressor. This concept was assessed through comparisons of perceived workload demand across both sessions.

The study also allowed for further analyses of the proposed S-IgA reserve model. That is, the second stressor (day two) should add further stress to the individual and therefore differences in reactivity to the stressor with regards to other factors should be more apparent.

A complex pattern of results were observed. Firstly, with regards to S-IgA concentrations, the current results were analogous with those observed in study one. However, following the observation that saliva volume could mask the true influence of the stressor upon S-IgA, analyses were conducted using S-IgA secretion rates. These analyses also yielded results analogous with those observed in study one. Although the saliva volume data indicated that S-IgA change could be attributed to the stressor, not changes in saliva volume, a decision was made to control for the effects of saliva

volume at all stages of analysis, as such, all remaining analysis incorporated this method of S-IgA assessment.

With regards to personality, volunteers in poor health demonstrated greater negative affect, neuroticism and openness, whilst there was a trend for volunteers in good health to be more agreeable. With regards to states / traits and S-IgA reactivity, the most salient patterns were observed with regards to negative affect and neuroticism. Volunteers high in negative affect demonstrated greater S-IgA reactivity, while volunteers with high neuroticism demonstrated reduced reactivity. Despite the expected similarity between patterns of reactivity in neurotic volunteers and volunteers high in negative affect, these differences are explained in terms of over-attendance to stimuli, and the chronic nature of neuroticism (a trait measure). That is, both states / traits are associated with over-attendance to stimuli, resulting in greater S-IgA reactivity. However, owing to the chronic nature of neuroticism, the reduced reactivity can be attributed to a diminished reserve brought about by frequent over-attendance and S-IgA reactivity.

Finally, further support was provided for the S-IgA reserve model. That is, although diminished reactivity in all volunteers on day two could be attributed to familiarity to the stressor, no differences were observed with regards to perceptions of workload. It was therefore suggested that day two reductions could be attributed to a diminished reserve, which is more pronounced in volunteers in poor health.

All contributory factors are viewed as a complex network, combinations of which can predispose individuals to reduced S-IgA reactivity to acute stress, perhaps through a diminished reserve. The results are therefore discussed in relation to the S-IgA reserve model. The shortcomings of the current study, in particular, the time delay between the

stressor being too small to efficiently test the S-IgA reserve, are discussed in relation to future recommendations.

6.1 Introduction

6.1.1 Reinforcement of Study One (stability of immune reactivity)

Study one assessed the S-IgA stress response over one session. The current study assessed S-IgA in response to two repeated exposures to the same stressor on consecutive days in order to assess whether people demonstrate the same S-IgA stress response on two separate occasions. Similar to the first study, stress responses over the two sessions were then assessed in relation to previous episodes of minor health complaints and perceived workload. Study one suggested the possible existence of an S-IgA reserve. That is, there was trend for volunteers in poor health to perceive greater demands from the stressor. Furthermore, these volunteers demonstrated reduced S-IgA reactivity to acute stress. This concept was assessed further in the current study. That is, if some sort of S-IgA reserve is in operation, these patterns should be apparent in both sessions. Further, given the nature of a reserve, the patterns should be more salient on following exposure to the second stressor.

6.1.2 Saliva Flow Rates and S-IgA

The previous study demonstrated significant increases in S-IgA concentrations following five minutes on the Synwork battery. The use of S-IgA concentrations as an effective measure of immune functioning has come under much debate. The focus of the debate is based upon the influence of saliva flow upon subsequent measures of S-IgA concentration, i.e., the risk of the dilution ratio between S-IgA concentration and saliva volume giving rise to artificial increases or decreases in S-IgA concentration.

It has been suggested that a negative correlation exists between S-IgA concentration and saliva volume (Evans *et al.*, 1993, Bristow *et al.*, 1997). That is, observed S-IgA concentrations are influenced by the amount of saliva secreted. When attempting to observe S-IgA change in response to a stressor, it is therefore difficult to determine whether changes in S-IgA concentration occur as a result of the manipulated stressor, or whether they exist as an artefact of the amount of saliva produced. For example, increases in S-IgA may be observed following exposure to a stressor. However, if the same stressor also decreases the secretion of saliva, then S-IgA increases cannot be solely attributed to the stressor, but also to the reduction in saliva volume and the apparent inflation of S-IgA within the sample.

As a result of this negative relationship, Stone *et al.*, (1987), suggest that S-IgA concentration in saliva may not be an efficient immune parameter. However, although stress is often associated with a reduction in saliva flow, not all acute stressors elicit this reduction, and if a reduction is observed, the effect on S-IgA concentration is minimal, e.g., Jemmott and Magloire (1988) observed a non-significant negative relationships (ranging from r = -.05 to -.22). Further, McClelland and Kirshnit (1988), observed no effect of saliva flow on S-IgA concentration following motivational arousal. Moreover, Jemmott and McClelland (1989), suggest that the influence of saliva flow upon S-IgA concentrations is most apparent in studies which measure stimulated saliva, where saliva is stimulated over and above those levels produced in normal circumstances.

Saliva samples in study one were used for the measurement of S-IgA concentrations only. Although, volunteers were asked to fill a vial with saliva up to a specific point (1ml), no time period was stipulated. As such, there are no data regarding saliva flow for study one. The evidence regarding the influence of saliva flow on S-IgA concentration rates appears to be contradictory. However, the specific relationship

between S-IgA concentration and saliva volume is very much dependent upon the effects of the manipulated stressor upon saliva volume. Although it is clear that a range of psychosocial variables can alter saliva flow, and therefore influence measures of S-IgA, it is likely that different stressors will exert differential effects upon saliva production and secretion. In the absence of saliva flow data in study one, it is impossible to detect whether IgA changes in response to the stressor can be attributed to the direct action of the stressor, or to a reduction in saliva flow. Given the observed negative relationships, and the apparent variations in response to different stressors, the saliva flow response to the Synwork battery must be measured in an attempt to assess the relative influences of the stressor and saliva flow rates upon S-IgA concentrations in previous, current and future studies.

6.1.3 Personality, Mood and S-IgA

Relatively few studies have assessed the moderating role of personality traits and immune stress responses (see Chapter 2). However, specific characteristics have been suggested to moderate immune reactivity in response to acute stress. In particular, Ohira *et al.*, (1999) demonstrated that Type A individuals (characterised by; an intense drive to succeed, hostility and competitiveness) had high levels of S-IgA prior to stress exposure than did Type B individuals. However, whereas Type B individuals demonstrated the normal pattern of up-regulation following the stressor, Type A individuals varied very little from pre to post-stress.

The authors suggest that pre-test elevation of S-IgA could be attributed to the Type A lifestyle. That is, such individuals constantly perceive stress in the environment, and as such continually stimulate their immune system, subsequently, they have elevated levels of S-IgA. Further, the absence of any immune reactivity in these individuals could be explained in terms of the S-IgA reserve model suggested in study two. That is, because

S-IgA levels are consistently high as a result of chronic lifestyle stimulation, the S-IgA reserve is diminished. As such, individuals are unable to produce a supply of S-IgA to protect the mucosa at times of acute stress.

In contrast, Coons *et al.*, (1995) demonstrated higher S-IgA levels following a musical examination in individuals high in confidence and low in denial. Conversely, lower post-stress S-IgA levels were observed in individuals who were low in confidence and high in denial. The authors relate their finding to the toughness formulation model (Dienstbier, 1989), which suggests that individuals who have adapted to coping with stress, through psychological coping skills, develop a "toughness", which corresponds with positive performance even in complex tasks and immune enhancement.

More research has been conducted involving the effects of mood upon S-IgA and S-IgA reactivity (*c.f.* Chapter 2). Mixed results have been observed in the relationships between S-IgA and mood. For example, Evans *et al.* (1993) observed higher (although not significantly higher) S-IgA in individuals reporting either high positive or low negative mood. However, with-in subject comparisons demonstrated that negative mood was significantly associated with S-IgA secretion rates. The authors suggest that in the short-term, negative mood (perhaps elicited by undesirable events) are associated with a rise in S-IgA. However, in the long term, evidence suggests that high negativity / low positivity is related to lower S-IgA.

It is therefore apparent that different personality traits and mood states can moderate immune reactivity. In particular, traits or states could influence S-IgA reactivity owing to the perceptions of events prior to the experimental manipulation. That is, certain personality / mood characteristics may predispose individuals to demonstrate specific patterns of S-IgA reactivity. For example, if an individual possesses a trait where they

over-perceive stimuli in the environment, they may already be aroused before entering the lab, and therefore demonstrate reduced S-IgA reactivity owing to the limitations of their S-IgA reserve. The current study therefore utilised the NEO-5 Factor Inventory and the PANAS (see Chapter 4) in order to assess both the individual and interactive relationships between personality, mood and health status, and the influence of a combination of these factors upon S-IgA reactivity.

6.1.4 Familiarity to the Stressor

Willemson *et al.*, (2000) suggest that familiar stressor induce less immune reactivity than novel stimuli. Although no physiological measures of arousal or stress which may reflect familiarity with the stimulus can be taken in the present study, comparisons can be made between data regarding the perceived workload demands of the stressors in both sessions to assess whether familiarity reduces perceived workload demands. That is, at the second session the stressor is more familiar, and as such, reductions in perceived workload demands would be expected. The design of the study therefore allows for the assessment of familiarity and the S-IgA reserve model as explanations of diminished S-IgA reactivity following subsequent stressor.

6.1.5 Aims and Hypotheses

Reinforcement of Study One

Aim: To attempt to reinforce the findings of Study One, demonstrating stable patterns of immune reactivity across the two sessions and similar relationships between immune reactivity and previous episodes of minor health complaints and perceived workload demands. Further, assessment across the two sessions will allow further evaluation of the S-IgA reserve model. That is, the second stressor will put greater stress on the individual and therefore deplete their reserve.

Hypothesis 1: That the patterns of immune reactivity will be similar across the two sessions, i.e., if reduced reactivity or down regulation is observed in the first session, a further decrease will be apparent in session two.

Hypothesis 2: That the perceived workload demands will be greater in volunteers classified as being in poor health.

Hypothesis 3: That volunteers classified as in poor health will demonstrate reduced S-IgA reactivity in both sessions. Reactivity will be most reduced in the second session as a result of a diminished S-IgA reserve.

Saliva Flow Rates

Aim: To assess the effects of the Synwork battery on saliva flow rates in order that changes in S-IgA reactivity can be attributed to manipulations of the stressor and not merely changes in saliva volume. If the stressor is observed to influence saliva flow rates, this influence will be taken into consideration in subsequent analyses through the use of S-IgA secretion rates.

Personality & Mood Characteristics

Aim: To assess whether specific personality and mood characteristics predispose volunteers to perceive stimuli in specific ways and therefore mediate their S-IgA reactivity to the manipulated stressor.

Hypothesis 4: That the volunteers classified as high for states / traits where over-perception to stimuli is likely (e.g., negative affect, neuroticism and conscientiousness) will demonstrate reduced S-IgA reactivity as they will have a diminished S-IgA reserve owing to continual arousal and depletion of S-IgA.

Familiarity to Stressor

Aim: To assess the changes in perceived workload demands in response to the stressor across both sessions in relation to changes in S-IgA reactivity. If the stressor is more familiar, reduced S-IgA reactivity should be demonstrated, however, study one demonstrated greater S-IgA reactivity in those who reported lower workload demands.

Hypothesis 5: That the perceived workload demands will be reduced in the second session when the stressor is more familiar.

6.1.6 Summary

As discussed this study has many purposes. Primarily, attempts were made to replicate the previous study and test further the potential for the existence of an 'S-IgA-reserve' in relation to the relationships between health status and perceived workload. In addition, the effects of personality and mood will be added to the model, in an attempt to monitor the state of volunteers prior to experimental manipulation On a methodological note, this study also assessed the effects of the stressor upon saliva flow rates in order that observed S-IgA reactivity can be attributed to the stressor, or other factors, not to increases or decreases in saliva volume alone.

6.2 Methods

6.2.1 Sample

Fifty volunteers were recruited through advertisements across the university campus. As such the majority of volunteers were related to the university in some way, i.e., students and university staff. Interested volunteers were asked to contact the experimenter to arrange convenient times for testing. All volunteers were tested individually during the months of August September and October, 2000.

6.2.2 Materials

6.2.2.1 Questionnaire Methods

Minor health complaints were assessed and classified using the MHCQ, and perceived workload assessed using the NASA-TLX Perceived Workload Questionnaire. In addition, personality characteristics were assessed using the NEO-5-Factor Inventory and mood state assessed using the PANAS (Full details can be seen in Chapter 4).

6.2.2.2 Stressor Task

The Synwork multi-tasking battery was used as the stressor task in the current study (Full details can be seen in Chapter 4)

6.2.3 Experimental Protocol

Volunteers were asked to select two experimental sessions. Each session took place at the same time of day 24 hours apart. At the first session volunteers were asked to complete the MHCQ, NEO-5 Factor Inventory and PANAS. Volunteers were then given a demonstration of the Synwork battery. Following the demonstration, volunteers provided the first saliva. Volunteers were asked to empty their mouth of saliva, before collecting saliva (without moving the tongue or jaw) saliva in the base of the mouth for a period of two minutes.), Volunteers then commenced a five minute session on the

Synwork session, Immediately following the task, volunteers provided their second saliva sample, before completing the NASA-TLX in relation to their perceived workload during the Synwork session. The second session followed the same procedure as session one with the exception of the MHCQ and the NEO-5 Factor Inventory. Prior to commencing the second Synwork session, volunteers were informed of their previous session score and told to try and score a greater score during the second session. This information was passed on in an attempt to maintain a similar level of arousal in the second session. That is, the task would be more familiar at the second session, as a result of previous exposure. A specific target would therefore increase arousal and perceived stress elicited by the task.

6.2.4 Treatment of Results

6.2.4.1 Classification of Data

Volunteers were classified as being in either good or poor health for each of the MHC clusters (Chapter 3), and either low or high for both PA and NA, and NEO-FFI traits.

6.2.4.2 Statistics

Several of the S-IgA samples in the current study demonstrated significant deviations from the normal distribution (S-IgA distributions are presented in Appendix D). Positively skewed distributions of raw S-IgA data are usually corrected using square root transformations (e.g., Bristow *et al.*, 1997), however, such transformations are problematic when applied to reactivity data. That is, transformed differences at the lower end of the scale are increased in relation to differences of the same magnitude higher up the scale. In this thesis, differences in S-IgA reactivity in relation to other factors are assessed using t-tests. Student's t-tests are extremely robust, and with current sample sizes, the assumption of normality can be violated without affecting the validity of the hypothesis test (Gravetter & Wallnau, 1996). All analyses were therefore performed on the raw data.

6.3 Results

6.3.1 Overview

The results section is divided into three main sections. Firstly, section 6.3.2 contains analyses regarding the demographics of the sample. Section 6.3.3 replicates the analyses conducted in study one. This section therefore comprises, S-IgA reactivity (concentrations) to the stressor on both occasions, and S-IgA reactivity and perceived workload demands in volunteers classified as in either good or poor health with regards to frequencies of health complaints for each of the identified MHC clusters.

Section 6.3.4 presents analyses regarding saliva flow rates and subsequent effects upon the accuracy and reliability of S-IgA measurements. Comparisons were made between S-IgA measurements both with and without the influence of saliva volume.

Following analyses concerning the effects of saliva volume upon S-IgA concentrations, section 6.3.5 presents analyses regarding S-IgA secretion rates. This section comprises replications of the analyses conducted on S-IgA concentration, S-IgA reactivity to the stressor, and S-IgA reactivity in volunteers classified as in either good or poor health with regards to frequencies of health complaints for each of the identified MHC clusters. In addition, this section comprises analyses regarding personality and mood characteristics. Specifically, personality and mood were assessed in relation to S-IgA reactivity, health status classifications and perceived workload demands.

6.3.2 Sample Demographics

The sample was selected from a university population, comprising both undergraduates and staff members from the University of Plymouth. Table 6.1 demonstrates that approximately equal numbers of males (n = 23) and females (n = 26) took place in the

study. Table 6.2 demonstrates that the majority of the sample were aged between 20 and 40 years of age, comprising 79.6% of the total sample.

	Frequency	Percent
Males	23	46.9
Females	26	53.1
Total	49	100.0

Table 6.1 Sex of Volunteers

	Number	Percent
< 20	3	6.1
20 - 30	21	42.9
31 - 40	18	36.7
41 - 50	3	6.1
51 - 60	2	4.1
> 61	2	4.1
Total	49	100.0

Table 6.2 Age of Volunteers

6.3.3 Reinforcement of Study One

6.3.3.1 S-IgA Reactivity (concentrations) to Acute Stress

Figure 6.1 presents the pre-post changes in S-IgA concentrations across both sessions. At session one, post-stress S-IgA concentrations (132.10 μg/ml) were not significantly greater than pre-stress levels (135.00 μg/ml). At session two post-stress S-IgA concentrations (102.68 μg/ml) were significantly lower ($t_{(48)} = 2.70$, $p < 0.01$) than pre-stress levels (117.17 μg/ml) . Pre-stress S-IgA concentrations across both sessions were significantly different ($t_{(48)} = 2.30$, $p = 0.03$). That is, pre-stress S-IgA concentrations at session one (132.10 μg/ml) were significantly greater than pre-stress levels at session two (117.17 μg/ml).

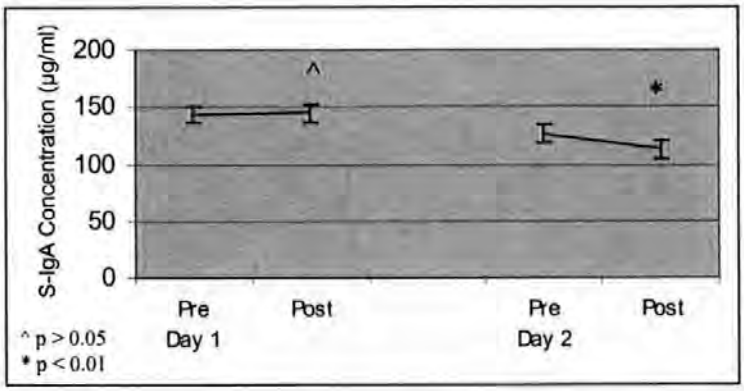


Figure 6.1 S-IgA Reactivity to Acute Stress (and S.E.M)

6.3.3.2 Health Status and S-IgA Reactivity

The relationships between S-IgA reactivity and scores for each of the MHC clusters for both day one and day two were assessed using Spearman's Rho. The correlation coefficients are presented in Table 6.3. With the exception of the relationship between S-IgA reactivity and microflora complaint scores on day one ($r_{(49)}$ .30, $p < 0.05$), and gastric complaints on day two ($r_{(49)}$ -.30, $p < 0.05$) no other significant relationships were observed. Distributions of MHC cluster scores are presented in Appendix D.

		TOTAL	STRESS	INDICATE	PSYCH	IMMUNE
S-IgA Reactivity Day 1	Correlation Coefficient	.062	.043	-.120	-.026	-.001
	Sig. (2-tailed)	.674	.768	.411	.857	.992
	N	49	49	49	49	49
S-IgA Reactivity Day 2	Correlation Coefficient	-.069	.013	-.085	-.209	-.059
	Sig. (2-tailed)	.638	.930	.563	.149	.686
	N	49	49	49	49	49

		ATOPY	GASTRIC	URINARY	FLORA	FUNGUS
S-IgA Reactivity Day 1	Correlation Coefficient	.105	.091	.099	.295	.162
	Sig. (2-tailed)	.473	.535	.498	.040	.266
	N	49	49	49	49	49
S-IgA Reactivity Day 2	Correlation Coefficient	.008	-.304	.017	.016	.042
	Sig. (2-tailed)	.957	.034	.909	.914	.773
	N	49	49	49	49	49

Table 6.3 Relationships (Spearman's Rho) Between S-IgA Reactivity and MHC Cluster Scores on Days One and Two

Using the pre-determined means, volunteers were classified as being in either good or poor health with regards to frequencies of complaints for each of the identified MHC clusters. S-IgA reactivity was subsequently compared between these groups.

Total Ill-health

The differences in S-IgA reactivity between volunteers with good and poor total ill-health on days one and two are presented in Figure 6.2. On day one there was a near significant trend for volunteers classified as in poor health (n = 22) with regards to frequencies of total ill-health complaints to demonstrate greater S-IgA reactivity to the stressor (mean = 9.87µg/ml, SEM = 6.37) when compared to volunteers in good health who demonstrated down-regulation (n = 27, mean = -5.33µg/ml, SEM. = 7.2). On day two volunteers in poor health (mean = 15.29µg/ml, SEM = 7.65) demonstrated greater S-IgA down regulation following the stressor than volunteers in good health (mean = -12.80µg/ml, SEM = 7.83), however there were no significant differences between the groups.

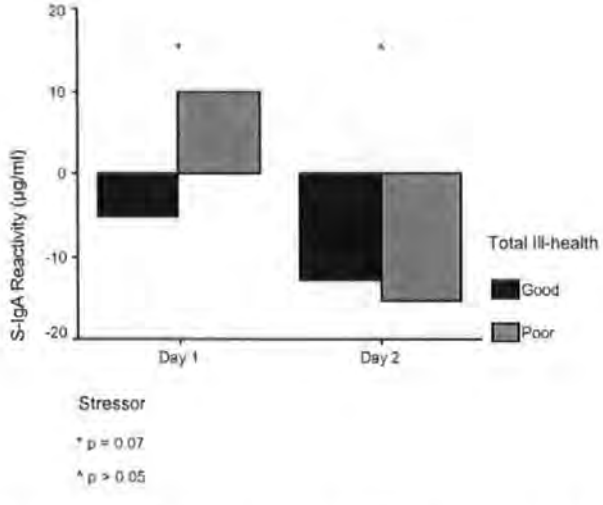


Figure 6.2 S-IgA Reactivity in Volunteers with Good and Poor Total Ill-health

Generalised Stress-related Complaints

No significant differences in S-IgA reactivity were observed between the groups. However, on day one there was a trend for volunteers in poor health (n = 24) with regards to frequencies of generalised stress-related complaints to demonstrate greater S-IgA reactivity (mean = 7.52µg/ml), SEM = 6.40) following the stressor than did volunteers in good health who demonstrated post-stress down-regulation (n = 25, mean = -4.10µg/ml, SEM = 7.51. On day two volunteers in good health demonstrated marginally greater down-regulation of S-IgA (mean = -14.58, SEM = 8.31) following the stressor than did volunteers in poor health (mean = -13.22, SEM = 7.20), although not significantly so. The differences in S-IgA reactivity on days one and two between volunteers classified as in good and poor health with regards to frequencies of stress-related complaints are presented in Figure 6.3.

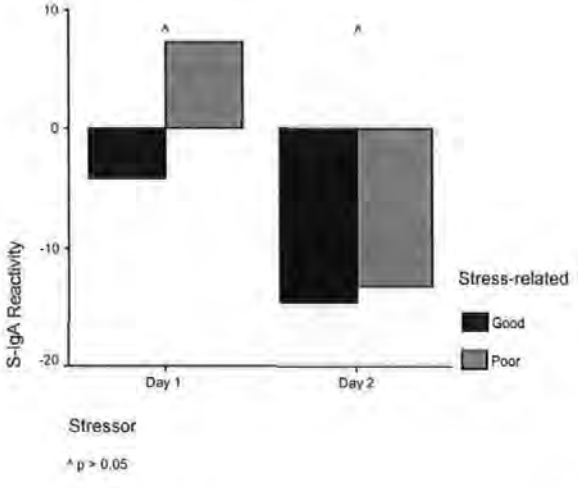


Figure 6.3 S-IgA Reactivity in Volunteers with Good and Poor Stress-related Complaints

Indicators of Ill-health

The differences in S-IgA reactivity between volunteers with good and poor indicators of l ill-health on days one and two are presented in Figure 6.4. No significant differences between the groups were observed on either day one or two. On day one volunteers in good health (n = 25, mean = 1.62µg/ml, SEM = 7.99) and poor health (n = 24, mean =

1.37μg/ml, SEM = 5.98) with regards to frequencies of indicators of ill-health demonstrated up-regulation of S-IgA following the stressor. On day two there was trend for volunteers in poor health to demonstrate greater S-IgA down-regulation (mean = -16.49μg/ml, SEM = 7.87) following the stressor than did volunteers in good health (mean = -11.44μg/ml, SEM = 7.71).

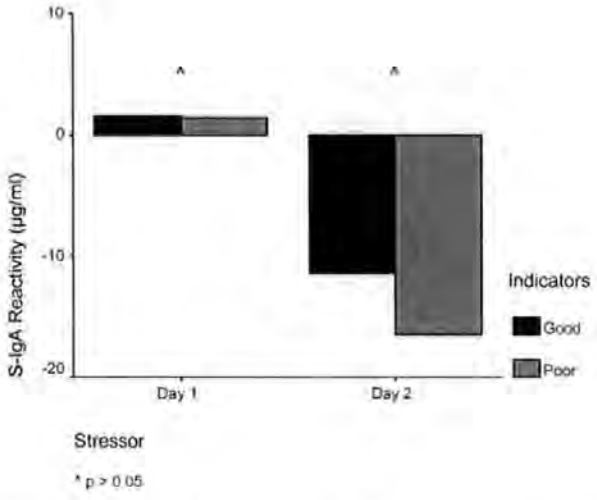


Figure 6.4 S-IgA Reactivity in Volunteers with Good and Poor Indicators of Ill-health

Psychological Complaints

No significant differences in S-IgA reactivity were observed between the groups. However, on day one there was a trend for volunteers in good health (n = 32) with regards to frequencies of generalised stress-related complaints to demonstrate greater S-IgA reactivity (mean = 2.93μg/ml), SEM = 6.43) following the stressor than did volunteers in poor health who demonstrated post-stress down-regulation (n = 17, mean = -1.22μg/ml, SEM = 7.84). On day two there was a trend for volunteers in poor health to demonstrate greater S-IgA down-regulation following the stressor (mean = -22.73μg/ml, SEM = 10.24) than did volunteers in good health (mean = -9.23, SEM = 6.32). The differences in S-IgA reactivity on days one and two between volunteers

classified as in good and poor health with regards to frequencies of psychological complaints are presented in Figure 6.5.

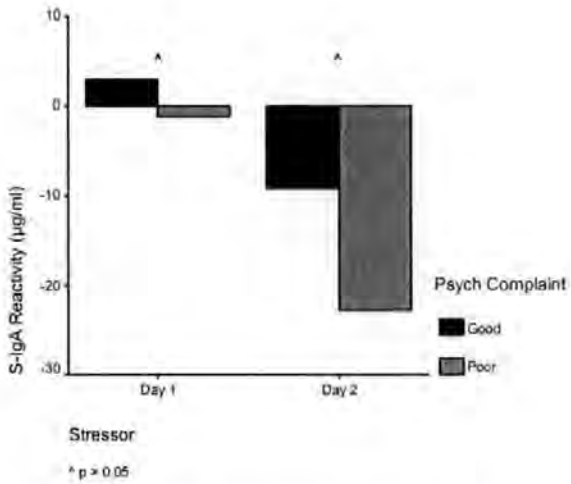


Figure 6.5 S-IgA Reactivity in Volunteers with Good and Poor Psychological Complaints

Immune Challenge Complaints

The differences in S-IgA reactivity between volunteers with good and poor immune challenge complaints on days one and two are presented in Figure 6.6. No significant differences between the groups were observed on either day one or two. On day one volunteers in good health (n = 35) and volunteers in poor health (14) with regards to frequencies of immune challenge complaints demonstrated S-IgA up-regulation following the stressor. However, there was a trend for volunteers in good health to demonstrate greater S-IgA reactivity (mean = 2.06µg/ml, SEM = 6.40) following the stressor than those in poor health (mean = 0.07µg/ml, SEM = 7.10). On day two both groups demonstrated S-IgA down-regulation following the stressor. However, there was a trend for volunteers in poor health to demonstrate greater down-regulation following the stressor (mean = -23.54µg/ml, SEM = 10.58) than did those in good health (mean = -10.06µg/ml, SEM = 6.35).

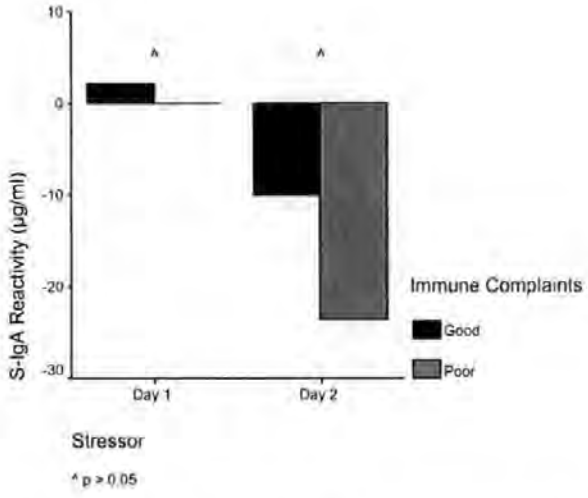


Figure 6.6 S-IgA Reactivity in Volunteers with Good and Poor Immune Challenge Complaints

Atopic Complaints

No significant differences in S-IgA reactivity were observed between the groups. On day one there was trend for volunteers in poor health (n = 22) with regards to frequencies of atopic complaints to demonstrate greater S-IgA up-regulation (mean = 6.14µg/ml, SEM = 6.89) following the stressor than volunteers in good health (n = 27) who demonstrated slight down-regulation (mean = -2.30µg/ml, SEM = 7.09). On day two both groups demonstrated S-IgA down-regulation following the stressor. However, there was a trend for volunteers in good health to demonstrate greater down-regulation (mean = -15.03µg/ml, SEM = 7.90) than those in poor health (mean = -12.55µg/ml, SEM = 7.54). The differences in S-IgA reactivity on days one and two between volunteers classified as in good and poor health with regards to frequencies of atopic complaints are presented in Figure 6.7.

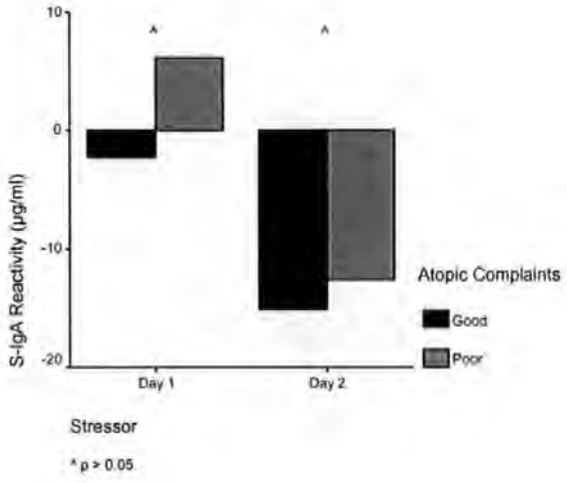


Figure 6.7 S-IgA Reactivity in Volunteers with Good and Poor Atopic Complaints

Gastric Complaints

The differences in S-IgA reactivity between volunteers with good and poor gastric complaints on days one and two are presented in Figure 6.8. On day one there was a trend for volunteers in poor health (n = 23) with regards to frequencies of gastric complaints to demonstrate greater S-IgA reactivity (mean = 6.94µg/ml, SEM = 7.25) following the stressor than those classified as in good health (n = 26) who demonstrated down-regulation (mean = -3.33µg/ml, SEM = 6.81). On day two both groups demonstrated S-IgA down regulation following the stressor. However, there was trend for volunteers in poor health to demonstrate considerably greater S-IgA down-regulation (mean = -26.43µg/ml, SEM = 7.50) than did those in good health (mean = -2.85µg/ml, SEM = 7.33).

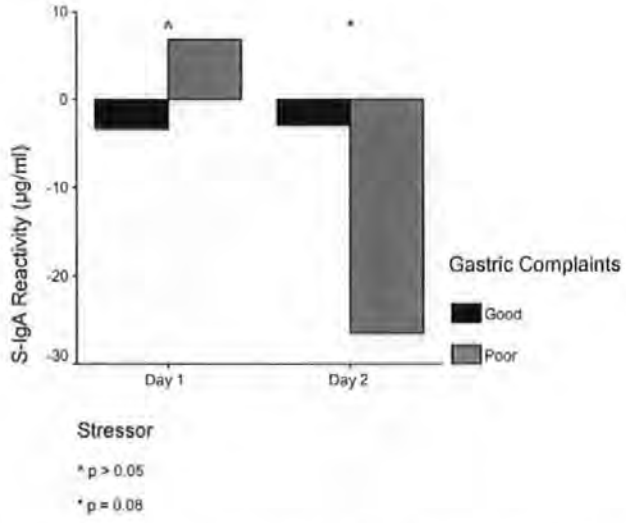


Figure 6.8 S-IgA Reactivity in Volunteers with Good and Poor Gastric Complaints

Urinary-tract Complaints

No significant differences in S-IgA reactivity were observed between the groups. On day one there was trend for volunteers in poor health (n = 30) with regards to frequencies of urinary-tract complaints to demonstrate greater S-IgA up-regulation (mean = 7.69µg/ml, SEM = 6.19) following the stressor than volunteers in good health (n = 19) who demonstrated down-regulation (mean = -8.29µg/ml, SEM = 7.97). On day two both groups demonstrated S-IgA down-regulation following the stressor. However, there was a trend for volunteers in good health to demonstrate greater down-regulation (mean = -21.34µg/ml, SEM = 9.90) than those in poor health (mean = -9.21µg/ml, SEM = 6.33). The differences in S-IgA reactivity on days one and two between volunteers classified as in good and poor health with regards to frequencies of urinary-tract complaints are presented in Figure 6.9.

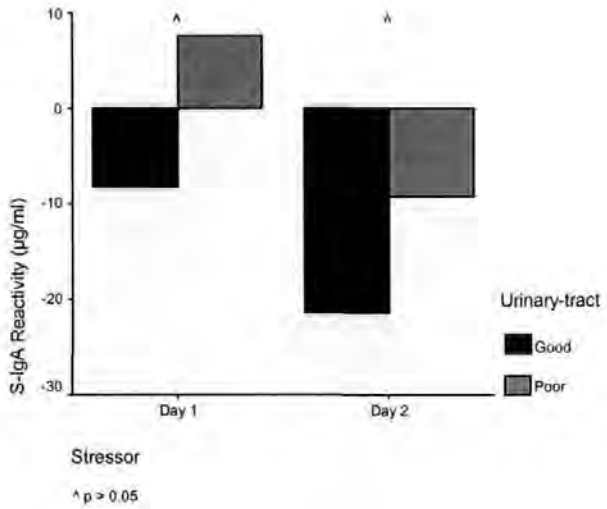


Figure 6.9 S-IgA Reactivity in Volunteers with Good and Poor Urinary-tract Complaints

Microflora Complaints

The differences in S-IgA reactivity between volunteers with good and poor microflora complaints on days one and two are presented in Figure 6.10. On day one there was a near significant trend ($t_{(47)}$ 1.92, $p = 0.06$) for volunteers in poor health ($n = 16$) with regards to frequencies of microflora complaints to demonstrate greater S-IgA reactivity (mean = 14.09µg/ml, SEM = 8.05) following the stressor than those classified as in good health ($n = 33$) who demonstrated down-regulation (mean = 4.61µg/ml, SEM = 6.06). On day two both groups demonstrated S-IgA down regulation following the stressor. However, there was trend for volunteers in good health to demonstrate considerably greater S-IgA down-regulation (mean = -17.65µg/ml, SEM = 7.39) than did those in poor health (mean = -6.21µg/ml, SEM = 6.82).

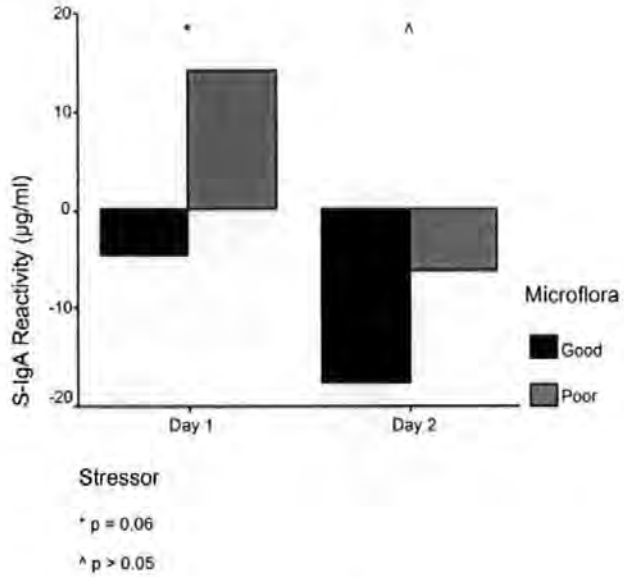


Figure 6.10 S-IgA Reactivity in Volunteers with Good and Poor Microflora Complaints

Fungal Complaints

No significant differences in S-IgA reactivity were observed between the groups. On day one volunteers in good health (n = 43, mean = 1.49µg/ml, SEM = 5.29) and poor health (n = 6, mean = 1.50µg/ml, SEM = 15.88) with regards to frequencies of fungal complaints demonstrated S-IgA up-regulation following the stressor. On day two there was a trend for volunteers in good health to demonstrate considerably greater S-IgA down-regulation (mean = -16.56µg/ml, SEM = 6.04) than those in poor health who demonstrated up-regulation (mean = 5.05µg/ml, SEM = 7.89). The differences in S-IgA reactivity on days one and two between volunteers classified as in good and poor health with regards to frequencies of fungal complaints are presented in Figure 6.11

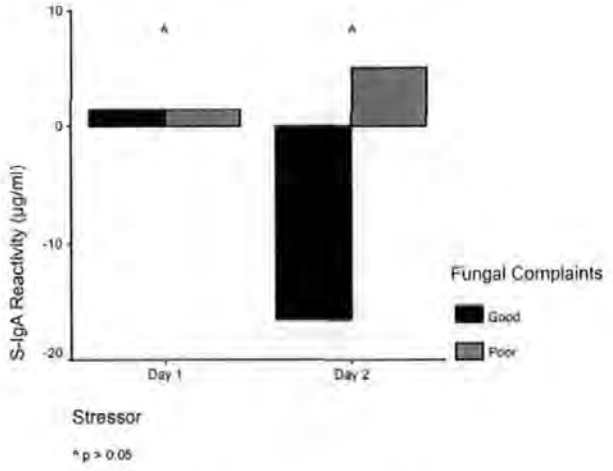


Figure 6.11 S-IgA Reactivity in Volunteers with Good and Poor Fungal Complaints

6.3.3.3 S-IgA Reactivity, Health Status and Perceived Workload

The relationships between S-IgA reactivity and perceived workload on both days were assessed using Spearman's Rho. The observed relationships are presented in Table 6.4. No significant relationships were observed between S-IgA reactivity and scores of perceived workload immediately following the task.. Moreover, the relationships are very weak and there is little consistency across both days.

		Mental Demand	Physical Demand	Temporal Demand	Effort	Performance	Frustration
S-IgA Reactivity Day 1	Correlation Coefficient	-.012	058	120	032	-.040	-.225
	Sig. (2-tailed)	932	690	411	828	787	121
	N	49	49	49	49	49	49

		Mental Demand	Physical Demand	Temporal Demand	Effort	Performance	Frustration
S-IgA Reactivity Day 2	Correlation Coefficient	.129	158	-.133	-.135	-.133	-.042
	Sig. (2-tailed)	376	279	364	355	362	776
	N	49	49	49	49	49	49

Table 6.4 Relationships (Spearman's Rho) Between S-IgA Reactivity and Perceived Workload on Days One and Two

Using the pre-determined means, volunteers were classified as being in either good or poor health with regards to frequencies of complaints for each of the identified MHC clusters. These two groups were subsequently compared with regards to their perceived

workload demands immediately following the stressor on both day one and two. Tables of means comprising perceived workload demands by health status are presented in Appendix D.

Total Ill-health

There was a consistent trend for volunteers in poor health with regards to total ill-health ($n = 22$) to report greater perceived workload. Moreover, poor health volunteers reported significantly greater frustration ($t_{(47)} -2.34$, $p < 0.05$) than did good health volunteers ($n = 27$). In contrast, good health volunteers reported marginally greater perceived performance than did poor health volunteers.

Similarly, there was a consistent trend for volunteers in poor health to report greater workload demands on day two. No significant differences were observed between the groups, however, there was near significant trend for volunteers in poor health to report greater effort ($t_{(47)} -1.80$, $p = 0.08$) immediately following the task than those in good health.

Generalised Stress-related Complaints

There was a consistent trend for volunteers classified as in poor health with regards to frequencies of stress-related complaints ($n = 24$) to report greater workload demands immediately following the task on day one than did those in good health ($n = 25$). Moreover, poor health volunteers perceived the task to elicit significantly greater frustration ($t_{(47)} -1.99$, $p < 0.05$) and demonstrated a trend to perceive greater physical demand ($t_{(47)} -1.84$, $p = 0.07$).

These trends were also apparent on day two. That is, there was trend for volunteers in poor health to perceive greater workload demands immediately following the stressor.

Moreover, poor health volunteers perceived the task to elicit significantly greater effort ($t_{(47)} -1.99$, $p < 0.05$). Further, these volunteers demonstrated a near significant trend to perceived greater temporal demand ($t_{(47)} -1.84$, $p = 0.07$) than did good health volunteers.

Indicators of Ill-health

On day one, volunteers classified as in poor health (n = 24) with regards to indicators of ill-health perceived the task to be significantly more mentally demanding ($t_{(47)} -2.03$, $p < 0.05$), temporally demanding ($t_{(47)} -2.17$, $p < 0.05$), requiring more effort ($t_{(40.2)} -2.10$, $p < 0.05$, and more frustrating ($t_{(47)} -2.49$, $p < 0.05$) than did those classified as in good health (n = 25). In contrast, good health volunteers perceived greater performance than did those in poor health ($t_{(47)}$ 2.48, $p < 0.05$).

Similarly, on day two there was a trend for volunteers classified as in poor health to perceive greater mental demand, physical demand and effort following the tasks than did those in good health. Moreover, poor health volunteers perceived the task to be significantly greater temporally demanding ($t_{(47)} -2.10$, $p < 0.05$), and more frustrating ($t_{(47)} -2.44$, $p < 0.05$). In contrast, good health volunteers perceived greater performance following the task than did those in poor health ($t_{(38.04)}$ 1.99, $p < 0.05$).

Psychological Complaints

There was a trend for volunteers in poor health (n = 17) with regards to psychological complaints to perceived greater workload demands following the task than did those in good health (n = 37). Moreover, volunteers in poor health perceived the task to be significantly more frustrating ($t_{(47)} -2.99$, $p < 0.01$) than did those in good health.

These trends were also evident on day two and there was a near significant trend for volunteers in poor health to perceive the task as more temporally demanding ($t_{(47)}$ – 1.80, $p = 0.08$). Further, poor health volunteers perceived the task as requiring significantly more effort ($t_{(47)}$ –2.40, $p < 0.05$) than did those in good health.

Immune Challenge Complaints

On day one there was trend for volunteers classified as being in poor health ($n = 35$) with regards to frequencies of immune challenge complaints to perceived greater workload demands for the facets of mental demand, physical demand ($t_{(17.06)}$ –1.95, $p = 0.06$), and effort. Further, poor health volunteers perceived significantly greater temporal demand ($t_{(47)}$ –2.22, $p < 0.05$) and frustration ($t_{(47)}$ –3.17, $p < 0.01$) following the stressor than did those in good health. ($n = 14$).

Similar trends were observed on day two with volunteers in poor health perceiving greater workload following the task than those in good health. Further, volunteers in poor health perceived significantly greater effort ($t_{(47)}$ 2.86, $p < 0.01$) following the stressor than those in good health.

Atopic Complaints

Volunteers in poor health with regards to frequencies of atopic complaints ($n = 22$) perceived marginally greater perceived workload demand than did those in good health ($n = 27$). Moreover, volunteers in poor health perceived the task to be significantly more frustrating ($t_{(47)}$ –2.02, $p < 0.05$) than did those in good health.

Similar trends were observed on day two, with those in poor health perceiving marginally greater workload, and significantly greater effort ($t_{(47)}$ –2.01, $p < 0.05$) immediately following the stressor than did those in good health.

Gastric Complaints

No significant differences were observed between volunteers in good and poor health with regards to frequencies of gastric complaints. Moreover, a mixed pattern was observed, i.e., with the exception of physical demand, there was a trend for volunteers in poor health (n = 23) to perceive greater workload demands than those in good health (n = 26).

Similarly, a mixed pattern of non-significant differences were observed on day two. There was a trend for volunteers in poor health to perceive greater physical and temporal demand, but perceive less demand with regards to mental demand, effort and frustration when compared to those in good health.

Urinary-tract Complaints

No significant differences were observed, although there was a trend for volunteers in poor health with regards to frequencies of urinary-tract complaints (n = 19) to perceive greater workload demands following the stressor than those classified as in good health (n = 30).

A mixed pattern of non-significant differences were observed on day two. With the exception of mental and temporal demand, there was trend for volunteers in poor health to perceive greater workload demands. In contrast, volunteers in good health perceived significantly better performance ($t_{(47)}$ 2.02, $p < 0.05$) than those in poor health.

Microflora Complaints

Volunteers in poor health with regards to frequencies of microflora complaints (n = 14) perceived marginally greater physical and temporal demand and frustration, but less demands with regards to mental demand and effort. In contrast, volunteers in good

health (n = 33) perceived significantly greater performance ($t_{(47)}$ 2.04, $p < 0.05$) following the task than did those in poor health.

No significant differences were observed on day two. However, in contrast to day one, there was a trend for volunteers in good health to perceive marginally greater mental and temporal demand, effort and frustration following the stressor than those in poor health.

Fungal Complaints

A mixed pattern of results were observed between volunteers in good and poor health with regards to frequencies of fungal complaints. There was a trend for volunteers in good health (n = 43) to perceive marginally greater mental demand and effort, and significantly greater frustration ($t_{(47)}$ 2.040, $p < 0.05$) following the task than those in poor health (n = 6).

Similarly, no significant differences were observed between the groups on day two. However, there was trend for volunteers in good health to perceive greater temporal demand, following the task when compared with those in poor health.

6.3.4 Saliva Flow Rates, S-IgA Concentrations and S-IgA Secretion Rates

The following section comprises analyses regarding the effects of the stressor on both days one and two upon saliva volume, and subsequent influences upon S-IgA concentrations. If post-stress reductions in saliva volume are observed, post-stress increases in S-IgA concentrations cannot be attributed to the effects of the stressor alone. That is, if saliva volume decreases, S-IgA within the given volume will be artificially elevated. In contrast, if the stressor is observed to have no effect, or moreover and increasing effect on saliva volume, it can be assumed that post-stress increases in S-IgA can be attributed to the stressor, not as an artefact of reductions in saliva volume.

Figure 6.12 presents pre and post-stressor mean saliva volume (µl/min) on days one and two. Significant post-stress increases in saliva volume were observed on both day one ($t_{(48)}$ -2.7, $p < 0.01$), and day two ($t_{(48)}$ -3.20, $p < 0.001$).

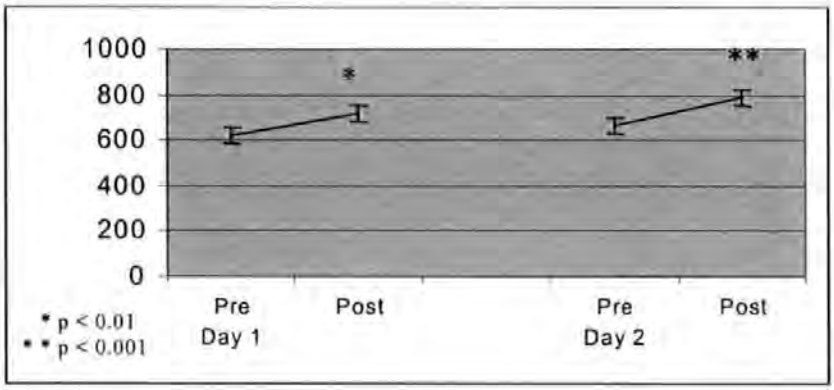


Figure 6.12 Pre and Post Saliva Volume (and SEM) on Days One and Two

6.3.4.1 Summary

The post-test increases in saliva volume in both sessions suggest that changes in S-IgA concentrations (in both the present study and study two) are not an artefact of reduced saliva volume. That is, if post-stress reductions in saliva volume are observed, it would

follow that S-IgA concentration within the sample would be artificially elevated. The stress induced by the stressor consistently increased saliva volume, and as such, any increases in S-IgA concentration can be confidently attributed to the stressor not reductions in saliva volume.

However, figures 6.12 and 6.1 (S-IgA reactivity to acute stress) demonstrate an influence of saliva volume upon S-IgA concentrations. That is, although post-stress changes in S-IgA concentrations cannot be attributed to the effects of change in saliva volume, the volume of saliva in pre-test measures appears to influence the pre-stress S-IgA concentrations. Pre-stress S-IgA concentrations in session one are relatively high, and as a result, it appears that the stressor does not induce significant S-IgA reactivity. However, the pre-test saliva volume in session one is relatively low, and as such, S-IgA concentrations in a smaller volume of saliva will be artificially elevated. The same mechanism is apparent in the post-stress measures in session two. That is, post-stress S-IgA concentrations are relatively low (and are observed to decrease following exposure to the stressor), however, post-stress saliva volume is significantly increased. As such, post-stress S-IgA concentrations in session two appear to be reduced in relation to the observed elevated increase in saliva volume.

Although saliva volume does not account for post-stress increases in S-IgA concentrations *per se*, it is likely that the amount of saliva produced can effect the relative concentration of S-IgA observed in both pre and post-stress samples. As such, all future analyses will utilise S-IgA secretion rates (expressed as amount of S-IgA released per minute of sampling time). This technique will take account of the influence of saliva volume upon S-IgA concentrations in both pre- and post-stress measures, and subsequent observations concerning pre- and post stress changes in S-IgA. That is, saliva volume will be accounted for in every sample of S-IgA.

Concentrations of S-IgA and observed changes can therefore be attributed directly to the effects of the manipulated stressor, not to the over-riding influence of changes in saliva volume.

6.3.5 S-IgA Secretion Rates

Although it is apparent that increases in S-IgA concentration can be attributed to the stressor, comparisons of saliva flow rates in relation to S-IgA concentrations have demonstrated that saliva volume influences all measurements of S-IgA. As such, and in order to create a 'cleaner' measurement of S-IgA, S-IgA secretion rates will be utilised in all subsequent analyses. S-IgA secretion rates give an efficient measure of S-IgA concentrations whilst accounting for fluctuations in saliva volume. The use of S-IgA secretion rates therefore allow for greater stringency and more confidence in the assumption that S-IgA concentrations are influenced by the stressor. S-IgA secretion rates are derived by multiplying S-IgA concentration (μg) by saliva volume (μl), and dividing this figure by total collection time (2 minutes in the current research). The final measurement is expressed as the amount of S-IgA secreted in saliva over a given time period (μg/min).

6.3.5.1 S-IgA Reactivity to Acute Stress

Figure 6.13 presents the pre-post changes in S-IgA secretion rates in response to the stressor on both days one and two. On day one, post-stress S-IgA (mean = 104.32μg/min) was significantly greater ($t(49) = -2.78$, $p < 0.01$) than the pre-stress measurement (mean = 80.59μg/min). On day two, there was a trend for post-stress S-IgA (mean = 84.96μg/min) to be greater than pre-stress measurements (mean = 78.89μg/min), although not significantly so. Further, there were no significant differences between pre-stress S-IgA secretion rates on days one and two.

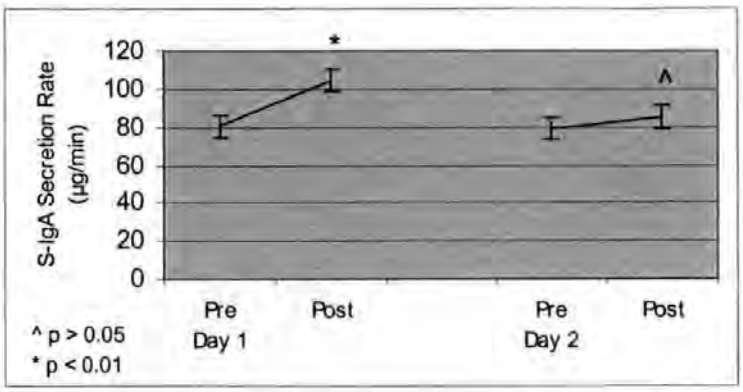


Figure 6.13 S-IgA Reactivity (and SEM) to Acute Stress on Days One and Two (Secretion Rates)

6.3.5.2 Health Status and S-IgA Reactivity

Using the previously identified classifications, S-IgA reactivity (secretion rates) was assessed on both days one and two in volunteers classified as in good and poor health with regards to frequencies of health complaints for each MHC cluster. These analyses are therefore analogous with those conducted in section 6.3.3.2, and as such, classification as either good or poor health are the same. However, these analyses account for the influence of saliva volume upon S-IgA reactivity in the two groups. No significant differences were observed between the groups, however, the patterns of reactivity in those classified as in good or poor health are analogous with the patterns observed with S-IgA concentrations in both the current and previous study. Mean pre- and post-stress data by health status are presented in Appendix D.

Total Ill-health.

Figure 6.14 presents the means S-IgA reactivity on day one and day two in volunteers classified as in either good or poor health with regards to frequencies of total Ill-health complaints. On day one there was a trend for volunteers in poor health to demonstrate greater S-IgA reactivity to the stressor. In contrast, on day two S-IgA reactivity was

reduced in both groups, however the greater reduction was observed in those volunteers classified as in poor health.

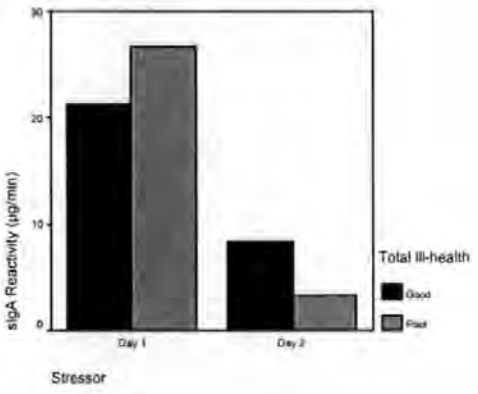


Figure 6.14 S-IgA Reactivity to Acute Stress in Volunteers With Good or Poor Total Ill-health

Generalised Stress-related Complaints

Both groups demonstrated greater S-IgA reactivity on day one, when compared with reactivity on day two. Moreover, on both days there was a trend for volunteers in poor health to demonstrate lower S-IgA reactivity following the stressor, than those classified as in good health. The mean S-IgA reactivity in volunteers classified as in either good or poor health with regards to frequencies of generalised stress-related complaints are presented in Figure 6.15.

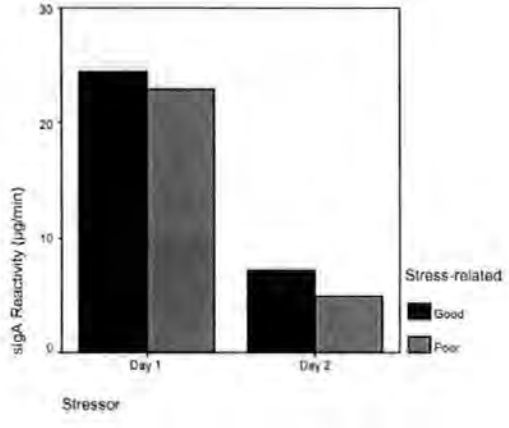


Figure 6.15 S-IgA Reactivity to Acute Stress in Volunteers With Good or Poor Stress-related Complaints

Indicators of Ill-health

Figure 6.16 presents mean S-IgA reactivity in volunteers classified as in either good or poor health with regards to frequencies of indicators of ill-health. Both groups demonstrated greater S-IgA reactivity on day one when compared with day two. Further, on both days, volunteers classified as in poor health demonstrated reduced S-IgA reactivity when compared to those in good health.

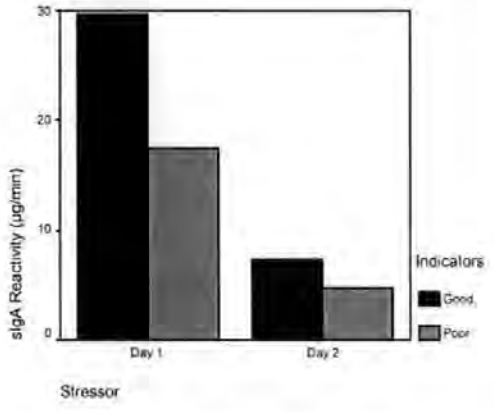


Figure 6.16 S-IgA Reactivity to Acute Stress in Volunteers With Good or Poor Indicators of ill-health

Psychological Complaints

Both groups demonstrated greater S-IgA reactivity on day one than on day two. Volunteers in poor health demonstrated reduced S-IgA reactivity on day one when compared to volunteers in good health. Further, poor health volunteers demonstrated post-stress down-regulation of S-IgA on day two. The mean S-IgA reactivity in volunteers classified as in either good or poor health with regards to frequencies of psychological complaints are presented in Figure 6.17.

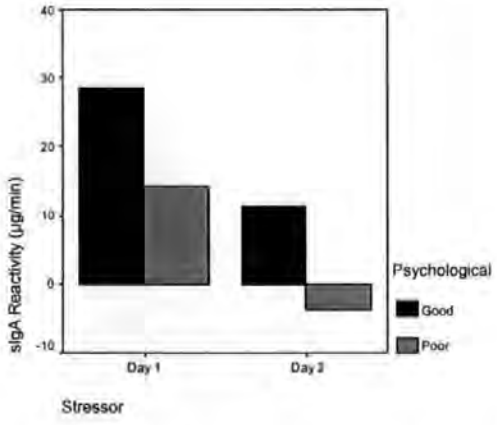


Figure 6.17 S-IgA Reactivity to Acute Stress in Volunteers With Good or Poor Psychological Complaints

Immune Challenge Complaints

Figure 6.18 presents mean S-IgA reactivity in volunteers classified as in either good or poor health with regards to frequencies of immune challenge complaints. On day one both groups demonstrated almost identical S-IgA reactivity to the stressor. On day two both groups demonstrated reduced S-IgA reactivity when compared with day one, however, volunteers in poor health demonstrated post-stress down-regulation of S-IgA.

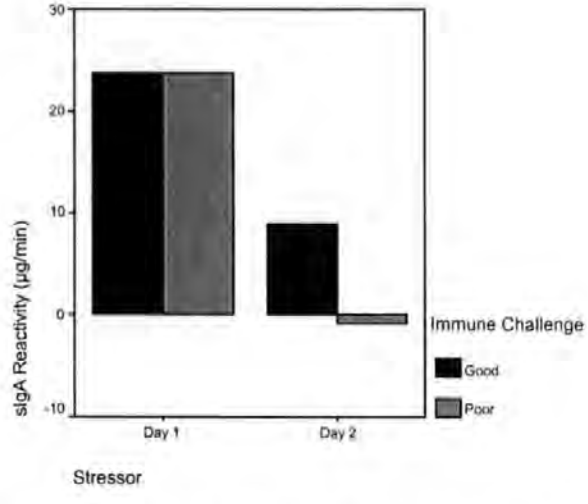


Figure 6.18 S-IgA Reactivity to Acute Stress in Volunteers With Good or Poor Immune Challenge Complaints

Atopic Complaints

Both groups demonstrated greater S-IgA reactivity on day one than on day two. Volunteers in poor health demonstrated greater S-IgA reactivity on day one when compared to volunteers in good health. Similarly, poor health volunteers also demonstrated greater post-stress S-IgA day two. The mean S-IgA reactivity in volunteers classified as in either good or poor health with regards to frequencies of atopic complaints are presented in Figure 6.19.

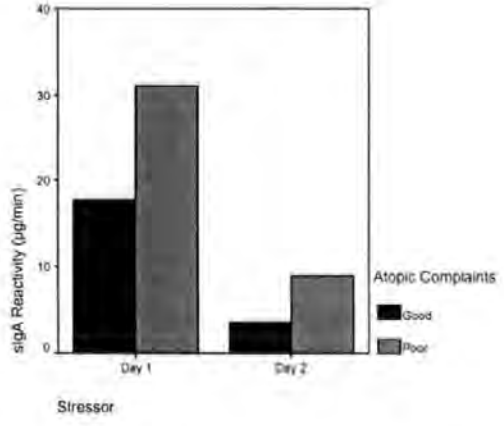


Figure 619 S-IgA Reactivity to Acute Stress in Volunteers With Good or Poor Atopic Complaints

Gastric Complaints

Figure 6.20 presents mean S-IgA reactivity in volunteers classified as in either good or poor health with regards to frequencies of gastric complaints. There was a near significant trend for volunteers classified as in poor health to demonstrate greater S-IgA reactivity ($t_{(47)} -1.82$, $p = 0.07$) on day one than those in good health. Both groups demonstrated reduced S-IgA reactivity on day two when compared to day 1, however there was very little differences between the groups.

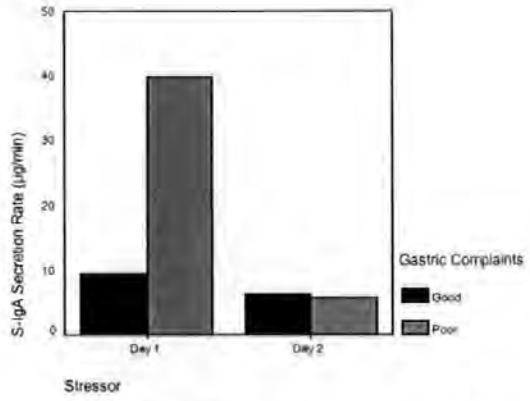


Figure 6.20 S-IgA Reactivity to Acute Stress in Volunteers With Good or Poor Gastric Complaints

Urinary-tract Complaints

Figure 6.21 presents mean S-IgA reactivity in volunteers classified as in either good or poor health with regards to frequencies of urinary-tract complaints. On day one both groups demonstrated greater S-IgA reactivity than on day two. Further, on both days, volunteers in poor health demonstrated greater post-stress S-IgA reactivity than did those in good health.

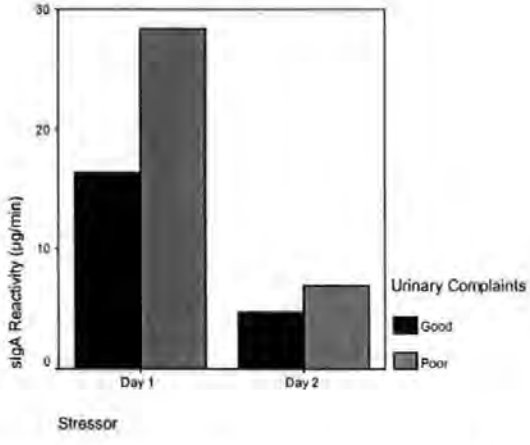


Figure 6.21 S-IgA Reactivity to Acute Stress in Volunteers With Good or Poor Urinary-tract Complaints

Microflora Complaints

Both groups demonstrated greater S-IgA reactivity on day one than on day two. Volunteers in poor health demonstrated greater S-IgA reactivity on day one when

compared to volunteers in good health. In contrast, volunteers in good health demonstrated marginally greater S-IgA reactivity on day two when compared with poor health volunteers. The mean S-IgA reactivity in volunteers classified as in either good or poor health with regards to frequencies of atopic complaints are presented in Figure 6.22.

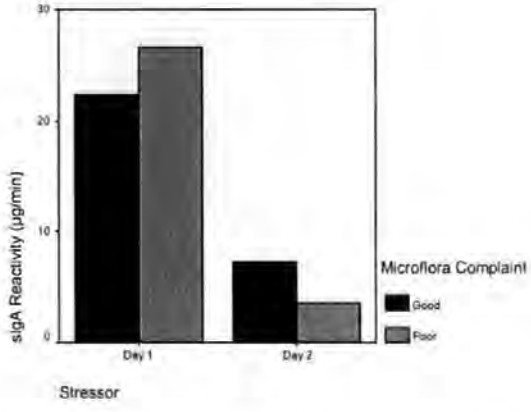


Figure 6.22 S-IgA Reactivity to Acute Stress in Volunteers With Good or Poor Microflora Complaints

Fungal Complaints

Figure 6.23 presents mean S-IgA reactivity in volunteers classified as in either good or poor health with regards to frequencies of fungal complaints. On day one both groups demonstrated greater S-IgA reactivity than on day two. Further, on both days, volunteers in poor health demonstrated considerably greater, although not significantly so, S-IgA reactivity than did those in good health.

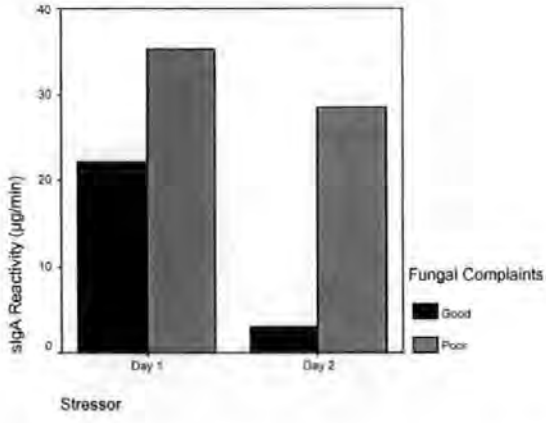


Figure 6.23 S-IgA Reactivity to Acute Stress in Volunteers With Good or Poor Fungal Complaints

6.3.5.3 S-IgA Reactivity and Perceived Workload

The relationships between S-IgA reactivity and perceived workload on both days were assessed using Spearman's Rho. The observed relationships are presented in Table 6.5. All relationships were negative, that is, those volunteers with the greatest S-IgA reactivity perceived the least workload demands, but also the lowest performance. Further, on day one there was a significant negative relationship between S-IgA reactivity and frustration ($r_{(49)}$ -.37, $p < 0.01$), and on day two, a near significant relationship between reactivity and perceived performance ($r_{(49)}$ -.28, $p = 0.06$). To be reminded of the differences in perceived workload between volunteers classified as in good and poor health for each MHC cluster, the reader is directed to section 6.3.3.3.

		Mental Demand	Physical Demand	Temporal Demand	Effort	Performance	Frustration
S-IgA Reactivity Day 1	Correlation Coefficient	-.092	-.132	-.034	-.004	-.014	-.366
	Sig. (2-tailed)	.530	.365	.815	.978	.923	.010
	N	49	49	49	49	49	49

		Mental Demand	Physical Demand	Temporal Demand	Effort	Performance	Frustration
S-IgA Reactivity Day 2	Correlation Coefficient	.089	-.179	-.044	-.064	-.216	-.211
	Sig. (2-tailed)	.543	.218	.766	.660	.137	.146
	N	49	49	49	49	49	49

Table 6.5 Relationships (Spearman's Rho) Between S-IgA Reactivity and Perceived Workload on Day One and Two (Secretion Rates)

6.3.5.4 Health Status, Personality and Mood

Table 6.6 presents the means personality and mood scores for volunteers classified as in good and poor health for each of the MHC clusters.

Total Ill-health

There was a trend for volunteers classified as in poor health with regards to frequencies of total ill-health complaints to demonstrate greater neuroticism, extraversion and openness. Further, volunteers in poor health demonstrated significantly greater negative affect ($t_{(27.02)} -2.08$, $p < 0.05$).

Stress-related Complaints

There was a trend for volunteers in poor health with regards to frequencies of stress-related complaints to demonstrate greater extraversion and openness. Further, poor health volunteers demonstrated significantly greater neuroticism ($t_{(47)} -3.47$, $p < 0.01$) and negative affect ($t_{(31.22)} -2.2$, $p < 0.05$) than did those in good health.

Indicators of Ill-health

There was a trend for volunteers classified as in poor health with regards to frequencies of ill-health indicators to demonstrate greater neuroticism. In contrast, there was a trend for volunteers in good health to demonstrate greater agreeableness ($t_{(47)} -2.01$, $p = 0.07$). Further, volunteers in poor health demonstrated significantly greater negative affect ($t_{(27.62)} -4.23$, $p < 0.001$) and openness ($t_{(47)} -2.01$, $p < 0.05$) than did good health volunteers.

Psychological Complaints

Volunteers in poor health with regards to frequencies of psychological complaints demonstrated significantly greater neuroticism ($t_{(47)} -5.24$), $p < 0.001$), openness ($t_{(47)}$

-2.12, $p < 0.04$), and negative affect ($t_{(47)} -3.34$, $p < 0.05$) than did those in good health.

Immune Challenge Complaints

Volunteers in poor health with regards to frequencies of immune challenge complaints demonstrated significantly greater neuroticism ($t_{(47)} -2.63$, $p < 0.01$) and negative affect ($t_{(47)} -1.85$), $p < 0.05$) than did those in good health.

Atopic Complaints

No significant differences were observed between volunteers classified as in good and poor health with regards to atopic complaints, although there was a trend for volunteers in poor health to demonstrate marginally greater neuroticism, extraversion, openness and negative affect.

Gastric Complaints

Volunteers classified as in poor health with regards to frequencies of ill-health complaints demonstrated significantly greater neuroticism ($t_{(47)} -2.15$, $p < 0.05$) and openness ($t_{(47)} -3.48$, $p < 0.01$) than did those in good health.

Urinary-tract Complaints

No significant differences were observed between volunteers classified as in good and poor health with regards to frequencies of urinary-tract complaints, although, there was trend for volunteers in poor health to demonstrate greater neuroticism and negative affect.

Microflora Complaints

There was near significant trend for volunteers classified as in poor health with regards to frequencies of microflora complaints to demonstrate greater neuroticism ($t_{(47)} -1.80$, $p = 0.07$), than did those in good health. In contrast there was a trend for volunteers in good health to demonstrate greater agreeableness than those in poor health.

Fungal Complaints

Volunteers classified as in poor health with regards to frequencies of poor health with regards to frequencies of fungal complaints demonstrated significantly greater conscientiousness ($t_{(47)} -1.98$, $p < 0.05$) than those in good health.

		N	E	O	A	C	PA	NA
Total Ill-health	Good	19.3 (7.27)	27.96 (6.38)	31.07 (5.59)	29.78 (7.07)	31.11 (5.26)	33.39 (5.84)	**15.91 (3.49)**
	Poor	25.86 (8.61)	28.91 (4.98)	33.14 (5.45)	27.91 (5.83)	31.05 (7.38)	33.00 (6.58)	**19.84 (8.32)**
Stress-related	Good	**18.52 (6.68)**	27.88 (6.63)	30.72 (5.58)	29.28 (7.11)	31.52 (5.26)	33.94 (5.60)	**15.76 (3.50)**
	Poor	**26.13 (8.53)**	28.92 (4.75)	33.33 (5.35)	28.58 (6.03)	30.63 (7.20)	32.46 (6.66)	**19.67 (8.01)**
Indicators	Good	18.68 (7.38)	29.08 (5.07)	**30.48 (4.89)**	**30.56 (5.05)**	31.08 (4.63)	32.56 (7.02)	**14.38 (2.41)**
	Poor	25.96 (8.07)	27.67 (6.42)	**33.58 (5.88)**	**27.25 (7.54)**	31.08 (7.67)	33.90 (5.09)	**21.10 (7.41)**
Psychological	Good	**18.53 (7.11)**	28.03 (6.04)	**30.81 (5.58)**	29.31 (6.80)	31.41 (5.88)	33.19 (5.90)	**15.66 (5.53)**
	Poor	**29.24 (6.19)**	29.06 (5.27)	**34.24 (4.96)**	28.24 (6.17)	30.47 (7.00)	33.26 (6.07)	**21.47 (6.28)**
Immune-Challenge	Good	**20.34 (7.67)**	27.97 (5.99)	31.80 (5.72)	29.26 (6.50)	31.43 (5.87)	33.17 (6.75)	**16.37 (4.71)**
	Poor	**27.00 (8.81)**	29.43 (5.17)	32.50 (5.33)	28.14 (6.81)	30.21 (7.22)	33.32 (4.36)	**20.93 (8.75)**
Atopy	Good	21.30 (7.55)	27.15 (6.05)	31.93 (5.34)	29.30 (7.20)	31.93 (5.92)	33.70 (6.55)	17.15 (4.98)
	Poor	23.41 (9.55)	29.91 (5.09)	32.09 (5.96)	28.50 (5.77)	30.05 (6.59)	32.61 (5.64)	18.32 (7.86)
Gastric	Good	**19.88 (8.76)**	28.77 (6.35)	**29.65 (4.70)**	29.58 (7.05)	32.50 (4.59)	34.08 (5.75)	16.40 (5.09)
	Poor	**24.91 (7.46)**	27.96 (5.10)	**34.65 (5.36)**	28.22 (5.99)	29.48 (7.47)	32.24 (6.51)	19.11 (7.44)
Urinary-tract	Good	19.68 (7.98)	29.16 (5.58)	32.63 (4.83)	28.52 (8.15)	30.47 (6.38)	34.13 (6.42)	16.79 (5.58)
	Poor	23.87 (8.52)	27.90 (5.90)	31.60 (6.03)	29.20 (5.43)	31.47 (6.22)	32.63 (5.96)	18.23 (6.88)
Microflora	Good	20.76 (8.58)	29.00 (5.51)	31.91)5.84)	39.58 (7.08)	30.54 (5.80)	34.15 (5.16)	17.02 (5.09)
	Poor	23.31 (7.65)	27.13 (6.21)	32.19 (5.14)	27.63 (5.24)	32.38 (7.06)	31.28 (7.56)	19.03 (8.50)
Fungal	Good	23.00 (8.40)	28.67 (5.43)	31.72 (5.43)	29.00 (6.68)	**30.44 (6.25)**	32.65 (6.22)	17.91 (6.53)
	Poor	16.83 (7.57)	26.33 (8.02)	34.00 (6.63)	28.50 (5.96)	**35.67 (3.98)**	37.25 (3.59)	15.92 (5.33)

N = Neuroticism, E = Extraversion, O = Openness, A = Agreeableness, C = Conscientiousness, PA = Positive Affect, NA = Negative Affect
Bold = $p < 0.05$

Table 6.6 Mean Personality and Mood Scores in Volunteers Classified as in Good and Poor health

6.3.5.5 Personality, Mood and S-IgA Reactivity

Volunteers were classified with regards to each of the five personality traits and positive and negative affect. The NEO-FFI enables volunteers to be classified into either three or five categories. With regards to the former classification technique, the sample sizes were not sufficient to classify volunteers into the sample. Further, although for the

majority of personality traits classification into three categories was appropriate, this technique led to extremely unequal sample sizes for some traits, particularly that of openness. In order to conduct consistent analyses across all five personality traits and positive and negative affect, volunteers were classified into one of two groups. Firstly, with regards to the NEO-FFI, the mid-point score within the middle (average) category was used to classify volunteers for each of the traits, those volunteers with a score greater than the mid-point being classified as high, those below as low. With regards to the PANAS, there is no method of classification. Volunteers were therefore classified using a within-sample mean split. Although it is acknowledged that this classification technique will result in a loss of variation within the groups, and will therefore reduce the ability to distinguish between extreme scores, it was important to apply a consistent method of classification throughout.

Using the classification of personality and mood, S-IgA reactivity on both days was subsequently assessed between the two derived groups. Mean reactivity between volunteers classified as either low or high for each of the personality traits and positive and negative affect are presented in Table 6.7.

No significant differences in S-IgA reactivity were observed between volunteers classified as low or high for each mood and personality characteristic. However, a mixed pattern of results were observed. The most salient differences were observed between volunteers classified as either low or high for negative affectivity (NA). Volunteers classified as high in NA demonstrated considerably greater S-IgA reactivity on both days, however, as with all traits, S-IgA reactivity was greater on the first day. This pattern was also evident for those classified as highly agreeable. In contrast, volunteers classified as high for the remaining traits demonstrated lower S-IgA reactivity than those classified as low.

		S-IgA Reactivity (µ/min)	
		Day 1 (SEM)	Day 2 (SEM)
PA	low	25.73 (14.16)	10.89 (8.12)
	high	21.46 (8.98)	0.62 (7.77)
NA	low	16.97 (8.51)	2.41 (6.27)
	high	36.44 (18.71)	12.96 (11.26)
N	low	29.58 (16.12)	13.83 (8.97)
	high	19.33 (8.97)	0.25 (7.15)
E	low	28.67 (19.76)	20.56 (9.87)
	high	21.10 (8.06)	-1.63 (6.94)
O	low	23.55 (14.02)	6.27 (7.58)
	high	23.89 (10.23)	5.87 (7.58)
A	low	13.01 (7.93)	5.08 (6.94)
	high	32.46 (14)	6.89 (8.65)
C	low	22.88 (15.34)	10.61 (9.48)
	high	24.53 (8.30)	1.17 (6.36)

PA = Positive Affect, NA = Negative Affect, N = Neuroticism,
E = Extraversion, O = Openness, A = Agreeablenss, C = Conscientiousness

Table 6.7 Mean S-IgA Reactivity on Days One and Two Between Volunteers Classified as Low and High for Personality and Mood Characteristics.

6.3.5.6 Personality, Mood and Perceived Workload

Using the same classification technique as in section 6.3.5.5, mean perceived workload demands were assessed in volunteers classified as either low or high for each of the traits.

Significant differences in workload were observed for negative affectivity. That is, there was a trend for volunteers classified as high in negative affectivity to perceive greater workload demand, and as a result significant reductions in perceived performance on day one ($t_{(47)}$ 2.72, $p < 0.01$) and day two ($t_{(47)}$ 2.40, $p < 0.05$). Similarly, volunteers classified as high in neuroticism demonstrated a trend to perceive greater workload demands, and significantly greater effort ($t_{(47)}$ –2.00, $p < 0.05$) and frustration ($t_{(47)}$ –2.41, $p < 0.05$), and in contrast, near significant reductions in performance ($t_{(47)}$ 1.83, $p = 0.05$) on day one when compared to those classified as low.

In contrast, the reverse pattern was observed for the trait of agreeableness. That is, there was a trend for volunteers classified as low in agreeableness to perceive greater workload demands, and therefore perceive significantly greater performance on day two

(t (47) –2.28, $p < 0.05$) when compared to those classified as high. This pattern was more evident for the trait of conscientiousness. That is, there was a trend for volunteers classified as low in conscientiousness to perceive greater workload demands, and significantly greater temporal demand on day one ($t_{(47)}$ 2.36, $p < 0.05$) and day two ($t_{(47)}$ 2.67, $p < 0.01$). The mean perceived workload scores for each facet on both days one and two in volunteers classified as either low of high are presented in Table 6.8.

		Trait	PA		NA		N		E		O		A		C	
			Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Day 1	Mental Demand	low	89.01	5.04	84.79	5.14	83.22	6.50	91.46	5.49	82.38	5.52	91.89	4.91	91.59	5.44
		high	88.32	5.99	96.03	5.09	92.79	4.56	87.22	5.15	94.75	5.17	86.08	5.74	85.90	5.47
	Physical Demand	low	15.78	3.71	15.30	3.34	13.38	3.06	14.99	5.32	19.02	4.40	11.80	2.93	13.58	3.75
		high	15.19	3.55	15.88	3.95	17.10	3.85	15.78	2.77	12.13	2.61	18.52	3.92	17.35	3.51
	Temporal Demand	low	82.55	6.16	74.17	5.29	72.14	6.82	77.64	7.04	71.99	6.18	80.52	6.50	**88.37**	5.20
		high	74.76	5.35	87.76	6.10	83.95	4.97	79.56	5.16	85.51	5.26	77.57	5.37	**89.80**	5.88
	Effort	low	76.27	4.92	73.63	4.85	**68.62**	6.21	76.58	5.87	73.43	5.48	76.49	4.93	79.92	4.87
		high	77.01	5.40	82.23	4.82	**82.61**	3.97	76.64	4.61	79.68	4.72	76.72	5.23	73.44	5.46
	Performance	low	51.77	5.26	**62.85**	4.10	63.66	5.32	48.96	6.02	62.30	5.18	48.76	7.35	56.09	6.83
		high	59.33	6.25	**41.14**	7.81	49.06	5.63	58.69	5.27	48.62	5.97	60.66	4.10	54.58	4.60
	Frustration	low	44.89	6.30	36.78	4.72	**33.06**	6.10	38.71	7.82	45.45	6.83	51.45	7.73	49.91	6.04
		high	44.61	6.30	59.82	6.12	**53.53**	5.78	47.96	5.34	44.10	6.20	39.30	4.84	39.82	6.39
Day 2	Mental Demand	low	82.55	6.33	79.01	5.02	78.54	6.25	80.18	6.80	82.99	5.00	86.95	5.69	86.66	5.91
		high	86.30	5.30	94.29	6.87	88.64	5.49	88.52	5.26	85.58	6.65	80.53	5.92	82.06	5.90
	Physical Demand	low	18.48	3.21	14.46	2.29	14.75	2.64	15.61	3.47	14.76	2.43	13.82	3.03	17.17	3.58
		high	14.53	3.51	20.71	5.24	18.04	3.65	17.17	3.15	18.42	4.02	18.92	3.49	16.11	3.17
	Temporal Demand	low	79.67	6.22	71.46	5.43	71.52	6.86	75.27	6.65	69.71	4.94	85.15	7.35	**88.42**	5.63
		high	73.09	6.93	86.22	8.22	80.37	6.21	77.28	6.18	83.17	7.56	69.60	5.01	**85.21**	6.57
	Effort	low	71.33	5.43	72.13	4.73	68.38	6.01	68.13	6.61	72.69	5.40	73.35	6.12	76.65	5.01
		high	80.29	7.37	81.94	9.46	80.90	6.36	79.47	5.88	78.26	7.20	77.32	6.55	74.46	7.49
	Performance	low	64.65	4.90	**71.52**	3.61	71.89	4.07	59.41	6.97	68.16	4.88	**56.40**	6.13	67.91	5.85
		high	66.15	5.72	**53.75**	7.61	60.46	5.60	68.52	4.27	62.67	5.58	**72.66**	4.07	62.90	4.66
	Frustration	low	39.27	5.34	35.75	4.83	33.54	6.46	35.72	6.93	42.09	5.76	43.41	7.24	44.13	5.54
		high	43.29	7.20	51.35	8.38	48.87	5.79	44.05	5.80	40.25	6.66	39.32	5.41	38.31	6.78

PA = Positive Affect, NA = Negative Affect, N = Neuroticism, E = Extraversion, O = Openness, A = Agreeableness, C = Conscientiousness
Bold = $p < 0.05$

Table 6.8 Mean Perceived Workload Demands on Days One and Two Between Volunteers Classified as Low and High for Personality and Mood Characteristics

6.3.5.7 Familiarity to the Stressor

Table 6.9 presents the mean perceived workload demands for volunteers on days one and two. No significant differences were observed for any facet of perceived workload, further, very little differences in mean scores were observed. Perceived performance on day two was however significantly greater ($t_{(48)}$ –3.09, $p < 0.01$).

		Mean	N	Std. Deviation
Mental Demand	Day 1	88.6878	49	26.8885
	Day 2	84.3122	49	29.0171
Physical Demand	Day 1	15.5041	49	17.8688
	Day 2	16.6286	49	16.5282
Temporal Demand	Day 1	78.8912	49	28.8302
	Day 2	76.5796	49	32.2736
Effort	Day 1	76.6163	49	25.1876
	Day 2	75.5341	49	31.4945
Performance	Day 1	**55.3184**	49	28.3093
	Day 2	**65.3571**	49	25.9112
Frustration	Day 1	44.7592	49	30.9191
	Day 2	41.1592	49	30.6073

Table 6.9 Familiarity to the Stressor: Perceived Workload Demands on Days One and Two

6.4 Discussion

6.4.1 Overview

This study attempted to address several factors. Firstly, replications of the study one analyses were conducted. Secondly, the effects of saliva volume upon S-IgA reactivity were assessed. Although results indicate that increases in S-IgA can be attributed to the stressor, the influence of saliva volume was viewed as influential on all S-IgA measurements. As such, all analyses were re-conducted using S-IgA secretion rates. These analyses included the relationships with health status, perceived workload personality and mood characteristics.

The study also assessed S-IgA reactivity (in relation to the aforementioned factors) on two separate occasions. This design allowed for further assessment of the S-IgA reserve model developed following study one. This model would therefore predict that reactivity would be reduced on the second day due to a reduction in S-IgA in the reserve. These reductions would be greater in individuals who possessed specific characteristics (either health, or personality related). That is, following the first stressor, subsequent reactivity will be further diminished.

The current study, like the thesis as a whole, has assessed many factors and as such has yielded a great deal of data. This discussion will therefore discuss the findings from each of the parts of the results section separately. An attempt will then be made to discuss the relationships between the findings, independently and then in relation to the findings from study one and the developed model.

6.4.2 Sample Demographics

The current sample was taken from a population of undergraduates and staff at the University of Plymouth. As such, the age range is greater than that sampled in study

one. Further, approximately equal numbers of males and females were used in the current study. The majority of the sample were aged between 20 and 40 years. As with study one, the classification of health status was derived using the younger sample health status means. This classification method was deemed appropriate as the within-sample means for the current sample did not differ significantly from the standardised health status means.

6.4.3 Reinforcement of Study One

All of the secondary analyses conducted in study one were replicated using the current data. Although the findings will be briefly discussed here, detail will be limited. That is, section 6.4.4 details the influence of saliva volume upon S-IgA concentrations. Given the finding that saliva volume could mask the true concentrations within a sample, it was decided that all analyses should be conducted upon S-IgA secretion rates. As such, study two analyses were conducted on S-IgA secretion rates and will be discussed in section 6.4.5 – 6.4.8.

With regards to S-IgA concentrations, as with study one, no significant relationships were observed between health and S-IgA in the whole sample. However, a mixed pattern of results were observed with regards to severity of health on each of the MHC clusters and S-IgA reactivity. It was hypothesised that volunteers classified in poor health with regards to frequencies of complaints would demonstrate reduced S-IgA reactivity than did those in good health. Further, on day two, these reductions would be more apparent due to an accumulation of stress and hence greater reductions in the S-IgA reserve.

In study one, there was trend for volunteers in poor health to demonstrate reduced S-IgA reactivity for the majority of MHC clusters. A replication of this pattern was

therefore expected in the current data. Moreover, this pattern was expected on day two, but owing to an accumulation of stress, the reactivity was expected to be reduced still further.

With regards to the observed reactivity on day one, for the clusters of; total ill-health, stress-related, gastric, urinary-tract and microflora complaints, volunteers classified as in poor health with regards to frequencies of contributory complaints demonstrated greater S-IgA reactivity than did those in good health. Further, this pattern was also evident for the cluster of atopy, however, as in study one, this pattern could be attributed to differential effects upon the Th1-Th2 balance. In contrast the expected pattern of reactivity was observed for the clusters of psychological and immune challenge complaints.

These mixed patterns can be attributed to the influence of saliva volume (see section 6.4.4). That is, it was noted that saliva volume was very low at pre-test on day one. As a result, S-IgA concentrations were artificially elevated, and further, masked the true magnitude of post-stress reactivity. Such an influence undoubtedly influenced the observed patterns of reactivity on day one.

On day two however, the influence of saliva volume was not as great and as such, expected patterns of reactivity were observed. As expected, all volunteers demonstrated reduced reactivity on day two. Further, the expected pattern of reactivity (reduced reactivity in volunteers in poor health) was apparent for the clusters of, total ill-health, ill-health indicators, psychological, immune challenge, and gastric complaints.

However, with the exception of reduced reactivity in all volunteers on day two, no consistency was observed. Moreover, the influence of saliva volume has created

potentially spurious patterns of reactivity. As such it seems inappropriate to compare the current findings with those in study one. Although the same argument could be applied to study one, i.e., saliva volume could create spurious patterns of reactivity, the observed patterns conformed to the predicted patterns of reactivity. Although no saliva volume data is available for study one it is plausible to accept this explanation. Further, given the known influence of saliva volume in the current data, it is more appropriate to compare the patterns of reactivity in study one, with S-IgA secretion rates from the current data. That is, if it is accepted that the trends observed in study one are real, and not an artefact of unknown factors, similar patterns should be observed with regards to S-IgA secretion rates in the current study. The following section therefore details the influence of saliva volume upon S-IgA concentrations. In response to the findings, the remaining sections focus upon S-IgA secretion rates, and patterns of reactivity in relation to the other assessed factors.

6.4.4 Saliva Flow Rates, S-IgA Concentrations and S-IgA Secretion Rates

Post-test increases in saliva volume were observed following a five minute exposure to the Synwork battery. The Synwork protocol used in study one is identical to that used in the current study. It is therefore likely that similar patterns of saliva volume (i.e., post-test increases) would have been observed in the study one sample. Although there is no saliva volume data from study two, the increase in saliva volume observed in the present study indicate that increases in S-IgA cannot be attributed to decreased saliva flow.

Post-stress increases in saliva volume suggest that increases in S-IgA concentrations in response to the stressor are not simply an artefact of reduced saliva flow. However, in the absence of saliva flow data regarding S-IgA concentrations should be viewed cautiously. That is, although the present finding suggests an increase in saliva volume

following the stressor, pre-test IgA concentrations could also be influenced by saliva volume. For example, even in the absence of a stressor, an individual's S-IgA concentrations could be influenced by the amount of saliva that they are producing at the time of sampling. Saliva flow and S-IgA secretion rate are independent processes (Hucklebridge *et al.*, 2000), and as such it is the interaction of both of these process that are observed following exposure to a stressor. The modulation of these processes will vary between individuals, i.e., some people may demonstrate post-exposure increases in saliva volume whereas others may demonstrate decreases, and apparent reductions or increases in S-IgA levels respectively in response to the stressor. Given the individuality of saliva flow and S-IgA concentrations at rest and in response to stressors, subsequent analyses will assess S-IgA secretion rates, therefore accounting for both S-IgA concentration and saliva volume.

6.4.5 S-IgA Reactivity (Secretion Rates)

A significant post-stress increase in S-IgA secretion rate was observed on day one. On day two, reduced reactivity was observed, however, the stressor still elicited an increase in S-IgA albeit not significant. The increase on day one is analogous with that observed in study one, lending further weight to the argument that saliva volume did not influence the observed reactivity unduly in study one. The stressor therefore demonstrated the expected increases in S-IgA, however, mean reactivity was diminished in all volunteers, regardless of status on day two. This reduction in reactivity could be attributed to the action of the S-IgA reserve. That is, owing to an accumulation of stress, there is a reduction in the availability of S-IgA. Alternatively, this reduction in reactivity could be due to familiarity to the stressor. That is, on day two, the stressor is more familiar to the volunteers, as such, it is not as stressful, and therefore does not elicit the same magnitude of S-IgA reactivity as that following first exposure. These explanations will be discussed further in relation to other factors later in the chapter.

6.4.5.1 S-IgA Reactivity and Health Status

Using S-IgA secretion rates, classifications of volunteers by health status demonstrated similar patterns of reactivity to those observed in study one. That is, on day one, for the clusters of; stress-related, indicators and psychological complaints, there was a trend for volunteers classified as in poor health with regards to frequencies of contributory complaints to demonstrate reduced S-IgA reactivity. However, for the clusters of; total ill-health, atopy, gastric, urinary-tract, microflora and fungal complaints, volunteers in poor health demonstrated greater S-IgA reactivity. As with study one, the greater S-IgA reactivity in volunteers in poor atopic health may be explained in terms of differential effects upon the Th1-Th2 balance (i.e., a shift away from mucosal activation to Th2 in atopic individuals). This concept can also be applied to the total ill-health cluster. That is, atopic individuals behave contrary to prediction. As such, if atopic scores are taken away from the total ill-health cluster, then those classified as in poor health with regards to frequencies of total ill-health complaints (less atopy) demonstrate reduced reactivity.

Regardless of S-IgA reactivity on day one, reactivity on day two conforms, in the main, to prior predictions. That is, mean S-IgA reactivity on day two was reduced when compared to reactivity on day one. These reductions on day two can be explained in terms of the S-IgA-reserve concept or through familiarity to the stressor. Both are plausible, but the latter cannot account for conformity to a second prediction. That is, as predicted, in the majority of clusters there was a trend for volunteers in poor health to demonstrate reduced S-IgA reactivity when compared to those in good health. Further, for the clusters of psychological and immune-challenge complaints, volunteers in poor health demonstrated post-stress down-regulation of S-IgA on day two. Whilst familiarity to the stressor can account for a general reduction in S-IgA following subsequent exposures, there is no obvious explanation as to why this reduction should be more pronounced in volunteers classified as in poor health.

The S-IgA reserve concept can account for this discrepancy. That is, poor health volunteers have a reduced S-IgA reserve and as such have a diminished capacity to respond to a stressor. Further, this discrepancy is greatest, or more apparent on day two, owing to an accumulation of stress. That is, poor health volunteers are more sensitive to challenges to the reserve, and following an accumulation of stress, the diminished reserve becomes apparent.

With regards to the remaining clusters, S-IgA is influential in the common mucosa, and as such, these discrepancies should be apparent in all clusters. However, it is of note that these discrepancies are most apparent in the clusters where S-IgA is very influential, i.e., immune-challenge complaints (the focus of the bulk of prior research in this area), psychological and stress-related complaints (both manifested following stress), and changes in S-IgA appear either directly or as a bi-product of the complaints. It could therefore be the case that those clusters that do not conform to prior predictions do not efficiently represent the action of S-IgA to the same extent as those clusters where S-IgA is dominant. This could he the case for those clusters that have shown inconsistent patterns of reactivity in both the current and former study (i.e., urinary-tract, microflora and fungal complaints). However, given the exploratory nature of this research, it is viewed as appropriate to maintain these clusters as a basis for further analyses.

6.4.5.2 S-IgA Reactivity, Perceived Workload and Health Status

Perceived workload was assessed in relation to S-IgA reactivity and health status. Although the latter of these analysis omits S-IgA, it creates a consistent approach to analysis. That is, health status is used as the basis for all analyses, therefore all factors can be assessed in those classified as in either poor or good health, and relationships subsequently developed between factors. The reader is reminded that analyses

regarding perceived workload demands assessed by health status are presented in section 6.3.3 (replication of study one). That is, although S-IgA secretion rates are now the focus of analyses, classifications by health status, in relation to all other factors are the same regardless of the S-IgA measurement technique.

Firstly, in the main, consistent relationships were observed between magnitude and direction of S-IgA reactivity (secretion rates) and facets of perceived workload on both days one and two. That is, negative relationships were observed, i.e., those volunteers who reported the greatest workload demonstrated the lowest S-IgA reactivity. In the main, these relationships were not significant, but this pattern was particularly salient for frustration on day one, that is, those volunteers who demonstrated the greatest S-IgA reactivity perceived the task to be less frustrating. This pattern was also apparent on day two, but the relationship was not as salient, possibly as a result of a reduction in perceived frustration.

Secondly, consistent differences were observed between volunteers classified as in either good or poor health with regards to their perceived workload demands on both days one and two. With the exception of the clusters of microflora and fungal complaints, consistent differences were observed between the clusters across both days. That is, volunteers classified as in poor health perceived greater workload demands following the stressor on both days one and two. In particular, on day one, volunteers in poor health for each of the clusters perceived greater frustration than did those in good health. Further, perceived workload was particularly great for those volunteers classified as in poor health with regards to immune challenge complaints and ill-health indicators, who perceived the tasks to be more mentally and temporally demanding and requiring more effort. Similar salient patterns were observed on day two, however,

volunteers in poor health for each of the clusters consistently reported the task to require more effort.

Greater perceptions of demand were therefore associated with poor health for the majority of ill-health clusters. That is, individuals in poor health generally perceived the tasks to be more demanding, in particular, poor health volunteers perceived the task to be more frustrating. As previously discussed, poor health is related to reduced S-IgA reactivity to acute stress. Although relationships between S-IgA and reactivity and perceived workload revealed no consistent trends, it can be stated that volunteers in poor health perceive greater workload demands and subsequently demonstrate reduced S-IgA reactivity. These relationships are complex as it may be assumed that regardless of health status, more demanding tasks may elicit greater reactivity owing to activation of the immune system. The observed reductions in these volunteers could occur as a result of the tasks being more demanding than arousing, that is, arousal may elicit up-regulation of S-IgA, however, in this instance, the task is being perceived as more than arousing, hence some kind of immune suppression has led to reductions in reactivity.

It must be remembered that none of these factors are being viewed in isolation (this will become more apparent when the influence of mood and personality are introduced). That is, all of these contributory factors are interacting to produce the observed patterns of S-IgA reactivity. As such, at this point, the only clear observation that can be made is that volunteers in poor health perceive the task to be more demanding, and also demonstrate reductions in S-IgA reactivity. The interaction of all the contributory factors will be discussed in a summary at the end of this chapter.

6.4.5.3 S-IgA Reactivity, Personality & Mood

Firstly, S-IgA reactivity was assessed in relation to personality and mood factors independently of health status. A mixed pattern of significant results were observed. A trend was apparent for volunteers scoring high in agreeableness to demonstrate greater S-IgA reactivity. This finding is analogous with the findings of Coon *et al.*, (1995). The authors reported higher S-IgA in volunteers classified as being high in confidence and low in denial following a musical examination. They attribute higher S-IgA to possession of 'toughness' (Deinstbier, 1989), that is, possession of traits that predispose individuals to either positive or negative immune reactivity to stress. Individuals who demonstrate 'toughness' possess traits / characteristics that make them more resilient to stress. They find stress arousing and demonstrate enhanced immune activity. The trait of agreeableness is likely to be characterised by such enhancement, i.e., such agreeable individuals are not defensive in response to stimuli, and as such demonstrate a trend for greater S-IgA reactivity following acute stress.

The most salient differences in S-IgA reactivity occurred between volunteers classified as either low or high in negative affect (NA) and neuroticism. That is, on both days, volunteers classified as high in NA demonstrated greater S-IgA than those classified as low in NA. This finding is analogous with that of Evans *et al.*, (1993), where following within-sample analyses, higher S-IgA was related to greater frequencies of daily hassles. That is, although the present study assessed reactivity to a stressor, the increased reactivity in high NA volunteers is analogous with the higher S-IgA observed in those who had experienced more hassles in Evans *et al.*, i.e., Evans' hassles could be viewed as stressors, and therefore elicit similar reactivity to the current stressor, hence higher S-IgA observed in Evans *et al.*, and greater reactivity observed in the present study. In contrast however, volunteers classified as high in neuroticism demonstrated lower S-IgA reactivity than those classified as low.

Although NA and neuroticism are closely related, and further, neuroticism is a large contributor to NA (Costa & McCrae, 1988), classifications on these factors elicited very different patterns of reactivity. It could be assumed that greater reactivity is associated with lower pre-stress S-IgA, and therefore a greater capacity to respond to stress. That is, the greater reactivity in high NA and low neuroticism volunteers could be attributed to lower pre-stress S-IgA. However, inspection of the pre-stress S-IgA levels reveals very little difference in pre-stress levels between those classified as either low or high in either trait, or further, between the traits (e.g., those classified as low in NA and low in neuroticism). Further, it was assumed that volunteers high in such traits would have higher S-IgA levels due to the arousing nature of the traits (i.e., both NA and neuroticism can result in over attendance to stimuli, and therefore constant S-IgA reactivity in response to the stimuli).

It is this over attendance to stimuli which is forwarded as an explanation of the differing patterns of reactivity observed in these supposedly similar factors. Firstly, differences between NA and neuroticism must be established in order that the elicited differences in S-IgA reactivity can be assessed. NA is a 'state' measurement and therefore reflects how volunteers felt at the time of experimentation. Moreover, NA is context specific and therefore reflects individuals' specific response to the particular situation, not life in general. Further, the measurement of NA in this study may also be specific to the stressor. That is, S-IgA reactivity in high NA volunteers may reflect how they respond to a particular kind of stress, e.g., work stress, and as such, may not be an adequate reflection of how they deal with stressors in general. Unfortunately, the only way to adequately assess this suggestion would be to make comparisons between high NA volunteers and low neurotic volunteers. As previously mentioned, these factors are highly associated, and as such, a classification of this kind would not be of significance owing to the small sample sizes.

In contrast, neuroticism is a trait measurement and is therefore context independent. That is, unlike NA, neuroticism reflects how individuals may respond to life in general. As such, neuroticism is less specific than NA especially given the suggestion that reactivity in high NA volunteers could be attributed to their response to a work related stressor only. As such, the general effect of neuroticism appears to be reduced reactivity, or moreover, a reduced capacity to respond to acute stress. This could occur as a result of continual over-attendance to stimuli and a subsequent depletion of their S-IgA reserve, hence the reduced reactivity observed in neurotic volunteers.

6.4.6 Health Status, Personality & Mood

Using the health status classifications personality and mood scores were compared in those volunteers classified as in either good or poor health with regards to each of the MHC clusters. With regards to mood, as would be expected, there were no differences in positive affect (PA) between those classified as in either good or poor health. However, in line with previous research (e.g., Costa & McCrae, 1988, Watson & Pennybaker, 1990), volunteers classified as in poor health, for the majority of MHC clusters reported greater levels of NA. These differences were most apparent for the clusters of; total ill-health, stress-related, indicators of ill-health, psychological and immune challenge complaints. Similarly, this pattern was also observed for the trait of neuroticism, i.e., volunteers in poor health were more neurotic than those in good health.

These differences are analogous with previous research which has attempted to establish the direction of causality between negative affectivity (including neuroticism) and ill-health (*c.f.*, Chapter 2). One argument for the link is that of the symptom perception hypothesis, whereby, negative affect serves as a nuisance variable in health related research. That is, volunteers with high negative affectivity are more likely to perceive

symptoms, and moreover report them. In the current study, there was a consistent trend for volunteers in poor health to be more open. One interpretation of this finding is that the observed differences lend further weight to the symptom perception hypothesis.. That is, the observed relationships between trait / state and health could simply be an artefact of likelihood to report - if poor health volunteers are more open, then they are more likely to disclose information regarding their health status. However, a true link between trait / state and health cannot be totally discounted. That is, poor health volunteers may actually experience these illnesses, and being more open simply leads to them being more likely to report and discuss such matters. However regardless of the direction of causality between ill-health and facets of NA, the previous section demonstrated that these states / traits also play a role in S-IgA reactivity. That is, specific traits have been implicated in both poor health and reduced S-IgA reactivity. Although these links have been assessed independently the relationships between these factors have also been assessed through classification by health status. Although this method of classification may seem convoluted, and the direct link between state / trait and S-IgA reactivity is lost, this method of classification has been consistently applied. Further, the reader is reminded that the degree of association between ill-health and facets of NA is high, as such, there is much overlap between individuals classified as in poor health, and those classified as high in NA and neuroticism.

6.4.7 Familiarity to the Stressor

In the current study, the same stressor was administered on two occasions 24 hours apart. This design attempted to test the suggestion that novel stimuli produce greater S-IgA reactivity than those which are more familiar (Willemson *et al.*, 2000). Willemson and colleagues reported greater S-IgA and cardiovascular reactivity following exposure to a novel stressor (mental arithmetic) than following subsequent exposures. Similarly, the S-IgA reserve model would also predict diminished reactivity following subsequent

stressors. That is, owing to a reduction in S-IgA available at times of acute stress, subsequent stressors will elicit reduced S-IgA reactivity. Further, the current data has demonstrated that these reductions are more pronounced in individuals classified as in poor health, possibly owing to greater reductions in their reserve. Combined with measures of perceived workload following both stressors, the current design allowed for further assessment of both of these concepts, both of which would suggest reduced S-IgA reactivity following the second stressor.

As predicted S-IgA reactivity was significantly reduced following the stressor on day two. However, reports of perceived workload did not differ greatly between days one and two. Further, no differences were apparent when perceived workload was assessed in relation to personality and mood characteristics. Although the S-IgA data fits that of Willemson and colleagues, the perceived workload data is at odds with their findings. That is, the authors reported no effect of task difficulty on S-IgA, although increases in difficulty were met with increases in perceived difficulty. While there were no differences in perceived workload between the days in the current study, there was a consistent trend for volunteers to perceive greater performance on day two. That is, although there were no differences in perceived workload, volunteers considered their performance to be better on the second day. Although S-IgA reductions on day two can be attributed to familiarity to the stressor, this concept is not supported by subjective reports of the workload demands required by the task.

S-IgA reactivity *per se* is undoubtedly a sub-conscious activity, however, much psychoneuroimmunological research is based upon the premise that conscious activity can moderate S-IgA reactivity. As such, if S-IgA reductions on day two are to be attributed to familiarity to the stressor, then it should follow that volunteers perceptions of stress should also be reduced on day two. Further, data regarding perceived

workload with regards to health status consistently demonstrated that volunteers in poor health perceived the task to be more demanding. It is these poor health volunteers that demonstrate reduced S-IgA reactivity to acute stress, however, as this chapter has highlighted, it is the combination of the assessed factors that contribute to diminished reactivity in certain individuals.

6.4.8 Summary, Conclusions & Recommendations

The current chapter has assessed several factors, all of which were hypothesised to interact and subsequently moderate S-IgA reactivity to acute stress. In this summary the findings will be briefly discussed in relation to the original hypothesis. However, the reader is reminded that many of the findings were not a direct result of the research hypotheses, however, given the exploratory nature of the research it was necessary to discuss the influence of all factors, regardless of whether they were the basis of a priori predictions.

Firstly, with regards to replicating the findings of study one, similar patterns of reactivity were observed in relation to classification of health status. That is, in the main, volunteers in poor health demonstrated reduced S-IgA reactivity to both stressors. However, this pattern was not all evident for all MHC clusters. This inconsistency is therefore attributed to the inappropriateness of several factors both in terms of their comprising items and their association with S-IgA. That is, although it is acknowledged that all MHC clusters should demonstrate similar patterns of reactivity through the action of S-IgA on the common mucosa, it is appreciated that S-IgA is more predominant in response to certain clusters (e.g., total ill-health, stress-related, psychological, indicators and immune challenge complaints). It is these clusters that have demonstrated the most salient and consistent patterns of reactivity that conform to the original hypotheses. Further, the precise action of S-IgA is not known in relation to

many complaints and it is known that the immune system can often compensate for deficiencies in the system. This concept has been forwarded as an explanation of the patterns of reactivity observed in relation to atopic complaints, but it is likely that such processes are also evident in other clusters where reactivity inconsistent with the hypotheses have been observed.

Secondly, the effects of saliva volume upon measurements of S-IgA were assessed. This was a purely exploratory process. That is, the precise influence of stress upon saliva volume is specific to both the stressor and the individuals. The effects of the Synwork battery upon saliva volume were therefore assessed in order that changes in S-IgA concentrations in the previous and current study could be attributed to the effects of the stressor not changes in saliva volume. Increases in saliva volume were observed following the stressor allowing for the conclusion that changes in S-IgA can be attributed to the stressor and not increase of S-IgA in a reduced volume of saliva. However, it was noted that both pre and post-stress measurements of S-IgA could be masked by the effects of saliva volume. As such, S-IgA secretion rates were adopted as the primary dependent variable with regards to S-IgA. Using secretion rates, patterns of reactivity with regards to health status conformed to preliminary hypotheses, that is, in the main, S-IgA reactivity was diminished in volunteers classified as being in poor health. However, as with S-IgA concentrations, these trends were not apparent for all MHC clusters. Again it is argued that these discrepancies in findings are due to the inappropriateness, or insensitivity of some health complaints towards the action of S-IgA. This point is further emphasised by the clarity of the findings concerning those clusters where S-IgA is expected to be predominant.

Thirdly, patterns of reactivity with regards to mood and personality were mixed. It was hypothesised that states / traits where over-attendance to stimuli, and therefore increased

arousal, would lead to reduced reactivity to acute stress. That is, a key factor in such traits (e.g., negative affect and neuroticism) is over-attendance to stimuli. This over-attendance leads to increased arousal and in the short term increased S-IgA reactivity. However, using the S-IgA reserve concept, continual arousal will deplete the S-IgA reserve. That is, the need for S-IgA in response to acute stress will outweigh production, and as such the S-IgA reserve will be depleted. Indeed, if this is the case, such a model can account for the increased frequencies of ill-health in individuals high in these states / traits, i.e., such individuals deplete their reserve and are therefore more susceptible to subsequent complaints. Specific S-IgA will be produced in response to these subsequent infections, however, continual arousal will deplete this reserve, resulting in a vicious circle of ill-health in such individuals.

Although it is generally acknowledged that neuroticism is a large contributor to negative affect, different patterns of reactivity were observed in volunteers classified as high in these traits. That is, while neurotic volunteers conformed to the hypothesis that they would demonstrate reduced S-IgA reactivity, volunteers high in negative affect demonstrated greater S-IgA reactivity. This discrepancy can be explained using the concept of continual depletion. That is, neuroticism is a trait measure and as such, neurotic volunteers frequently over-attend to stimuli, therefore depleting their reserve. In contrast, negative affect is a state measure and is recording negative affect at the time of testing. Volunteers high in negative affect are also likely to over-perceive to stimuli, in this case, the manipulated stressor. As such, they demonstrate greater S-IgA reactivity due to increased arousal. However, it is proposed that it is this pattern of reactivity which will eventually deplete the S-IgA reserve, i.e., continual arousal to an individual stressor. It is therefore suggested that volunteers high in negative affect are in fact analogous with those high in neuroticism, however, the sensitivity of the PANAS and NEO-FFI have picked up on the differences between state and trait, and as such,

differences in S-IgA reactivity. The short-term differences in reactivity, and the proposed longer term similarities between negative affectivity and neuroticism can be explained in terms of over-attendance to stimuli leading to increased arousal. However, the chronic trait of neuroticism can be viewed as the result of an accumulation of negative affect. That is, continual arousal as a state measurement leads to continual depletion of the S-IgA reserve. This depletion is manifested in the reduced reactivity observed in neurotic volunteers, i.e., the chronic result of negative affectivity.

The argument that these individuals are similar in nature is further compounded by the personality and mood data. That is those volunteers classified as high in negative affect and those high in neuroticism are characterised by increased frequencies of health complaints. Such susceptibility to ill-health could be brought about by the depletion of the S-IgA reserve and therefore increased vulnerability to subsequent ill-health. Both groups therefore being caught in the previously mentioned vicious circle of ill-health.

With regards to perceived workload demands, no direct relationships were observed between S-IgA reactivity and perceived workload. However, consistent trends were observed with regards to both personality and mood and health status, both of which have demonstrated both direct and indirect moderating effects upon S-IgA reactivity. In general, volunteers in poor health perceived greater workload demands from the task than those classified as in good health. Similarly, the state / traits of negative affectivity and neuroticism demonstrated consistent trends with regards to perceived workload. That is, volunteers high in these factors consistently perceived greater workload demands. In contrast, the traits of agreeableness and conscientiousness demonstrated the opposite. That is, volunteers high in these traits perceived lower workload demands. This is somewhat surprising for the trait of conscientiousness, i.e., it would be expected that conscientious individuals would perceive greater demands and they would be

striving to perform at a consistently high level, however, it is apparent that those low in conscientiousness found the task harder and perceived greater demands accordingly.

Although complex, all of these factors are seen as contributing either directly or indirectly to S-IgA reactivity to acute stress, the direction and magnitude of which will subsequently influence subsequent health and mood / personality. As previously discussed, all of these factors have been assessed in direct relation to S-IgA reactivity. However, in order that a consistent method of classification is applied (i.e., as far as possible, the same volunteers are compared in each analysis), at time, the links can appear to be very convoluted. Despite these process adding to the complexity of the overall picture, this method does allow an overall assessment of factors, that in combination, predispose individuals to specific patterns of immune reactivity in response to acute stress. As such, the findings of the current study can be added to the S-IgA reserve model. As mentioned in Chapter five, the model was initially developed in attempt to clarify the often complex relationships between factors and how they interact to moderate S-IgA reactivity. However, the current health status data have provided more support for the concept of an S-IgA reserve. Further, the current data on mood and personality can also be viewed as making valuable contributions to the model. That is, consistent trends have been observed between personality and mood both directly with S-IgA reactivity, and indirectly through associations with health status. Figure 6.24 presents the S-IgA reserve model which now includes the moderating effects of mood and personality. Although the model should primarily be viewed as a graphical representation of the complex data, increasing support is being provided for the interaction between factors and their moderating effect upon S-IgA reactivity to acute stress.

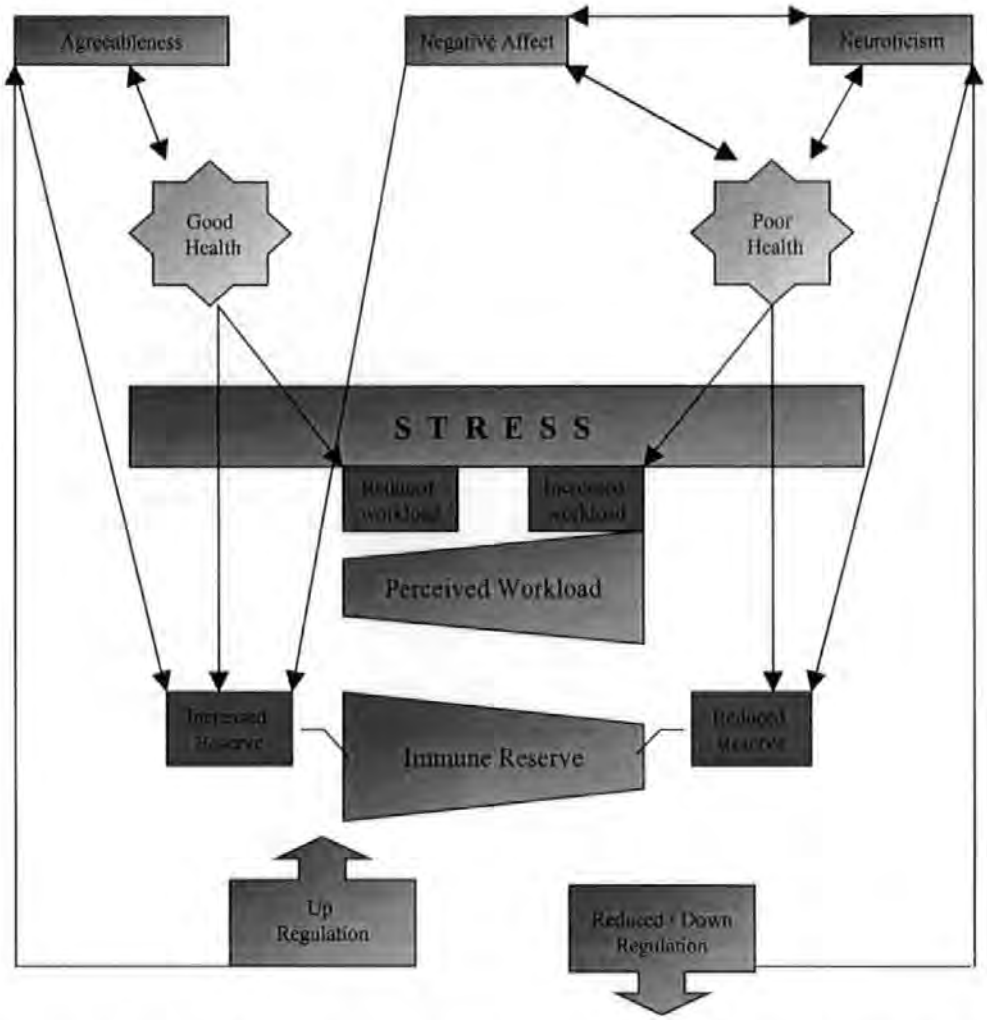


Figure 6.24 S-IgA Reserve Model (Including health status, personality, mood and perceptions of stress

Figure 6.24 shows the observed relationships between the assessed factors. In addition, the dashed line connecting negative affect to increased capacity represents a hypothesised temporary relationship, i.e., it is suggested that such individuals will deplete their reserve due to over attendance to stimuli, thus leading to ill-health. This is emphasised by the observed link between negative affect and poor health.

It must be remembered that many of the discussed relationships are merely trends, however, their consistency both within factors and with regards to preliminary hypotheses suggest their potential importance. Although many highly significant results have been observed (most notably with regards to S-IgA reactivity, and relationships between personality and health status) the basis of the S-IgA reserve model is based on

trends alone. Although to reiterate, the consistency of these trends in both the previous and the current study, and moreover similar patterns of reactivity between health clusters, suggest that the observed relationships are of theoretical importance. Given this support for the model, in particular the importance of retrospective health status upon S-IgA reactivity, potential causes of lack of significance must be addressed.

Firstly, the derived clusters in the MHCQ could be one cause. As previously discussed, several factors do not demonstrate the expected pattern of S-IgA reactivity, namely, atopy, gastric, urinary-tract, microflora and fungal complaints. While the reverse reactivity in the atopic cluster may be explained in terms of the Th1-Th2 balance (c.f. chapter 5), no obvious explanation can be offered for the other clusters. However, one plausible explanation could be that S-IgA in not equally influential in all the derived clusters of ill-health. That is, the most salient (albeit not significant) trends were observed in clusters where S-IgA is known to play an influential role, either through moderation by psychosocial factors or as a direct link in the protection against specific complaints. In contrast, less research has been conducted upon S-IgA and vulnerability towards gastric, urinary-tract, microflora and fungal complaints. Although S-IgA is active in the common mucosa, and as such should play a role in defence against a wide variety of clusters, S-IgA is undoubtedly more influential in defence against certain pathogens / complaints(most likely those complaints present in the clusters demonstrating the most salient patterns of expected reactivity). The immune system also compensates for deficits in the network, some of which may be more apparent in these clusters, i.e., other immune activity may be primarily involved with dealing with such complaints (e.g., macrophage activity in response to microflora complaints). These are therefore potential causes for reactivity contrary to prediction in these clusters. However, as previously mentioned, this is very exploratory research and as such, the

knowledge gained from the inclusion of all clusters, and indeed all factors of theoretical interest could yield beneficial results.

Secondly, the concept of an S-IgA reserve is, as the name suggests, based upon the action of a reserve of immune resources, in this case S-IgA. Moreover, the concept is based upon what factors may moderate the depletion of a reserve, and what consequences this has for subsequent health status. This concept was developed using data from one stressor. In order to assess the model further the current study administered two stressor in an attempt to deplete the reserve. There was a trend for reactivity to be reduced following the second stressor, and the fact that this reduction was greater in poor health volunteers implies some influence other than that of familiarity to the stressor. However, despite consistency in the trends (in those clusters now considered to be most appropriate for S-IgA assessment), no significance was observed.

The reader is reminded that the second stressor was administered 24 hours after the first stressor. Whilst this design is appropriate in assessing consistency of reactivity on two separate occasions, the time delay was too long to effectively exhaust an S-IgA reserve. That is, fluctuations in S-IgA are very transient, and as such S-IgA is likely to have fluctuated in response to a wide variety of other environmental stressor in the 24 hour period. For example, some volunteers may have spent the 24 hours engaged in arousing activity, whereas other may have participated in relaxing activities. Despite these interim activities, the design still elicited consistent patterns of reactivity on both occasions (i.e., those in poor health demonstrated lower reactivity on both days), and therefore to some extent the results can be interpreted as a depletion of a reserve. However, it is now acknowledged that to fully assess the S-IgA reserve model,

volunteers must be exposed to cumulative stress, that is, in the absence of any other stimuli.

Such a design has been utilised in rat research (Carpenter, Garrett, Hartley & Proctor, 1998), and indeed their results could be viewed as a depletion of some sort of reserve. Rats were repeatedly exposed to nervous stimulation by way of bipolar electrodes. Following initial stimulation high outputs of IgA were observed. This output is attributed to the release of an accumulation of IgA in the ductal system. However, IgA release is greatly reduced following subsequent exposures to nerve impulse. Their research also provides further support for the concept of an S-IgA reserve. That is, they suggest that in the absence of stimulation, IgA may be synthesised and secreted at a rate that exceeds demand and as such accumulates in the ductal system until stimulation evokes release. It is this accumulation in the ductal system that is analogous with the hypothesised S-IgA reserve in humans.

As such, the next study will attempt to conduct a cumulative stress study on human volunteers. Such a design will effectively test the S-IgA reserve concept, that is volunteers, will be in isolation and exposed to repeated stress. As such, other factors that may have exerted effects in the interim period in the current study will be eliminated. Further, the hypothesised effects of over-attendance on S-IgA reactivity, can also be fully assessed using a cumulative stress design. This point is particularly pertinent for negative affectivity, where it is predicted that following an accumulation of stress, high negative affectivity will demonstrate patterns analogous with neuroticism, i.e., reduced S-IgA reactivity.

7. Study Three

7.0 Chapter Overview

The primary objective of the current study was to explore further the concept of an S-IgA reserve. This concept was developed in order to explain the findings of study one, and further assessed in study two. However, given that this research is dealing with acute changes in S-IgA, the 24 hour period between sessions in study two was considered to be too long when attempting to deplete an S-IgA-reserve. The current study therefore adopted a cumulative stress paradigm. The paradigm was adapted from that used by Carpenter *et al.*, (1998). That is, given the existence of a deficiency in the ability to respond to stress, perhaps brought about by a reduced S-IgA reserve / capacity, it is essential to attempt to deplete this reserve *in situ*. Such a design would therefore avoid the influence of other environmental stimuli that could moderate reserve activity, which are likely to have played an influential role in the previous study. As with the previous studies, the current study also assessed the differences between good and poor health volunteers with regards to mood and perceived workload characteristics, as well as the independent influence of these factors upon S-IgA reactivity.

As predicted by the S-IgA reserve model, volunteers in poor health demonstrated reduced S-IgA reactivity when compared to volunteers in good health. Moreover, poor health volunteers demonstrated progressively poor S-IgA reactivity and in some cases, down-regulation of S-IgA following cumulative stress. The differences in S-IgA reactivity between good and poor health volunteers are discussed in relation to the S-IgA reserve model. That is, volunteers in poor health demonstrate reduced S-IgA reactivity to acute stress, however, S-IgA reactivity becomes poorer following cumulative stress. The combination of the current stressor and the cumulative stress

paradigm seems to be sensitive enough to tease out differences between healthy volunteers with regards to S-IgA reactivity.

7.1 Introduction

7.1.1 The S-IgA Reserve & Cumulative Stress

The previous studies have demonstrated a consistent, but non-significant trend. That is, volunteers in poor health have consistently demonstrated reduced S-IgA reactivity to acute stress when compared to volunteers in good health. It is acknowledged that in the previous study, all volunteers demonstrated reduced S-IgA reactivity to the second stressor when compared with the stressor on day one, and as such, reduced S-IgA reactivity can in part be attributed to familiarity to the stressor, and a subsequent reduction in ANS activation. However, familiarity to the stressor cannot account for the fact that greater reductions in S-IgA reactivity were observed in poor health volunteers. Information regarding health status and mood / personality suggests that the observed reductions could be attributed to a complex interaction between perceptions of workload (i.e., how individuals perceive the task) mood, personality and health status. That is, poor health volunteers also possess other characteristics that are independently associated with lower S-IgA reactivity to acute stress (negative traits and greater perception of workload demands). The association of these factors as mediators in the S-IgA response to stress will be discussed in more detail in the final chapter.

Regardless of the interactive nature of the assessed factors, the S-IgA reserve model can still be applied to data regarding health status and to some extent, mood. Study two attempted to exploit this reserve by administering the same stressor on two occasions. However, as previously discussed, such a design was not adequate in exploiting the reserve owing to the possible influence of external factors between stressor one and two. In order to fully exploit a reserve, volunteers must be assessed in one session, therefore

avoiding the influence of external factors. Further, the S-IgA reserve model is based upon the premise that certain individuals have a reduced reserve (or an inability to respond perhaps as a result of a dysregulated system). To fully exploit this reserve, volunteers must therefore be subjected to continuous stress, in order that a depletion can be observed. As the current research is dealing primarily with acute stress, it would not be appropriate to extend the duration of the stressor (*c.f.*, Chapter Two). That is, in order to fully explore the effects of the current acute stressor, it is more appropriate to continue with the same time duration of stress utilised in previous studies. The concept of cumulative stress in the current study comprises the cumulative effects of several acute stressors (of the same duration as administered in the previous studies).

Although such a paradigm has not been used in human research before, the paradigm is analogous to that utilised by Carpenter *et al., (1998)*. Moreover, the concepts, and further, the findings of Carpenter *et al.*, (1998) are analogous with the findings and concepts of the current body of research. In an attempt to isolate the mechanisms driving S-IgA release in response to stress, Carpenter *et al.*, (1998) utilised a cumulative stress paradigm (of sorts) to rats. The authors wished to explore the effects of sympathetic and parasympathetic nervous stimulation upon S-IgA reactivity. Although such a concept could be explored using continuous stimulation, previous research (Anderson, Garrett & Proctor, 1988) demonstrated that continual sympathetic stimulation usually resulted in damage to S-IgA secreting glands, and could mask the true mechanism of S-IgA secretion. Part of their final paradigm was analogous with that developed in the current study. That is, while parasympathetic stimulation was delivered continuously, sympathetic stimulation comprised bursts of high frequency activation (once every 10 seconds).

With regards to the sympathetic stimulation (most analogous with the current stressor), the authors observed a high concentration of S-IgA following the first stimulation, and reduced S-IgA reactivity following subsequent stimulation periods. The authors attributed the high S-IgA concentration following the first stimulation to an accumulation of S-IgA in the period of anaesthesia prior to stimulation. They further suggest that the basal secretion of S-IgA, in the absence of stimulation, is responsible for the accumulation of IgA within the ductal system. With regards to S-IgA secretion, they suggest that IgA is continually synthesised and secreted at a rapid rate by plasma cells, however, the rate of secretion increases during stimulation. Further, in the absence of stimulation, IgA production and secretion may always be in excess of demand, resulting in an accumulation of S-IgA. During stimulation, when demand increases, this S-IgA is rapidly transported into saliva. That is, the accumulated IgA is "washed out" of the ductal system into saliva.

The concepts and moreover the terminology arising from the work of Carpenter *et al.*, (1998) is of vital importance and could be interpreted in terms of an S-IgA-reserve. Thus far, the difficulties in describing the reserve are of mechanism and location. As a consequence, no information regarding the location of the reserve has been put forward, moreover, the model has been used as a method of explaining the data observed in the current body of work, i.e., certain individuals (usually those in poor health or in possession of negative traits / states) demonstrate a consistent inability to secrete S-IgA of the same magnitude of those in good health (or with positive states / traits). However, the work of Carpenter *et al.*, (1998) has demonstrated the existence of specific mechanism that are key to the existence of some sort of S-IgA reserve. Most importantly, Carpenter *et al.*, describe the accumulation of S-IgA (under anaesthesia) in the ductal system. It is this ductal system which is analogous with the hypothesised S-IgA reserve in the current body of work. Further, following initial stimulation, the

authors describe the high S-IgA concentration as a "wash out" from the ductal system into saliva, in response to stimulation. Following this "wash out" further stimulation results in reduced S-IgA release into saliva, i.e., the accumulation of IgA in the ductal system has been depleted. These concepts are therefore key to the existence of an S-IgA reserve. Further, the work of Carpenter *et al.*, (1998) suggest that the S-IgA reserve, described thus far in this body of work, may be more accurately described as the ductal system. If this is indeed the case, then the S-IgA reactivity observed following acute stress reflects the accumulation of S-IgA (prior to experimentation) in the ductal system (S-IgA reserve).

The work of Carpenter *et al.*, (1998) is therefore of vital importance to the current body of work, and more importantly, to the hypothesised existence of an S-IgA reserve. However, this being the case, the questions remains, what factors are mediating the rate of depletion from the reserve, or indeed, the rate of accumulation in the reserve prior to stimulation. That is, the S-IgA reserve model is based upon a reserve, or accumulation of IgA, which is secreted into saliva (and other secretions of the common mucosa) following stimulation, in this case acute stress. However, the S-IgA-reserve model was developed in response to the finding that, in the main, volunteers in poor health demonstrate reduced S-IgA reactivity to acute stress when compared with those in good health. This difference therefore implies that firstly, there is a reserve of some kind (analogous with the ductal system suggested by Carpenter *et al.*, 1998), and secondly, for one reason or another, the reserve is dysregulated in poor health volunteers. This dysregulation could lie in a lack of accumulation of IgA, or a fault in the mechanism driving the release from the reserve to saliva following acute stress in poor health volunteers. Further, the dysregulation may occur as a result of an interaction between a host of factors which effect perceptions of stress (i.e., mood, personality, coping styles) and therefore influence S-IgA activity and subsequent susceptibility to illness.

The work of Carpenter *et al.*, (1998) therefore provides strong support for the existence of an S-IgA reserve. As such, a cumulative stress paradigm will allow for this reserve to be depleted *in situ*. Further, if, for whatever reason, volunteers in poor health (or in possession of other negative characteristics associated with reduced S-IgA reactivity) have a dysregulated reserve, the discrepancy in S-IgA reactivity to stress between good and poor health volunteers should be more apparent using the cumulative stress paradigm.

7.1.2 Health Status & S-IgA Reactivity

With regards to the derived clusters of ill-health, all clusters have given rise to consistent trends regarding S-IgA reactivity to acute stress. This pattern of S-IgA reactivity was discussed in detail in the previous chapter, however, to reiterate, it is likely that those clusters demonstrating the most salient and consistent patterns in S-IgA reactivity, comprise those complaints where S-IgA plays a predominant role in illness protection. However, given the exploratory nature of this research, and further, the use of a new paradigm in the current study, it is appropriate to assess all of the derived MHC clusters. That is, it may be the case that consistent trends will manifest in all clusters using the cumulative stress paradigm.

7.1.3 Health Status & Mood

In support of previous work (*c.f.*, Watson & Pennybaker, 1982), study two demonstrated significant relationships between ill-health and negative affect and neuroticism. In contrast, there was a consistent trend for good health to be associated with agreeableness. Given this well established relationship between negative traits / states and ill-health, and conversely, although to a lesser extent, positive states and traits and good health, similar patterns are expected in the current study. That is, positive

relationships are expected between NA and ill-health and PA and good health. It should also be noted, that while the emphasis of the current study is the use of the cumulative stress paradigm, such a paradigm should not effect the expected relationships in any way. That is, measurements of state mood and health status were obtained prior to the administration of the cumulative stress paradigm.

7.1.4 Mood & S-IgA Reactivity

In study two, good health and agreeableness were both associated with increased S-IgA reactivity following acute stress. However, patterns of S-IgA reactivity with regards to negative affect and neuroticism were not consistent. That is, neuroticism was associated with reduced S-IgA reactivity, while negative affect was associated with increased S-IgA reactivity. The increased S-IgA reactivity observed in high negative affect volunteers is analogous with the findings of Evans et al., (1993). That is, Evans *et al.*, (1993) observed higher S-IgA on days with the greatest frequency of daily hassles (analogous with negative affect). Moreover, the contrasting findings in study two were explained in terms of over-attendance to stimuli, and the differences between neuroticism and negative affect with regards to measures of trait and state. That is, negative affect is a state measure, and therefore reflects how volunteers feel at the time of experimentation. Moreover, it was suggested that this measure could be even more specific, i.e., the measurement may reflect how volunteers respond to a particular type of stressor,, in this instance, work stress. Using this concept, certain volunteers demonstrated greater S-IgA reactivity in response to a work stressor.

Thus far, this concept is still analogous with the findings of Evans et al., (1993) that is, in the short-term, negative stimuli (daily hassles), or perhaps more appropriately, arousing stimuli (work stress) elicit up-regulation of S-IgA. However, using the S-IgA reserve model, volunteers would be unable to continue responding in this manner, i.e.,

each response would reduce their reserve, and therefore result in progressive reductions in S-IgA, and possible down-regulation following continued exposure to the stressor. This explanation was therefore forwarded as an explanation of the reduced S-IgA reactivity observed in neurotic volunteers – a trait measure, and therefore analogous with longer-term exposure and response to the stressor.

The current study therefore combines the concepts of the S-IgA reserve, and the possibility of reduced S-IgA reactivity / down-regulation in response to the cumulative effects of stress. That is, the S-IgA reserve model suggests that volunteers would be unable to demonstrate continual S-IgA reactivity (at a similar rate) in response to cumulative stress. As hypothesised with poor health volunteers, following cumulative stress, high NA volunteers should also deplete their S-IgA reserve. Moreover, this reduction could be even greater than that observed in poor health volunteers. That is, high NA volunteers demonstrated great positive S-IgA reactivity in response to the stressor. Such reactivity would leave a deficit in the reserve, resulting in reduced S-IgA reactivity following subsequent stressors, or an accumulation of stress. The adoption of a cumulative stress paradigm therefore enable the reserve to be depleted (or otherwise) *in situ*, that is, in the absence of any external variable which may have influenced S-IgA reactivity between stressors in Study two.

7.1.5 Aims & Hypotheses

Aims: To use a cumulative stress paradigm in order that the hypothesised depletion of the S-IgA reserve (either as a result of poor health or possession of states characterised by over-attendance to stimuli) can be observed *in situ*.

S-IgA Reactivity

Hypothesis One: The greatest S-IgA reactivity will be observed in response to the first stressor. Subsequent stressor will elicit S-IgA reactivity, but will be reduced when compared with S-IgA reactivity to the initial stressor.

Health Status & S-IgA reactivity

Hypothesis Two: All volunteers will demonstrate reduced S-IgA reactivity to cumulative stress, when compared with the initial stressor. However, volunteers in poor health will demonstrate greater reductions / down-regulation following cumulative stress.

Mood & S-IgA Reactivity

Hypothesis Three: High negative affect volunteers will demonstrate reduced S-IgA reactivity following cumulative stress when compared to those with low negative affect.

Health Status & Mood

Hypothesis Four: Volunteers in poor health will demonstrate greater negative affect, whereas volunteers in good health will demonstrate greater positive affect.

Health Status & Perceived Workload

Hypothesis Five: Poor health volunteers will perceive the stressors as requiring greater workload than those in good health.

7.2 Methods

7.2.1 Sample

Twenty undergraduate volunteers were recruited using a departmental participation scheme, whereby, student must participate in experiments for course credit. All experimentation was conducted between the hours of 1000 and 1500 during the month of February, 2001.

7.2.2 Materials

No new materials were utilised in the current study compared with other studies described in this thesis. Retrospective health status was assessed using the MHCQ, state mood was assessed using the PANAS, and perceived workload assessed following each stressor using the NASA-TLX. Further, the Synwork multi-tasking battery was used as the stressor task. Full details of all materials can be seen in Chapter Four.

7.2.3 Experimental Protocol

All volunteers were tested individually between the hours of 1000 and 1500. Upon entry, volunteers were briefed and asked to complete the MHCQ and the PANAS. Volunteers were then given a demonstration of the Synwork battery, and given the opportunity to ask any questions of the experimenter. The rest of the session was divided into three mini-sessions, each one comprising five minutes stressor task, followed by five minutes of passive relaxation in order that volunteers were engaged in stressful activity and relaxation for equivalent periods of time.. Timed saliva samples were obtained before and after each stressor and relaxation period, further, the final stressor was followed by another rest, or recover period of five minutes. The experiment therefore yielded 7 saliva samples for each volunteer. A graphical representation of the experimental protocol can be seen in figure 7.1.

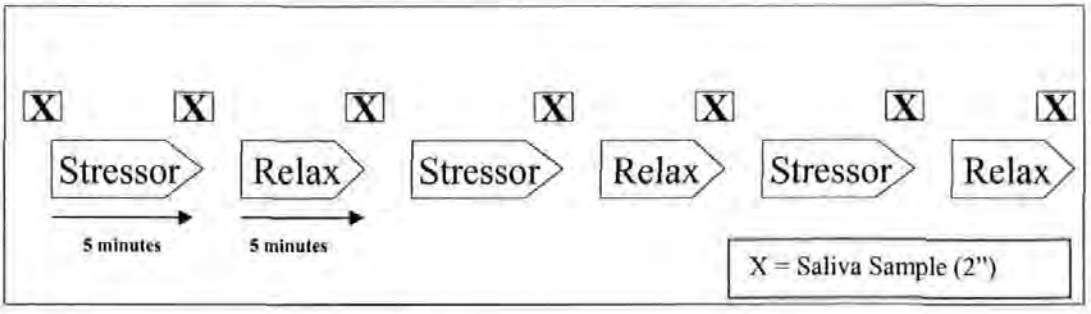


Figure 7.1 Cumulative Stress Procedure

7.2.4 Treatment of Results

As with the previous studies, health status were established using the standardised means for each cluster. However, within sample means were compared with the standardised means to ensure similarity in classification. This is especially important given the smaller sample size in the current study. That is, inappropriate application of standardised means to the current sample could give rise to unequal groups sizes. With regards to mood data (derived from the PANAS), volunteers were classified as either high or low in both negative and positive affect using within sample mean splits.

Within sample S-IgA reactivity was assessed using t-tests for related samples, and differences in S-IgA reactivity with regards to other factors (e.g., Health status) were assessed using t-tests for unrelated samples. Although S-IgA data was positively skewed, distributions did not significantly differ from normal (S-IgA distributions are presented in Appendix E). It is further acknowledged that t-tests are extremely robust and as such, are insensitive to minor violations of normality in distribution.

7.3 Results

7.3.1 Results Overview

The results section comprises analyses regarding S-IgA reactivity to acute stress, and the individual and combined moderating effects of health status and mood upon reactivity individually. Further, the analyses regarding perceived workload demands were assessed in relation to S-IgA reactivity and health status and mood. As with the previous studies the primary method of classification is health status. As such, although other variables were assessed in direct relation to S-IgA reactivity, their association with S-IgA is assessed indirectly through health status classification.

7.3.2 Sample Demographics

The sample was selected from stage one psychology undergraduates. Table 7.1 presents the sex composition of the sample (males = 7, females = 13). As would be expected from an undergraduate population, all volunteers were aged under 40 years, moreover, the majority of the sample (65%) was aged under 20 year. The age composition of the sample is presented in Table 7.2.

	Number	Percent
Male	7	35.0
Female	13	65.0
Total	20	100.0

Table 7.1 Sex of Volunteers

	Number	Percent
< 20	13	65.0
20 - 30	6	30.0
31 - 40	1	5.0
Total	20	100.0

Table 7.2 Age of Volunteers

7.3.3 S-IgA Reactivity to Cumulative Acute Stress

Figure 7.2 presents the pre-post changes in S-IgA secretion rate for each of the three stressors (stressor 1 = pre & post 1, stressor 2 = pre and post 2, stressor 3 = pre and

post 3, rest = 5 minutes post-stress). Each of the three stressors elicited increases in S-IgA secretion rate, however, this increase was only significant following the first stressor ($t_{(19)}$ –2.27, $p < 0.05$). In contrast, each resting period of five minutes (between post-stress and next pre-stress sample) elicited a return to baseline. This return to baseline was only significant following the final stressor ($t_{(19)}$ 2.29, $p < 0.05$).

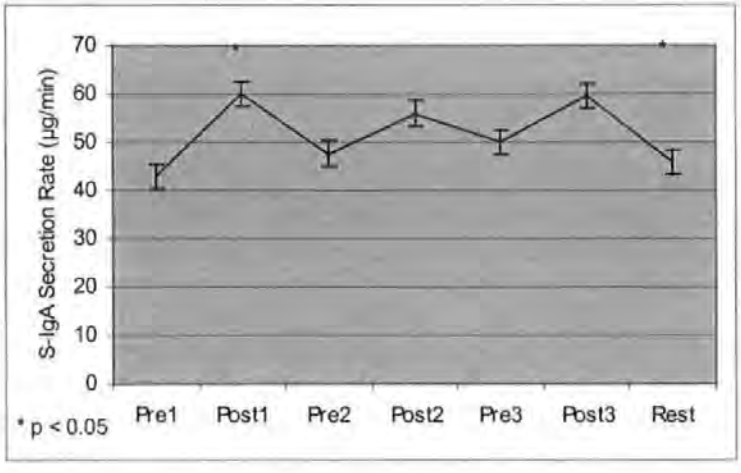


Figure 7.2 S-IgA Reactivity(and SEM) to Cumulative Acute Stress

7.3.4 Health Status and S-IgA Reactivity

As with the previous studies, S-IgA reactivity was assessed in response to all three stressors. Volunteers were classified as being in either good or poor health using the standardised means with regards to the MHC clusters. However, it should be noted that volunteers could not be classified by health status for the clusters of urinary-tract, microflora and fungal complaints. That is, owing to the relatively small sample size (n = 20), there was not enough variation with scores for each of these clusters for classifications to be made. Such a situation is an obvious risk when using standardised means, moreover, the risk is greater in smaller samples, where the chance of variation is reduced. one alternative was to classify volunteers using

within sample means. However, variations in frequencies of complaints in these clusters was so limited that within sample classification was also inappropriate. Differences in S-IgA reactivity for each of the stressor in good and poor health volunteers are therefore presented for the clusters of; total ill-health, stress-related complaints, ill-health indicators, psychological health, immune challenge complaints, atopy and gastric complaints only. However, the issue of sample size is also an important issue with regards to those MHC clusters where classification was appropriate. That is, given the small sample, the power of the design to detect a difference is small. As such, as with the previous studies, consistency in trends in the hypothesised direction is a theoretically salient factor. Distributions of MHC cluster scores are presented in Appendix E.

Total Ill Health

Figure 7.3 presents the mean S-IgA reactivity in volunteers classified as in good and poor health with regards to total ill-health following each of the three stressors. Following stressor one, there was little or no difference in S-IgA reactivity between good ($n = 10$) and poor health volunteers ($n = 10$), with both groups demonstrating positive reactivity. However, following stressor two, discrepancies between the groups began to emerge. Good health volunteers demonstrated positive reactivity, whereas, poor health volunteers demonstrated slight down-regulation. This discrepancy was even greater following the third and final stressor. Good health volunteers demonstrated significantly greater ($t_{(18)}$ 2.33, $p < 0.05$) S-IgA reactivity than those in poor health who demonstrated down-regulation.

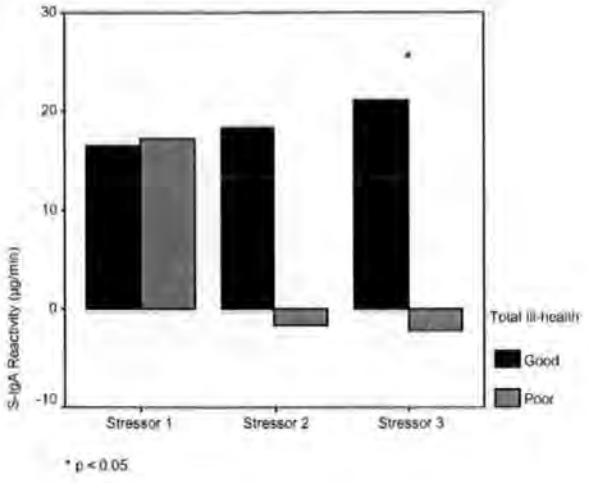


Figure 7.3 S-IgA Reactivity to Cumulative Acute Stress in Volunteers With Good and Poor Total Ill-health

Stress-Related Complaints

Following each of the stressors, volunteers in good health (n = 10) with regards to frequencies of stress-related complaints, demonstrated greater S-IgA reactivity than those in poor health. The discrepancy between good and poor health volunteers was greatest following the second stressor, where volunteers in good health demonstrated significantly greater ($t_{(18)}$ 2.16, $p < 0.05$) S-IgA reactivity than those in poor health who demonstrated post-stress down-regulation. Following, stressor three, volunteers in good health again demonstrated greater S-IgA reactivity than those in poor health, although not significantly so. The mean S-IgA reactivity in volunteers in good and poor health with regards to frequencies of stress-related complaints is presented in Figure 7.4.

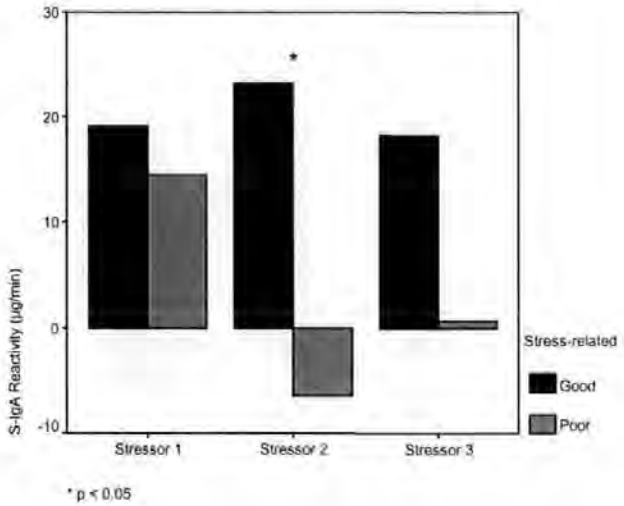


Figure 7.4 S-IgA Reactivity to Cumulative Acute Stress in Volunteers With Good and Poor Stress-related Complaints

Indicators of Ill-health

Figure 7.5 presents the mean S-IgA reactivity to each of the three stressors in volunteers classified as in good and poor health with regards to frequencies of ill-health indicators. Volunteers in good health (n = 12) consistently demonstrated positive S-IgA reactivity to each of the three stressors. However, in contrast, volunteers in poor health (n = 8) demonstrated reduced and then progressive down-regulation following each of the stressors. The discrepancy between good and poor health volunteers was greater following stressor two ($t_{(18)}$ 1.91, $p < 0.05$), and greater still following the third and final stressor ($t_{(18)}$ 3.59, $p < 0.01$).

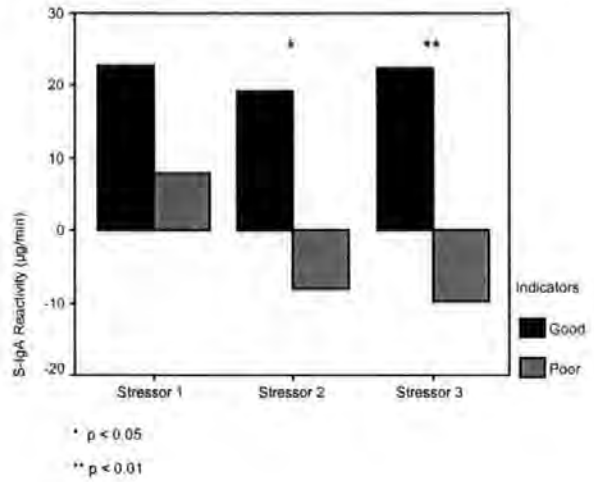


Figure 7.5 S-IgA Reactivity to Cumulative Acute Stress in Volunteers With Good and Poor Indicators of Ill-health

Psychological Complaints

As with the previously presented clusters, similar patterns of reduced S-IgA reactivity were demonstrated between volunteers classified as in good and poor health with regards to frequencies of psychological complaints. Following stressor one, volunteers in poor health (n = 8) and volunteers in good health (n = 12) demonstrated positive reactivity, although reactivity was reduced in those in poor health. Following stressor two, volunteers in good health demonstrated significantly greater S-IgA reactivity ($t_{(18)}$ 2.74, $p < 0.05$) than those in poor health who demonstrated post-stress down-regulation of S-IgA. A similar discrepancy was observed following the third and final stressor. That is, volunteers in good health demonstrated positive reactivity, and those in poor health demonstrated down-regulation. This difference was statistically significant ($t_{(18)}$ 3.02, $p < 0.01$). It is noted that while the discrepancy following stressor three is not as great as that observed following stressor two, greater statistical significance was achieved. This can be attributed to greater variation within S-IgA levels following the second stressor. That is following the second stressor variation in both groups (good health

s.d. = 32.53 µg/min, poor health s.d. = 26.40 µg/min), was greater than that observed following the third stressor (good health s.d. = 20.50 µg/min, poor health s.d. = 21.13 µg/min). This is issue is especially pertinent in the current study where the total sample size is relatively small. Figure 7.6 presents the mean S-IgA reactivity to each of the three stressors in volunteers classified as in good and poor health with regards to frequencies of psychological complaints.

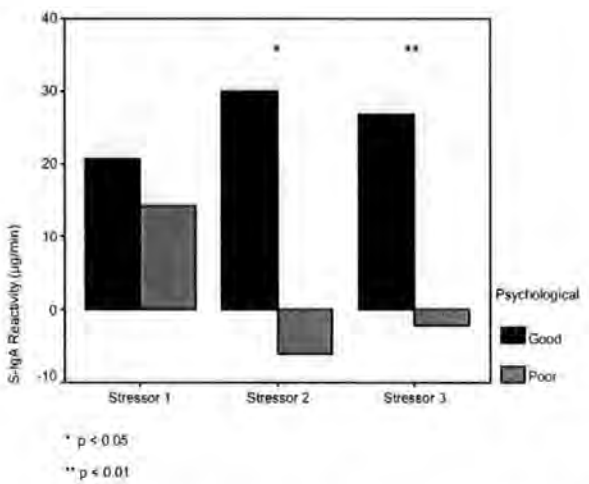


Figure 7.6 S-IgA Reactivity to Cumulative Acute Stress in Volunteers With Good and Poor Psychological Health

Immune Challenge

Figure 7.7 presents the mean S-IgA reactivity following each of the three stressors in volunteers classified as in good and poor health with regards to frequencies of immune-challenge complaints. Following the first stressor a near significant ($t_{(18)} = 1.99$, $p = 0.06$) difference in S-IgA reactivity was observed. That is volunteers in poor health (n = 13)demonstrated greater post-stress reactivity than those in good health (n = 7). Very little difference between the groups were observed following stressors two and three, where both groups demonstrated low but positive post-stress reactivity.

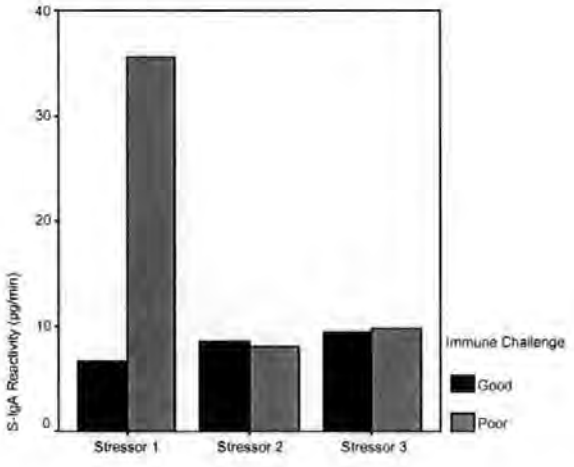


Figure 7.7 S-IgA Reactivity to Cumulative Acute Stress in Volunteers With Good and Poor Immune Challenge Complaints

Atopic Complaints

A mixed pattern of S-IgA reactivity was observed following each of the stressor in volunteers classified as in good and poor health with regards to frequencies of atopic complaints. Following the first stressor, volunteers in poor health (n = 8) demonstrated greater post-stress reactivity than those in good health (n = 12). This pattern was reversed following the second stressor, with volunteers in good health demonstrating near significant ($t_{(18)}$ 2.04, p = 0.06) greater reactivity than those in poor health who, demonstrating post-stress down-regulation. There was very little difference in post-stress reactivity between the groups following the third and final stressor. That is, both groups demonstrated small positive S-IgA reactivity. The mean S-IgA reactivity following each of the stressors in volunteers in good and poor health volunteers with regards frequencies of atopic complaints are presented in Figure 7.8.

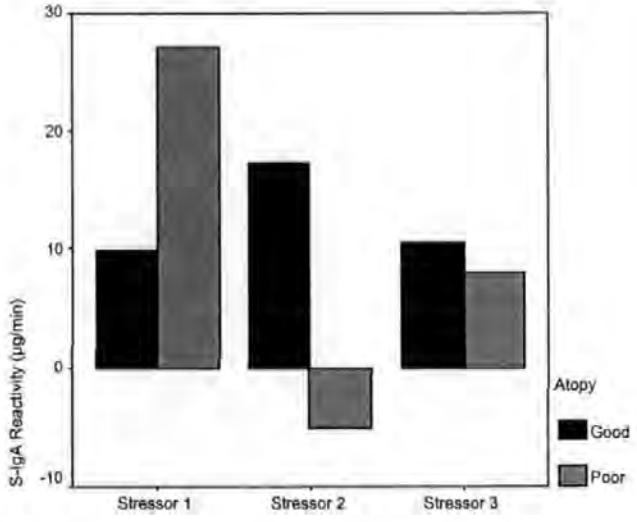


Figure 7.8 S-IgA Reactivity to Cumulative Acute Stress in Volunteers With Good and Poor Atopic Complaints

Gastric Complaints

Figure 7.9 presents the mean S-IgA reactivity following each of the three stressor in volunteers classified as in good and poor health with regards to frequencies of gastric complaints. It should be noted that the standardised classification could not be appropriately applied to the cluster of gastric complaints. Using the standardised classification resulted in only three volunteers being classified as in poor health. This discrepancy occurred as a result of a lower within sample mean (when compared with the standard mean) in the current sample. As such, it was appropriate to apply a classification based upon the sample data. Within sample classification gave rise to a mixed pattern of post-stress S-IgA reactivity between the groups. Following the first stressor, there was very little difference in S-IgA reactivity between volunteers classified as in good health (n = 12) and those in poor health (n = 8), both groups demonstrating positive post-stress reactivity. A discrepancy between the groups emerged following the second stressor. That is volunteers in poor health demonstrated considerable post-stress reactivity, compared to those in good health who demonstrated slight down-regulation. This pattern was also apparent following

the third and final stressor, where volunteers in poor health demonstrated slightly higher positive reactivity than those in good health.

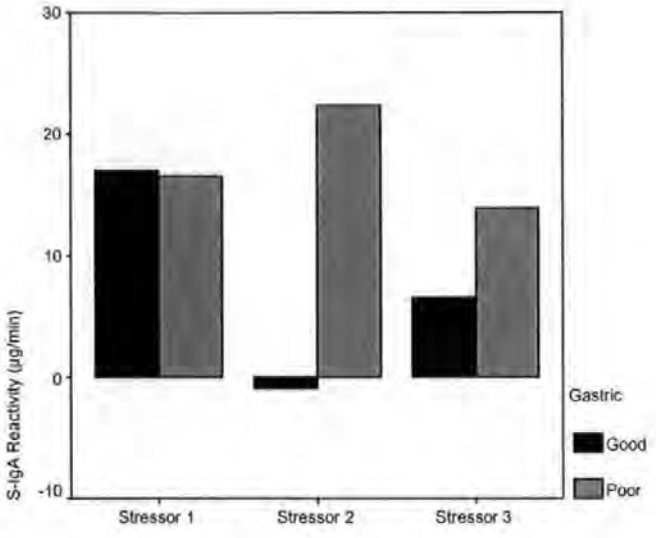


Figure 7.9 S-IgA Reactivity to Cumulative Acute Stress in Volunteers With Good and Poor Gastric Complaints

Urinary-Tract, Microflora Complaints & Fungal Complaints

As discussed at the beginning of this section, neither the standardised or within-sample classification techniques could be appropriately applied to these clusters. That is, in the main, mean scores for these clusters were extremely low, and as such, any classification would be of no benefit.

7.3.5 Health Status, Perceived Workload & S-IgA Reactivity

7.3.5.1 S-IgA Reactivity & Perceived Workload

The relationships between S-IgA reactivity and perceived workload following each of the three stressors are presented in Table 7.3. No significant correlations were observed, however, as with other analyses in the current study, the absence of significance can in part be attributed to reduced power brought about by small sample sizes. A mixed pattern of results were observed, however, it should be noted that consistent patterns were observed between S-IgA reactivity and perceived

workload following the first and last stressor. That is, negative associations were observed between S-IgA reactivity and facets of; mental physical and temporal demand, effort and frustration.

		Mental Demand	Physical Demand	Temporal Demand	Effort	Performance	Frustration
S-IgA Reactivity	Correlation Coefficient	-.146	-.381	-.074	-.123	.090	-.351
	Sig. (2-tailed)	.539	.097	.755	.604	.705	.130
	N	20	20	20	20	20	20

Stressor 1

		Mental Demand	Physical Demand	Temporal Demand	Effort	Performance	Frustration
S-IgA Reactivity	Correlation Coefficient	.180	-.294	.000	.015	-.056	-.005
	Sig. (2-tailed)	.448	.208	1.000	.950	.816	.982
	N	20	20	20	20	20	20

Stressor 2

		Mental Demand	Physical Demand	Temporal Demand	Effort	Performance	Frustration
S-IgA Reactivity	Correlation Coefficient	-.209	-.392	-.299	-.186	-.041	-.265
	Sig. (2-tailed)	.376	.087	.200	.431	.863	.259
	N	20	20	20	20	20	20

Stressor 3

Table 7.3 Relationships (Spearman's Rho) between S-IgA Reactivity and Perceived Workload Demands

7.3.5.2 Health Status & Perceived Workload

Perceived workload demands following each of the three stressors were subsequently compared in volunteers classified as in good or poor health with regards to each of the MHC clusters. Tables of means comprising perceived workload demands by health status are presented in Appendix E. As with studies one and two, there was a consistent trend for volunteers in poor health to report greater workload demands following each of the stressors than did those classified as in good health. In contrast, volunteers in poor health tended to have greater perceptions of performance, regardless of their actual performance attainment. Further, this consistency was greatest in those clusters previously identified as being most associated with S-IgA reactivity (total ill-health,

stress-related complaints, indicators of ill-health and psychological complaints). The most salient differences are presented with reference to each of the MHC clusters.

Total Ill-health

Volunteers in poor health with regards to total ill-health reported the first stressor to require greater effort ($t_{(18)}$ 5.91, $p < 0.05$), and the second task to be more temporally demanding ($t_{(18)}$ 7.48, $p < 0.01$) than did those classified as in good health.

Generalised Stress-related Complaints

Volunteers in good health with regards to frequencies of stress-related complaints perceived greater performance ($t_{(18)}$ 4.99, $p < 0.05$) regardless of their actual performance when compared with those in poor health.

Indicators of Ill-health

The cluster of indicators of ill-health demonstrated widespread increases in workload demands for volunteers classified as in poor health with regards to ill-health indicators. Volunteers in poor health demonstrated significant (or near significant) increases in mental demand for each of the three stressors ($t_{(18)}$ 3.66, $p < 0.07$, $t_{(18)}$ 5.78, $p < 0.05$, $t_{(18)}$ 3.56, $p < 0.07$ respectively). Similarly, poor health volunteers reported greater physical demand ($t_{(18)}$ 4.32, $p < 0.05$) and temporal demand ($t_{(18)}$ 20.80, $p < 0.001$) following stressor two. Further, volunteers in poor health demonstrated significant increases in the effort required following each of the stressors ($t_{(18)}$ 5.35, $p < 0.05$, $t_{(18)}$ 9.75, $p < 0.01$, $t_{(18)}$ 3.79, $p < 0.05$ respectively).

Psychological Complaints

Volunteers in poor health with regards to frequencies of psychological complaints reported the task to be more temporally demanding ($t_{(18)}$ 5.77, $p < 0.05$) and requiring more effort ($t_{(18)}$ 4.58, $p < 0.05$) than those in good health following the second stressor.

Immune Challenge Complaints

No significant differences were observed between volunteers classified as in good and poor health with regards to frequencies of immune challenge complaints, however, the trends are consistent with the other health clusters.

Atopic Complaints

In contrast to the other health clusters, volunteers classified as in good health with regards to frequencies of atopic complaints perceived the tasks to be generally more demanding. Poor health volunteers perceived the first and last stressor to be more physically demanding ($t_{(18)}$ 4.28, $p < 0.05$, $t_{(18)}$ 6.88, $p < 0.05$). However, in contrast, following the same stressors (1 and 3) these individuals perceived greater performance ($t_{(18)}$ 3.82, $p < 0.07$, $t_{(18)}$ 4.84, $p < 0.05$).

Gastric Complaints

As with atopic volunteers, volunteers classified as in good health with regards to gastric complaints perceived greater performance than did those in poor health following the first stressor ($t_{(18)}$ 5.82, $p < 0.05$).

As previously discussed, volunteers could not be classified as good or poor health with regards to the clusters of; urinary-tract, microflora and fungal complaints owing to a lack of variation within the cluster scores.

7.3.6 Health Status & Mood

Table 7.4 presents the mean positive and negative affect scores for volunteers classified as in good and poor health with regards to each of the MHC clusters. With the exception of gastric complaints, there was a consistent trend for volunteers in good health to have greater positive affect. Further, positive affect was significantly greater in volunteers in good health for the clusters of total ill-health ($t_{(18)}$ 2.30, $p < 0.05$) and atopy ($t_{(18)}$ 2.73, $p < 0.01$). In contrast, with the exception of immune challenge complaints, there was a consistent trend for volunteers in poor health to demonstrate greater negative affect. Furthermore, negative affect was significantly greater in poor health volunteers for the cluster of psychological complaints ($t_{(18)}$ –2.01, $p < 0.05$).

		PA	NA
Total Ill-health	Good	32.20 (3.88)*	18.21 (5.90)
	Poor	26.60 (6.67)*	22.61 (7.88)
Stress-related	Good	32.40 (3.86)*	18.01 (5.52)
	Poor	26.40 (6.48)*	22.80 (8.03)
Indicators	Good	30.67 (4.06)	17.78 (5.24)
	Poor	28.36 (7.31)	22.55 (7.98)
Psychological	Good	32.38 (4.37)	16.75 (5.06)*
	Poor	27.29 (6.87)	22.83 (7.46)*
Immune-challenge	Good	29.46 (5.84)	21.01 (8.03)
	Poor	29.29 (6.87)	19.29 (5.50)
Atopy	Good	32.00 (3.81)*	18.67 (4.87)
	Poor	25.51 (6.87)*	23.01 (9.40)
Gastric	Good	28.25 (6.30)	19.58 (7.32)
	Poor	31.13 (5.27)	21.63 (7.17)

*$p < 0.05$

Table 7.4 Mean Positive and negative Affect Scores in Good and Poor Health Volunteers

7.3.7 Mood & S-IgA Reactivity

Figure 7.10 presents the mean S-IgA reactivity following each of the stressors in volunteers classified as low and high in positive affect. No significant differences were observed between the groups, however, there was a consistent trend for volunteers classified as high in positive affect to demonstrate greater post-stress S-IgA reactivity than those low in positive affect.

Figure 7.11 presents the mean S-IgA reactivity following each of the stressors in volunteers classified as low and high in negative affect. Following stressor one, all volunteers demonstrated positive S-IgA reactivity, however, this reactivity was marginally greater in those volunteers classified as low in negative affect. A similar pattern was observed following stressor two, where volunteers with low negative affect demonstrated significantly greater ($t_{(18)}$ 3.06, $p < 0.01$) than those with high negative affect, who demonstrated negative S-IgA reactivity. Following the third and final stressor, all volunteers demonstrated positive S-IgA reactivity, however, again volunteers with low negative affect demonstrated greater reactivity than those with high negative affect.

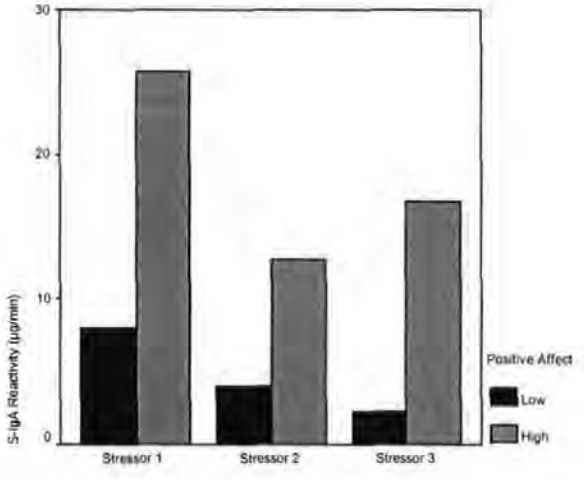


Figure 7.10 Mean S-IgA Reactivity in Volunteers With Low & High Positive Affect

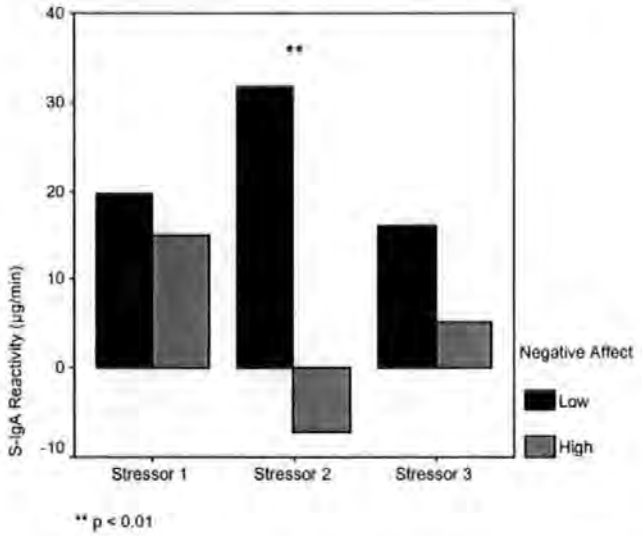


Figure 7.11 Mean S-IgA Reactivity in Volunteers With Low & High Negative Affect

7.3.8 Familiarity to the Stressor

Table 7.5 presents the mean perceived workload scores in all volunteers following each of the three stressors. No significant differences in mean perceived workload were observed (although as with other analyses in this study, the power of the experiment is reduced owing to the small sample size). In the main there was a trend for perceptions of workload to be reduced following each subsequent stressor (i.e., stressor two was perceived as less demanding than stressor one, and so on). This reduction in workload was especially the case for facets of mental and temporal demand, and effort. In contrast, as expected, increases in perceptions of performance co-varied with reductions in workload. However, for the facets of physical demand and frustration, the second stressor elicited greater workload demands than did either the first or last stressor.

		N	Mean	Std. Deviation
Mental Demand	Stressor 1	20	93.5895	21.0481
	Stressor 2	20	84.7375	32.0848
	Stressor 3	20	77.0890	34.6493
Physical Demand	Stressor 1	20	17.1835	13.6866
	Stressor 2	20	27.3770	28.8370
	Stressor 3	20	23.8400	24.4126
Temporal Demand	Stressor 1	20	81.9475	31.2785
	Stressor 2	20	76.2625	35.2623
	Stressor 3	20	73.4405	30.3517
Effort	Stressor 1	20	82.5590	20.3294
	Stressor 2	20	80.6970	29.2779
	Stressor 3	20	74.3565	27.0718
Performance	Stressor 1	20	51.9005	30.7385
	Stressor 2	20	61.6485	29.0228
	Stressor 3	20	67.1970	29.4266
Frustration	Stressor 1	20	44.7265	24.0502
	Stressor 2	20	55.8045	26.9109
	Stressor 3	20	40.6680	22.7944

Table 7.5 Mean Perceived Workload Demands Following Each Stressor

7.4 Discussion

7.4.1 Sample Demographics

As will be discussed with regard to the findings of the current study, due to financial restraints, the sample size was low (n = 20). As such, the power of the study was also low. As in previous studies, and as would be expected from a psychology undergraduate population, approximately two thirds of the sample was female, further, all but one of the volunteers was under the age of 30 years.

7.4.2 S-IgA Reactivity to Cumulative Acute Stress

Volunteers were exposed to three five minute doses of the stressor, interspersed with five minutes rest period. All three stress – recover periods followed the same pattern of S-IgA reactivity. That is, each stressor elicited an increase in S-IgA reactivity followed by a return to baseline during the recover period. It is noted that S-IgA never reached absolute baseline during the first and second recover periods, although, S-IgA reached near baseline following the final rest period of the session. This near return to baseline following the final stressor could be attributed to expectancy of the conclusion to the experiment. Expectancy of the experimental situation was discussed in Chapter Two. That is, given that it is important to assess the effects of the stressor per se upon S-IgA reactivity, lab-based stressors were discussed as a method of reducing expectation and therefore stemming arousal prior to experimentation. A similar phenomena could be responsible for the greater recover following the final stressor. That is, following stressor one and two, volunteers were aware that another stressor would be administered following the five minute recovery period. As such, arousal levels were still relatively high. Following the final stressor however, volunteers were aware that the end of the experiment was in sight. As such, the final recovery period can be viewed as a "true" period of recovery. That is, no further stressor would be administered, and as such, S-IgA reactivity was able to return to a base level analogous with pre-experiment levels.

This being the case, patterns of reactivity analogous with stressor two would be observed following subsequent stressors, until administration of the final stressor. However, to explore fully the influence of expectancy of the final stressor, volunteers should be totally naïve as to the periods of stress they were about to face. That is, using the current protocol, volunteers should not have been informed that, prior to stressor three, they were going to be administered the final stressor of the session. Given the potential influence of expectancy, which indeed provides a rationale for the use of lab-based stressors when assessing S-IgA reactivity, such a procedure must therefore be implemented in any further studies of this kind.

Although each of the stressors elicited up-regulation of S-IgA, significant reactivity was only observed following the first stressor. The reduction in S-IgA reactivity following the second and final stressors could be explained through familiarity to the stressor. That is, the initial presentation of the stressor was novel, and as such would invoke greater arousal, greater ANS activity, and therefore an increase in S-IgA. Subsequent stressors were therefore more familiar to the volunteer, and therefore elicited less S-IgA reactivity. This notion is also supported by the perceived workload data, which in the main indicated a a non-significant, but consistent reduction in workload demands with each subsequent stressor.

However, it is important to acknowledge that the magnitude of reactivity is influenced by the baseline measure (in this case each of the pre-stress measures) It has already been discussed that S-IgA only reached a near baseline level following the last stressor. As such, the pre-stress levels prior to stressors two and three were already elevated. Given a longer time period, it is likely that following each of the stressors, S-IgA would return to levels analogous to the pre-stressor one baseline. However, the higher pre-stress levels prior to stressors two and three indicate that volunteers are still aroused in

some way. The concept of an S-IgA reserve would suggest that owing to this elevated pre-stress state, the capacity to respond following stress is diminished. However, the overall picture concerning S-IgA reactivity to cumulative acute stress, in the sample as a whole, is one of robustness. That is, in each case, volunteers demonstrate positive reactive to each of the stressors, and a return towards baseline levels during each recover period. As will be discussed in more detail later on, the concept of an S-IgA reserve, or moreover a dysregulated reserve in some individuals, is more appropriately applied to data regarding S-IgA reactivity and health status.

Before discussing S-IgA reactivity and health status, it is appropriate to briefly compare the current findings with those of Carpenter *et al.*, (1998). As previously discussed, the current paradigm was loosely based upon a protocol employed by Carpenter *et al.*,(1998) on rats. In rats, the greatest S-IgA reactivity was also observed following initial stimulation. Subsequent stimulations also elicited S-IgA release, but of a much reduced magnitude. Further, periods of stimulation following the initial stressor all elicited very similar concentrations of S-IgA. The reductions could be attributed to habituation to the stressor, however, the observed patterns of reactivity are also of importance to the concept of an S-IgA-reserve. That is, the authors describe the high concentrations following the first stressor as a 'wash-out' from the ductal system (S-IgA-reserve). Following this initial wash-out, the reserve is considerably depleted, and as such, the reduced S-IgA reactivity observed is representative of a reduction in the availability of S-IgA during periods of stress. The reduced reactivity in the current study following stressors two and three (when compared with stressor one reactivity), could be viewed as analogous with the initial wash-out and subsequent reduced reactivity observed by Carpenter *et al.*(1998). Although the analogy is evident, it must be remembered that the current study is assessing human volunteers, and as such, S-IgA reactivity is likely to be moderated by a host of other factors. For example, although it

is possible that rats desensitised to the stimulation, the influence of such a process upon S-IgA reactivity in animal volunteers would be minimal. Further, as previously discussed, the S-IgA reserve, or perhaps more importantly, dysregulation of the reserve in some individuals, is more appropriately discussed in relation to other factors that can moderate S-IgA reactivity. Specifically, thus far in this body of work, classifications by health status have provided a consistent pattern of reactivity following stress. The concept of an S-IgA reserve will now be discussed in relation to health status.

7.4.3 Health Status & Cumulative Acute Stress Reactivity

The previous two studies have consistently demonstrated reduced S-IgA reactivity to stress in those volunteers classified as in poor health. Further, the S-IgA reserve model has thus far been developed as a graphical representation of the data regarding health status (and mood / personality) and S-IgA reactivity to acute stress. In order to observe the depletion of the reserve *in situ*, the cumulative stress paradigm was utilised. Given the observed reductions in S-IgA reactivity in poor health volunteers, it was therefore hypothesised that those in poor health would demonstrate the greatest reductions in S-IgA reactivity following cumulative acute stress. In support of the hypothesis, S-IgA reactivity was reduced in poor health volunteers. Further, as predicted, the discrepancy in S-IgA reactivity between good and poor health volunteers became more apparent following an accumulation of stress. This was specifically the case for the health clusters of; total ill-health, stress-related complaints, indicators of ill-health and psychological complaints. Further, it is these clusters that thus far have provided the most support for the consistent reductions in S-IgA in poor health volunteers. For each of these clusters, the pattern of reactivity was similar. Following all stressors, volunteers in good health demonstrated positive reactivity, greater than that demonstrated by the poor health volunteers. In the main, poor health volunteers demonstrated positive S-IgA reactivity to stressor one (but reduced in comparison with

good health volunteers). However, following the second stressor, poor health volunteers demonstrated considerably reduced S-IgA reactivity, and in many cases, down-regulation from pre-stress levels. Similar, but more salient patterns of reactivity were observed following the third and final stressor. That is, as predicted, poor health volunteers demonstrated progressively reduced S-IgA reactivity to cumulative acute stress when compared with good health volunteers. However, this predicted pattern was not evident in all health clusters. As such, these contrary clusters will be discussed before the predicted patterns are discussed in more detail with regards to the S-IgA reserve model.

In the previous chapter it was suggested that S-IgA reactivity was as predicted in those health clusters most associated with the action of S-IgA. Furthermore, in the current study, it was again, these identified clusters (total ill-health, stress-related, indicators and psychological) that demonstrated the predicted patterns of reactivity. In contrast, the cluster of gastric complaints demonstrated a mixed pattern of reactivity. Although S-IgA should be influential in all complaints comprising the health clusters by virtue of the common mucosa, it is likely that the spurious reactivity observed within the gastric cluster can be attributed to the fact that the action of S-IgA is not dominant in gastric complaints. This explanation has already been discussed in relation to the gastric cluster, as well as clusters of urinary-tract, fungal and microflora complaints (*c.f.*, Chapter Six). Unfortunately owing to unequal distribution of volunteers to either good or poor health with regards to these latter clusters, no comparisons could be made in the current study. However, the reader is reminded once more that patterns in the predicted direction have been consistently observed for the clusters of total ill-health, stress-related, indicators and psychological complaints.

With regards to health complaints closely associated with the action of S-IgA, the cluster of immune-challenge complaints is somewhat paradoxical. That is, the complaints comprising the immune-challenge cluster are all related (in the main) to the action of S-IgA upon the upper respiratory tract. As such, it should be expected that classifications within this cluster should be as predicted (i.e., poor health volunteers demonstrate reduced reactivity to acute stress). However, a very different pattern of reactivity was observed. Following the first stressor volunteers in poor health with regards to immune-challenge complaints demonstrated significantly greater S-IgA reactivity than those in good health, whilst there was very little difference in reactivity following the second and final stressors. As previously discussed, the complaints comprising the immune-challenge cluster are in the main, complaints of the upper respiratory tract (in addition, the cluster contains mouth ulcers). As such, volunteers classified as in poor health with regards to frequencies of these complaints are likely to have an abundance of previously specific S-IgA present in their S-IgA reserve (ductal system). This being the case, the greater S-IgA concentrations observed in poor health volunteers following the first stressor may represent a "wash-out" of the ductal system, as detailed by Carpenter *et al.*(1998). As a result of this initial washout, subsequent S-IgA secretion is greatly reduced in comparison.

Despite demonstrating patterns of reactivity contrary to prediction, the cluster of immune-challenge complaints still provides support for the existence of an S-IgA reserve. That is, like the work of Carpenter *et al.*, (1998) initial stimulation is followed by secretion with high S-IgA concentration (possibly as a result of greater previously specific S-IgA present in the ductal system as a result of previously encountered antigens). Subsequently, the second and final stressor elicited consistently lower reactivity (approximately 10μg/min following both stressor one and two). This pattern was also apparent in the work of Carpenter *et al*, (1998) who following initial

stimulation observed consistent (between 5 & 10μg/min) secretions of S-IgA following subsequent stressors. This observation suggests the role of some sort of feedback mechanism. That is, the consistent, but reduced reactivity following stressor two and three implies that some mechanism is preventing the absolute depletion of S-IgA from the reserve / ductal system. In functional terms, such a feedback mechanism is important to ensure that the reserve always maintains a store of S-IgA for the purposes of protection following subsequent antigen or psychosocial stressors.

Further evidence for some sort of feedback mechanism preventing the total depletion of the reserve is provided through assessment of the pre-stress S-IgA levels. As previously stated, the preferred S-IgA measurement in the current research is reactivity to the stressor (i.e., post-stress change in S-IgA from the baseline). Such a measurement is therefore influenced by the pre-stress measurement. Unlike studies one and two, pre-stress levels of S-IgA in the current study were higher in poor health volunteers than in those in good health (see Appendix E for mean pre-stress S-IgA levels). These higher levels indicate a reduction in the capacity to respond to stress, perhaps brought about by a finite supply of S-IgA and some sort of feedback mechanism preventing too much S-IgA being released from the ductal system, i.e., total depletion and therefore increases susceptibility to subsequent illness. That is, poor health volunteers start with higher pre-stress levels of S-IgA, and therefore in response to stress demonstrate a reduced capacity to respond, i.e., in order that the reserve is not totally depleted, a reduction in S-IgA reactivity occurs. This being the case, the potential causes for the higher pre-stress S-IgA in these volunteers must be evaluated. As such, this concept will be discussed with regard to health status and mood.

7.4.4 Health Status, Mood & S-IgA Reactivity to Cumulative Acute Stress

As in study two, with the exception of immune-challenge complaints, there was a consistent trend for volunteers in poor health to demonstrate greater negative affect. In contrast, volunteers in poor health demonstrated greater positive affect than those in poor health. Whilst this notion is not new, in the context of this body of work, evidence regarding the relationships between affect and health status, as well as the independent moderating effects of both health and affect on S-IgA reactivity, are essential when attempting to explain the pattern of reactivity observed in certain individuals. That is, in this body of work it has been demonstrated that health status is indicative of reduced S-IgA reactivity, whilst high negative affect is associated with increased S-IgA reactivity. However, it was predicted that such high reactivity in high negative affect volunteers was representative of how they dealt with work stress. Further, given an accumulation of work stress, such levels of reactivity could not be maintained. It was therefore predicted that high negative affect volunteers would demonstrate reductions in S-IgA following cumulative stress due to depletion of their reserve. Moreover, it is this depletion that leads to the high association between negative affect and ill-health, i.e., volunteers cannot maintain their high levels of reactivity. As such, the reactivity of high negative affect volunteers following cumulative stress would be analogous with the reactivity demonstrated by highly neurotic volunteers, i.e., continual arousal leads to depletion of the reserve, and therefore reduced S-IgA reactivity.

This pattern was apparent using the cumulative stress paradigm. That is, volunteers classified as high in negative affect demonstrated lower S-IgA reactivity than those classified as low. This was particularly apparent following the second stressor, where volunteers with high negative affect demonstrated post-stress down-regulation of S-IgA. It was predicted that the discrepancy in reactivity would become greater following cumulative stress, however, while high negative affect volunteers again demonstrated

reduced reactivity following stressor three, the discrepancy was not as great as that demonstrated following the second stressor.

Although not totally as predicted, high negative affect volunteers did demonstrate reduced S-IgA reactivity when compared to low negative affect volunteers. However, data regarding affect is also of vital importance with regard to the S-IgA reactivity observed in poor health volunteers. That is, it has been established that in the main, poor health volunteers have greater negative affect. Negative affect is associated with over-attendance to stimuli, and as such, expectancy of the stressor is likely to be higher in high negative affect volunteers. This increase in expectancy could therefore account for the higher pre-stress levels of S-IgA in poor health volunteers (who are also high in NA). The S-IgA reserve model would therefore suggest that high pre-stress S-IgA is indicates a depletion of the reserve. Subsequently, following stress, there is a reduction in S-IgA available. This could therefore account for the post-stress reductions observed in poor health volunteers. That is, the combination of poor health status and high negative affect (both characteristics of each other and both independently associated with depletion of the reserve) contribute to a dysregulation in the system, resulting in a lack of availability available at times of stress.

7.4.5 Familiarity to the Stressor

In study two there was very little difference in perceptions of workload between day one and day two. However, in a shorter time period, perceptions of workload were indeed reduced with each subsequent stressor. This therefore implies that, as with the work of Willemson *et al.*,(2000) as the task became more familiar, perceptions of workload were reduced. However, unlike the work of Willemson *et al.*, (2000) these reductions in workload do not seem to have any effect upon S-IgA reactivity. That is, other mechanism seems to be more influential in moderating S-IgA reactivity regardless of

novelty or familiarity to the stressor. In particular, volunteers in poor health consistently perceived the task to be more demanding. Taken together, the assessed factors all seem to be contributing to reductions in S-IgA reactivity to acute stress. It is the combination of factors that will now be discussed in the final section of this chapter.

7.4.6 Summary, Conclusion & Recommendations

The S-IgA reserve model was developed in response to the observation that poor health volunteers demonstrated reduced S-IgA reactivity to acute stress. This concept was explored further in study two, where poor health volunteers demonstrated reduced reactivity to the same stressor administered on two occasions. However, it was acknowledged that in order to fully explore the concept of an S-IgA reserve, a cumulative stress paradigm must be utilised, in order that any potential depletion could be observed in situ. That is, it was hypothesised that cumulative stress would deplete the S-IgA reserve, however, based on the findings of studies one and two, reactivity would be most reduced in poor health volunteers.

As hypothesised, several clusters demonstrated the predicted reactivity. That is, for the health clusters of; total ill-health, stress-related complaints, ill-health indicators and psychological complaints, volunteers classified as in poor health demonstrated reduced S-IgA reactivity when compared with those in good health. Further, the discrepancy in S-IgA reactivity between good and poor health volunteers became greater with each successive stressor. It is these four clusters that have consistently demonstrated reactivity in the predicted direction. However, as previously discussed (Chapter Six), given the exploratory nature of this body of work, it was seen as appropriate to assesses all of the identified clusters, especially considering that a new paradigm was introduced to this study. Unfortunately, given the small sample size of the current study, comparisons between good and poor health volunteers with regards to frequencies of

urinary-tract, fungal, and micro-flora complaints could not be calculated due to lack of variation in frequencies of these clusters. As such, it is now acknowledged, that future work of this kind should perhaps focus upon the four clusters that have demonstrated predicted reactivity. That is, it is likely that the complaints comprising these clusters are most sensitive to S-IgA. However, this argument should be followed with caution with regards to the reactivity observed with the immune challenge cluster (i.e., a huge "wash-out" following initial stimulation).

With regards to the cumulative stress paradigm, the administering of cumulative acute stress has emphasised the discrepancies in S-IgA reactivity between good and poor health volunteers. It is therefore suggested that the combination of the current stressor, and the cumulative stress paradigm are sensitive enough to tease out the underlying differences between good and poor health volunteers with regards to S-IgA reactivity. As discussed in Chapter two, there is increasing evidence for a negative relationship between health and S-IgA levels, however, this body of work has demonstrated that retrospective health status can mediate S-IgA reactivity to stress, magnitude of which influences susceptibility to subsequent illness. Further, this body of work clearly demonstrates individual differences in S-IgA reactivity in healthy volunteers. That is, although volunteers are classified as either good or poor health, the samples are essentially health, i.e., classification are based on frequencies of minor health complaints. Further, with regards to the sample, the volunteers can be described as normal healthy adults and as such, these findings demonstrate the link between health status and S-IgA reactivity in "non-vulnerable" individuals.

With reference to the methodology adopted in the current study, the potential problem of expectancy has been briefly discussed. That is, it was suggested that the near return to baseline levels following the final stressor only occurred as a result of expectancy of

the end of the experiment, or moreover, lack of expectancy of the next stressor. As such, in order to avoid potential problems of expectancy, in future studies of this kind, volunteers should be totally naïve as to the structure of the experiment. Such naivety would allow for the true assessment of S-IgA reactivity to the stressor. That is, volunteers will still demonstrate expectancy to the stressor, but total naivety will allow for exploration of the suggestion that, in the absence of stressor expectancy, S-IgA levels return to near baseline levels.

The cumulative stress paradigm was utilised in an attempt to assess the concept of an S-IgA reserve. That is, thus far, volunteers in poor health have demonstrated reduced reactivity to one stressor and then two stressors. In order to observe this reserve or moreover differences in the depletion of the reserve between good and poor health volunteers, cumulative acute stress was administered. As predicted for specific health clusters, volunteers in poor health demonstrated progressively reduced S-IgA reactivity. this provides further evidence for the existence of an S-IgA reserve. That is, as Carpenter *et al.*, (1998) suggest, S-IgA accumulates in the ductal system and is released following stimulation. In the current body of work, volunteers in poor health demonstrate greater depletion than those in good health. The current and previous studies in this thesis have also demonstrated the importance of state and trait factors, which are associated with both health status and S-IgA reactivity. The combination of these factors will be discussed in the final discussion (Chapter 8), which will detail the S-IgA reserve with regards to health status and the mediating roles of trait and state upon the action of the reserve.

Part Three

Conclusions

Part Three comprises the conclusions and wider implications chapter. The findings of the three experimental studies are discussed in light of previous research, current methodologies and theoretical and practical implications of this thesis.

8. Conclusions and Wider Implications

8.1 Conclusions

The main theme of this thesis is the factors, or combination of factors that moderate S-IgA reactivity. The first main finding in this thesis was that the chosen stressor, in the main, elicited up-regulation of S-IgA. Although the current series of studies could not assess what mechanisms are driving the S-IgA response to acute stress, other lab-based studies have assessed this concept. The level of stimulation from the current stressor is analogous with many of the previously used lab-based stressors, and moreover, the gross effects upon S-IgA are similar. As such, it is appropriate at this point to discuss the evidence regarding driving mechanisms in terms of the current findings.

Proposed Mechanisms

The observed increase in S-IgA concentration could be attributed to increased activation of the autonomic nervous system as a result of the acute stress induced by the manipulated stressor. This mechanism has been proposed by Willemson *et al.*, (1998), who observed alpha and beta-adrenergic cardiovascular activity following mental arithmetic tasks. Although the link is not direct (i.e., stimulation of the autonomic nervous system does not lead directly to S-IgA production and / or release), S-IgA release can occur as a function of adrenergic activity, which is stimulated by tasks such as mental arithmetic. The tasks comprising the Synwork battery are analogous with tasks such as mental arithmetic. It is therefore likely that post-stress increases in S-IgA could be caused by stimulation of the autonomic nervous system through the Synwork battery.

Attempts have been made to identify the mechanism and location responsible for S-IgA release in response to acute stress through the manipulation of different tasks known to elicit specific activation of either the sympathetic or parasympathetic branches of the

autonomic nervous system. Willemson *et al.*, (1998) observed S-IgA changes in response to both a cold pressor task and mental arithmetic tasks which elicited alpha-adrenergic activity and a mix of alpha and beta-adrenergic activity respectively. Adrenergic activity is stimulated by the sympathetic branch of the autonomic nervous system, and as such, S-IgA increases to both stressor were attributed to activity in this location.

Further support for the role of the sympathetic branch is provided by Ring *et al.*, (1999), who administered tasks of mental arithmetic and paced breathing, known to stimulate the sympathetic and parasympathetic nervous systems respectively. They observed a mixed pattern of alpha and beta-adrenergic activity in response to the mental arithmetic task, and a reduction in parasympathetic activity. In contrast, the paced breathing task elicited an increase in parasympathetic activity. Further, increases in S-IgA concentrations were observed following the mental arithmetic but not the paced breathing tasks. Using these observations the authors propose that the S-IgA response to acute stressors is activated by stimulation of the sympathetic nervous system.

Although this evidence does suggest the role of the sympathetic nervous system responsible for S-IgA release in response to acute stressor, the role of the parasympathetic nervous system cannot be dismissed. In contrast to acute stress, S-IgA increases have also been observed following relaxation tasks. Moreover, anecdotal evidence proposes the benefits of relaxation, not stressor tasks as being beneficial to immune enhancement. Green and Green (1987, 1988) observed increases in S-IgA concentration rates and S-IgA secretion rates following short term (20 minutes) and longer term daily session (3 weeks) respectively. Similarly, Janoski and Kugler (1987) observed higher S-IgA concentrations in those individuals assigned to a progressive

relaxation schedule than in those assigned to a positive control condition using an auditory discrimination task.

Relaxation tasks such as those utilised by Green and Green (1987, 1988), and Janoski and Kugler (1987), are known to stimulate the parasympathetic nervous system. Similar relaxation tasks were used by Ring *et al.*, (1999). However, they reported no increases in S-IgA following stimulation of the parasympathetic nervous system through paced breathing tasks, and therefore proposed that S-IgA responses are mediated by the sympathetic nervous system. It is important to note that, whilst Green and Green (1987) implemented relaxation for a minimum of 20 minutes (with a subsequent prolonged relaxation schedule), Ring *et al* (1999) assigned relaxation for only 20 minutes. The two findings are therefore not comparable given the differences in time period. It could be the case that the sympathetic nervous system responds to short term acute stress, but the parasympathetic system only responds over prolonged periods of time especially given the nature of the tasks that elicit a response (i.e., relaxation)

If S-IgA release is caused by activation of the autonomic nervous system, the contradiction in findings does not suggest the location of the mechanism as present in either the sympathetic or parasympathetic nervous systems. Bristow *et al.*, (1997) suggests that conventional antibody production takes days to complete and as such, post-stress S-IgA increases are far too rapid to be accounted for in these terms. Instead, it is suggested that rapid increases reflect modulation of the secretion, not production processes. Further, Hucklebridge *et al.*, (2000), suggest that such rapid changes are likely to reflect modulation of the transepithelial secretory process. Morse, Schahterle, Espisoto, Chod, Furst, Di Ponziano and Zedenberg (1983), suggest that salivary glands are densely innervated by the autonomic nervous system, and as such many aspects of

salivary gland activity are regulated by the system. Stimulation of the autonomic nervous system, be it sympathetic or parasympathetic, will therefore result in stimulation of the secretory glands and invoke salivary related activity such as S-IgA release. Although, attempts have been made to identify the exact location of the mechanism, stimulation of the transepithelial secretory process can account for up-regulation of the immune system following both sympathetic and parasympathetic stimulation.

Given the observed up-regulation of the immune system following both acute stress and prolonged relaxation it is important to assess why these changes occur and what potential benefits can result. As the nature, and therefore perceptions of tasks of acute stress and relaxation are very different, the purpose of such changes cannot be explained from the same perspective.

Firstly, S-IgA increases have been observed following acute stress. This response is very rapid, and is often very transient. That, is, S-IgA concentrations often return to normal in the time following the stressor. Although when subjected to a stressor, individuals are capable of making a conscious judgement of the nature and perceived risk of the stressor, analogous physiological judgements cannot be made in this way.. Any stressor is perceived as a potential threat, and as such, up-regulation of the immune system results to ensure no increase in susceptibility occurs following exposure to the stressor. The stressor could be biological or psychological in nature, however, the main function of the immune system is to protect against viral or bacterial antigens. As a result, S-IgA secretion is increased immediately following a stressor to ensure that the body is not more susceptible to antigens during and immediately following the period of stress.

In terms of the current findings, the stress induced by the Synwork battery is sufficient to provoke an increase in S-IgA. That is, the body perceives a potential threat and as such increases regulation of S-IgA to protect against the stressor and any susceptibility to viral and bacterial antigens immediately following the stressor.

Although the potential benefits of S-IgA increases following prolonged periods of relaxation are the same as those following acute stress, the underlying function is likely to be different. That is, S-IgA increase following acute stress is immediate in response to the potential immediate effects of a stressor. Following relaxation, there is no potential threat, in fact the body is in a relaxed state and as such does not require immediate immune protection. During its relaxed state the body therefore has the opportunity to replenish immune reserves. This explanation can account for the fact that immune up-regulation occurs following prolonged periods of relaxation. It is therefore likely that relaxation stimulates the production, not secretion of S-IgA. That is, IgA molecules are produced through the process of B-cell activation and subsequent plasma cell differentiation. This process is not immediate and can take days to complete. As such, S-IgA increases will only be apparent following prolonged periods of relaxation, where the body has the opportunity to focus upon antibody production without the interruption of immediate responses following immune challenges be they viral / bacterial or psychological in nature. The proposed mechanisms for S-IgA increases following both acute stress and prolonged relaxation are illustrated in Figure 8.1.

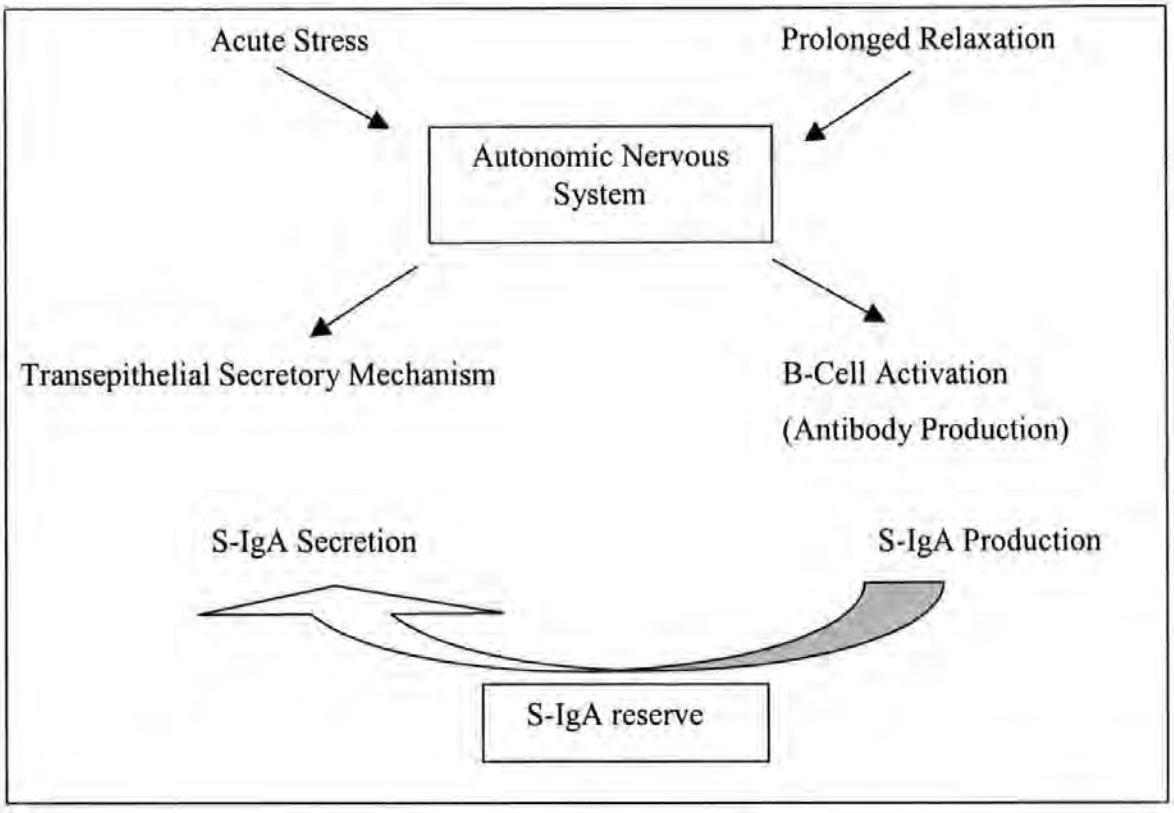


Figure 8.1 Proposed mechanisms responsible for S-IgA increases following both acute stress and prolonged relaxation

The right side of Figure 8.1 illustrates the likely cause of S-IgA increases following prolonged periods of relaxation. It is this process that is responsible for the production of IgA molecules. The left side of the figure illustrates the likely mechanism responsible for the S-IgA release following acute stress, however, the magnitude of this response may be dependent upon the S-IgA production process. That is, when an individual experiences either physiological or psychological challenges, S-IgA will be released accordingly. However, if these challenges are occurring frequently, the demand for S-IgA release will be in more frequent demand. These challenges are also likely to interrupt the production of IgA, and therefore the supply or reserve of IgA available in response to stressor will diminish. Using the terminology of Carpenter *et al.*, (1998) the flow from IgA production to IgA secretion can be seen as the accumulation in the ductal system. Moreover, introducing the concept of an S-IgA reserve model, this flow can be seen as replenishing the reserve.

The current research then assessed the concept of an S-IgA reserve in relation to health status, and furthermore the influence of other moderating factors, i.e., state, trait and perceptions of workload in relation to the flow into the reserve, and further, the rate of release into saliva (and therefore subsequent susceptibility to infection).

With regards to health status a consistent trend was observed demonstrating reduced S-IgA reactivity to acute stress in volunteers classified as being in poor health with regards to frequencies of minor health complaints. This concept was explored further in study two, where S-IgA reactivity was assessed on two occasions following administration of the same stressor. In addition, personality and mood factors were assessed both in relation to health status and as individual moderators of S-IgA reactivity. Findings demonstrated that again, poor health was associated with reduced S-IgA reactivity. Further, specific traits and states were also associated with reduced reactivity. In addition, further data was provided regarding the relationships between poor health and personality and mood factors. That is, negative traits (neuroticism) were associated with both reduced S-IgA reactivity, and poor health. To maintain consistency in this body of work, the major method of classifying volunteers was by health status (good and poor health). As such, it was possible to highlight specific factors that were associated with reduced S-IgA reactivity to acute stress. That is, poor health is predictive of reduced reactivity, but also associated with negative traits, which in turn are independently associated with reduced reactivity.

It is tempting to speculate that the reduced S-IgA reactivity observed in those with negative characteristics (e.g., neuroticism) is brought about by the chronic nature of the characteristics they possess. That is, it could be the case that the chronic nature of neuroticism results in HPA axis activation, subsequent release of cytokines and immune suppression. Further, this immune suppression could account for the greater incidence of illness in neurotic volunteers. This concept could further be applied with regard to

the affective state of volunteers before they arrived at the lab. That is, such volunteers were likely to be anticipating the study, and as such could have been in a high state of arousal before being exposed to the stressor. The level of stress elicited in the lab could have been sufficient to elicit HPA activation and subsequent immune suppression. This could also account for the post-stress S-IgA reductions observed by Coons *et al.*, (1995). They suggested that certain volunteers were already highly aroused, and as such could only demonstrate down-regulation following a stressor. This seems like a plausible concept, and moreover, could explain why certain volunteers demonstrate down-regulation, and further, why these same volunteers experience greater frequencies of illness. However, in the absence of further data (i.e., cortisol data which could indicate HPA axis activation), such a concept is purely speculative, but worthy of further investigation.

In response to the findings of studies one and two, a model was developed. This model suggested the existence of an S-IgA reserve. That is, a reserve or pool of S-IgA that is drawn upon during times of stress. It was further suggested that this reserve, or mechanisms driving this reserve were dysregulated in certain individuals. That is, volunteers in poor health demonstrated reduced reactivity to acute stress as a result of a depleted reserve. Further evidence for an S-IgA reserve was provided by Carpenter *et al.*, (1998) who following repeated administration of stimulation (stress) observed and initial "wash-out" of S-IgA from the ductal system, followed by reduced but consistent secretion of S-IgA following subsequent stimulation. The authors suggest that during rest, IgA accumulates in the ductal system. Further, the S-IgA release observed following stimulation represents a "wash-out" of the ductal system, the reserve is depleted, and subsequent reductions in S-IgA are observed, i.e., there is less S-IgA available for secretion in response to stress.

In order to observe the potential depletion of an S-IgA-reserve (or ductal system) in humans, a cumulative stress paradigm was adopted (Study three). That is, volunteers were subjected to repeated stress (three stressors interspersed with recovery periods), in order that the reserve could be depleted in situ, i.e., in the absence of any external stimuli. Using classification by health status, poor health volunteers demonstrated reduced S-IgA reactivity when compared to good health volunteers. Furthermore, the discrepancy in reactivity between good and poor health volunteers became greater with each subsequent stressor, i.e., following an accumulation of acute stress. These findings provided more evidence for the existence of some sort of reserve. That is, reduced reactivity in poor health volunteers can be attributed to either a depleted reserve, or a dysfunctional mechanism driving release from the reserve into secretions.

The relationships between health status and S-IgA are however, complicated by the influences of personality and mood. The relationships between health and states and traits are well recorded in the literature, in particular, the relationships between negative factors (negative affect and personality) and poor health. Given the interactive nature of PNI as a discipline, it therefore seemed appropriate to assesses the direct relationship between these factors and S-IgA reactivity, as well as the their association with health status. That is, the S-IgA reserve model could be used as a method of explaining the reduced reactivity observed in poor health volunteers, and to these ends, the model is quite coherent. However, this body of work, also attempted to assess other factors which may moderate S-IgA reactivity to acute stress. The concept of stress in itself is not a simplistic one. That is, different people perceive stress in different ways. Immune reactivity to stress influences subsequent susceptibility to illness, and as such it is prudent to assess other factors that may influence individual effects of stress. The assessed factors in this body of work can therefore be rationalised with ease. That is, stress is related to illness, both of which are associated with S-IgA reactivity (the former

influencing reactivity, the latter being a product of reactivity). However, individual perceptions of stress are influenced by factors such as personality and mood, which can therefore moderate immune reactivity to the stressor.

It is suggested that the influences of personality and mood, including perceptions of stress in relation to both health status and S-IgA reactivity to acute stress all contribute to characteristics of an "illness-prone individual". That is, the S-IgA-reserve model is best applied to classification by health status, however, state and trait factors should be seen as influential in moderating the individual stress response. Negative affect and neuroticism are both associated with ill-health. However, whilst neuroticism is independently associated with reduced reactivity, negative affect is associated with higher S-IgA in the short-term However, it is suggested that this is a temporary up-regulation owing to the reduced reactivity observed in high NA volunteers following the second stressor. Further, negative states and traits are associated with increased perceptions of workload. When drawing all these factors together a pattern emerges. That is, poor health volunteers demonstrate greater neuroticism and negative affect. Further, they perceive tasks to be more demanding and also demonstrate reduced S-IgA reactivity to acute stress. It is this reduced reactivity that is suggested as causing increased susceptibility to post-stress illness, however, the direct relationships between neuroticism and negative affect (following cumulative acute stress) and S-IgA suggest that levels of these characteristics also mediate the S-IgA stress response.

The series of studies within this thesis highlight the complex relationships between factors that account for the interactive processes that link the brain and the immune system. Although complex, this thesis has attempted to provide further information regarding these factors. A model has been provided to demonstrate the discrepancy between good and poor health volunteers with regards to S-IgA reactivity to stress. In

addition, the roles of state and trait characteristics have been assessed, and should be viewed as having moderating effects upon the depletion of the reserve.

8.2 Wider Implications

This section will be divided into two parts. The first part will assess the implications of the adopted methodologies, and the second part will discuss the wider implications of the findings.

8.2.1 Methodologies

Minor Health Complaints Questionnaire (MHCQ)

The MHCQ was developed in an attempt to classify frequencies of minor health complaints in a healthy population. In addition to the MHCQ as a measurement tool, data has also been collected regarding mean frequencies of complaints (for each cluster) in both younger (mean age = 30 years) and older (mean age = 55 years) healthy adults. The development was primarily focused upon classifying health complaints with regards to subsequent classification regarding S-IgA reactivity. To these ends, the MHCQ can be viewed as successful in its' ability to classify volunteers with regards to health status and demonstrate subsequent differences in S-IgA reactivity, state and trait factors and perceptions of workload. However, it is acknowledged that some clusters were more successful in eliciting differences regarding S-IgA reactivity.

Although the MHCQ was developed specifically for the current research focus, it can be used as a classification tool in a variety of other applications. That is, the MHCQ can be used to create scores with regards to frequencies of health complaints for nine distinct clusters of minor ill-health. As such, the MHCQ can be applied to any research area where there is a need for data regarding frequencies of minor health complaints. Data can be collated to form a reliable total-ill-health score, or alternatively for any of

the comprising ill-health clusters to create ill-health profiles for respondents. In addition, the data regarding mean frequencies can be used as a benchmark for subsequent research involving the MHCQ, or as a means of classification in subsequent sample populations.

The Stressor (Synwork)

The choice of the Synwork battery as a stressor in this thesis, was made in response to the stressors utilised in previous research of this kind (*c.f.*, Chapter 2). That is, a range of stressors have been previously utilised all varying in external validity. Early studies utilised examination periods as a means of stressing the volunteer. While this can be seen as externally valid, the use of examination stress proved complex with regards to the distinction between acute and chronic stress. In order to assess more precisely the effects of stressors upon S-IgA reactivity, laboratory stressors were then utilised. Although previously used lab based stressors have been lacking in external validity, their use has been invaluable in assessing specific stressor effects and potential mechanisms involved in S-IgA reactivity. In contrast, several studies have assessed S-IgA reactivity to naturalistic stressors. Such studies have obvious external validity, however, results may be specific to the utilised stressors, and further, the role of expectancy of the stressor is likely to have played an influential role in the observed reactivity, i.e., the stressors are integral parts of the volunteer's lives, and as such, observed reactivity could not be entirely attributed to the action of the stressor, moreover, a complex interaction between many other psychosocial factors.

The current stressor was therefore seen as a compromise between lab-based stressors and naturally occurring stressors. That is, the stressor can be administered in the lab and as such can be rigorously controlled and reduces the potential effects of expectancy dominant in examination studies and studies utilising naturalistic stressors. However,

the advantage of the Synwork battery is that it was developed as a multi-tasking performance battery, and as such provides an efficient simulation of any working environment where individuals are required to attend and respond to several stimuli simultaneously. S-IgA reactivity observed following this stressor is therefore more analogous with other working environments, and as such, the current findings can provide reliable baseline data with regards to reactivity that might be expected in a variety of other working situations.

The current stressor is also capable of demonstrating individual differences with regards to S-IgA reactivity. That is, the stressor itself seems to be sensitive enough to highlight individual differences in S-IgA reactivity, i.e., owing to individual perceptions of stress and individual differences in the way people respond to this kind of stressor. Furthermore, the combination of the current stressor and the cumulative stress paradigm creates further sensitivity. That is, in healthy volunteers, the combination of these factors is capable of teasing out individual differences with regards to frequencies of minor health complaints. The cumulative stress paradigm also increases external validity. That is, the paradigm can be viewed as representative of how individuals deal with an accumulation of acute stress in everyday life. The working day can be seen as being made up of continual acute stressors. As such, repeated administration of a stressor which simulates a working environment is analogous with build up of acute stressors in a normal working situation. Using this concept, it should follow that during the working day, volunteers in good and poor health with regards to frequencies of minor health complaints should demonstrate similar discrepancies in S-IgA reactivity as the working day progresses and the accumulation of stress builds up.

8.2.2 Implications of the Research Findings

One of the major findings of this thesis is the difference in S-IgA reactivity between those classified as in either good or poor health with regards to frequencies of minor health complaints. Given that the Synwork battery is analogous with many working environments, then as previously discussed, it is reasonable to assume that volunteers would demonstrate similar S-IgA reactivity in everyday life. In the sample as a whole (and in good health volunteers), the stressor elicited up-regulation of S-IgA. The clinical relevance of this S-IgA secretion into saliva is contentious, however, the general consensus suggests that following stress, S-IgA is released into saliva (and the common mucosa) in an attempt to protect against potential infection. Previous literature regarding health status and S-IgA indicates that good health is associated with higher S-IgA, and conversely, poor health associated with lower S-IgA. In relation to the current findings, volunteers in good health demonstrate greater S-IgA reactivity. If it is assumed that this S-IgA release has a general protective effect against infection, then it follows that the these volunteers are better equipped to maintain their good health status. That is, they demonstrate positive S-IgA reactivity to acute stress, this S-IgA serves to protect against potential post-stress infection, and thus they are less susceptible to ill-health. In contrast, volunteers in poor health could be described as being stuck in a vicious circle of ill-health. That is, they demonstrate reduced S-IgA reactivity to stress, and as a result are more susceptible to post-stress infection, maintaining a cycle of ill-health.

These cycles of good and poor health appear to be mediated by trait characteristics, including perceptions of stress. That is, neuroticism and negative affect are associated with poor health status, and reduced S-IgA reactivity (this is especially the case for the trait of neuroticism, but the reader is reminded that although high negative affect leads to short-term up-regulation of S-IgA, reactivity is reduced following an accumulation of

stress). It is difficult to establish the precise roles of state and trait characteristics, but they undoubtedly play an influential role in how stressors are perceived by the individual. These perceptions of stress in turn influence S-IgA reactivity, magnitude and direction of which influences post-stress susceptibility to ill-health, and the maintenance of either a good or poor health cycle.

The potential moderating effects of state and trait characteristics are therefore essential when attempting to suggest strategies that may alleviate the negative effects of stress. That is, if individuals can be instructed in efficient ways to cope with the stressors they encounter (i.e., coping strategies that attempt to reduce the negative perceptions of stress), the deleterious effects upon immune reactivity could be reduced. As a result, poor health volunteers could break out of their poor-health cycle, i.e., stress would elicit positive S-IgA reactivity and these individuals would be less susceptible to post-stress infection.

References

Alabadie, M.S., Kent, G.G. & Gawkrodger, D.J. (1994). The relationship between stress and the onset and exacerbation of psoriasis and other skin conditions. *British Journal of Dermatology*, 130(2), 199-203.

Booth-Kewley, S., & Friedman, H.S. (1987). Psychological predictors of heart disease: A quantitative Review. *Psychological Bulletin*, 101, 343-362.

Bosch, J., Brand, A., Ligtenburg, B., Bermond, B., Hoogstraten, J., & Nieuw-Amerongen, A. (1998). The response of salivary protein levels and s-IgA to an academic examination are associated with daily stress. Journal of Psychophysiology, 12: 384-391.

Bristow, M, Hucklebridge, F., Clow, A., & Evans, P. (1997). Modulation of secretory immunoglobulin A in students undergoing assessment. *Journal of Psychophysiology*, 35: 575-576.

Brodman, K., Erdman, A.J., & Wolff, H.G. (1949). The Cornell Medical Index: An adjunct to medical interview. , 140: 530-534.

Caplan, R.P. (1994). Stress, anxiety and depression in hospital consultants, general practitioners, and senior health service managers. *British medical Journal*, 309 (6964), 1261-1263, Nov. 12.

Carpenter, G.H., Garrett, J.R., Hartley, R.H., & Proctor, G.B. (1998). The influence of nerves on the secretion of immunoglobulin A into submandibular saliva in rats. *Journal of Physiology*, 512.2, 567-573.

Carroll, D., Ring, C., Shrimpton, J., Evans, P., Willemson, G., & Hucklebridge, F. (1996). Secretory immunoglobulin-A and cardiovascular responses to acute physiological challenge. *International Journal of Behavioral Medicine*, 3, 266-279.

Child, D. (1970). *The Essentials of Factor Analysis*. London: Holt, Rinehart & Winston.

Cochrane, R. (1969). Neuroticism and the discovery of high blood pressure. Journal of *Psychosomatic Research*, 13: 21-25.

Cochrane, R. (1973). Hostility and neuroticism among unselected essential hypertensives. *Journal of Psychosomatic Research*, 17: -215-218.

Coe, R.M. (1978). *Sociology of Medicine* (2nd Ed.). New York: McGraw-Hill.

Coons, E.E., Montello, L., & Perez, J. (1995). Confidence and denial factors affect musicians' post-performance immune response. *International Journal of Arts Medicine*, 4(1) 4-14.

Costa, P.T., Jr., McCrae, R.R., Andres, R., & Tobin, J.D. (1980). Hypertension, somatic complaints, and personality. In M.F. Elias & D. Streeten (Eds.), *Hypertension and Cognitive Processes* (pp. 95-110). ME: Beech-Hill.

Cronbach, L.J. (1951). Coefficient Alpha and internal structure of tests. *Psychometrika*, 16, 297-334.

Dancey, C.P., Taghavi, M., & Fox, R.J. (1998). The relationship between daily stress and symptoms of irritable bowel: A time-series approach. *Journal of Psychosomatic Research*, 44 (5): 537-545.

Deinzer, R., & Schuller, N. (1998) Dynamics of stress-related decrease in salivary immunoglobulin A (sIgA): Relationship to symptoms of the common cold and studying behaviour. *Behavioral Medicine*, 23: 161-169.

Deinzer, R., Kleineidam, C., Stiller-Winkler, R., Idel, H., & Bachg, D. (2000). Prolonged reduction of salivary immunoglobulin A (sIgA) after a major examination. *International Journal of Psychophysiology*, 37: 219-232.

DeLongis, A., Coyne, J.C., Dakof, G., Folkman, S., & Lazarus, R.S. (1982). Relationships of daily hassles, uplifts, and major life events to health status. *Health Psychology*, 1: 119-136.

Derogatis, L.r., Lipman, R.S., Rickels, K., Uhlenhute, E.H., & Covi, L. (1974). A measure of primary symptom dimensions. *Psychological Measurement in Psychopharmacology*, 7: 79-114.

Dillon, K.M., Minchoff, B., & Baker, K. (1985). Positive emotional states and enhancement of the immune system. *International Journal of Psychiatry and Medicine*, 15: 13-18.

Elsmore, T.F. (1990). *A Synthetic Work Environment for the PC.* Walter Reed Army Institute of Research, Washington, DC.

Evans, P, Bristow, M., Hucklebridge, F., Clow, A., & Walters, N. (1993). The relationship between secretory immunity, mood and life events. *British Journal of Clinical Psychology*, 32: 227-236.

Evans, P., Bristow, F., Hucklebridge, F., Clow, A., & Pang, F-Y (1994). Stress, arousal, cortisol and secretory immunoglobulin A in students undergoing assessment. British *Journal of Clinical Psychology*, 33: 575-576.

Evans, P., Hucklebridge, F., & Clow, A. (2000*). Mind, Immunity and Health: The Science of Psychoneuroimmunology.* London: Free Association Books.

Gleeson, M. (2000). Mucosal immunity and respiratory illness in elite athletes. *International Journal of Sports medicine*, 21 (suppl 1): S33-44.

Gravetter, F.J., & Wallnau, L.B. (1996). *Statistics for the Behavioral Sciences* (4th Ed.). West Publishing Company, Minneapolis.

Gray, J.A. (1985). Issues in the neuropsychology of anxiety. In A. H Tuma & J.D. Maser (Eds.), *Anxiety and Anxiety Disorders* (pp.5-25). New Jersey: Erlbaum.

Green, M.L., Green, R.G., & Santoro, W. (1988). Daily relaxation modifies serum and salivary immunoglobulins and psychophysiological symptom severity. *Biofeedback and Self-Regulation*, 13: 187-199.

Green, R., & Green, M.L. (1987). Relaxation increases salivary immunoglobulin A. *Psychological Reports*, 61: 623-629.

Harrison, L.K., Carroll, D., Burns, V.E., Corkill, A.R., Harrison, C.M., Ring, C., & Drayson, M. (2000). Cardiovascular and secretory immunoglobulin A reactions to humurous, exciting, and didactic film presentations. *Biological Psychology*, 52: 113-126.

Hart, S.G., and Staveland, L.E. (1988). Development of a multi-dimensional workload rating scale: Results of empirical and theoretical research. In: P.A. Hancock and N. Meshkati (Eds.), *Human Mental Workload.* Amsterdam. The Netherlands: Elsevier.

Holroyd, K.A., Stensland, M., Lipchik, G.L., Hill, K.R., O'Donnell, F.S. & Cordingley, G. (2000). Psychosocial correlates and impact of chronic tension-type headaches. *Headache*, 40 (1): 3-16 JAN.

Hucklebridge, F, Clow, A., & Evans, P. (1998) The relationships between salivary secretory immunoglobulin A and cortisol: Neuroendocrine response to awakening and the diurnal cycle. *International Journal of Psychophysiology*, 31: 69-76.

Hucklebridge, F., Lambert, S., Clow, A., Warburton, D.M., Evans, P.D. and Sherwood, N. (2000). Modulation of secretory immunoglobulin-A in saliva; response to manipulation of mood. *Biological Psychology*, 53, 25-35.

Huwe, S., Hennig, J., & Netter, P. (1998). Biological, emotional, behavioural, and coping reactions to examination stress in high and low state anxious subjects. *Anxiety, Stress and Coping*, 11: 47-65.

Hyland, M.E. & Sodergren, S.C. (1997). Dysfunctional gut syndrome investigated by association of symptoms in the general population. *Journal of Nutritional and Environmental Medicine*, 7, 253-265.

Hyland, M.E. & Sodergren, S.C. (1998). Relationship between lifestyle and minor health complaints: Evidence for two clusters of association. *Journal of Nutritional and Environmental Medicine*, 8, 233-246.

Jasnoski, M.L., & Kugler, J. (1987). Relaxation, imagery, and neuroimmodulation. *Annals of the New York Academy of Science*, 496: 722-730.

Jemmot, J.B., & Magloire, K. (1988). Academic stress, social support, and secretory immunoglobulin A. *Journal of Personality and Social Psychology*, 55: 803-810.

Jemmott, J.B., & McClelland, D.C. (1989). Secretory IgA as a measure of resistance to infectious disease: Comments on Stone, Cox, Valdimarsdottir, and Neale. *Behavioural Medicine*, 15(2), 63-71.

Jemmott, J.B., Borysenko, J.Z., Borysenko, M., McClelland, D.C., Chapman, R. & Meyer, D. (1983). Academic stress, power motivation and decrease in secretory rate of salivary immunoglobulin A. *Lancet*, 1:1400-1402.

Kasl, S.V., Cobb, I.D., & Brooks, G.D. (1968). Changes in serum uric acid and cholesterol level in men undergoing job stress. *Journal of the American Medical Association*, 206: 1500-1507.

Keehn, R.J., Goldberg, I.D., & Beebe, G.W. (1974). Twenty-four year mortality follow-up of army veterans with disability separations for psychoneurosis in 1944. *Psychosomatic Medicine*, 36: 27-46.

Kellner, R. (1994). Psychosomatic syndromes, somatization and somatoform disorders. *Psychotherapy and Psychosomatics*, 61: 4-24.

Kiecolt-Glaser, J.K., Ricker, D., George, J., Messick, G., Speicher, C.E., Garner, W., & Glaser, R. (1984). Urinary cortisol levels, cellular immunocompetency , and loneliness in psychiatric inpatients. *Psychosomatic Medicine*, 46: 15-23.

Kuby, J. (1997) *Immunology* (3rd edition). New York: Freeman and Co.

Kugler, J., Reitjes, F., Tewes, V., & Schedlowski, M. (1996). Competition stress in soccer coaches increases salivary immunoglobulin A and salivary cortisol concentrations. *Journal of Sports Medicine and Physical Fitness*. 36: 117-120.

LaRue, A., Bank, L., Jarvik, L., & Hetland, M. (1979). Health in old age: How do physicians' ratings and self-ratings compare? *Journal of Gerontology*, 34: 687-691.

Lefcourt, H.M., Davidson-Katz, K., & Keuneman, K. (1990). Humor and immune system functioning. *Humor*, 3: 305-321.

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.

Loewenthal, K.M., Eysenck, M., Harris, D., Lubitsh, G. and Gorton, T. (2000). Stress, distress and air traffic incidents: job dysfunction and distress in airline pilots in relation to contextually-assessed stress. *Stress Medicine*, 16 (3): 179-183.

Magnam, A. & Vervloet, D. (2000). Natural history of atopy. *Revue Des Maladies Respiratoires*, 17 (1BIS), 235-244, Feb.

Martin, R.A., & Dobbin, J.P. (1988) Sense of humor, hassles and immunoglobulin A: Evidence for stress-moderating effect of humor. *International Journal of Psychiatry and Medicine*, 18: 93-105.

McClelland, D., Floor, E., Davidson, R.J., & Saron, C. (1980). Stressed power motivation, sympathetic activation, immune function and illness. *Journal of Human Stress*, 6: 11-19.

McClelland, D., Ross, G., & Patel, V. (1985). The effects of academic examination on salivary norepinephrine and immunoglobulin levels. *Journal of Human stress*, 11: 52-59.

McClelland, D.C. & Kirshnit, C. (1988). The effects of motivational arousal through films on salivary immunoglobulin A. *Psychology of Health*, 2: 31-52.

McCrae, R.R., Bartone, P.T., & Costa, P.T., Jr. (1976). Age, anxiety, and self-reported health. *Aging and Human Development*, 7: 29-58.

McCrae, R.R. & Costa, P.T. Jr. (1987). Validation of the 5-factor model of personality across instruments and observers. *Journal of Personality and Social Psychology*, 52(1), 81-90, Jan.

McCraty, R., Atkinson, M., Rein, G., & Watkins, A.D. (1996). Music enhances the effect of positive emotional states on salivary IgA. *Stress Medicine*, 12: 167-175.

Michel, F.B. Psychology of the allergic patient.(1994) *Allergy*, 29 (18), 28-30, supplement S.

Morse, D.R., Schacterle, G.R., Espisoto, J.V., Chod, S.d., Furst, M.L., Di Ponziano, J., and Zedenberg, M. (1983). Stress, meditation and saliva: A study of separate salivary gland secretions in endodontic patients. *Journal of Oral Medicine*, 38, 150-160.

Mouton, C., Fillion, L., Tawadros, E., & Rejean, T. (1989). Salivary IgA is a weak stress marker. *Behavioral Medicine*, 15: 179-185.

Myers, J.L., (1972). *Fundamentals of Experimental Design*. Boston: Allyn and Bacon,.

Navazesh, M. (1993). Methods for collecting saliva. *Annals of New York Academy of Sciences*, 694,: 72-77.

Ohira, H., Watanabe, Y., Kobayashi, K., & Makiko, K. (1999). The type A behaviour pattern and immune reactivity to acute stress: change in volume of secretory immunoglobulin A in saliva. *Perceptual and Motor Skills*, 89: 423-430.

Pearlin, L.I., Menaghan, E.G., Lieberman, M.A., & Mullan, J.T. (1981). The Stress response. *Journal of Health and Social Behavior*, 22 (4): 337-356.

Pennybaker, J.W., Burnam, M.A., Schaeffer, M.A., & Harper, P. (1977). Lack of control as a determinant of perceived physical symptoms. *Journal of Personality and Social Psychology*, 35: 167-174.

Rider, M.S., & Welden, C. (1990). Imagery, improvisation, and immunity. *The Arts in Psychotherapy*, 17: 211-216.

Ring, C., Carroll, D., Willemson, G., Cooke, J., Ferraro, A., & Drayson, M. (1999) Secretory immunoglobulin A and cardiovascular activity during mental arithmetic and paced breathing. *Psychophysiology*, 36: 602-609.

Robinson, J.O. (1969). Symptoms and the discovery of high blood pressure. *Journal of Psychosomatic Research*, 13: 157-161.

Shwartz, G.E., Davidson, R.J., & Goleman, D.J. (1978). Patterning of cognitive and somatic processes in the self-generation of anxiety: Effects of meditation versus exercise, *Psychosomatic Medicine*, 40: 321-328.

Spangler, G. (1997) Psychological and physiological responses during an exam and their relation to personality characteristics. *Psychoneuroendocrinology*, 22(6): 423-441.

Stone, A.A., Cox, D.S., Valdimarsdottir, H., Jandorff, L. & Neale, J.M. (1987). Evidence that secretory IgA is associated with daily mood. *Journal of Personality and Social Psychology*, 52(5): 988-993.

Tomasi, T.B. (1976). Mucosal immune system. A general review. *Annals of Otology, Rhinology & Laryngology.* 85(Suppl 25 Pt 2):87-9.

Valdimarsdottir, H.B., & Stone, A.A. (1997). Psychosocial factors and secretory immunoglobulin A. *Critical Review of Oral Biological Medicine*, 8: 461-474.

Van Rood, Y.R., Bogaards, M., Goulmy, E., & Van Houwelingen, H.C. (1993). The effects of stress and relaxation on the in vitro immune response in man: A meta-analytic study. *Journal of Behavioral Medicine*, 16: 163-181.

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., & Pennybaker, J.W. (1989). Health Complaints, stress and distress: Exploring the central role of negative affectivity. *Psychological Review*, 96 (2): 234-254.

Wetherell, M.A. The Common Cold: The effects of infection on perceived and actual cognitive performance. *The Proceedings of the British Psychological Society, 2001.*

Wheatley, D. (1997). Stress, anxiety and depression. *Stress-Medicine*, 13 (3): 173-177.

Willemson, G., Ring, C., Carroll, D., Evans, P., Clow, A., & Hucklebridge, F. (1998). Secretory immunoglobulin A and cardiovascular reactions to mental arithmetic and cold pressor. Psychophysiology, 35: 252-259.

Willemson, G., Ring, C., McKeever, S., & Carroll, D. (2000). Secretory immunoglobulin A and cardiovascular activity during mental arithmetic: effects of task difficulty and task order. *Biological Psychology*, 52: 127-141.

Winzer, A., Ring, C., Carroll, D., Willemson, G., Drayson, M., & Kendall, M. (1999) Secretory immunoglobulin A and cardiovascular reactions to mental arithmetic, cold pressor, and exercise: Effects of beta-adrenergic blockade. *Psychophysiology*, 36: 591-601.

Zeier, H., Brauchli, P., & Joller-Jemelka, H. (1996). Effects of work demand on immunoglobulin A and cortisol in air traffic controllers. *Biological Psychology*, 42: 413-423.

Appendix A

MHCQ

Research by: **Plymouth Health-Related Quality of Life Research Centre**
Director: **Professor Michael Hyland**
Project Co-ordinator: **Mark A. Wetherell MSc.**

Health in Plymouth

- **We are conducting research into minor health complaints - the sorts of complaints that everyone has from time to time. This survey measures how often minor health complaints occur in people living in the Plymouth area.**

- **Your name has been selected at random from the electoral register. We hope you will take the five minutes needed to answer these questions.**

- **Every response is important to us. The questions are straightforward - you just need to tick boxes.**

- **All replies are anonymous and confidential, but the overall findings will be published in due course.**

Thank you in advance for your help

Please return your responses in the FREEPOST envelope provided.

No Stamp is Needed

General Questions

- **Age**

Please tick one box

Below 20 ❑
20-30 ❑
31-40 ❑
41-50 ❑
51-60 ❑
Above 60 ❑

- **Sex**

Please tick one box

Male ❑
Female ❑

Health Questions

a) How many times have you had each of the following health complaints in the *last year* ?

Please tick a box for each complaint

	1 or less	2 or 3	4 or 5	6 or 7
Colds or Flu	❑	❑	❑	❑
Athletes foot	❑	❑	❑	❑
Wheeze	❑	❑	❑	❑
Mouth ulcers	❑	❑	❑	❑
Sore throats	❑	❑	❑	❑
Fungal infection of groin or scalp	❑	❑	❑	❑
Cystitis	❑	❑	❑	❑
Thrush (answer only if female)	❑	❑	❑	❑

b) On how many days in the *last month* have you had each of the following problems ?

Please tick a box for each question

	Never	Once	2 or 3	4-6	7 +
Headaches or migraines	❑	❑	❑	❑	❑
Constipation (hard pellety stools)	❑	❑	❑	❑	❑
Watery diarrhoea (loose stools running out like water)	❑	❑	❑	❑	❑
Explosive diarrhoea (loose stools mixed with wind)	❑	❑	❑	❑	❑
Heartburn (indigestion pain)	❑	❑	❑	❑	❑
Itchy eyes	❑	❑	❑	❑	❑
Difficulty sleeping	❑	❑	❑	❑	❑
Feeling very tired for no reason	❑	❑	❑	❑	❑
Thirsty for no reason	❑	❑	❑	❑	❑

c) Please answer these questions

Please tick a box for each question

	No	A little	Yes
Are you prone to accidents?	❑	❑	❑
Would you consider yourself a clumsy person?	❑	❑	❑
Do you get anxious easily?	❑	❑	❑
Do you get depressed easily?	❑	❑	❑
Do you urinate at a slower rate than normal?	❑	❑	❑
Do you often feel hungry shortly after you have eaten a large meal?	❑	❑	❑
Do you have patches of dry itchy skin (eczema)?	❑	❑	❑
Do you sneeze a lot even when you do not have a cold?	❑	❑	❑
Do you have a blocked nose even when you do not have a cold?	❑	❑	❑
Do you have vivid daydreams that seem almost real?	❑	❑	❑

Other Health Questions

- Do you smoke ?

Please tick one box

Never ❑
Occasionally ❑
Everyday ❑

- Compared with other people, do you ?

Please tick one box

Easily become too hot (often need less clothes) ❑

Easily become too cold (often need more clothes) ❑

Both of the above ❑

Neither of the above ❑

- On average, how many times do you urinate during the night?

Please tick one box

Never / almost never ❑
Once ❑
Twice ❑
Three or more ❑

Medication

In the last year

- How many courses of antibiotics have you taken ? _____

- How often have you taken painkillers ?

Please tick a box

every day	once a week	once a month	once every 3-4 months	once a year	Never
❑	❑	❑	❑	❑	❑

- How often do you take vitamin supplements ?

Please tick a box

every day	once a week	once a month	once every 3-4 months	once a year	Never
❑	❑	❑	❑	❑	❑

- How often have you taken mineral supplements ?

Please tick a box

every day	once a week	once a month	once every 3-4 months	once a year	Never
❑	❑	❑	❑	❑	❑

- How often do you take energy drinks or glucose tablets ?

Please tick a box

every day	once a week	once a month	once every 3-4 months	once a year	Never
❑	❑	❑	❑	❑	❑

Family Income and Education

a) **Education**

Please tick a box

No formal education	❑
GCSE or O-Level	❑
A-Level or HND	❑
Degree or Professional Level	❑
Other (Please specify)	❑ _ _ _ _ _ _ _ _ _ _ _ _ _

b) **Family Income**

Please tick a box

Below £10, 000	❑
£10, 000 - £20, 000	❑
£20, 000 - £30, 000	❑
£30, 000 - £40, 000	❑
Above £40, 000	❑

Appendix B

Methods & Materials

Adaptation of the PANAS

How do you feel ?

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 the word. Indicate to what extent you feel this way at the moment.

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

_____	Interested	_____	Irritable
_____	Distressed	_____	Alert
_____	Excited	_____	Ashamed
_____	Upset	_____	Inspired
_____	Strong	_____	Nervous
_____	Guilty	_____	Determined
_____	Scared	_____	Attentive
_____	Hostile	_____	Jittery
_____	Enthusiastic	_____	Active
_____	Proud	_____	Afraid

NEO-FFI: Items, Domains & Scoring

1. I am not a worrier (N*)
2. I like to have a lot of people around me (E)
3. I don't like to waste my time daydreaming (O*)
4. I try to be courteous to everyone I meet (A)
5. I keep my belongings clean and neat (C)
6. I often feel inferior to others (N)
7. I laugh easily (E)
8. Once I find the right way to do something, I stick to it (O*)
9. I often get into arguments with my family and co-workers (A*)
10. I'm pretty good about pacing myself so as to get things done on time (C)
11. When I'm under a great deal of stress, sometimes I feel like I'm going to pieces (N)
12. I don't consider myself especially "light-hearted" (E*)
13. I am intrigued by the patterns I find in art and nature (O)
14. Some people think I'm selfish and egotistical(A*)
15. I am a very methodological person (C*)
16. I rarely feel lonely or blue (N*)
17. I really enjoy talking to people (E)
18. I believe letting students hear controversial speakers can only confuse and mislead them (O*)
19. I would rather co-operate with others than compete against them (A)
20. I try to perform all the tasks assigned to me conscientiously (C)
21. I often feel tense and jittery (N)
22. I like to be where the action is (E)
23. Poetry has little or no effect on me (O*)
24. I tend to be cynical and sceptical of others' intentions (A*)
25. I have a clear set of goals and work toward them in an orderly fashion (C)
26. Sometimes I feel completely worthless (N)
27. I usually prefer to do things alone (E*)
28. I often try new and foreign foods (O)
29. I believe that most people will take advantage of you if you let them (A*)
30. I waste a lot of time before settling down to work (C*)
31. I rarely feel fearful or anxious (N*)
32. I often feel as if I'm bursting with energy (E)
33. I seldom notice the moods or feelings that different environments produce (O*)
34. Most people I know like me (A)
35. I work hard to accomplish my goals (C)
36. I often get angry at the way people treat me (N)
37. I am a cheerful, high-spirited person (E)
38. I believe we should look to our religious authorities for decisions on moral issues (O*)
39. Some people think of me as cold and calculating (A*)
40. When I make a commitment, I can always be counted on to follow through (C)
41. Too often, when things go wrong, I get discouraged and feel like giving up (N)
42. I am not a cheerful optimist (E*)
43. Sometimes when I am reading poetry or looking at a work of art, I feel a chill or wave of excitement (O)
44. I'm hard-headed and tough-minded in my attitudes (A*)
45. Sometimes I'm not as dependable or reliable as I should be (C*)
46. I am seldom sad or depressed (N*)
47. My life is fast-paced (E)

48. I have little interest in speculating on the nature of the universe or the human condition (O*)
49. I generally try to be thoughtful and considerate (A)
50. I am a productive person who always gets the job done (C)
51. I often feel helpless and want someone else to solve my problems (N)
52. I am a very active person (E)
53. I have a lot of intellectual curiosity (O)
54. If I don't like people, I let them know it (A*)
55. I never seem to be able to get organised (C*)
56. At times, I have been so ashamed I just wanted to hide (N)
57. I would rather go my own way than be a leader of others (E)
58. I often enjoy playing with theories or abstract ideas (O)
59. If necessary, I am willing to manipulate people to get what I want (A*)
60. I strive for excellence in everything I do (C)

N = Neuroticism
E = Extraversion
O = Openness
A = Agreeableness
C = Conscientiousness

* = Items with reverse scoring

Respondents are asked to specify the degree to which they agree or disagree with each item using the following scale

SD = Strongly Disagree
D = Disagree
N = Neutral
A = Agree
SA = Strongly Agree

NASA – TLX Perceived Workload Questionnaire

Mark each line at the point which matches your experience of the tests you have just completed.

1. **MENTAL DEMAND** – How much mental demand and perceptual activity was required (thinking, deciding, calculating, remembering, looking etc) ? Was your task easy or demanding, simple or complex ?

Low _____ High

2. **PHYSICAL DEMAND** – How much physical activity was required (pulling, turning, controlling activating etc) ? Was your task easy or demanding, slow or brisk, slack or strenuous, restful or laborious ?

Low _____ High

3. **TEMPORAL DEMAND** – How much time pressure did you feel due to the rate of the task ? Was the pace slow and leisurely or rapid and frantic ?

Low _____ High

4. **EFFORT** – How hard did you have to work, mentally and physically, to achieve your level of performance ?

Low _____ High

5. **PERFORMANCE** – How successful do you think you were in performing the tests? How satisfied were you with your performance ?

Low _____ High

6. **FRUSTRATION** – How insecure, discouraged, irritated, stressed and annoyed versus secure, gratified, content, relaxed and complacent did you feel ?

Low _____ High

NASA – TLX Perceived Workload Questionnaire (Continued)

So far you have rated your workload after the tests according to six factors. Now I would like you to say how important each factor was to you in all of the tests you have completed. There are no right or wrong answers, it is you opinion would like.

The factors are arranged below in pairs. For each pair, circle the factor which was most important to you in doing the tests.

MENTAL DEMAND VS PHYSICAL DEMAND

EFFORT VS FRUSTRATION

PHYSICAL DEMAND VS EFFORT

FRUSTRATION VS TEMPORAL DEMAND

TEMPORAL DEMAND VS MENTAL DEMAND

PHYSICAL DEMAND VS PERFORMANCE

PERFORMANCE VS EFFORT

EFFORT VS MENTAL DEMAND

FRUSTRATION VS PHYSICAL DEMAND

PHYSICAL DEMAND VS TEMPORAL DEMAND

MENTAL DEMAND VS FRUSTRATION

TEMPORAL DEMAND VS EFFORT

FRUSTRATION VS PERFORMANCE

PERFORMANCE VS MENTAL DEMAND

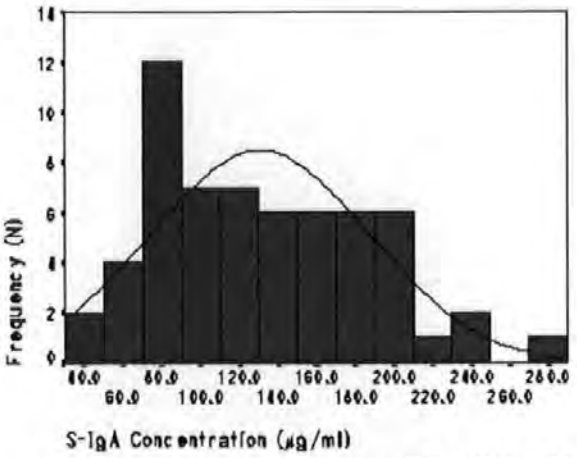
TEMPORAL DEMAND VS PERFORMANCE

Appendix C

Study One

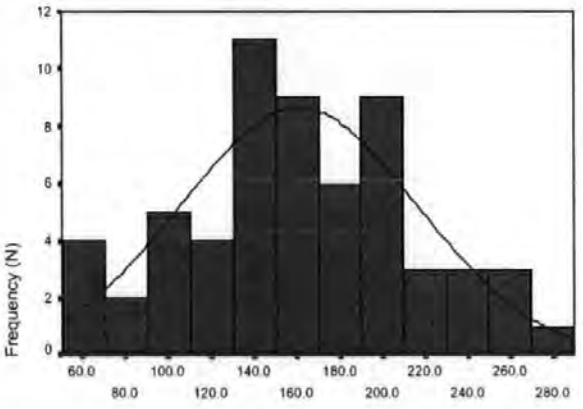
S-IgA Distributions

Pre-stress S-IgA Concentrations



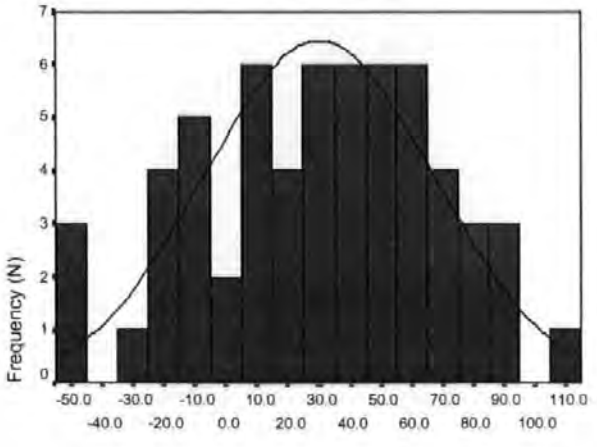
Skew Statistic = .44

Post-stress S-IgA Concentrations



Skew Statistic = - 43

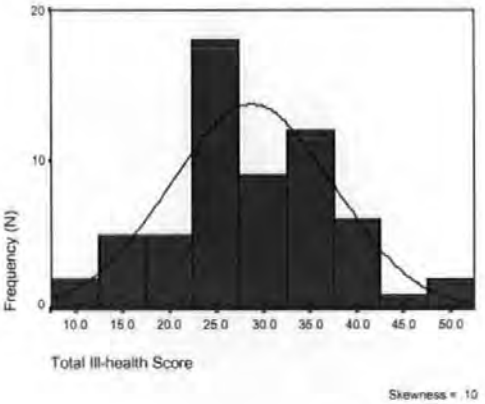
S-IgA Reactivity



Skew Statistic = -0.17

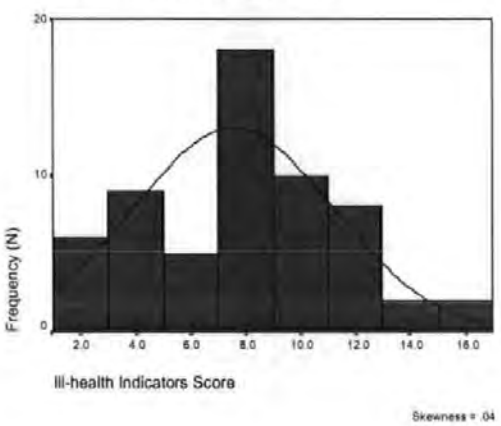
MHC Cluster Score Distributions

Total Ill-health

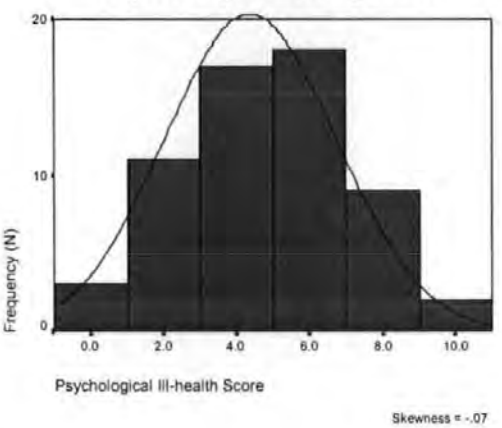


Stress-related Complaints

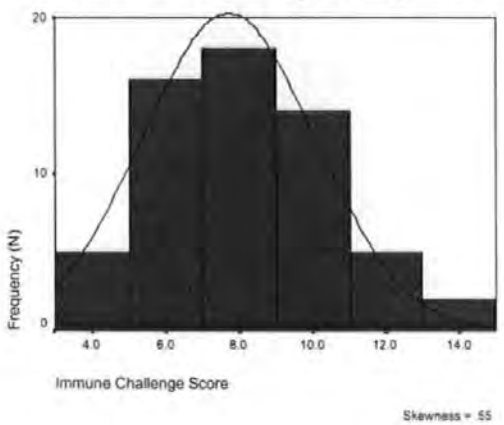
Ill-health Indicators



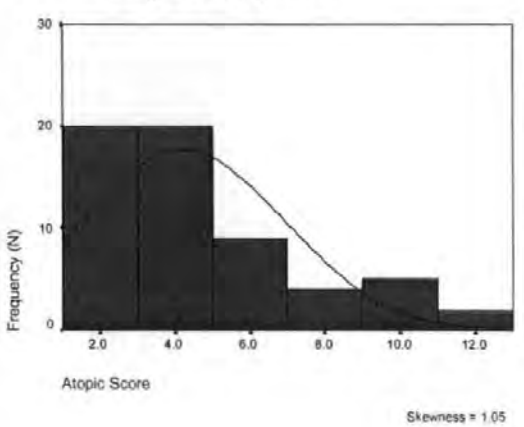
Psychological Complaints



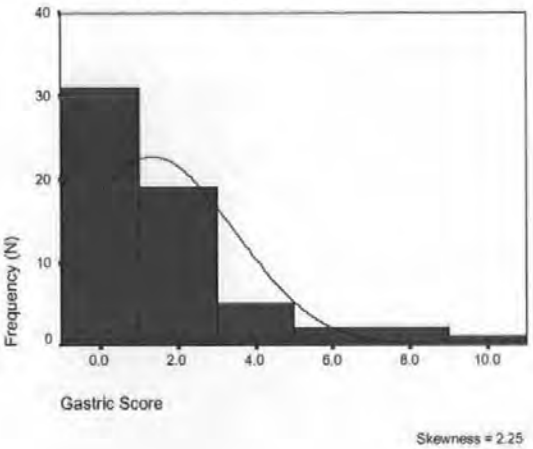
Immune Challenge Complaints



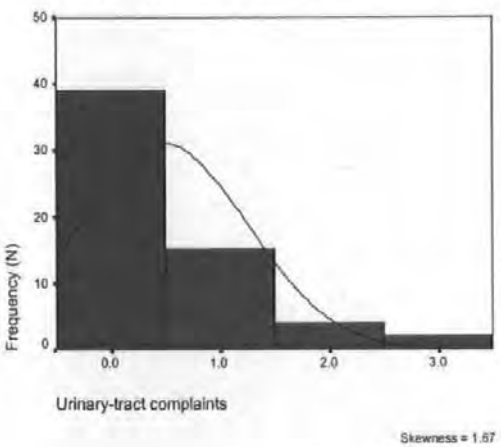
Atopic Complaints



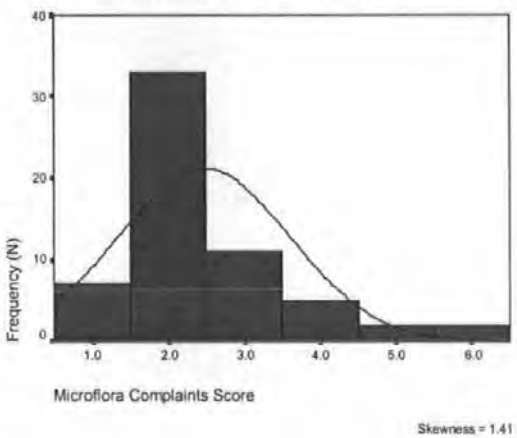
Gastric Complaints



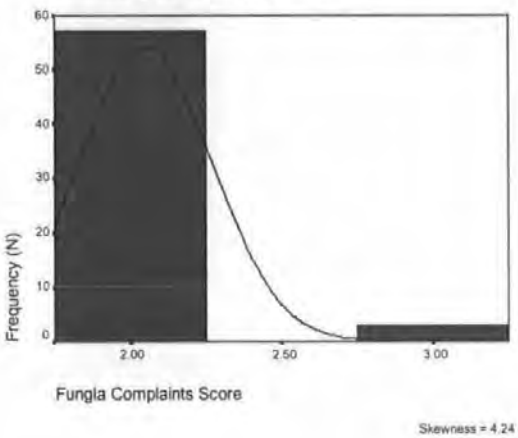
Urinary-tract Complaints



Microflora Complaints



Fungal Complaints



Perceived Workload by Health Status

Total Ill-health

	Total Ill-health	N	Mean	Std. Deviation
Mental Demand	Good	26	92.5077	24.5244
	Poor	34	101.8471	22.2008
Physical Demand	Good	26	27.0808	23.3815
	Poor	34	22.6647	20.0962
Temporal Demand	Good	26	74.3923	34.4099
	Poor	34	73.1647	25.9180
Effort	Good	26	74.4038	26.6238
	Poor	34	83.4029	24.7902
Performance	Good	26	50.2154	24.9628
	Poor	34	49.7794	24.1934
Frustration	Good	26	37.3769	25.2025
	Poor	34	59.4941	22.8584

Stress-Related Complaints

	Stress-related	N	Mean	Std. Deviation
Mental Demand	Good	29	95.5379	23.9154
	Poor	31	99.9161	23.2984
Physical Demand	Good	29	24.0793	19.5076
	Poor	31	25.0452	23.5320
Temporal Demand	Good	29	77.7793	31.6056
	Poor	31	69.8774	27.6199
Effort	Good	29	77.6483	21.6012
	Poor	31	81.2387	29.4029
Performance	Good	29	51.7655	24.4090
	Poor	31	48.2871	24.5175
Frustration	Good	29	40.2241	22.7281
	Poor	31	58.9710	26.2038

Indicators of Ill-health

	Indicators	N	Mean	Std. Deviation
Mental Demand	Good	20	92.1750	25.5295
	Poor	40	100.6125	22.2190
Physical Demand	Good	20	24.3900	19.9240
	Poor	40	24.6725	22.4970
Temporal Demand	Good	20	82.3250	31.8107
	Poor	40	69.3825	27.8875
Effort	Good	20	81.6150	22.1017
	Poor	40	78.4475	27.6327
Performance	Good	20	51.6950	24.5078
	Poor	40	49.1050	24.4926
Frustration	Good	20	40.6300	25.5619
	Poor	40	54.5500	25.4853

Psychological Complaints

	Psychological Complaints	N	Mean	Std. Deviation
Mental Demand	Good	20	95.4850	26.5445
	Poor	40	98.9575	22.0941
Physical Demand	Good	20	28.1200	20.0025
	Poor	40	22.8075	22.2479
Temporal Demand	Good	20	78.9500	28.0092
	Poor	40	71.0700	30.4063
Effort	Good	20	75.4900	21.0684
	Poor	40	81.5100	27.8629
Performance	Good	20	52.3050	25.0401
	Poor	40	48.8000	24.1895
Frustration	Good	20	44.3100	25.0632
	Poor	40	52.7100	26.5315

Immune Challenge Complaints

	Immune Challenge	N	Mean	Std. Deviation
Mental Demand	Good	21	89.8238	24.6091
	Poor	39	102.0949	22.0159
Physical Demand	Good	21	29.5190	24.0514
	Poor	39	21.9179	19.8175
Temporal Demand	Good	21	68.2095	31.8053
	Poor	39	76.6513	28.3709
Effort	Good	21	66.8524	26.3969
	Poor	39	86.3154	22.9917
Performance	Good	21	48.9619	25.0620
	Poor	39	50.5103	24.2260
Frustration	Good	21	43.2095	29.5191
	Poor	39	53.5179	23.7591

Atopic Complaints

	Atopy	N	Mean	Std. Deviation
Mental Demand	Good	40	96.9000	22.9677
	Poor	20	99.6000	25.0430
Physical Demand	Good	40	27.9000	23.3698
	Poor	20	17.9350	15.6579
Temporal Demand	Good	40	71.0750	32.3549
	Poor	20	78.9400	23.1008
Effort	Good	40	76.1975	25.2889
	Poor	20	86.1150	26.0980
Performance	Good	40	50.4625	22.5391
	Poor	20	48.9800	28.1493
Frustration	Good	40	44.4050	26.0733
	Poor	20	60.9200	23.1463

Gastric Complaints

	Gastric Complaints	N	Mean	Std. Deviation
Mental Demand	Good	40	95.4800	26.6642
	Poor	20	102.4400	14.8891
Physical Demand	Good	40	25.0200	22.4156
	Poor	20	23.6950	20.0823
Temporal Demand	Good	40	70.8625	30.8377
	Poor	20	79.3650	26.8910
Effort	Good	40	75.3300	26.7407
	Poor	20	87.8500	22.0246
Performance	Good	40	53.4000	25.0521
	Poor	20	43.1050	21.7729
Frustration	Good	40	48.7075	27.5990
	Poor	20	52.3150	23.4508

Urinary tract complaints

	Urinary-tract	N	Mean	Std. Deviation
Mental Demand	Good	39	96.4821	25.8194
	Poor	21	100.2476	18.7885
Physical Demand	Good	39	24.6154	22.5303
	Poor	21	24.5095	19.9905
Temporal Demand	Good	39	72.4692	30.5867
	Poor	21	75.9762	28.3479
Effort	Good	39	76.1564	28.0793
	Poor	21	85.7190	19.9811
Performance	Good	39	51.2692	23.4318
	Poor	21	47.5524	26.3102
Frustration	Good	39	45.1615	25.5496
	Poor	21	58.7286	25.5055

Microflora Complaints

	Microflora	N	Mean	Std. Deviation
Mental Demand	Good	7	84.0143	36.4662
	Poor	53	99.6208	21.0775
Physical Demand	Good	7	29.4571	20.8724
	Poor	53	23.9340	21.6970
Temporal Demand	Good	7	77.9143	21.5746
	Poor	53	73.1396	30.6522
Effort	Good	7	73.4286	33.1596
	Poor	53	80.3057	24.9246
Performance	Good	7	51.3571	29.5286
	Poor	53	49.7849	23.8787
Frustration	Good	7	52.1143	26.2329
	Poor	53	49.6189	26.3706

Pre & Post-Stress S-IgA Means

Health Status

Total Ill-health

	Total Ill-health	Mean	Std. Error Mean
Pre	Good	125.0192	10.7584
	Poor	135.2059	9.9210
Post	Good	162.6154	10.1106
	Poor	159.6029	10.0895

Indicators

	Indicators	Mean	Std. Error Mean
Pre	Good	128.1250	12.3733
	Poor	132.1250	9.0866
Post	Good	159.9750	12.3488
	Poor	161.3750	8.8731

Immune-challenge

	Immune	Mean	Std. Error Mean
Pre	Good	135.262	13.4577
	Poor	128.385	8.6342
Post	Good	169.524	10.5004
	Poor	156.269	9.4408

Gastric

	Gastric	Mean	Std. Error Mean
Pre	Good	131.063	9.1273
	Poor	130.250	12.2716
Post	Good	165.488	8.3713
	Poor	151.750	13.4709

Microflora

	Microflora	Mean	Std. Error Mean
Pre	Good	126.071	13.7786
	Poor	131.415	8.0601
Post	Good	151.071	17.9660
	Poor	162.208	7.7720

Stress-related

	Stress-related	Mean	Std. Error Mean
Pre	Good	123.7241	11.4982
	Poor	137.4032	9.1014
Post	Good	157.0862	10.7739
	Poor	164.4839	9.6007

Psychological

	Psychological	Mean	Std. Error Mean
Pre	Good	128.73	13.6312
	Poor	131.82	8.6402
Post	Good	160.07	11.8677
	Poor	161.32	9.0318

Atopy

	Atopy	Mean	Std. Error Mean
Pre	Good	129.625	8.7086
	Poor	133.125	13.4479
Post	Good	158.400	8.7754
	Poor	165.925	12.5559

Urinary-tract

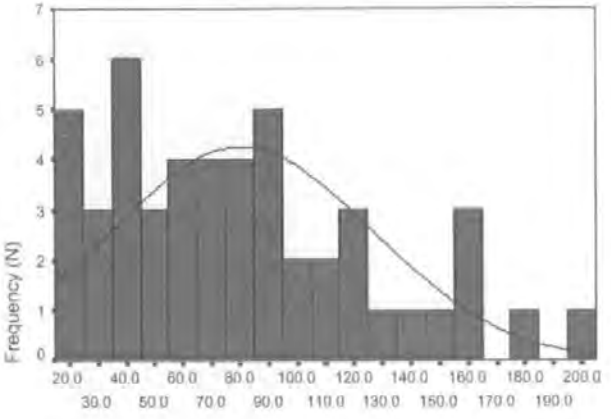
	Urinary-tract	Mean	Std. Error Mean
Pre	Good	141.9103	9.2452
	Poor	110.1429	10.5500
Post	Good	168.1282	9.0061
	Poor	147.5000	11.4208

Appendix D

Study Two

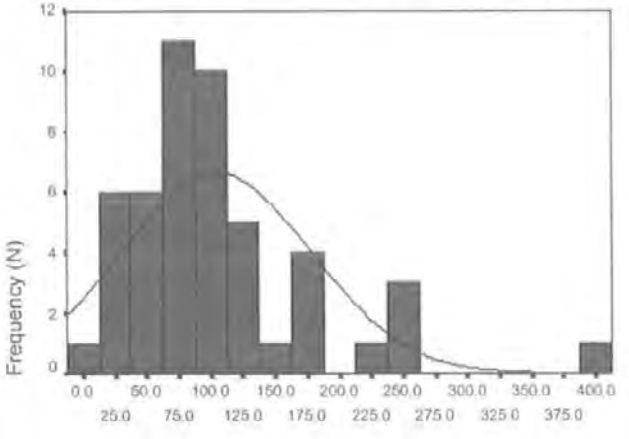
S-IgA Distributions

Day One Pre-stress S-IgA Secretion Rate



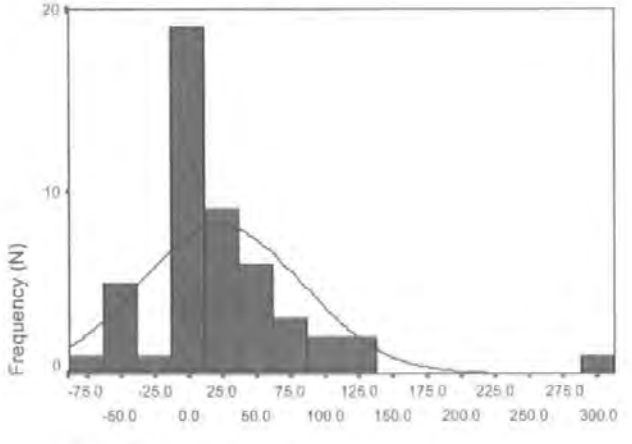
Skewness = .87

Day Two Post-stress S-IgA Secretion Rate



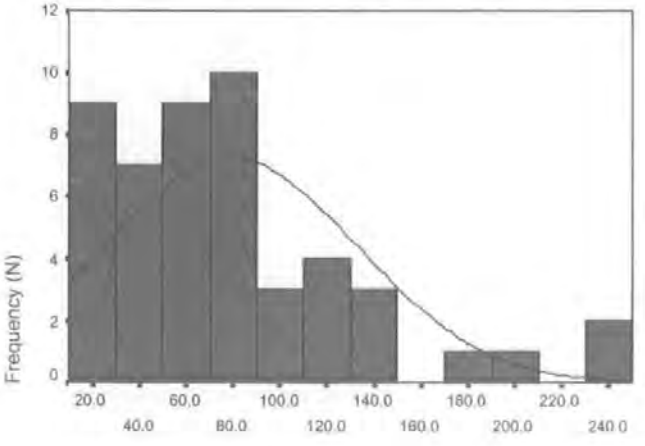
Skewness = 1.96

Day One S-IgA Reactivity



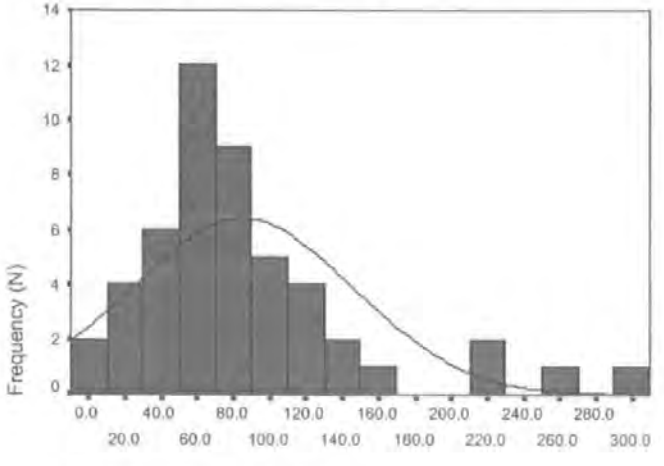
Skewness = 2.23

Day Two Pre-stress S-IgA Secretion Rate



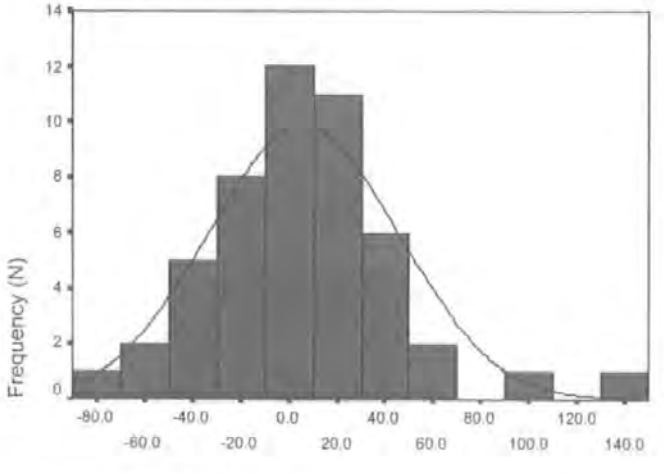
Skewness = 1.39

Day Two Post-stress S-IgA Secretion Rate



Skewness = 1.81

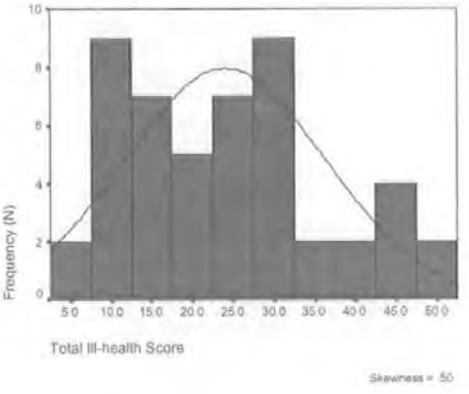
Day Two S-IgA Reactivity



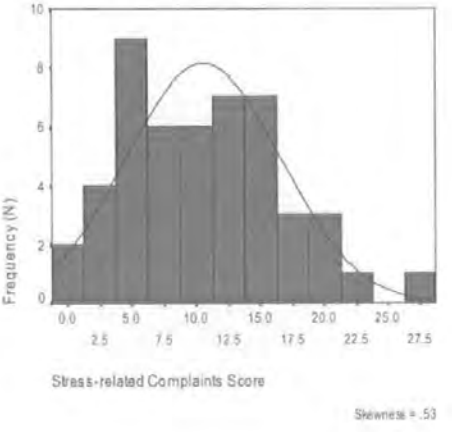
Skewness = .72

MHC Cluster Distributions

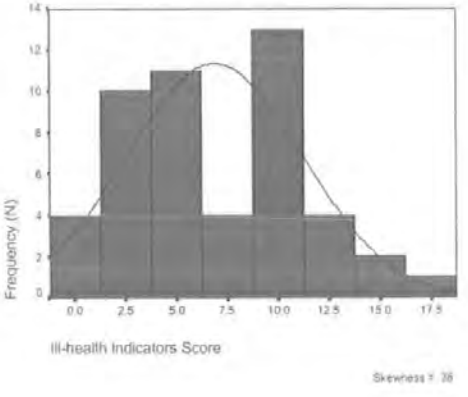
Total Ill-health



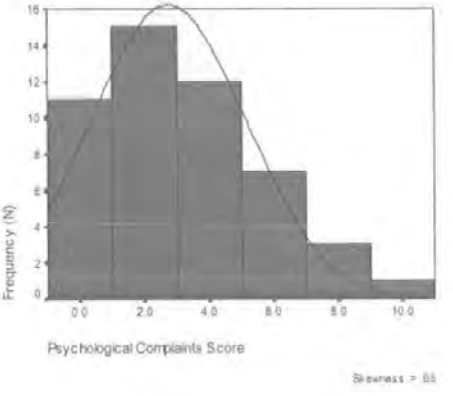
Stress-related Complaints



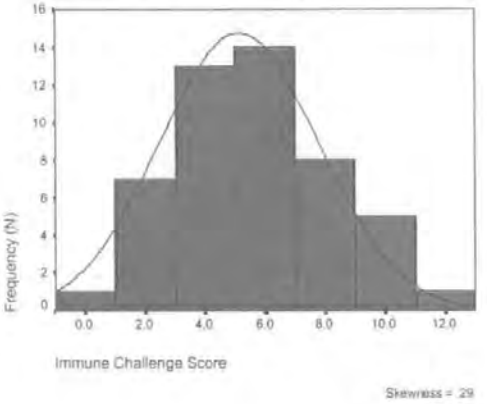
Ill-health Indicators



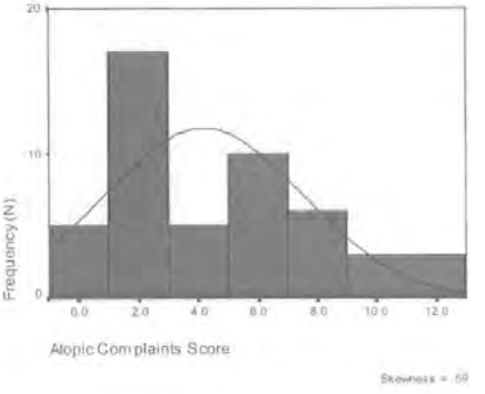
Psychological Complaints



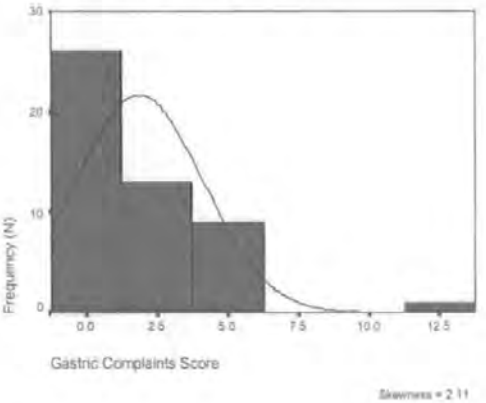
Immune-Challenge Complaints



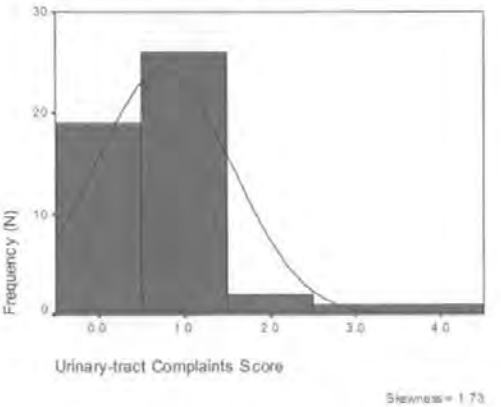
Atopic Complaints



Gastric Complaints

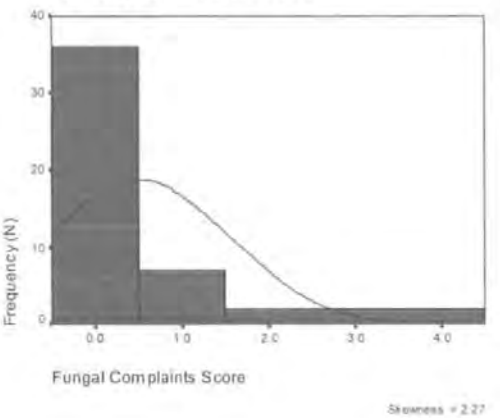


Urinary-tract Complaints



Microflora Complaints

Fungal Complaints



Perceived Workload by Health Status

Total Ill-health

	Total Ill-health	Day 1 Mean (SD)	Day 2 Mean (SD)
Mental Demand	Good	85.90 (28.57)	82.66 (32.30)
	Poor	92.10 (24.90)	86.34 (25.01)
Physical Demand	Good	13.28 (15.36)	14.63 (13.74)
	Poor	18.23 (20.58)	19.09 (19.47)
Temporal Demand	Good	73.83 (26.63)	72.58 (29.65)
	Poor	85.10 (30.80)	81.49 (35.31)
Effort	Good	72.68 (28.03)	68.40 (29.74)
	Poor	81.45 (20.80)	84.29 (32.03)
Performance	Good	57.66 (24.66)	68.00 (23.25)
	Poor	52.45 (32.61)	62.12 (29.08)
Frustration	Good	35.90 (28.23)	36.57 (26.67)
	Poor	55.63 (31.21)	46.79 (34.66)

Stress-Related Complaints

	Stress-related	Day 1 Mean (SD)	Day 2 Mean (SD)
Mental Demand	Good	83.98 (28.82)	81.13 (33.02)
	Poor	93.60 (24.34)	87.63 (24.42)
Physical Demand	Good	11.97 (13.49)	12.48 (9.86)
	Poor	19.18 (21.19)	20.95 (20.74)
Temporal Demand	Good	71.24 (25.78)	69.88 (28.09)
	Poor	86.86 (30.19)	83.55 (35.36)
Effort	Good	72.20 (28.79)	67.01 (30.50)
	Poor	81.21 (20.40)	84.42 (30.63)
Performance	Good	59.82 (24.24)	68.63 (22.76)
	Poor	50.62 (31.84)	61.95 (28.93)
Frustration	Good	34.54 (28.89)	35.88 (27.57)
	Poor	55.40 (29.87)	46.66 (33.16)

Indicators of Ill-health

	Indicators	Day 1 Mean (SD)	Day 2 Mean (SD)
Mental Demand	Good	81.30 (29.71)	78.26 (30.69)
	Poor	96.39 (21.60)	90.62 (26.32)
Physical Demand	Good	15.70 (19.43)	15.15 (12.66)
	Poor	15.30 (16.50)	18.17 (19.95)
Temporal Demand	Good	70.47 (29.69)	67.41 (28.31)
	Poor	87.66 (25.65)	86.13 (33.92)
Effort	Good	69.46 (21.16)	68.29 (28.75)
	Poor	84.07 (17.98)	83.08 (33.03)
Performance	Good	64.66 (20.21)	72.38 (18.80)
	Poor	45.58 (32.44)	58.04 (30.38)
Frustration	Good	34.53 (29.11)	31.22 (22.96)
	Poor	55.42 (29.64)	51.51 (34.43)

Psychological Complaints

	Psych Complaints	Day 1 Mean (SD)	Day 2 Mean (SD)
Mental Demand	Good	86.28 (28.80)	82.18 (29.06)
	Poor	93.22 (22.99)	88.32 (29.39)
Physical Demand	Good	13.68 (17.74)	14.87 (11.61)
	Poor	18.93 (18.13)	19.94 (23.25)
Temporal Demand	Good	75.53 (30.51)	70.70 (30.68)
	Poor	85.21 (25.00)	87.65 (33.19)
Effort	Good	72.93 (28.05)	68.03 (27.52)
	Poor	83.81 (17.15)	89.66 (34.40)
Performance	Good	59.43 (23.88)	69.15 (22.35)
	Poor	47.58 (34.67)	58.22 (31.04)
Frustration	Good	35.83 (29.65)	36.68 (29.95)
	Poor	61.56 (26.54)	49.58 (30.94)

Immune Challenge Complaints

	Immune Challenge	Day 1 Mean (SD)	Day 2 Mean (SD)
Mental Demand	Good	87.45 (27.87)	82.83 (30.38)
	Poor	91.78 (24.97)	88.03 (25.96)
Physical Demand	Good	12.45 (14.38)	15.96 (14.46)
	Poor	23.14 (23.44)	18.29 (21.38)
Temporal Demand	Good	73.32 (28.71)	72.88 (32.70)
	Poor	92.81 (24.93)	85.84 (30.35)
Effort	Good	73.21 (26.65)	67.97 (28.73)
	Poor	85.14 (19.37)	94.46 (31.04)
Performance	Good	58.50 (25.26)	64.51 (22.13)
	Poor	47.36 (34.57)	67.49 (34.52)
Frustration	Good	36.63 (27.45)	36.44 (27.05)
	Poor	65.08 (30.60)	52.96 (36.53)

Atopic Complaints

	Atopic Complaints	Day 1 Mean (SD)	Day 2 Mean (SD)
Mental Demand	Good	88.21 (29.76)	84.46 (32.06)
	Poor	89.27 (23.56)	84.13 (25.54)
Physical Demand	Good	12.89 (15.05)	14.83 (13.60)
	Poor	18.71 (20.31)	18.84 (19.65)
Temporal Demand	Good	74.13 (30.30)	72.78 (34.26)
	Poor	84.73 (26.43)	81.24 (29.78)
Effort	Good	74.78 (28.99)	67.62 (32.17)
	Poor	78.87 (20.00)	85.25 (28.40)
Performance	Good	53.47 (22.43)	65.05 (24.03)
	Poor	57.58 (34.63)	65.73 (28.62)
Frustration	Good	36.95 (25.71)	38.63 (29.53)
	Poor	54.35 (34.43)	44.27 (32.30)

Gastric Complaints

	Gastric Complaints	Day 1 Mean (SD)	Day 2 Mean (SD)
Mental Demand	Good	88.21 (27.28)	89.84 (24.22)
	Poor	89.23 (27.04)	78.07 (33.06)
Physical Demand	Good	19.07 (20.38)	16.28 (14.74)
	Poor	11.47 (13.89)	17.02 (18.68)
Temporal Demand	Good	74.35 (26.89)	73.02 (29.63)
	Poor	84.03 (30.66)	80.60 (35.25)
Effort	Good	73.66 (27.05)	76.51 (23.70)
	Poor	79.96 (23.03)	74.43 (39.03)
Performance	Good	57.40 (25.01)	68.30 (24.82)
	Poor	52.97 (32.04)	62.03 (27.52)
Frustration	Good	39.79 (28.68)	41.75 (29.37)
	Poor	50.37 (32.99)	40.49 (32.60)

Urinary-tract Complaints

	Urinary-tract	Day 1 Mean (SD)	Day 2 Mean (SD)
Mental Demand	Good	82.68 (30.07)	87.93 (35.90)
	Poor	92.49 (24.43)	82.02 (24.08)
Physical Demand	Good	13.06 (13.25)	13.78 (13.31)
	Poor	17.05 (20.32)	18.43 (18.26)
Temporal Demand	Good	76.92 (29.41)	78.05 (32.28)
	Poor	80.14 (28.89)	75.65 (30.80)
Effort	Good	69.81 (28.38)	73.75 (32.02)
	Poor	80.93 (22.37)	76.66 (31.65)
Performance	Good	62.82 (24.83)	74.48 (21.41)
	Poor	50.57 (29.73)	59.58 (27.16)
Frustration	Good	36.11 (31.58)	35.55 (33.34)
	Poor	50.24 (29.72)	44.71 (29.46)

Microflora Complaints

	Microflora	Day 1 Mean (SD)	Day 2 Mean (SD)
Mental Demand	Good	89.12 (29.76)	88.05 (29.60)
	Poor	87.79 (20.56)	46.61 (27.03)
Physical Demand	Good	16.12 (19.22)	14.87 (16.37)
	Poor	14.23 (15.21)	20.26 (16.79)
Temporal Demand	Good	78.42 (27.22)	77.75 (31.00)
	Poor	79.85 (32.83)	74.16 (35.39)
Effort	Good	78.74 (28.48)	78.58 (33.27)
	Poor	72.23 (16.43)	69.25 (28.39)
Performance	Good	60.88 (28.24)	69.32 (27.03)
	Poor	43.84 (25.58)	57.19 (21.98)
Frustration	Good	43.59 (28.38)	41.98 (28.32)
	Poor	47.18 (36.49)	39.47 (35.81)

Fungal Complaints

	Fungal Complaints	Day 1 Mean (SD)	Day 2 Mean (SD)
Mental Demand	Good	86.01 (26.97)	82.41 (29.66)
	Poor	107.87 (18.06)	97.95 (20.92)
Physical Demand	Good	15.42 (18.27)	16.27 (15.62)
	Poor	16.10 (16.14)	19.20 (23.73)
Temporal Demand	Good	79.90 (29.50)	77.59 (31.18)
	Poor	78.80 (25.86)	69.35 (35.07)
Effort	Good	75.10 (25.07)	72.49 (28.21)
	Poor	87.47 (25.46)	97.37 (46.35)
Performance	Good	55.05 (28.46)	65.41 (25.20)
	Poor	57.25 (29.73)	64.97 (33.29)
Frustration	Good	47.63 (30.30)	41.11 (29.18)
	Poor	24.20 (29.75)	41.53 (42.92)

Pre & Post-Stress S-IgA Means

Health Status

Total Ill-health

	Total Ill-health	Mean	Std. Error Mean
Pre1	Good	90.2004	9.8750
	Poor	68.8023	7.6930
Post1	Good	111.457	17.2178
	Poor	95.5582	10.1729
Pre2	Good	81.2600	12.1803
	Poor	75.9882	8.6637
Post2	Good	89.6341	14.2530
	Poor	79.2305	8.6441

Indicators

	Indicators	Mean	Std. Error Mean
Pre1	Good	82.0692	10.7843
	Poor	79.0554	7.5490
Post1	Good	111.804	18.7507
	Poor	96.5213	9.0460
Pre2	Good	74.8108	11.9460
	Poor	83.1454	9.7698
Post2	Good	82.2224	15.2269
	Poor	87.8179	8.3995

Immune-Challenge

	Immune	Mean	Std. Error Mean
Pre1	Good	82.6957	8.3796
	Poor	75.3364	9.7352
Post1	Good	106.4383	13.4891
	Poor	99.0200	15.1744
Pre2	Good	82.1800	10.2254
	Poor	70.6757	8.5849
Post2	Good	91.0354	11.7782
	Poor	69.7821	6.9699

Stress-related

	Stress-related	Mean	Std. Error Mean
Pre1	Good	91.2120	10.6195
	Poor	69.5317	7.1130
Post1	Good	115.734	18.3312
	Poor	92.4275	9.5850
Pre2	Good	82.5024	12.9281
	Poor	75.1333	8.3206
Post2	Good	89.6556	15.2218
	Poor	80.0750	8.3294

Psychological

	Psychological	Mean	Std. Error Mean
Pre1	Good	80.7528	8.7243
	Poor	80.2924	9.7472
Post1	Good	109.46	14.9257
	Poor	94.6324	11.4319
Pre2	Good	76.4947	10.4260
	Poor	83.4076	10.6411
Post2	Good	87.7628	12.7597
	Poor	79.6929	7.7201

Atopy

	Atopy	Mean	Std. Error Mean
Pre1	Good	87.1770	9.8183
	Poor	72.5127	8.2006
Post1	Good	105.0037	12.7735
	Poor	103.4782	17.6802
Pre2	Good	85.6663	11.7097
	Poor	70.5805	9.3220
Post2	Good	89.3148	12.2251
	Poor	79.6223	12.5174

Gastric

	Gastric	Mean	Std. Error Mean
Pre1	Good	78.8335	10.5206
	Poor	82.5822	7.6009
Post1	Good	88.2646	12.3244
	Poor	122.4670	16.9852
Pre2	Good	66.5377	11.7088
	Poor	92.8600	9.0665
Post2	Good	72.7712	12.6323
	Poor	98.7452	11.4627

Mircroflora

	Mircroflora	Mean	Std. Error Mean
Pre1	low	89.1218	8.3176
	high	63.0025	9.3411
Post1	low	111.444	14.4834
	high	89.6238	11.6077
Pre2	low	85.0979	10.6948
	high	66.0956	7.7697
Post2	low	92.4118	12.2964
	high	69.6000	7.4066

Urinary-tract

	Urinary	Mean	Std. Error Mean
Pre1	Good	99.3179	12.1991
	Poor	68.7340	6.7443
Post1	Good	115.69	16.4515
	Poor	97.1150	13.6629
Pre2	Good	91.8800	15.4499
	Poor	70.6680	7.7529
Post2	Good	96.5611	16.0316
	Poor	77.6177	9.9637

Fungal

	Fungal	Mean	Std. Error Mean
Pre1	Good	78.1702	6.5844
	Poor	97.9567	26.4511
Post1	Good	100.30	11.0740
	Poor	133.14	32.5718
Pre2	Good	78.2958	7.7133
	Poor	83.1733	32.4494
Post2	Good	81.2272	8.4447
	Poor	111.74	38.6218

Mood / Personality

Positive Affect

	PA	Mean	Std. Error Mean
Pre1	Low	85.0312	8.6448
	High	75.5761	10.1063
Post1	Low	110.7604	17.0078
	High	97.0370	11.6490
Pre2	Low	71.4454	10.6883
	High	87.3122	11.0545
Post2	Low	82.3338	12.7851
	High	87.9352	11.9131

Negative Affect

	NA	Mean	Std. Error Mean
Pre1	Low	85.5706	9.0541
	High	71.2235	8.0765
Post1	Low	102.5434	11.3172
	High	107.6606	21.9993
Pre2	Low	84.2228	10.9489
	High	68.8606	8.0493
Post2	Low	86.6322	11.6144
	High	81.8212	12.7722

Neuroticism

	Neuroticism	Mean	Std. Error Mean
Pre1	Low	88.4605	12.2695
	High	74.6925	6.8860
Post1	Low	118.041	21.8135
	High	94.0268	8.3107
Pre2	Low	95.0486	15.3511
	High	66.7764	6.3616
Post2	Low	108.880	18.0994
	High	67.0250	5.1219

Openness

	Openness	Mean	Std. Error Mean
Pre1	Low	75.3025	9.4507
	high	85.6720	9.1950
Post1	Low	98.8550	16.9895
	high	109.564	12.7965
Pre2	Low	70.1554	9.9393
	high	87.2812	11.6218
Post2	Low	76.4350	11.8538
	high	93.1500	12.7448

Conscientiousness

	Conscientiousness	Mean	Std. Error Mean
Pre1	Low	83.9013	7.4028
	High	77.4172	10.8462
Post1	Low	106.783	16.7055
	High	101.953	13.2067
Pre2	Low	73.5471	9.3944
	High	84.0252	12.1862
Post2	Low	84.1525	12.0557
	High	85.7412	12.7922

Extraversion

	Extraversion	Mean	Std. Error Mean
Pre1	Low	87.2694	12.3115
	High	77.0463	7.7095
Post1	Low	115.940	22.5252
	High	98.1450	10.8815
Pre2	Low	70.0582	12.6126
	High	83.5866	9.7272
Post2	Low	90.6206	19.0731
	High	81.9575	8.9047

Agreeableness

	Agreeable	Mean	Std. Error Mean
Pre1	Low	77.5759	10.9711
	High	83.0515	8.0398
Post1	Low	90.5859	11.5525
	High	115.509	16.4428
Pre2	Low	75.3309	11.9762
	High	81.7956	10.1549
Post2	Low	80.4123	13.8473
	High	88.6711	11.2614

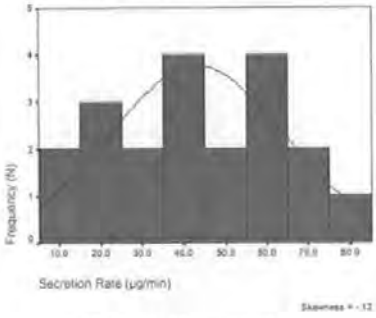
Appendix E

Study Three

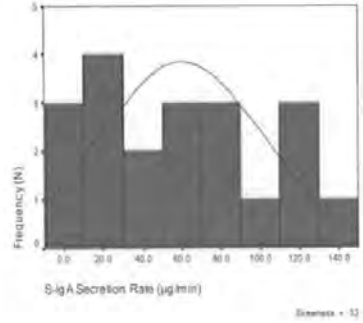
S-IgA Distributions

Stressor One

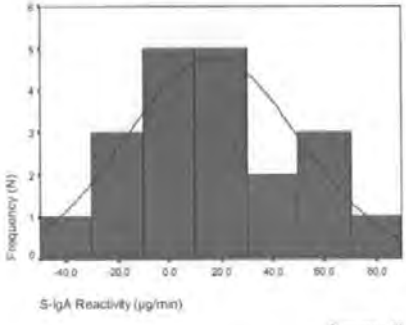
Pre-stress S-IgA Secretion rate



Post-stress S-IgA Secretion Rate

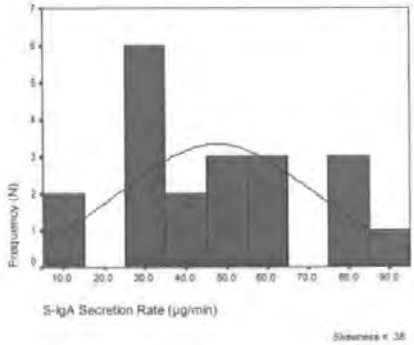


S-IgA Reactivity

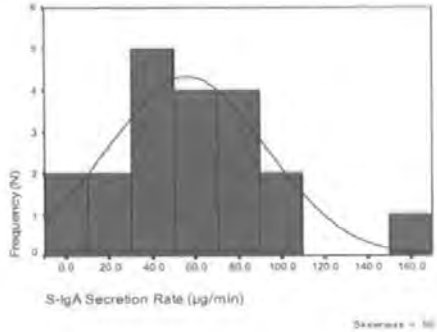


Stressor Two

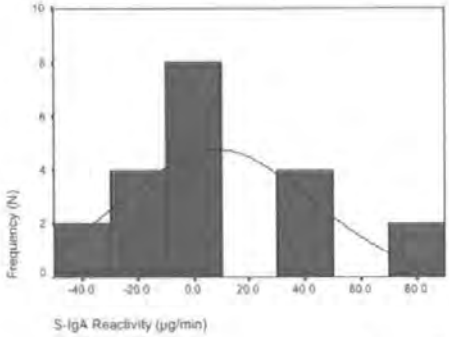
Pre-stress S-IgA Secretion Rate



Post-stress S-IgA Secretion rate

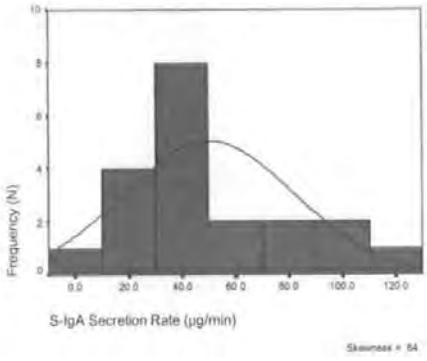


S-IgA Reactivity

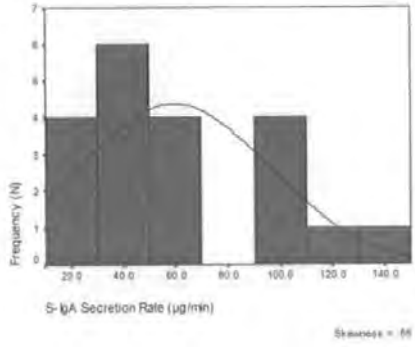


Stressor Three

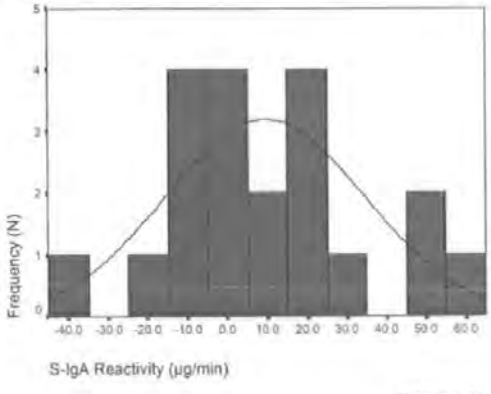
Pre-stress S-IgA Secretion Rate



Post-stress S-IgA Secretion rate

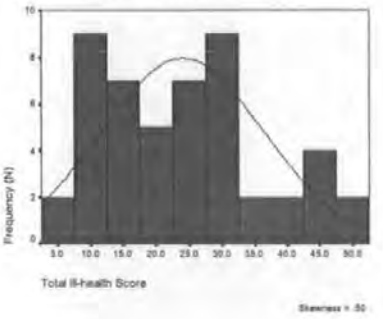


S-IgA Reactivity

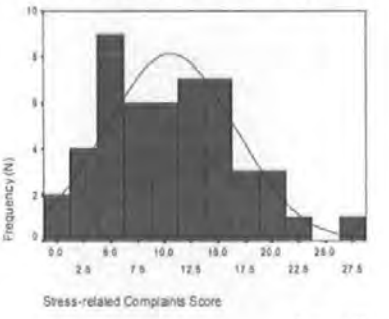


MHC Cluster Distributions

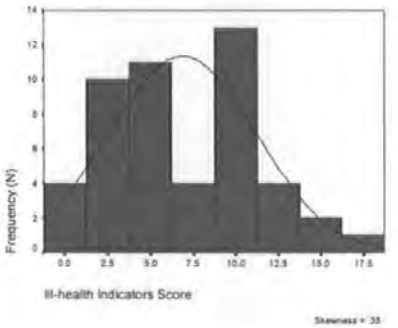
Total Ill-health



Stress-related Complaints

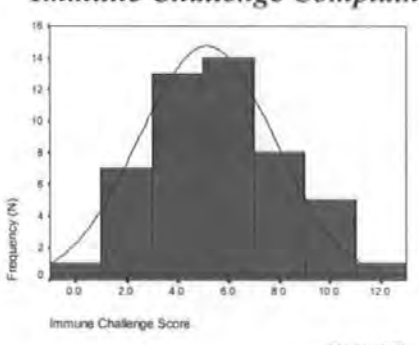


Ill-health Indicators

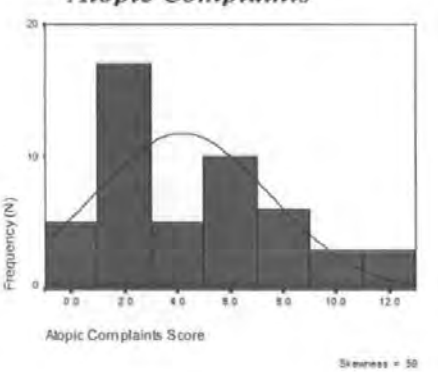


Psychological Complaints

Immune Challenge Complaints



Atopic Complaints



Gastric Complaints

Perceived Workload Demands by Health Status

Total Ill-health

		N	Mean	Std. Deviation	Std. Error
MD1	Good	11	87.3691	23.4937	7.0836
	Poor	9	101.1922	15.6207	5.2069
MD2	Good	11	78.9345	35.7159	10.7687
	Poor	9	91.8300	27.3403	9.1134
MD3	Good	11	73.8282	36.8563	11.1126
	Poor	9	81.0744	33.4797	11.1599
PD1	Good	11	17.8364	16.3699	4.9357
	Poor	9	16.3856	10.4223	3.4741
PD2	Good	11	21.5945	24.2856	7.3224
	Poor	9	34.4444	33.6983	11.2328
PD3	Good	11	25.2000	27.4280	8.2699
	Poor	9	22.1778	21.6660	7.2220
TD1	Good	11	76.7091	29.3170	8.8394
	Poor	9	88.3500	34.1374	11.3791
TD2	Good	11	59.4173	33.3220	10.0470
	Poor	9	96.8511	26.4224	8.8075
TD3	Good	11	69.3336	32.0072	9.6505
	Poor	9	78.4600	29.2539	9.7513
EFF1	Good	11	74.3509	19.5591	5.8973
	Poor	9	92.5911	17.2479	5.7493
EFF2	Good	11	67.8673	28.3934	8.5609
	Poor	9	96.3778	22.9165	7.6388
EFF3	Good	11	70.3327	29.5076	8.8969
	Poor	9	79.2744	24.5509	8.1836
PERF1	Good	11	50.5755	24.0240	7.2435
	Poor	9	53.5200	38.9515	12.9838
PERF2	Good	11	65.4909	18.6751	5.6307
	Poor	9	56.9522	38.9802	12.9934
PERF3	Good	11	74.2609	17.2177	5.1913
	Poor	9	58.5633	39.1604	13.0535
FRUS1	Good	11	41.5945	25.2843	7.6235
	Poor	9	48.5544	23.3375	7.7792
FRUS2	Good	11	48.9764	24.8075	7.4797
	Poor	9	64.1500	28.4295	9.4765
FRUS3	Good	11	41.5509	28.5770	8.6163
	Poor	9	39.5889	14.5199	4.8400

Stress-Related Complaints

		N	Mean	Std. Deviation	Std. Error
MD1	Good	10	88.8130	23.6837	7.4895
	Poor	10	98.3660	17.9904	5.6891
MD2	Good	10	75.0480	33.9221	10.7271
	Poor	10	94.4270	28.5291	9.0217
MD3	Good	10	67.7650	38.9219	12.3082
	Poor	10	86.4130	28.7478	9.0909
PD1	Good	10	21.2270	16.7000	5.2810
	Poor	10	13.1400	8.9576	2.8326
PD2	Good	10	26.0070	23.7371	7.5063
	Poor	10	28.7470	34.4662	10.8992
PD3	Good	10	24.9730	26.8812	8.5006
	Poor	10	22.7070	23.0806	7.2987
TD1	Good	10	80.1600	29.6323	9.3706
	Poor	10	83.7350	34.3543	10.8638
TD2	Good	10	62.8390	35.5088	11.2289
	Poor	10	89.6860	31.0438	9.8169
TD3	Good	10	68.2470	32.0434	10.1330
	Poor	10	78.6340	29.2931	9.2633
EFF1	Good	10	76.3190	20.5817	6.5085
	Poor	10	88.7990	19.0356	6.0196
EFF2	Good	10	70.5540	30.2040	9.5514
	Poor	10	90.8400	25.8599	8.1776
EFF3	Good	10	65.5460	32.5005	10.2776
	Poor	10	83.1670	17.8441	5.6428
PERF1	Good	10	49.4930	25.7637	8.1472
	Poor	10	54.3080	36.3048	11.4806
PERF2	Good	10	64.8330	18.9535	5.9936
	Poor	10	58.4640	37.3693	11.8172
PERF3	Good	10	80.5600	15.4355	4.8811
	Poor	10	53.8340	34.5396	10.9224
FRUS1	Good	10	42.8610	24.6904	7.8078
	Poor	10	46.5920	24.5711	7.7701
FRUS2	Good	10	50.6470	26.8252	8.4829
	Poor	10	60.9620	27.3889	8.6611
FRUS3	Good	10	37.5930	29.2066	9.2359
	Poor	10	43.7430	14.9288	4.7209

Indicators of Ill-health

		N	Mean	Std. Deviation	Std. Error
MD1	Good	12	86.7050	22.1952	6.4072
	Poor	8	103.9162	15.0080	5.3061
MD2	Good	12	72.1508	33.7570	9.7448
	Poor	8	103.6175	18.0145	6.3691
MD3	Good	12	65.8867	38.7312	11.1807
	Poor	8	93.8925	19.0675	6.7414
PD1	Good	12	16.6892	15.4494	4.4599
	Poor	8	17.9250	11.5034	4.0671
PD2	Good	12	17.2783	17.2137	4.9692
	Poor	8	42.5250	36.8024	13.0116
PD3	Good	12	19.9000	21.6661	6.2545
	Poor	8	29.7500	28.5210	10.0837
TD1	Good	12	73.5558	29.9165	8.6361
	Poor	8	94.5350	30.7780	10.8817
TD2	Good	12	55.7158	30.3238	8.7537
	Poor	8	107.0825	10.9888	3.8851
TD3	Good	12	67.4667	32.0691	9.2575
	Poor	8	82.4012	27.0448	9.5618
EFF1	Good	12	74.8158	18.4920	5.3382
	Poor	8	94.1737	18.0958	6.3978
EFF2	Good	12	66.8842	26.3426	7.6044
	Poor	8	101.4163	20.4579	7.2330
EFF3	Good	12	65.3658	29.9259	8.6389
	Poor	8	87.8425	15.3466	5.4258
PERF1	Good	12	46.6725	19.3905	5.5975
	Poor	8	59.7425	43.0885	15.2341
PERF2	Good	12	65.9000	19.8249	5.7230
	Poor	8	55.2713	39.8902	14.1033
PERF3	Good	12	73.3617	18.6013	5.3697
	Poor	8	57.9500	40.5433	14.3342
FRUS1	Good	12	39.5000	23.8592	6.8875
	Poor	8	52.5663	23.6295	8.3543
FRUS2	Good	12	47.2733	23.2123	6.7008
	Poor	8	68.6013	28.4087	10.0440
FRUS3	Good	12	37.1608	25.9893	7.5025
	Poor	8	45.9288	17.2099	6.0846

Psychological Health

		N	Mean	Std. Deviation	Std. Error
MD1	Good	8	87.9250	26.6389	9.4183
	Poor	12	97.3658	16.5754	4.7849
MD2	Good	8	74.1175	34.4062	12.1644
	Poor	12	91.8175	29.8008	8.6028
MD3	Good	8	70.8888	40.9571	14.4805
	Poor	12	81.2225	30.9779	8.9426
PD1	Good	8	17.7750	17.0111	6.0143
	Poor	12	16.7892	11.7893	3.4033
PD2	Good	8	19.9088	19.4583	6.8795
	Poor	12	32.3558	33.5828	9.6945
PD3	Good	8	18.8913	23.6054	8.3458
	Poor	12	27.1392	25.3995	7.3322
TD1	Good	8	79.7088	33.5820	11.8730
	Poor	12	83.4400	31.0827	8.9728
TD2	Good	8	55.5238	35.8306	12.6680
	Poor	12	90.0883	28.4504	8.2129
TD3	Good	8	64.1250	34.9172	12.3451
	Poor	12	79.6508	26.6487	7.6928
EFF1	Good	8	72.9325	21.7972	7.7065
	Poor	12	88.9767	17.2968	4.9932
EFF2	Good	8	64.9588	31.4459	11.1178
	Poor	12	91.1892	23.4759	6.7769
EFF3	Good	8	67.7575	34.4309	12.1732
	Poor	12	78.7558	21.4173	6.1827
PERF1	Good	8	45.8162	23.2452	8.2184
	Poor	12	55.9567	35.2604	10.1788
PERF2	Good	8	62.1825	19.5148	6.8995
	Poor	12	61.2925	34.8171	10.0508
PERF3	Good	8	77.3000	15.4231	5.4529
	Poor	12	60.4617	34.9369	10.0854
FRUS1	Good	8	41.5175	27.5413	9.7373
	Poor	12	46.8658	22.4476	6.4801
FRUS2	Good	8	45.3425	27.5705	9.7476
	Poor	12	62.7792	25.1891	7.2715
FRUS3	Good	8	35.0663	30.2165	10.6831
	Poor	12	44.4025	16.6856	4.8167

Immune Challenge Complaints

		N	Mean	Std. Deviation	Std. Error
MD1	Good	13	92.1738	22.9770	6.3727
	Poor	7	96.2186	18.2926	6.9140
MD2	Good	13	83.9292	33.4060	9.2652
	Poor	7	86.2386	31.9987	12.0944
MD3	Good	13	77.0400	33.1785	9.2021
	Poor	7	77.1800	40.0021	15.1194
PD1	Good	13	17.1900	11.1183	3.0837
	Poor	7	17.1714	18.6000	7.0301
PD2	Good	13	27.5077	29.9648	8.3107
	Poor	7	27.1343	28.9382	10.9376
PD3	Good	13	22.2769	21.7967	6.0453
	Poor	7	26.7429	30.3633	11.4763
TD1	Good	13	76.4062	32.2558	8.9461
	Poor	7	92.2386	28.7599	10.8702
TD2	Good	13	69.5377	37.9610	10.5285
	Poor	7	88.7514	27.8476	10.5254
TD3	Good	13	70.5700	33.0137	9.1564
	Poor	7	78.7714	26.1989	9.9023
EFF1	Good	13	79.2092	19.0102	5.2725
	Poor	7	88.7800	22.7265	8.5898
EFF2	Good	13	75.2569	30.3187	8.4089
	Poor	7	90.8000	26.3212	9.9485
EFF3	Good	13	72.1538	26.4443	7.3343
	Poor	7	78.4471	29.8689	11.2894
PERF1	Good	13	47.4462	29.3067	8.1282
	Poor	7	60.1729	33.9329	12.8254
PERF2	Good	13	58.0823	25.3672	7.0356
	Poor	7	68.2714	36.0782	13.6363
PERF3	Good	13	65.5185	27.3978	7.5988
	Poor	7	70.3143	34.9766	13.2199
FRUS1	Good	13	45.8923	25.5418	7.0840
	Poor	7	42.5614	22.7696	8.6061
FRUS2	Good	13	50.1908	20.0891	5.5717
	Poor	7	66.2300	35.9312	13.5807
FRUS3	Good	13	38.0346	16.2128	5.0513
	Poor	7	45.5586	30.6434	11.5821

Atopic Complaints

		N	Mean	Std. Deviation	Std. Error
MD1	Good	12	90.9883	22.3723	6.4583
	Poor	8	97.4912	19.6714	6.9549
PD1	Good	12	21.9558	14.3041	4.1292
	Poor	8	10.0250	9.4509	3.3414
TD1	Good	12	75.3675	30.8878	8.9165
	Poor	8	91.8175	31.1563	11.0154
EFF1	Good	12	80.5658	23.7820	6.8653
	Poor	8	65.5488	14.6961	5.1958
PERF1	Good	12	55.4675	31.6450	9.1351
	Poor	8	46.5500	30.6013	10.8192
FRUS1	Good	12	44.1283	23.3557	6.7422
	Poor	8	45.6238	26.6691	9.4290
MD2	Good	12	80.6625	33.2141	9.5881
	Poor	8	90.8500	31.4555	11.1212
PD2	Good	12	35.2725	30.5254	8.8119
	Poor	8	15.5338	22.9280	8.1063
TD2	Good	12	67.3883	36.3777	10.5013
	Poor	8	89.5738	30.9513	10.9430
EFF2	Good	12	76.2067	32.7180	9.4449
	Poor	8	87.4325	23.6243	8.3524
PERF2	Good	12	71.3108	22.6444	6.5369
	Poor	8	47.1550	32.8695	11.6211
FRUS2	Good	12	58.8117	25.9631	7.4949
	Poor	8	51.2938	29.4558	10.4142
MD3	Good	12	68.2600	32.4441	9.3658
	Poor	8	90.3325	35.6442	12.6021
PD3	Good	12	34.0558	26.9280	7.7734
	Poor	8	8.5163	5.5591	1.9654
TD3	Good	12	65.8450	28.9148	8.3470
	Poor	8	84.8338	30.6496	10.8363
EFF3	Good	12	67.2775	28.2757	8.1625
	Poor	8	84.9750	22.7619	8.0475
PERF3	Good	12	77.9725	18.7987	5.4267
	Poor	8	51.0338	36.0196	12.7349
FRUS3	Good	12	43.0883	26.2687	7.5831
	Poor	8	37.0375	17.3449	6.1324

Gastric Complaints

		N	Mean	Std. Deviation	Std. Error
MD1	Good	12	91.2550	22.8360	6.5922
	Poor	8	97.0913	18.9647	6.7051
PD1	Good	12	14.6333	11.7488	3.3916
	Poor	8	21.0088	16.2379	5.7410
TD1	Good	12	81.5450	31.6444	9.1349
	Poor	8	82.5512	32.8822	11.6256
EFF1	Good	12	87.0158	23.1208	6.6744
	Poor	8	75.8738	14.0215	4.9573
PERF1	Good	12	63.9892	33.0500	9.5407
	Poor	8	33.7675	14.8940	5.2658
FRUS1	Good	12	41.7550	13.5691	3.9171
	Poor	8	49.1838	35.2534	12.4639
MD2	Good	12	81.7558	35.8708	10.3550
	Poor	8	89.2100	27.0944	9.5793
PD2	Good	12	34.3725	33.0297	9.5348
	Poor	8	16.8838	18.2490	6.4520
TD2	Good	12	81.8267	33.5432	9.6831
	Poor	8	67.9162	38.3958	13.5750
EFF2	Good	12	79.8000	36.5816	10.5602
	Poor	8	82.0425	14.8434	5.2479
PERF2	Good	12	62.3142	27.8798	8.0482
	Poor	8	60.6500	32.6031	11.5269
FRUS2	Good	12	47.4008	22.8623	6.5998
	Poor	8	68.4100	29.0112	10.2570
MD3	Good	12	76.8717	35.4184	10.2244
	Poor	8	77.4150	35.8774	12.6846
PD3	Good	12	24.9275	25.6118	7.3935
	Poor	8	22.2087	24.1199	8.5277
TD3	Good	12	70.1500	34.3596	9.9187
	Poor	8	78.3763	24.4717	8.6520
EFF3	Good	12	77.2500	28.5917	8.2537
	Poor	8	70.0163	25.8602	9.1430
PERF3	Good	12	70.4725	28.4867	8.2234
	Poor	8	62.2838	32.0809	11.3423
FRUS3	Good	12	36.4775	18.6443	5.3822
	Poor	8	46.9538	28.0856	9.9298

Pre & Post-Stress S-IgA Means

Total Ill-health

	Total Ill-health	Mean	Std. Deviation	Std. Error Mean
Pre1	Good	43.0200	24.0147	7.5941
	Poor	43.0400	19.2083	6.0742
Post1	Good	59.5260	48.1461	15.2251
	Poor	60.2440	36.2495	11.4631
Pre2	Good	44.9010	26.4254	8.3564
	Poor	50.4140	22.2605	7.0394
Post2	Good	63.2370	44.8502	14.1829
	Poor	48.8100	26.8813	8.5006
Pre3	Good	46.6650	35.4929	11.2238
	Poor	53.5860	28.7107	9.0791
Pos3	Good	67.8570	44.7266	14.1438
	Poor	51.3540	25.4765	8.0564
Recovery	Good	47.6590	36.6228	11.5811
	Poor	44.2980	19.5788	6.1914

Stress-related

	Stress-Related	Mean	Std. Deviation	Std. Error Mean
Pre1	Good	38.1030	23.7439	7.5085
	Poor	47.9570	18.1092	5.7266
Post1	Good	57.2240	46.9255	14.8392
	Poor	62.5460	37.6115	11.8938
Pre2	Good	39.6110	22.9994	7.2731
	Poor	55.7040	23.1918	7.3339
Post2	Good	62.7880	45.1601	14.2809
	Poor	49.2590	26.6206	8.4182
Pre3	Good	42.3100	33.3914	10.5593
	Poor	57.9410	29.3247	9.2733
Post3	Good	60.5230	45.2699	14.3156
	Poor	58.6880	27.3787	8.6579
Recovery	Good	41.4910	28.9977	9.1699
	Poor	50.4660	29.0726	9.1936

Indicators

	Indicators	Mean	Std. Deviation	Std. Error Mean
Pre1	Good	39.6842	24.2276	6.9939
	Poor	48.0487	15.6677	5.5394
Post1	Good	62.4267	44.5382	12.8571
	Poor	56.0725	39.0554	13.8082
Pre2	Good	43.4800	24.8436	7.1717
	Poor	53.9238	22.6263	7.9996
Post2	Good	62.7825	41.1233	11.8713
	Poor	45.8850	28.4731	10.0668
Pre3	Good	45.0342	32.5791	9.4048
	Poor	57.7625	30.5729	10.8092
Post3	Good	67.3392	41.7915	12.0642
	Poor	48.0050	24.4987	8.6616
Recovery	Good	47.0567	33.2645	9.6026
	Poor	44.3613	21.9441	7.7584

Psychological

	Psychological	Mean	Std. Deviation	Std. Error Mean
Pre1	Good	38.6488	24.9518	8.8218
	Poor	45.9508	18.8218	5.4334
Post1	Good	59.3750	51.7644	18.3015
	Poor	60.2250	35.5872	10.2731
Pre2	Good	39.6488	25.6057	9.0530
	Poor	52.9967	22.2622	6.4265
Post2	Good	69.7175	48.4384	17.1256
	Poor	46.8942	24.7233	7.1370
Pre3	Good	43.6338	37.7282	13.3389
	Poor	54.4533	27.7491	8.0105
Post3	Good	70.3900	45.5537	16.1057
	Poor	52.4158	28.8109	8.3170
Recovery	Good	39.6000	32.5620	11.5124
	Poor	50.2308	26.3077	7.5944

Immune-Challenge

	Immune-challenge	Mean	Std. Deviation	Std. Error Mean
Pre1	Good	40.2885	23.7280	6.5810
	Poor	48.1214	15.6821	5.9273
Post1	Good	47.0362	42.8116	11.8738
	Poor	83.7471	27.5839	10.4257
Pre2	Good	47.7015	26.8632	7.4505
	Poor	47.5757	19.3079	7.2977
Post2	Good	56.2246	42.5593	11.8038
	Poor	55.6500	25.5308	9.6497
Pre3	Good	50.5392	35.2596	9.7793
	Poor	49.3571	26.0472	9.8449
Post3	Good	59.9077	39.3139	10.9037
	Poor	59.0443	33.3094	12.5898
Recovery	Good	43.0338	32.8865	9.1211
	Poor	51.4471	19.4816	7.3633

Atopy

	Atopy	Mean	Std. Deviation	Std. Error Mean
Pre1	Good	35.9908	22.3446	6.4503
	Poor	53.5888	14.7955	5.2310
Post1	Good	45.9875	42.3210	12.2170
	Poor	80.7313	32.0610	11.3353
Pre2	Good	37.2592	20.1131	5.8062
	Poor	63.2550	21.3901	7.5625
Post2	Good	54.6442	41.8351	12.0767
	Poor	58.0925	30.0927	10.6394
Pre3	Good	40.4783	30.2346	8.7280
	Poor	64.5963	29.6370	10.4783
Post3	Good	50.9808	37.9826	10.9646
	Poor	72.5425	31.8623	11.2650
Recovery	Good	33.9583	25.2229	7.2812
	Poor	64.0087	24.6253	8.7063

Gastric

	Gastric	Mean	Std. Deviation	Std. Error Mean
Pre1	Good	39.1667	20.1666	5.8216
	Poor	48.8250	22.6450	8.0062
Post1	Good	56.2658	43.4713	12.5491
	Poor	65.3138	40.5516	14.3371
Pre2	Good	47.8242	23.2310	6.7062
	Poor	47.4075	26.6167	9.4104
Post2	Good	46.9042	30.9220	8.9264
	Poor	69.7025	42.4857	15.0210
Pre3	Good	49.5658	28.1164	8.1165
	Poor	50.9650	38.3420	13.5559
Post3	Good	56.1300	31.2083	9.0091
	Poor	64.8188	44.9309	15.8855
Recovery	Good	42.9475	31.1301	8.9865
	Poor	50.5250	25.7516	9.1046

Positive & negative Affect

Positive Affect

	PA	Mean	Std. Deviation	Std. Error Mean
Pre1	Low	46.1057	20.0956	7.5954
	High	41.3738	22.3347	6.1945
Post1	Low	60.1186	44.0759	16.6591
	High	59.7592	41.8674	11.6119
Pre2	Low	59.3329	21.8019	8.2403
	High	41.3708	23.4103	6.4928
Post2	Low	53.3529	24.7372	9.3498
	High	57.4615	42.7196	11.8483
Pre3	Low	64.7086	26.6344	10.0669
	High	42.2731	32.2094	8.9333
Post3	Low	62.8671	23.1912	8.7654
	High	57.8492	42.6867	11.8392
Recovery	Low	47.6657	21.7808	8.2324
	High	45.0700	32.5326	9.0229

Negative Affect

	NA	Mean	Std. Deviation	Std. Error Mean
Pre1	Low	33.8988	24.4017	8.6273
	High	49.1175	17.1388	4.9475
Post1	Low	53.5775	45.7009	16.1577
	High	64.0900	39.9328	11.5276
Pre2	Low	41.8888	26.1869	9.2585
	High	51.5033	22.6684	6.5438
Post2	Low	73.6550	45.2659	16.0039
	High	44.2692	25.5130	7.3650
Pre3	Low	48.4325	38.8405	13.7322
	High	51.2542	27.6301	7.9761
Post3	Low	64.4850	43.1848	15.2681
	High	56.3525	32.8017	9.4690
Recovery	Low	41.9538	28.8663	10.2058
	High	48.6617	29.4320	8.4963