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**THE RELATIONSHIP BETWEEN DAILY MOOD**  
**AND SALIVARY IMMUNOGLOBULIN A.**

A thesis presented in fulfilment of the  
requirements for the degree of

MASTER OF ARTS

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PSYCHOLOGY

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## ABSTRACT

The aim of the present study was to examine the influence of daily positive and negative mood on secretory immunoglobulin A (S-IgA) concentrations in human saliva. An instrument was constructed for the measurement of daily mood, based on current theories in the psychobiology of affect, neuroendocrinology and behaviour. With this instrument the average intensity, peak intensity and duration of eight moods, two from each pole of positive and negative affect dimensions, were measured. From these scores three positive affect variables were created by combining scores on positive dimension moods, and three negative affect variables created by combining scores on negative dimension moods, and these variables were used for multivariate analysis. Twenty female subjects between the ages of 18 and 60 years were studied for 28 consecutive days. They were each required to capture 1.5 ml of free flowing parotid saliva, fill in the mood questionnaire, and record whether or not they had taken medication, exercise, alcohol, tobacco or menstruated on each evening of the study. These last variables were subsequently used as control variables in the multivariate analysis. Concentrations of S-IgA in the saliva were measured with an enzyme-linked immunosorbent assay (ELISA). No significant associations between S-IgA levels and positive or negative mood variables were detected. The lack of significant effects of mood variables on S-IgA is discussed in the context of the psychoneuroimmunological literature, and with particular emphasis on measurement issues.

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## INTRODUCTION

A large body of scientific evidence now exists in support of the long-held popular belief that physical health status is in some way affected by psychological factors. More specifically, an association between psychological stress and lowered resistance to disease has been reported in a number of studies, both experimental and observational, and in both animals and humans (Cohen and Williamson, 1991, and O'Leary, 1990).

The present thesis focuses on the effects of mood on one component of the immune system viz. salivary immunoglobulin A (S-IgA). In the introduction I provide a brief summary of the area of research on psychological stress, immunoglobulins, and health, and a critique of recent results on mood and S-IgA, from which research questions will be identified. A theoretical framework will be formulated to allow construction of a research instrument that is compatible with recent research in the areas of mood structure, psychoimmunology, and the psychobiology of emotion.

### A. The Immune System and Salivary IgA

#### (a) The place of S-IgA in immunity

The body protects itself against invasion by foreign agents with a battery of defenses. These can be broadly categorised into two main classes, conferring non-specific and specific immunity respectively (Bowry, 1984). Non-specific immunity provides means of controlling against invasion, and includes genetic control which determines species specificity, physical and mechanical barriers such as a thick skin, biochemical factors such as acid secretion in the stomach, interferon and complement production within body tissues, and cellular mechanisms such as phagocytosis and natural killer cell activity. Characteristically, these mechanisms do not involve specific recognition of an agent as foreign, or "non-self".

In contrast, specific immunity involves reaction against foreign "non-self" agents, when special immune products we produce react specifically with the stimulating foreign agent (antigen). The specific reaction may be either cell-mediated or humoral. In both



cases recognition of non-self (antigen) materials is achieved by immunoglobulin molecules. In cell mediated immunity these are incorporated into the cell surface as receptors and their reaction with an antigen induces a cellular response. In humoral immunity on the other hand the immunoglobulin molecules are free "antibodies" which react with antigens, such as molecules on the bacterial cell surface. Antibodies may have multiple combining sites and are therefore able to form cross-links between foreign particles. When antibodies form cross links between a number of these particles a mass is formed, in a process termed agglutination. Agglutination is the mechanism by which secretory immunoglobulins such as S-IgA neutralize bacteria at the mucosal surface.

Antibodies belong to a class of glycoproteins (protein with sugar attached) known as immunoglobulins, all of which have the same basic structure, including a pair of binding sites with which the antibody binds an antigen. A number of different antibody types have been identified, including IgA, IgG, IgM, IgE and IgD. IgA exists in two major forms, serum IgA which is found in the blood, and secretory IgA (s-IgA) which is the main form of IgA, found predominantly in external secretions. It is the s-IgA secreted in the saliva (S-IgA) which is the topic of this study.

(b) S-IgA in the first line of defence

Secretory IgA appears to have evolved specifically for the purpose of protecting body surfaces. It is thought that its most important function is to prevent the adherence of bacteria and viruses to epithelial surfaces (Calbrese et al., 1987) It does so by causing the agglutination of foreign particles.

The importance of the defensive role of secretory IgA in adults is uncertain, as only a third of secretory IgA-deficient subjects suffer from recurrent gastrointestinal or respiratory infections (Bowry, 1984). However, populations showing a depression of S-IgA levels in response to an influence such as stress, also show an increased incidence of upper respiratory tract infections (McClelland et al., 1982; Jemmott & McClelland, 1988). These associations have been cited as evidence in favour of a protective role for S-IgA, but unless the effect of changes in S-IgA levels can be isolated from the numerous other concurrent changes in the immune response, they cannot be regarded as constituting strong evidence that S-IgA is important in

resistance to infection. However, lack of evidence is not evidence of lack of a role for S-IgA. The mere existence of a molecule as specialized in its structure and function as S-IgA suggests that it has had an action of significance to survival.

(c) S-IgA as an exemplar for the immune response

As discussed above, S-IgA is only one of a number of antibodies produced, and antibodies are only one of a battery of defenses used by the body. To what extent then, can S-IgA be used as an exemplar of the immune response?

There is no doubt that antibodies are a major essential component of the humoral branch of the immune system, and their absence is associated with an ultimately fatal susceptibility to disease. It is also true that production of all antibodies is a function of B cells, irrespective of the anatomical site of production. Secretory IgA is produced by B cells beneath the epithelium, but is then transported through the epithelial cells before being released at the external surface with other components that make up the submucous secretions - such as saliva. But the B cells beneath the epithelium are subject to much the same (humoral) influences as other B cells within the body. The actual production of S-IgA may therefore be treated as a reasonable model for production of antibodies as a whole, under the assumption that stress does not differentially affect different specific antibodies (Jemmott & Magliori, 1988). One study has however found total plasma IgA to change independently of IgG, IgM and S-IgA in response to stress (Kiecolt-Glaser et al., 1984).

Use of changes in levels of S-IgA as measured in the saliva, as valid and quantitative indicators of changes in other non-secretory antibodies is not straightforward. The concentration of S-IgA has for instance been shown experimentally to be inversely related to the salivary flow rate, (Tomasi, 1976). Therefore, in theory salivary flow rate could be a confounding factor when S-IgA concentration is to be related to another variable (such as mood) which also affects flow rate, at least when interpretations regarding underlying mechanisms are to be made. Even when flow rate is measured, the interpretation is complicated by the fact that saliva produced under parasympathetic activation differs markedly in composition from that secreted when the sympathetic nervous system is active (Carlson, 1986). However, even when saliva

flow rate is statistically controlled, significant effects of psychological variables on S-IgA have been obtained (e.g. Jemmott & Magliori, 1988).

Some studies suggest that humoral immunity may not be closely coupled to cell-mediated immunity in responses to stress. Thus, changes in antibodies may not parallel (Locke, 1982), or may even be counter to (Kiecolt-Glaser et al., 1984) changes in natural killer cell activity, in response to stress. It is apparent, therefore, that one must be constrained in extrapolating from results of measurements on S-IgA to the immune system as a whole, to the humoral part of the immune system, or to the role of S-IgA as a mediator between stress and rates of upper respiratory infection. This should not be surprising given the complexity of the immune system, - compared in complexity to the brain itself by some - and the number of controls operating within it.

These caveats should not be seen to imply that there is little value in studying the relationship between psychological factors and S-IgA concentrations in saliva, but simply that there are problems in extrapolating from measures of S-IgA to conclusions regarding effects on processes involved in antibody production. Irrespective of the difficulties in understanding mechanisms involved, merely establishing that a relationship exists between psychological state and S-IgA levels has such implications for health that further study in the area is warranted.

#### B. Psychological Factors and Salivary IgA

Although there is now a large literature on the interactions between the central nervous, endocrine, and immune systems, the number of publications concerned directly with the influence of psychological factors on immunocompetence is relatively small. Of these only a portion relate S-IgA (rather than some other parameter of immune function) to psychological variables.

Nearly all of the studies of psychological influences on S-IgA to be described below have focussed on stress, and/or factors which might moderate the effects of stress. In most studies the independent variable has been examination stress. Two studies have examined relaxation alone, and one daily mood in relation to S-IgA. Of the

possible psychosocial moderators of stress, personality, social support, social readjustment, loneliness, and sense of humour have been studied. A general pattern to emerge from these studies has been that a negative relationship exists between stress or negative mood and S-IgA concentrations, and a positive relationship between relaxation, or positive mood and S-IgA.

McClelland, Jemmott, and co-workers have made a number of studies on the effects of stress and its moderation by personality characteristics and social support. These motivational-personality characteristics were termed "power motivation" (desire for impact on others through argument, persuasion, attack, or helping) and "affiliation motivation" (desire for close warm relationships). In a study of the effects of examination stress in students (McClelland and Jemmott, 1980; Jemmott *et al.*, 1983; McClelland *et al.*, 1985), rates of S-IgA secretion were depressed by stress, but more so in those with a high but inhibited power motivation than in those with a need for affiliation. The authors concluded that personality characteristics are important moderators of the effects of stress on S-IgA. Similar findings were obtained with male prisoners (McClelland *et al.*, 1982) when total number of stresses listed in an open ended questionnaire was used as a measure of stress. Those high in need for power and in reported stress showed higher levels of reported illness and lowest concentrations of S-IgA, but a high need for power (motivation) was associated with greater reported illness in the prisoners than in the students.

Those high in need for power, like the "Type A" individual, often show autonomic hyper-responsiveness. Therefore these studies relating stress and personality to S-IgA were extended to a search for parallel changes in the physiological systems which underlie the immune system response. Elevations in sympathetic activation (McClelland *et al.*, 1980) and salivary norepinephrine (McClelland *et al.*, 1985), and motivational arousal through films (McClelland and Kirshnit, 1988) were found to be related to S-IgA.

The finding that affiliation motivation is associated with higher S-IgA than the need for power, and that affiliation motivation arousal leads to increases in S-IgA (McClelland and Kirshnit, 1988), leads naturally to the possibility that social support would also increase S-IgA, particularly in light of evidence that the related variables such as

loneliness and bereavement have been shown to decrease activity in other immune functions (Tecoma and Huey, 1985). Jemmott and Magliori (1988) therefore hypothesised that social support would reduce the immunosuppressive impact of stress on S-IgA, and conducted a study of the effect of examination stress on S-IgA levels in students high and low in perceived social support. They found that students who reported more adequate social support at the pre-examination period had consistently higher S-IgA than did their peers reporting less adequate social support.

Since cognition mediates the perception of an event as stressful, it is reasonable to predict that cognitive coping strategies could moderate the effects of stress on suppression of S-IgA. In support of this proposition, Martin and Dobbin (1988) found that sense of humour had a significant moderating effect on the immunosuppressive action of daily hassles.

In some studies a relationship between stress and S-IgA has been found only under extreme conditions. For instance, Mouton *et al.* (1989) found S-IgA concentration to be only a weak stress marker, in a comparison of two stressful and two non-stressful occasions. Only in the most polarized contrast - final exam versus end of summer vacation - was a significant difference found. Similarly, one study suggests that S-IgA is a less effective marker of stress than other indicators of immune suppression. Thus, Kiecolt-Glaser *et al.* (1984) found that examination stress did not significantly affect S-IgA even though total plasma IgA, and natural killer cell activity were affected.

If activity of physiological stress systems reduces S-IgA levels, factors which moderate activity in these systems might also decrease the effect of stress on S-IgA. To test such an hypothesis, studies of several relaxation methods (Green and Green, 1987) and progressive muscle relaxation (Lammers *et al.*, 1987) were made in relation to S-IgA. Green and Green (1987) collected saliva immediately before and after a twenty minute relaxation exercise. S-IgA levels were significantly and substantially increased by relaxation - a more than threefold increase in only 20 minutes - despite the fact that relaxation is likely to lead to an increased flow-rate of saliva, due to reduced sympathetic nervous system activity (Carlson, 1986).

Many of the studies by McClelland, Jemmott and co-workers discussed above have involved use of rather sustained situational stressors, such as stress of examinations, or imprisonment, as independent variables. The studies of response to motivation arousal through films (McClelland & Kirshnit, 1988), daily hassles (Martin and Dobbin, 1988), and relaxation (Green and Green, 1987) however show that changes in S-IgA in response to psychological states can be rapid. S-IgA changes may therefore be sensitive to the normal fluctuations in psychological state, such as mood, which occur often as a result of individual shifts in cognitive focus and exposure to environmental challenge, even when superimposed upon a sustained situation which on average is perceived as stressful. As subjects perceive situations idiosyncratically, it is perhaps not surprising that the mean values for S-IgA concentration in saliva have very high standard deviations due to between subject variation.

### C. Mood and Salivary IgA

A more direct access to relatively transient psychological states which may be capable of influencing S-IgA, might be obtained through the use of mood, since mood is a state of awareness which is immediately responsive to cognitive and environmental stimuli. In support of this contention it is noteworthy that when psychological state was relatively controlled, and S-IgA was related to that state the variation was much lower than in studies relating S-IgA to ongoing situations such as examination stress. Thus, Green and Green (1987) in their study of relaxation effects obtained a mean (average mean for all relaxation groups) S-IgA concentration of 13.00 mg/dl before relaxation with a comparatively low SD of 3.21, increasing to a mean of 32.83 mg/dl with a SD of 4.65 after relaxation 20 minutes later. In contrast, in their study of examination stress and S-IgA, Jemmott and Magliori (1988) presented the following data on S-IgA concentrations: exam period (mean =33.82mg/dl, SD =17.39), pre-exam (mean =48.52mg/dl, SD =26.92), post exam (mean =54.51mg/dl, SD =30.78). Similarly, Mouton et al. (1988) give the following figures: midterm break (mean =11.50mg/100ml,SD =6.52), final examination (mean =11.46mg/100ml, SD =5.01), end of summer vacation (mean =15.0mg/100ml, SD =9.32), mid term exams (mean =14.61mg/100ml, SD =7.16).

As yet, there has been only one published study of the relationship between mood and S-IgA levels, which is outlined here but will be discussed in detail on page 17. Stone *et al.* (1987) compared days classified by predominant mood (e.g. high negative vs low negative) as measured on a mood adjective checklist, and found that S-IgA response to a specific orally administered antigen (rabbit albumen) was lower on days with a high negative mood relative to days with lower negative mood, and conversely higher on days with high positive mood relative to days of low positive mood. This research seems to establish that daily mood and S-IgA levels are related. It did not, however, use a fine grained analysis of mood which would be necessary to measure short-term, within-day fluctuations which might affect S-IgA levels at the time of saliva collection.

It is to be expected that mood would be related to S-IgA levels, since mood is a correlate of other factors - situational and psychological (e.g. depression) - which have been shown to influence immune function (O'Leary, 1990). The directness of mood as an index of psychological state also suggests that it would be worth building on the beginnings of research on S-IgA and mood made by Stone *et al.* (1987) to evaluate mood as a potentially useful independent variable in psychoimmunological research. And because this specific approach to psychoimmunology is in its infancy, it is important that it is developed on a firm foundation. That is, the approach should be integrated - make biological sense - and be developed with physiological mechanisms in mind, since it is these which provide the link between psychology and changes in immune function.

An integrated theory of the relationship between mood and S-IgA levels would benefit from psychological concepts based on biological principles, since it would then sit comfortably within a theoretical framework based on the operation of physiological systems and anchored in the concrete observation of biology. Within such a framework research involving mood will be able to add to an established body of knowledge in a systematic way, and by doing so will meet an important criterion of usefulness in scientific research. On this cautionary note, an integrated theoretical approach to the study of daily mood in relation to S-IgA will now be developed.

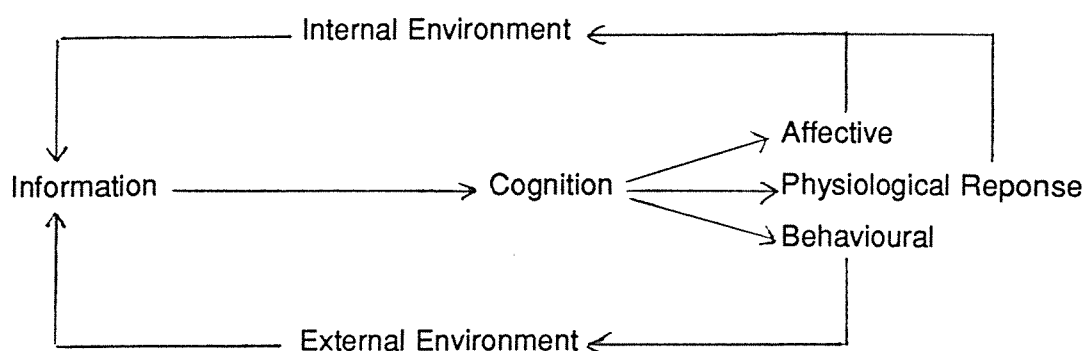
### A Psychobiological Theory of Mood

To be consistent with the great unifying principle of biology - evolution - emotions, like physiological and anatomical systems, must be regarded as having evolved, so they will be related to functions associated with survival (Plutchik,1980), and to discrete neurobiological systems which subserve these functions. Detailed consideration of the question of the function of mood is beyond the scope of this thesis; it is sufficient to say that since affect (as opposed to affective behaviour) is private and subjective, it probably has the function of providing the individual with feedback on his/her own response tendencies (their nature and intensity) at any time, and thus allows anticipation, control, and verbal communication of likely responses. It is perhaps for this reason that the frontal lobes, as the most recently elaborated brain region, are intimately involved in both affect and response inhibition in humans, in whom social complexity requires a high degree of self awareness and self-control over affective behaviour. Such control is indicative of the "Postponement (P) factor" of Stenhouse (1974) in the evolution of control over the instinctive emotional response in order to allow the more recent instinctive processes of thought (planning, reflection), and consciousness to emerge.

To be adaptive mood must be closely and immediately coupled to activity in the physiological systems which support behaviour and/or prepare the body for its consequences. Extension of this principle to the intimacy of contact between higher mental activity and immune function is justified by studies showing that the cerebral neocortex can modulate activity in the immune system in a site-specific manner. For instance, lesions of the left frontoparietal cortex depress T lymphocyte-linked immune functions including antibody production, whereas right frontoparietal lesions augment them (Neveu,1988). In general then, mood may be useful as a rapid guide to the general type of physiological response of a person to stimuli within the context of psychoimmunology.



The links between mood, physiological response and behavioural response can be summarized as follows:



It is now clear that a degree of specificity exists in the relationship between mood, brain systems, and physiological state. Negative moods, as in syndromes such as depression and anxiety, involving disengagement and withdrawal, are associated with increased activity in the pituitary adrenal cortical system (PACS), leading to elevated glucocorticoid levels. The neuroanatomical substrate appears to include the septohippocampal system, its monoaminergic afferents from the brain stem, and its neocortical projection to the frontal lobe - the so-called "Behaviour Inhibition System" (BIS) (Gray, 1982) - which connects with the endocrine system via the anterior hypothalamus. Moods associated with activity involving reward acquisition (including elimination of threat through attack, or escape) on the other hand appear to be accompanied by increased activity in the sympathetic adrenal medullary system (SAMS) and raised plasma norepinephrine. Gray (1982) had suggested that reward-directed behaviour was subserved by a brain system distinct from that involved in behaviour inhibition, but undefined at the time. The neuroanatomical substrate is now thought to involve the nucleus accumbens and dopaminergic projections from the ventral tegmental area - the "Behaviour Facilitation System" (BFS) (Depue and Iacono, 1989) - which activates the sympathetic nervous system via the posterior hypothalamus.

Anxiety involving attempts to escape is associated with a mixture of SAMS and PACS action, but as possibilities for a coping response diminish the neuroendocrine profile shifts to a predominance of PACS activity, typically associated with immunosuppressive elevations in glucocorticoids and opiates (Morley *et al.*, 1987), and the onset of a more depressive anxiety (Calabrese *et al.*, 1987). This represents a shift from a balance of BFS/BIS activities to a state in which activity in the BFS has declined and that in the BIS remains high. In practice a high degree of specificity in neuroendocrine response to stimuli is probably not often encountered. Most stimuli cause some activation of both the PACS and SAMS. However, the high degree of activity in the PACS with elevated plasma cortisol is characteristic of acute situations involving perceived loss of control, and superimposed on this there is an increase in sympathetic adrenal medullary activity where the response involves activity, or effort (Lundberg and Frankenhaeuser, 1980).

There is some evidence that activity in the SAMS may be modulated by the PACS, in that cortisol may stimulate conversion of norepinephrine to epinephrine in the adrenal medulla (Axelrod and Reisine, 1984). Thus situations involving activity and distress (loss of control), involving flight, may be associated with higher epinephrine / norepinephrine ratios than those involving other activities.

As a general rule the following is the pattern: sympathetic nervous activity, as indicated by plasma or urinary noradrenalin, is related to familiar attention-demanding activity, especially that involving muscular exertion, and regardless of the amount of distress; PACS and SAMS activity is elevated when conditions involve anxiety, limited coping options and unpredictability; PACS and parasympathetic activity predominates when distress continues while attempts to cope have ceased (Goldstein, 1989).

In the context of developing an integrative framework in which to link mood, physiology and immunocompetence, it is encouraging to note that recent factor analytic research on the structure of mood has isolated two orthogonal mood dimensions, which seem to correspond to positive and negative affect respectively (Watson and Tellegen, 1985). Gray (1981) had similarly earlier identified two dimensions of personality, also as a result of factor analysis, which were consistent with the operation of two brain systems, one the defined BIS and the other a then undefined reward acquisition

system. The latter would appear to be the BFS - since discussed by Depue and Iacono (1989).

Our understanding of how/if the activities in these systems could differentially modulate immune response as a function of mood is at this stage too limited to allow any prediction to be made regarding the relationship of mood to immunity. The point is, however, that any such links are unlikely to emerge from research unless it is designed to be congruent with known physiological systems. Details of any immunomodulatory differentiation between the two systems must await further research.

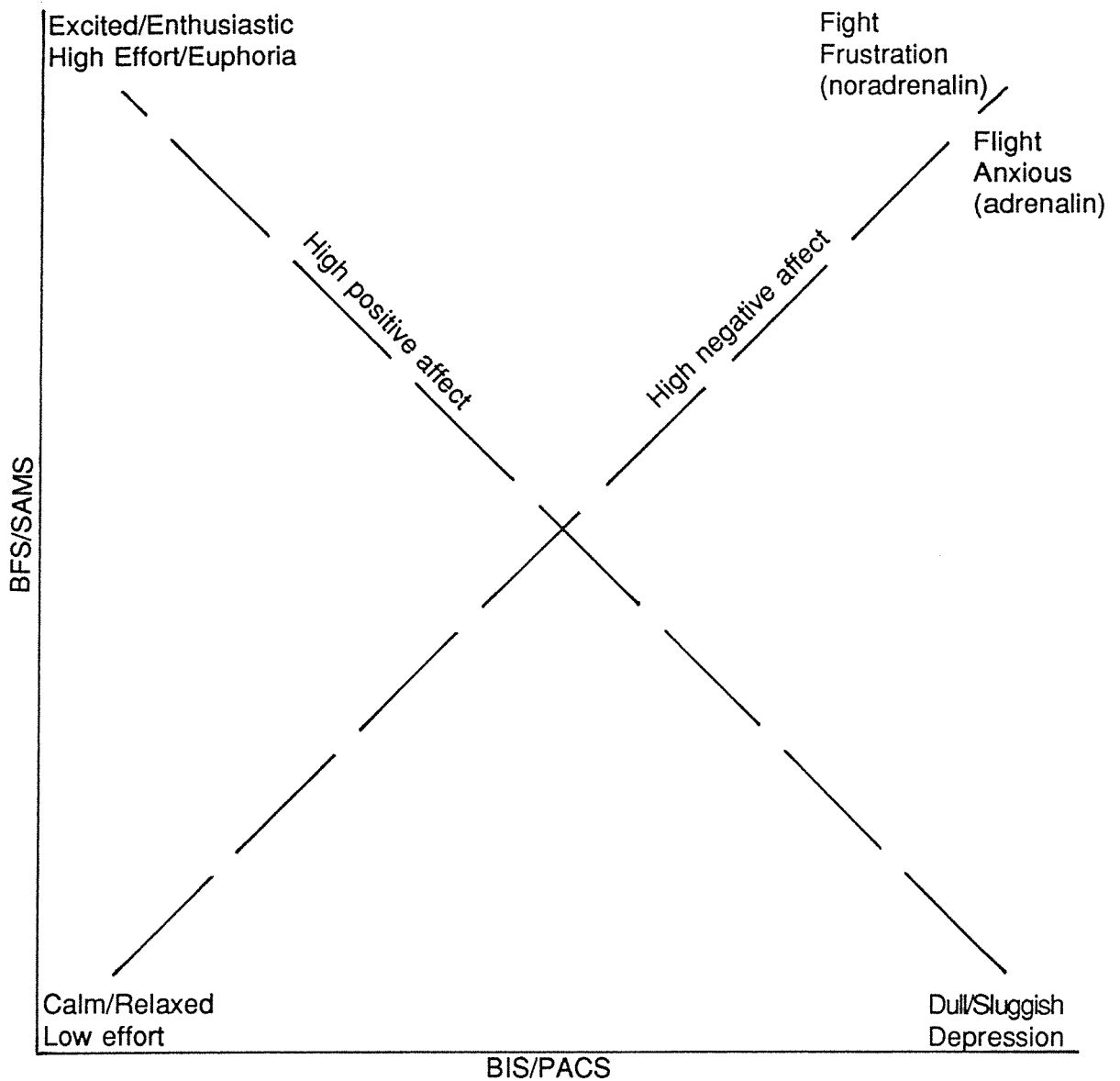
The linkage between Tellegen and Watson's findings on the structure of affect, and the more neurobiological studies of brain systems and personality is close, given that particular response tendencies defining personality are associated with the tendency to experience the particular classes of mood associated with the specific response types. For example, the negative affect scales are significantly correlated with trait measures of anxiety and neuroticism but not with extraversion, whereas the positive affect scales correlate strongly with extraversion but not with neuroticism (Watson, 1988). Neuroticism and extraversion can be successfully explained as trait manifestations of activities in the BIS and BFS (Eysenck, 1981; Gray 1981). Taken together, these studies suggest that a set of mood descriptors representing substantive domains may be related in theory to the activity of brain systems mentioned above, and hence to the way in which a change in the balance of activities within these systems is in turn reflected in the neuroendocrine responses involving the PACS and SAMS. To reiterate, both of these systems have been shown to be immunomodulatory (Dantzer and Kelly, 1989).

A connection between activation of specific physiological systems, emotions, and stress-related diseases (hypertension and cardiovascular) has already been suggested by Henry and Meehan (1981) in an extensive review. As part of this they proposed relationships between activities in the SAMS and PACS respectively and particular classes of affective state, which in some respects correspond well with those of Watson and Tellegen (1985), at least in the groupings of affect which they proposed. The similarity between the adjective clusters which Watson and Tellegen relate to the poles of their affect dimensions, and those with which Henry and Meehan describe the

emotional states associated with activity in the SAMS and PACS axes respectively, can be seen as follows:

Tellegen & Watson		Henry & Meehan	
Affect dimension pole	Mood	Physiological axis pole	Emotion
High negative	frustrated/anxious	High SAMS	Effort flight/fight
Low negative	relaxed/calm	Low SAMS	relaxation
High positive	enthusiastic/excited	Low PACS	euphoria security/control
Low positive	dull/sluggish	High PACS	depression helplessness

It appears reasonable at first sight to superimpose the factor analytically derived affect dimensions of Tellegen and Watson directly on to Henry and Meehan's schematic axes. Henry and Meehan's scheme could however be made to better accommodate the recent work on dimensionality of mood, as it does not include orthogonal (or perpendicular) axes associated with positive and negative mood respectively, but, rather, a single mood type axis running from EUPHORIA to DISTRESS, and another axis to do with activity, running from RELAXATION to EFFORT. Given the dynamic nature of fight/flight responses it would be more intuitively rational to confer both positive and negative affect dimensions with an activity component. The most reasonable positioning of affect dimensions in relation to the SAMS and PACS axes of Henry and Meehan therefore would seem to be achievable through a 45 degree clockwise rotation of the affect dimensions relative to Henry and Meehan's axes. Such a rotation is consistent with Gray's (1981) modification of Eysenck's theory of personality structure, achieved with a 45 degree rotation of his ANXIETY and IMPULSIVITY axes relative to Eysenck's NEUROTICISM and INTROVERSION-EXTROVERSION axes respectively. Given that Gray's Anxiety and Impulsivity axes reflect activity in the BIS and BFS respectively, and that Watson and Tellegen's affect axes relate to Neuroticism and Extroversion respectively, it is wholly consistent that the positive and negative affect dimensions should be between the BIS and BFS axes, and reflect the balance of activity in the two. This configuration has the advantage that the four major affect poles of Tellegen and Watson, with their associated mood descriptors, now coincide with the four combinations of extreme activity in the PACS and SAMS (high PACS/low SAMS =low positive; low PACS/low SAMS =low negative; high PACS/high SAMS =high negative; low PACS / high SAMS =high positive). In view of the evidence that fight-flight distress activates both the PACS and SAMS, and that distress without coping attempts, activates the PACS only (Goldstein, 1987), the relationship of the dimensions of Tellegen and Watson to those of Henry and Meehan, achieved with a 45 degree rotation of the former relative to the latter seems reasonable. The proposed relationships between the affect dimensions of Tellegen and Watson and activities in the SAMS and PACS with this arrangement are shown in Figure 1.



BIS = Behaviour Inhibition system  
 BFS = Behaviour Facilitation system  
 PACS = Pituitary Adrenal Cortical system  
 SAMS = Sympathetic Adrenal Medullary system

**FIGURE 1.** Proposed relationship between affect dimensions and activities in neuroendocrine and brain systems.

The proposed links discussed above have considerable support from the clinical literature which has established that elevated corticosteroids are typical of depressive disorders. Elevated sympathetic activity has on the other hand been associated with hyperactive states such as florid schizophrenia (van Kammen and Antelman, 1984) and mania (Lipinski *et al.*, 1987) which reflect hyperactivity in the BFS (Depue and Iacono, 1989). The latter is characterised by a high level of reward acquisition, and positive mood, but an extremely low capacity for behaviour inhibition if goal-directed activity is blocked. At the other extreme, a high tendency for reward acquisition coupled with a high degree of behaviour inhibition would be typified by the inhibited power motivation studied by McClelland, and discussed earlier. Subjects with inhibited power motivation show reduced S-IgA levels, increased sympathetic activation (McClelland *et al.*, 1980), increased salivary norepinephrine (McClelland *et al.*, 1985) and an increase in physical illness (McClelland & Jemmott, 1980).

On the basis of the above discussion it seems reasonable to accept as a working assumption that the mood dimensions of Tellegen and Watson (1985) may indeed relate to the operation of real, somewhat discrete neurological-physiological systems, and are therefore most appropriate to use in studying an association between mood and the physiological S-IgA response. The author appreciates that this particular approach to mood structure is only one of many. For example Diener *et al.* (1985) use different factor structures and Harre's (1983) social constructivist theory is based on radically different concepts, but what is attractive about Watson and Tellegens' theory is how it articulates so well with biology.

Apart from the close coupling of mood and physiology there are a number of other aspects of mood which make it a useful independent variable in the study of psychological affects on S-IgA. The dynamic responsiveness of mood to changing circumstances combined with its close coupling to physiology, means that mood provides access to causes of change in S-IgA. It is a rapid, immediate index of response tendency, free of the cognitive confusion of factors acting concurrently to both drive and inhibit reaction : it is an output from the information processing system. In other words, it is a proximal correlate of physiological response.

#### D. Studies Relating Mood to Salivary IgA

To the author's knowledge the relationship between mood and S-IgA has been the specific subject of only two studies to date; both similar longitudinal approaches looking for an association between daily mood and S-IgA levels. As noted earlier Stone et al. (1987) examined antigen-specific S-IgA response to ingested antigen (rabbit albumen) on days classed as high versus low positive, and high versus low negative mood respectively. Chamberlain and Spicer (1991) measured total S-IgA in relation to negative moods and a number of control variables; day of study, day of week, age, tobacco and alcohol consumption, exercise, medication and menstruation.

In the Stone et al. study saliva samples were collected thrice weekly (Monday, Wednesday, Friday) for eight weeks, and at the same time the subjects (30 dental students) were asked to rate the day's moods on a three point scale - the three response options were: definitely applied to today's mood, slightly applied, or did not apply - for each of twelve mood adjectives, six positive and six negative from the Nowlis checklist. Collection times were not given, but as the collections were made during scheduled classes they were probably made during the day, so that assessment of daily moods referred to only a portion of the day. Stone et al. collected daily mood checklists at the time of saliva sampling, so that explicit reference to scores of previous days was not encouraged. Total S-IgA was measured using an RID (radial immunodiffusion) assay, and antigen-specific S-IgA with an ELISA (enzyme-linked immunosorbent assay).

In contrast to Chamberlain and Spicer, there were no control variables measured or included in the analysis so that the level of uncertainty is high regarding causation of the significant but weak associations between S-IgA and mood recorded by Stone et al. Responses on the mood checklist of Stone et al. were used to calculate a positive and a negative mood score for each day, and after conversion of all variables to Z scores, to then classify days as relatively high or relatively low in both positive and negative mood, about a zero cutpoint. A oneway analysis of variance was then used to test for a relationship between mood and S-IgA. The primary hypothesis, that negative affective states should be inversely related to antigen-specific S-IgA was supported. Conversely antigen-specific S-IgA was higher on days with high positive



mood relative to days with lower positive mood. Total S-IgA antibody levels in whole saliva were not significantly related to mood.

Chamberlain and Spicer, (1991) measured total S-IgA concentration rather than antigen-specific (using a turbidimetric assay) in relation to daily mood, and in this respect used a similar approach to most of the studies discussed above in section B. They collected saliva thrice weekly from 27 subjects, including both male and female, for eight weeks. Five adjectives taken from Diener *et al.*'s (1985) factor analytic work were used to tap negative affect, namely worried, depressed, frustrated, angry and hostile. For each adjective subjects were asked to rate peak and average intensity, and duration of negative affect, using visual analogue scales. Subjects were also asked to record alcohol and tobacco consumption, medication, menstruation and exercise, which were used as control variables (coded 0=zero, 1=yes). The research was designed for a within-subjects multivariate analysis to be used, following Bolger *et al.*, (1989). Scores were converted to deviation scores for analysis by subtracting each individual's mean from each of their scores. Multiple regressions did not reveal a relationship between mood and S-IgA levels.

In their replication of the Stone *et al.* studies, Chamberlain and Spicer similarly sampled saliva on non consecutive days. However, they used an evening saliva collection (between 9 and 11pm) and asked subjects to score both peak and average intensities, and duration of daily mood, on visual analogue scales. By doing so they extended the work of Stone *et al.* in which mood intensity and duration were potentially confounded. In further contrast to Stone *et al.*, Chamberlain and Spicer explicitly requested that previous scores should be taken into account by referring to previous days ratings when assessing mood, so that where subjects felt some definite change they could consciously make their current rating accordingly. All ratings for any given mood were scored on a single page containing 24 vertical lines for visual analogue rating, one per day, so that direct referral back to the previous day, or indeed to any previous score was possible and encouraged. This provided a cumulative graph of each rating.

The studies of Stone et al., (1987), and Chamberlain and Spicer, (1991) are similar in that both use thrice weekly sampling of saliva and measurement of total S-IgA, although their total S-IgA assays differed. Stone et al also measured antigen-specific S-IgA. Both studies used a within subjects design but Stone et al used dichotomized standardized scores for ANOVA whereas Chamberlain and Spicer used deviation scores (Bolger, et al., 1989) for linear multiple regression analysis.

Stone et al. measured positive and negative moods, whereas Chamberlain and Spicer measured only negative mood on the basis of Diener et al.'s (1985) theory. In the former study only mood presence (definitely applied and slightly applied) versus absence (did not apply) was measured. However, Chamberlain and Spicer considerably developed the measurement of mood in relation to S-IgA by separate measurement of duration and intensity, and a further separation of intensity into peak and average. Thus duration, peak intensity and average intensity were measured on three separate VAS questionnaires. They therefore made possible much more detailed analysis in which the separate influence of several mood variables on S-IgA could be determined, in contrast to the study of Stone et al. in which they were confounded in a single measure.

Chamberlain and Spicer included a range of control variables whereas Stone et al. did not use control variables, although effects of menstruation were eliminated (immunological changes associated with women's menstrual cycles), by using only male subjects. Chamberlain and Spicer used both male and female subjects, but found no distinction even though menstruation was one of their control variables. Both studies were unable to detect an association between total S-IgA concentration and mood, although Stone et al did find a significant inverse relationship between negative mood and antigen-specific S-IgA.

In light of research on moods and S-IgA discussed above, the study of S-IgA in relation to mood could be extended in several respects.

(1) By daily sampling of mood and of S-IgA:

In the present study mood and S-IgA sampling will be made every day for 28 consecutive days. Stone *et al.* did not collect data on consecutive days, so that there is no possibility of controlling for the effect on S-IgA of moods occurring on these days. Given that the immune response to stress may differ according to the proximity of the stress to measurement of immune function (Dantzer and Kelley, 1989), a record of mood over the whole of the testing period would be advisable, and measurements on all days of the study should be taken.

(2) By detailed recording of mood duration and time within each day:

In contrast to the above studies (Stone *et al.*, 1987; Chamberlain and Spicer, 1991) the present research obtained a record of moods within each day on which S-IgA was measured, so the potential to conduct a fine grained analysis of the relationship between affect and S-IgA is possible. In view of the rapid response of S-IgA to psychological mood (Green and Green, 1987) a detailed time analysis is required to establish whether the influence of mood on S-IgA relates to its duration and/or proximity to S-IgA sampling, given the fluctuations to which mood is subject. However, it is recognised that there may also be a cumulative or averaged relationship between mood and S-IgA, as Stone *et al.* found. Recording of mood to the nearest hour may allow more accurate measurement of the duration of moods, as well as their relationship to S-IgA sampling time. Hedges, Jandorf and Stone, (1985) have partly justified their use of daily assessment of mood on the basis that this appeared to give a better approximation of daily mood reports than mood taken at four times throughout the day, although there was no indication of how mood at the time of filling out the daily mood assessment would affect daily mood score. It seems reasonable to suppose that assessment of daily mood might correlate with actual daily mood partly because actual mood at time of filling in the questionnaire was correlated with mood experienced earlier in the day, or because remembrance of mood is influenced by mood at the time of scoring. Although daily mood reports based on "momentary" mood reports taken at several times throughout the day were poor predictors of reported daily mood, there was no evidence collected as to which was the best index of actual mood. But given that moods immediately prior to S-IgA sampling may be able to have a profound effect on S-IgA, some way of measuring mood up to the time of saliva collection should be available, as well as daily mood score. A record of time

of moods throughout the day was therefore sought, although it is realised that the correspondence between remembered and clocktime of moods is a controversial issue requiring separate research. It may be argued that in order to estimate duration of mood in relation to previous days, a mental analysis of clocktime may be performed implicitly. Therefore it was considered appropriate to make this process explicit.

(3) By sampling of moods from both poles of major affect dimensions:

Mood descriptors should be chosen which adequately sample affect. Use of mood descriptors derived from recent research on the dimensionality of affect (Watson and Tellegen, 1985) and chosen to represent both poles of the negative and positive dimensions of affect, would probably sample the domain of affect more systematically than descriptors used by Stone et al. Stone et al. used six positive (playful, elated, energetic, kindly, self-centred and leisurely) and six negative (sad, angry, sceptical, clutched-up and concentrating) moods based on the mood adjective checklist of Nowlis (1965). The use of these descriptors by Stone et al. is not consistent with the more recent analyses of Watson and Tellegen, who found concentrating to be high positive, (rather than negative), and leisurely to be low negative (rather than positive). Given that miscategorization of a single mood will affect both positive and negative ratings, reducing its validity as a measure of actual affect, this may have influenced Stones results. Furthermore, the choice of adjectives does not appear to sample moods at both poles of both mood factors, i.e. high and low for both positive and negative mood. The sampling was therefore unbalanced. A better approach would be to use the same number of adjectives for each pole of each mood factor.

The adjectives used by Stone et al. do not all appear to be suitable for analysis of common moods in the present study population, as some (e.g. clutched up) are not part of local vernacular, and would best be substituted by others from the lists of Tellegen and Watson.

In this study choice of adjectives to represent both poles (high and low) of both dimensions of affect (positive and negative), in an attempt to achieve a balanced sampling of the mood domain, is consistent with the view that there is some specificity in the association of mood and physiological response (Henry and Meehan, 1981) as discussed above. It seems then that data on the relationship of moods on specific

affect dimensions to S-IgA may be of interest, especially in view of the influences of personality (usual ways of behaving) on S-IgA response to stress.

(4) By a more appropriate statistical analysis:

All three studies focus on within-person variation, and therefore use within-person deviation scores obtained by subtracting the within-person mean from the raw mood scores. Stone et al. then derived Z scores for analysis, and created dichotomized groupings of subjects, who differed in the ranges of their levels of moods. But by using Z scores all subjects were assumed for the sake of comparison, to have experienced approximately 50% medium to high, and 50% relatively medium to low days within similar ranges, for both positive and negative mood. A problem with this is that dichotomising subjects' standard scores distorts the element of biological variation, which may be an important source of real difference between subjects. Even if a person had experienced a very low and/or uniform level of negative mood on all days of the study, they were for analytical purposes grouped as having had the same number of relatively negative days, experienced with the same range of intensity as someone who had experienced many very negative days. Dichotomisation would be most applicable if the progression from very low to very high mood was associated with linear increase in S-IgA, but this cannot be assumed. In fact, given the feedback mechanisms which operate in biological systems the relationship is quite likely to be non-linear. Also, as evidence discussed above suggests that relatively stable "personality" factors (e.g. the Need for Power of McClelland and Jemmott, 1980) can influence response to events, which are related to daily mood (Stone, 1981; Stone and Neale, 1984), the strategy of "normalizing" all subjects through the use of Z scores may not therefore be the best research strategy to use, as it seems to ignore the possibility of real effects linked to individual difference. Use of pooled within-person variation (Bolger et al. (1989), as used by Chamberlain and Spicer (1991), is a better approach.

(5) By using control variables:

Inclusion of appropriate control variables in multivariate analyses will allow some confounding (non-mood) influences to be accounted for in the data analysis. The relevance of some of those used in the present study has been discussed by Kiecolt-Glaser and Glaser (1988), namely alcohol and tobacco consumption, medication,

exercise, menstruation. All of these were discussed as confounding factors because of their numerous affects not only on physiological systems (and therefore on immune function), but also because they often show a reciprocal interaction with mood. Menstruation is an important variable to be included because of the marked endocrinological fluctuations accompanying different phases of the menstrual cycle which could have immunological correlates. In addition, age, day of study and day of the week were recorded. Immune function is known to change with age. Inclusion of day of study would allow for practice effects on saliva production which might increase the flow rate, thereby reducing S-IgA concentration. Stone *et al.* controlled for practice effects by not using the first eight saliva sampling collected. Day of the week also seems worth recording because of the possible effects of systematic changes in stress levels, such as might be related to the structure of the week, imposed by employment. It has been argued that since mood might influence salivary flow rate through autonomic nervous activity, studies of mood in relation to S-IgA should include flow rate as a control variable (Stone *et al.*, 1987). Mouton *et al.* (1989) did not however find any association between salivary flow rate and S-IgA concentration in their study of the effects of examination stress on S-IgA levels. Apart from this, if one is interested in studying the association between mood and S-IgA concentrations, in the context of health, flow rate may simply be considered as one of the many immeasurable factors which mediate between mood and S-IgA concentration, so that inclusion of flow rate as a control variable is not essential. Measurement of flow rate raises practical difficulties and questions as to the reliability of flow-rate data when measured under non experimental conditions.

Stone *et al.* (1987) proposed that antigen-specific S-IgA should be measured in an attempt to control for the high background of total S-IgA, which they considered to be unrelated to disease incidence. This argument has been rebutted (Jemmott and McClelland, 1988) on the grounds that an association between S-IgA concentrations and disease incidence is demonstrable, and because total S-IgA provides a multiple defence against ongoing exposure to a host of potentially harmful agents, and is therefore a highly appropriate variable to measure.

### E. Hypotheses

Since Stone et al. (1987) found a significant relationship between negative mood and S-IgA with antigen-specific rabbit albumin and using an ELISA assay, and Chamberlain and Spicer (1991) did not find a significant correlation between negative mood and S-IgA using whole saliva and a turbidimetric assay, a study partially replicating the above seemed appropriate. The following hypotheses will be tested in the present study.

(1) That there is a positive relationship between the average intensity, peak intensity and duration of positive mood and S-IgA.

(2) That there is a negative relationship between the average intensity, peak intensity and duration of negative mood and S-IgA.

The possibility of both linear and non-linear relationships will be investigated because feedback loops often result in non-linear responses in living systems. It was originally intended that more detailed analyses be carried out, but because of problems encountered in mood measurement it was feasible but not advisable to do this. Such questions as the effect of mood on S-IgA levels as a function of the proximity of the mood to time of S-IgA sampling within the day; the sequencing of mood as a possible moderator of S-IgA levels; the effects that previous days moods may have on S-IgA levels, and the influence of sleep patterns and disturbances on S-IgA levels could have been addressed. The extension of mood research by the present study is thus more a potential methodological one, based on more detailed information and analysis of within day dynamics in relation to S-IgA, than an extension through new hypotheses and the testing of them.

## METHODS

### A. Procedure

Subjects were provided with a written explanation of what the study involved (Appendix B). Those agreeing to participate completed a health history questionnaire form to briefly provide their life-long medical history, including major illnesses and medical conditions which might influence antibody and/or hormone levels. Subjects were instructed in filling out daily questionnaires for mood and control variable measurement, and in the procedure for collecting saliva samples (Appendix C). They were provided with a written set of instructions and enough questionnaires for recording daily mood and control variables for 28 days; 28 plastic pipettes and labelled 1 ml capped centrifuge tubes for daily saliva collection, packaged in weekly lots. The subjects were instructed to take a saliva sample and fill in their mood forms between 9 and 11pm at night. Prior to the main study three subjects were recruited to test all of the proposed procedures over a two day duration. The result of the trial indicated that no difficulties were encountered.

### B. Subjects

Twenty adult females between 20-60 years of age were recruited. Females were chosen because they may experience greater fluctuations in mood than men in association with the menstrual cycle (Casey & Dwyer, 1987). Inclusion of menstruation as a variable in multivariate analyses could appropriately control for such changes. It was also felt that they may be more honest than males in reporting moods. The study was restricted to subjects in the 20-60 year old group because it was expected that this group would be more hormonally stable.



### C. Measurements

To test the hypotheses put forward in the introduction it was necessary to develop methods capable of measuring properties of daily moods, levels of S-IgA in the saliva, and control variables.

#### (a) Measurement of Mood

##### Construction of Time-mood log

Mood was measured on a Time-Mood Log (TML) which was subject to several pilot studies resulting in some modifications. Subjects reported that there were no problems in filling it out. This questionnaire, developed for measurement of daily mood, was required to provide data allowing quantification of intensity of mood (average and peak), actual duration of mood (permitting sequence/proximity), and change in mood intensity and duration between days.

It was decided that the questionnaire format should allow all of each days mood data to be recorded on a single sheet, with each daily sheet treated independently (the issue of whether or not to refer back to previous days is discussed below). This format was adopted partly because pilot studies had shown that subjects preferred to be able to record all of the data on a single sheet (one per day) rather than using three per mood, for peak intensity, average intensity, and duration (i.e. 8 moods x 3 = 24 sheets per day). It was hoped that this would encourage compliance and conscientiousness in the subjects. For the sake of clarity, and to overcome the space constraints of this arrangement, scoring of mood peak and average intensity respectively were carried out with reference to a ten point numerical scale with appropriate score entered in a box below the column used to record mood duration (see instructions and mood record form, Appendix A).

##### Choice of adjectives

Before measuring mood, peak and average intensity, and duration, it was necessary to choose appropriate mood adjectives to identify mood states which might be systematically related to the response of the immune system, as represented by S-IgA. As discussed in the Introduction, Tellegen & Watson have identified two main orthogonal mood factors ("positive" and "negative") which can be related to the

operation of physiological systems. These represent mood domains, rather than specific mood words, findings which have been corroborated by other recent research (Matthews, Jones & Chamberlain, 1990).

A number of adjectives were therefore used to access an area (or dimension) of feeling (a mood cluster), rather than single words, which may correspond to only a point within a domain. Use of several adjectives corresponding to both poles (high and low) of each mood dimension, overcomes the limitation of the specificity of a single word, which may be determined by the context of the mood and its intensity (i.e. different mood descriptors may correspond to different intensities or contexts of the same mood), rather than by any fundamental property of the mood itself. The problem of balancing adequate coverage of the mood dimensions with the practical need to limit the number of mood descriptors required a compromise. In the present study, the numbers of positive and negative mood descriptors, high and low in intensity, was kept balanced, so that responses in the different mood classes were not a function of the options for scoring them, as determined by the questionnaire design.

#### Measurement of Intensity of mood

Stone *et al.* (1987) measured mood "intensity" with a three point scale attached to mood adjectives within a checklist but in doing so confounded mood intensity and duration. Chamberlain and Spicer, (1991) attempted to overcome this problem by specifying mood duration and intensity separately, and furthermore elicited scores for both average and peak intensity. They used a visual analogue scale for each mood of interest. In the present study a numerical scale (0-10) was used. Such scales have often been used in measurement of affect, and there seemed to be no convincing arguments either against use of the numerical scale, or for use of a preferable scoring system.

A criticism of the numerical scale has been that subjects tend to use favourite numbers. But subjects may also favour a particular position on a visual analogue scale. Divergence from a mean score on the numeric scale may therefore be as valid for measurement indicating change in mood as variation about a mean position on the VAS. The questions remain, do VAS and numeric scales differ in variation about a favourite point, and how do they differ in providing valid measures of difference in the

mood construct? It is possible that use of favourite numbers will reduce response to small changes in mood if a subject moves from the preferred score only when definite changes are experienced.

Scores on the VAS in measurement of anxiety and depression have been correlated with scores on a number of other tests of the affective states (McCormack et al, 1988) in validating trials. But the VAS correlations with these tests are not high, even though statistically significant. Although lack of definition of intermediate points is a distinctive property of the VAS, it does however share certain properties with the validating tests. It is therefore not possible to say that lack of definition of intermediate points per se has made any positive contribution to the validity of the test in measurement of mood, but merely that it has not had a completely adverse effect. Published validation studies are therefore of limited relevance to the question of the advantage versus disadvantage of not defining intermediate points.

#### Measurement of actual mood duration

In the present study a twenty four hour chronological time scale was used in order to obtain a record of the approximate onset and termination of moods, from which actual duration of mood could be determined. For subjects to indicate actual time of mood they must be able to refer to a record form on which they can record chronological times, rather than to a VAS (recording relative times) or to some other form of non-chronological scale. In the context of the present study real time is a particularly relevant measure, because the S-IgA response is a physiological response and as such it will be affected by real physical/physiological factors, such as time or time-dependent processes. Therefore, it is better to provide an external time scale for recording time and durations of moods rather than rely on subjective estimation of relative duration. Use of relative scales necessitates subjective assessment which is not explicitly linked to real time, and therefore cannot provide the same level of correspondence between the remembered and the actual timing of mood. Subjects in pilot studies found that moods could generally be linked to real time through memory of events and other cues known to occur at particular times, because of the way the individual structures the day.

The chronological time scale of the TML, unlike the VAS, actually provided an aid to recall by encouraging the subjects to think back to particular times within the day, often linked to particular events, and so may reduce error in judgment of mood duration.

A possible advantage of a chronological time scale marked at equal time intervals, over an unmarked scale such as the VAS is that timing and therefore duration can use the hourly markings as an anchor points (three hourly multiple fixed points) throughout the scale. The VAS on the other hand is anchored only at its extremities, so that the subjective criterion or reference point against which duration is plotted may be subject to drift over the course of a study such as the present one, which last twenty-eight days.

Carlsson (1983, cited McCormack *et al.*, 1988) has questioned the assumption that an advantage of the VAS over other scales is its ease of use, pointing out that complex mental transformations may be required to score the VAS. In the context of the present study, when subjects record remembered time against the chronological time scale they need remember only the approximate times at which moods occur before placing their mark from which duration can be calculated against the scale. However, when subjects score mood duration against the VAS they must carry out a cognitive transformation from experienced time to a visio-spatial display requiring perceptual judgement and accuracy.

#### Measurement of change in mood between days

Whereas Stone *et al* used the difference between independent mood ratings to assess change, Chamberlain and Spicer used explicit change ratings, on the assumption that subjects will probably refer back to their previous scores anyway. The construction of an instrument for measurement of change in mood between days requires an answer to the question as to whether or not there is more error involved in the experimenter obtaining the arithmetic difference between two independent daily scores, or in the subject providing a single difference score by subjectively assessing the difference between days, while referring to his/her previous responses.

Guyatt *et al.* (1985) have investigated the effect of knowledge of previous questionnaire responses on subsequent scores on the same questionnaire. Compared with results

when previous responses were not available, knowledge of prior results led to a substantial decrease in within subject variance in judgement of the states of dyspnea, fatigue and emotional function in chronic but stable cardiovascular patients. The authors therefore suggested that by letting subjects see their previous responses sample size could be decreased in clinical trials.

Although this may be true, the primary objective of research is to obtain meaningful data, and this must not be sacrificed as a statistical expedient (validity should not be sacrificed for reliability). The patients in the Guyatt et al.(1985) study were judged to be in stable condition, knew their condition to be stable, and might therefore (like the experimenters) expect and indeed want the questionnaire scores on the differing testing occasions to be similar. The low variance is therefore quite possibly simply an expectancy effect, reflecting the intrinsic human need for consistency (Mower-White, 1982). The effect of expectations on questionnaire responses involving subjective judgments has been shown to lead to serious distortions when appropriate standards of comparison are used (Shweder and D'Andrade, 1980).

Dyspnea, fatigue and emotional function measured in the study of Guyatt et al.(1985) all have a strong subjective component and the effect of reference to prior results on the validity of measures provided by the questionnaire cannot be judged without separate, objective, and concurrent measures of the states. Not even measures of the patients physical state were made at the time of questioning by Guyatt et al.(1985).

Strictly speaking, it is impossible to definitively answer the question as to whether or not explicit reference should be made to previous mood scores, because judgment of moods by questionnaire is necessarily subjective, whereas independent objective measures of mood are required to settle the question. But given that prior knowledge of questionnaire results can influence subsequent responses, as indicated by Guyatt et al., (1985) it seemed better that subjects should be instructed not to refer back. Although Guyatt et al. showed that reference back was associated with an increase in reliability, their study design did not allow one to determine whether or not this was at the expense of validity, given the human need for cognitive consistency (Mower-White, 1982).

Error associated with recall is another reason why the present study avoided reference to previous days scores, in contrast to the study of Spicer and Chamberlain (1991). When a daily score is based on a subject's judgment of the difference between two days (s)he is doing exactly what the experimenter will do in obtaining an arithmetic difference, but subjectively, and based on memory of feeling over 48 hours or more rather than the 24 hour (usually longer) period involved in assessment of independent days. Memory has been shown to be notoriously unreliable as it usually involves inaccurate reconstruction from incomplete memory, (Shweder and D'Andrade, 1980), and is known to decay as a function of time. Studies should therefore be designed with the shortest possible recall times necessary to obtain the data required to answer the research questions and even then recall of moods within a day is likely to be subject to error. Due to the time dependent decay of memory the time of occurrence of key events is likely to be remembered better if recall does not have to extend beyond the preceding 24 hours than if it is required to extend over two to three days as in the study of Chamberlain and Spicer (1991). Mood questionnaires completed with reference to previous days require memory of the subjective, so that validity may be decreased.

Reference to previous days scores may lead to contamination of daily scores. Where the subject is asked to give scores relative to previous days each daily score is adjusted relative to that of the previous day scored; and so too, the previous daily score has been influenced by the one before. Therefore, neither a relationship purely between two consecutive daily scores nor an independent daily score is ever obtained, because contamination by previous days is an inevitable consequence of the procedures adopted. In practice, some reference to previous days may be difficult to avoid, but in principle it seems that the subjects should be instructed not to look back, for the reasons discussed above.

#### Formation of affect variables

Scores for average intensity, peak intensity, and duration of daily overall negative and positive moods respectively were obtained by combining the relevant scores on the negative and positive dimensions. This was achieved by adding scores on the high end of each dimension to reversed scores on the low end, i.e. those on the low end were subtracted from zero before addition to those on the high end. Six affect

variables, each based on four moods, were therefore created, for duration, peak and average intensity on both the positive and negative affect dimensions, representing the sum of scores on each of their respective items.

All of the separate high negative, high positive, reversed low negative, and reversed low positive mood scores (i.e. all moods together) for average mood intensity were factor analysed with both oblimin and varimax rotation. This exploratory factor analysis showed that the items with the exception of calm, separated clearly into the positive and negative groupings.

#### Reliability of affect variables

Time constraints did not permit extensive pilot studies to conduct reliability analysis of mood measures so the internal consistency of the measures was only examined once all data had been collected. Reliability and validity had been assumed in view of the thorough analyses of Watson and Tellegen (1985) in their studies of affect. As noted above, scores were transformed into deviation item scores around subject's individual means following Bolger *et al.* (1989), therefore there were 532 (19x28) data points representing person-days and including missing data (23 days missed across all subjects).

The six item affect scales derived from the mood questionnaire (positive and negative dimensions of average and peak intensity, and duration respectively) were subjected to a reliability analysis. Interitem correlations within each scale were low, with mean correlations below 0.2 for all scales except duration of negative affect (mean  $r=0.26$ ), and disappointingly low alpha values ranging from .21 to .57. (Table 1)

These alpha coefficients make it clear that the groupings of items used to create the affect variables did not produce reliable measures for these subjects. The implications of this are considered in the discussion section.

TABLE 1. Inter-Item Correlations and Alpha Coefficients  
for Six Mood Variables

<u>SCALE</u>	<u>Items</u>				ALPHA
	Anxious	Frustrated	Calm		
A.Negative Affect					
(1)Average intensity	Frustrated	.20			.44
	Calm	.18	.11		
	Relaxed	.12	.19	.19	
(2)Peak intensity	Frustrated	.11			.21
	Calm	.13	.05		
	Relaxed	.08	-.02	.03	
(3)Mood Duration	Frustrated	.29			.57
	Calm	.33	.22		
	Relaxed	.29	.25	.19	
		Excited	Enthusiast	Dull	
B.Positive Affect					
(1)Average intensity	Enthusiastic	.15			.46
	Dull	.08	.13		
	Sluggish	.15	.30	.26	
(2)Peak intensity	Enthusiastic	.22			.37
	Dull	.03	.08		
	Sluggish	.10	.18	.15	
(3)Mood Duration	Enthusiastic	.17			.49
	Dull	.10	.16		
	Sluggish	.12	.24	.34	



## (b) Measurement of S-IgA

### Collection of Saliva

Each subject was instructed to collect approximately 1.5 mls of saliva from passive accumulation under the tongue, and to transfer it with a pipette to the appropriate labelled centrifuge tube. The tubes were immediately placed in a freezer until collected by the researcher. At the end of the 28 day sampling period all samples were transported in ice, without thawing, to a freezer in the laboratory where analyses were to be conducted.

It was decided to measure changes in total S-IgA protein in the saliva as a measure of immunocompetence (see introduction) following Jemmott and McClelland (1988), rather than antigen-specific anti-rabbit albumen S-IgA as Stone *et al* (1987) had done. Flow rate was not measured as it has been found to be weakly related to S-IgA concentration (Jemmott & Magliori, 1988; McClelland and Kirshnit, 1988), or unrelated (Mouton *et al.*, 1989).

### S-IgA assay procedure

S-IgA analyses were performed using an enzyme-linked immunosorbent assay (ELISA). Preliminary runs were conducted with several serially diluted saliva samples and reagents to determine the optimal concentration range for S-IgA analysis. Analyses were finally conducted on four serial 1:1 dilutions of each sample, starting with 10 microlitres of a 0.01 dilution of saliva in a total volume of 150 microlitres.

The final ELISA procedure was as follows:

Microtitre plates were coated with goat anti-human IgA antibody (Sigma) at 0.5 microlitres/ml in 0.05 M carbonate/bicarbonate buffer pH 9.6 (50 microlitres per well).

Plates were then incubated for one hour at 37 degrees C, and after incubation they were flicked out and remaining sites blocked with 0.5% defatted milkpowder (BLOTTO) in carbonate/bicarbonate buffer. Blocking incubation was for one hour at 37 degrees C followed by an overnight incubation at 4 degrees C. Plates were stored at 4 degrees prior to use.

Saliva samples which had been stored at -20 degrees C were thawed and then diluted 1:100 in phosphate buffered saline (PBS) containing 0.5% Biotin, 0.1% Tween 20 and 0.1% NaN<sub>3</sub>, as a preservative. These diluted samples were then held at 4 degrees C until used in the assay.

The assay plate was laid out as follows:-

1) 100 microlitres of a 1 microl/ml solution of Human IgA was applied to well A1, 20 microlitres of this was then transferred, with thorough mixing, through the wells B1-H1 which had previously had 50 microlitres of PBS 0.5% Biotin 0.1% Tween 20 (PBS/B/T) added to them. This procedure gave half dilutions through the series of wells.

2) 10 microlitres of each saliva sample was then added to a well that contained 90 microlitres of PBS/B/T, this well was then thoroughly mixed and 50 microlitres transferred serially to three further wells each also containing 50 microlitres of PBS/B/T. This gave dilutions of the saliva of 1/1000, 1/2000, 1/4000 and 1/8000. Approximately 19 saliva samples were on each plate.

3) The procedure in (1) was repeated but using wells A12-H12. Plates were then incubated at 37 degrees C for one hour.

The plates were washed six times with PBS containing 0.1% Tween 20 (PBST) and then a 1/1000 dilution of Peroxidase labelled anti-human IgA in PBS/B/T (50 microl/well) was added followed by an incubation at 38 degrees C for one hour. The plates were washed in PBST, and 200 microlitres of substrate added (40 mg orthophenylene diamine/100 ml. of citrate/phosphate buffer pH 6.0, containing 40 microlitres of 30% hydrogen peroxide) and then incubated for 30 minutes at room temperature. The reaction was stopped by addition of 50 microlitres 4M sulphuric acid and the plate read at 492nm on a Titertek Multiscan plate reader. Optical Density values were read into TITERSOFT software which calibrates the plate and computes values relative to the two sets of standard curves contained in the plate (wells A1-H1 & A12-H12).

### (c) Control Variables

A number of control variables which may influence S-IgA levels (Kiecolt-Glaser and Glaser, 1988) were measured using a separate daily checklist on which the subjects recorded menstruation, medication, alcohol and tobacco consumption, and exercise during the preceding 24 hours. The presence of these was indicated by marking a box labelled "some" or absence by marking a boxed labelled "none". In the case of medication, alcohol, tobacco consumption and exercise, an indication of the amount was also requested. Day of study was included because of possible practice affects, and because diary respondents in longitudinal studies tend to report fewer stressors over time (DeLongis, Folkman and Lazarus, 1988) which may be reflected in their reporting of mood. Day of week was also included because research has documented systematic day-of-the-week variation in mood (Rossi and Rossi, 1977; Stone et al., 1985).

### D. Ethical Issues

This study was conducted under the guidelines of the New Zealand Psychological Society and Massey University. Care was taken to ensure safety in saliva collections and handling. Confidentiality of information was assured, as well as an understanding by the subjects that participation was voluntary and they were able to withdraw from the experiment at any time. Approval was given by the Massey University Ethics Committee.

### E. Statistical analysis

Statistical analysis was carried out using SPSS/PC statistical programme (SPSS/PC Manual, 1985).

Individual scores for mood duration were rounded to the nearest hour, and variables created for the average and peak intensity and duration of each mood. A value of zero was assigned to each occasion on which a mood was not experienced. Deviation scores around individual means were calculated by subtraction of the 28 day mean of

each individual from their daily mood item scores for each of the above variables, and for S-IgA. This is the procedure used by Bolger et al. (1989) for analysis of within-subject variation, independent of the effects of individual difference. The analysis then becomes one of pooled within-person variation, for analysis of group data.

Preliminary descriptive statistics on raw scores for mood items and S-IgA were obtained to determine patterns of response on each. The control variables menstruation, medication, alcohol and tobacco consumption were dichotomously coded as, some=1, and none=0. A variable representing day of study from 1 (start) to 28 (finish), and six dummy variables represented day of study from Monday (1) to Saturday (6) with Sunday as the comparison, were also created.

Three multiple-regression analyses were conducted, one for average intensity, one for peak intensity, and one for duration, each containing both positive and negative affect variables, regressed against S-IgA. The control variables were also included in all of these regression analyses for reasons discussed above (previous page). The regressions test the hypotheses proposed in the introduction, viz that there is a relationship between the averaged intensity of negative and/or positive mood and S-IgA, that there is a relationship between the peak intensity of negative and/or positive mood and S-IgA, and that there is a relationship between the duration of negative and/or positive mood and S-IgA.

Regressions were also run with each affect variable squared to test for the possibility of a non-linear relationship between mood and S-IgA. Regressions were also carried out using mood variables dichotomised at the median in order to approximate analyses used by Stone et al. (1987). Residual analysis suggested that assumptions for multiple regression were adequately met.

## RESULTS

Descriptive statistics on raw item scores for mood average intensity, peak intensity and duration, as well as for S-IgA concentration, are shown in Table 2. Scores for average and peak intensity covered most of the possible range (scale length 0-10) for all moods, but in all cases a very high percentage of zero scores were recorded, indicating that the distributions of mood items were highly skewed, although the S-IgA showed a normal distribution.

The percentage of control variables scored present, for all subjects, were: medication, 19.1; alcohol intake, 32.8; tobacco consumption, 12.0; exercise, 43.4, and menstruation, 14.5.

Correlation coefficients between S-IgA and the six affect variables are shown in Table 3. They were invariably low (all  $r < 0.1$ ) and non-significant ( $p > .05$ ).

Multiple linear regression analysis of average (negative average with positive average) and peak (negative peak with positive peak) intensity and mood duration (negative duration with positive duration) variables, with control variables included, gave the results summarized in Table 4. For all three regression analyses the results were similarly significant ( $R^2 = 0.06$ ,  $p < .05$ ) but inspection of the Beta and T values for component variables showed that none of the affect variables were contributing to the significant associations with S-IgA. There was a single source of significance which was the control variable, "Day of study" (Beta =  $-0.22$ ,  $T = 4.9$ ,  $P < .0001$ ).

TABLE 2.                      Descriptive Statistics for Mood Items and S-IgA.

		MEANS	S.D.	RANGE	% SCORING 0
Frustration:	average	1.85	2.63	0-9	63.3
	peak	1.42	2.95	0-10	81.2
	duration	1.59	2.96	0-16	63.7
Anxious	average	1.80	2.50	0-9	62.0
	peak	1.13	2.63	0-9	83.5
	duration	2.38	4.24	0-20	62.6
Calm	average	2.56	2.50	0-8	44.7
	peak	0.99	2.47	0-10	85.5
	duration	5.42	5.86	0-17	43.7
Relaxation	average	2.97	2.70	0-9	40.6
	peak	1.23	2.80	0-10	83.4
	duration	4.75	5.41	0-18	40.4
Enthusiastic	average	2.67	2.68	0-9	46.0
	peak	2.16	3.31	0-9	68.9
	duration	3.73	4.55	0-17	45.5
Excited	average	1.36	2.53	0-9	75.4
	peak	1.11	2.62	0-10	84.9
	duration	1.19	2.65	0-17	75.2
Dull	average	1.13	2.16	0-8	76.7
	peak	0.44	1.70	0-10	93.2
	duration	1.51	3.52	0-22	77.2
Sluggish	average	1.59	2.38	0-9	65.9
	peak	0.63	1.98	0-10	90.2
	duration	2.13	3.93	0-17	66.2
S-IgA concentration(mg/ml)		0.11	0.06	0-38	

TABLE 3.     Simple correlations between S-IgA and six affect variables.

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Negative Affect:	Average	-.00
	Peak	.02
	Duration	-.01

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Positive Affect:	Average	.02
	Peak	.01
	Duration	.02

---

**TABLE 4.** Regression Analyses of Affect and Control Variables against S-IgA.

Variable:	Affect Variables					
	Average		Peak		Duration	
	Beta	P	Beta	P	Beta	P
Positive Affect	-0.01	0.09	0.03	0.56	0.01	0.98
Negative Affect	0.13	0.95	0.01	0.80	0.01	0.85
Control Variables:						
Age	-0.01	0.86	-0.01	0.86	-0.01	0.86
Alcohol	0.02	0.67	-0.02	0.66	-0.02	0.68
Tobacco	0.01	0.79	0.01	0.80	0.01	0.79
Exercise	-0.01	0.79	-0.01	0.75	-0.01	0.78
Medication	0.07	0.11	0.07	0.10	0.07	0.11
Menstruation	0.08	0.09	0.08	0.09	0.08	0.10
Day of study	-0.22	0.01	-0.22	0.01	-0.22	0.01
Day of week:						
1.	0.02	0.70	0.02	0.67	0.02	0.69
2.	-0.04	0.55	-0.04	0.55	-0.04	0.55
3.	-0.06	0.33	-0.06	0.33	-0.06	0.33
4.	-0.05	0.40	-0.05	0.38	-0.05	0.41
5.	-0.04	0.47	-0.04	0.49	-0.04	0.47
6.	-0.05	0.35	0.05	0.37	0.05	0.35
Adjusted R squared		0.03		0.03		0.03
F Values		2.04		2.06		2.04
P Values		0.01		0.01		0.01



## DISCUSSION

None of the hypotheses tested in the present study, namely that there are associations between salivary S-IgA concentrations and mood average intensity, peak intensity and duration, for positive and/or negative moods, were supported by the results presented above. There are a number of possible reasons for these findings, relating to both the specific procedures used and more generally to the nature of mood - salivary S-IgA research.

### Measurement of Mood

The design of the questionnaire used for measurement of mood, discussed in the methods section, was apparently straight forward, involving remembrance of the experience of eight mood words, two representing each of the four affect poles, during the preceding 24 hours. The results of factor analysis showed that in general responses were consistent with the existence of positive and negative dimensions of affective experience. But the factor loadings and reliability analyses suggested that use of two four-mood sets, each representing a single linear dimension of affect was not very successful since the intercorrelations among scale items were low.

There are several possible explanations for the low reliabilities observed, including the usual problems of recall discussed in the introduction. Amongst other possibilities are:

- (1) Because of the large number of zero scores in the mood items, they may have contained insufficient variability for reasonable correlations among them to be achieved. Even so, the distributions of positive and negative affect scores were reasonably normal.
- (2) Aggregation of some moods along the same dimension may not have been an accurate reflection of reality. For example, anxious and frustrated may not have correlated well because they are descriptors of mood which reflected response tendencies to different types of situation in the present study population, even though they were associated on the same dimension in Tellegen and Watson's studies.

It was in fact argued in the introduction that anxious and frustrated lie in different regions of the affect domain and the reliability results are consistent with this, as the two moods showed a correlation of only .20, .11 and .27 for the item analysis of average intensity, peak intensity and duration respectively on the negative affect scale. Anxiety itself is a term which may be semantically heterogeneous. As discussed in the Introduction, (page 11) active "anxiety" may be associated with coping attempts, involving BIS/PACS - BFS/SAMS activation, or when coping attempts have ceased it may be a more depressive (learned helplessness, giving up) affect in which BFS/SAMS activity has dropped, and BIS/PACS activity remains high and therefore dominates. Although depression has been associated with the low positive pole (sluggish, dull) of the mood dimensions, anxiety is classed as a high negative mood, yet it has been argued that anxiety is a core component of depression (Gray, 1987). Perhaps what is known as anxiety covers a large segment lying between the high negative and low positive poles, with fluctuation between active and passive anxiety-induced behaviour. If this is the case, inclusion of all anxiety responses on the questionnaire as items in the negative affect scales may have produced a distorted reflection of reality, and lowered any chance of detecting an association between the mood and S-IgA responses. Conversely, "depression" is itself a hazy term, perhaps reflecting its syndromic nature. Clearly, semantics is a problem with the meaning of both mood descriptors and scores in studies of this type.

However, the fact that inter item correlations overall were low, and not merely those involving "anxious" and "frustrated", suggests a more fundamental problem with the questionnaire. "Enthusiastic" and "excited" for instance showed a very low correlation (0.15) for average mood intensity.

(3) A difference between the collection of affect data on this and the Tellegen and Watson studies is that the latter used single samplings of mood, rather than using data collected over 28 consecutive days in a single analysis. Their data may not therefore have been subject to time dependent influences which might have made the structure of affect which they derived an inappropriate basis for the affect variables used in this study.

(4) Moods on the low end of dimensions (e.g. dull, sluggish, calm, relaxed) may not have been experienced with the same awareness of feeling as those on the high end, because they correspond to a low rather than a high "quantity" of affect. In other words, they may have been experienced within a lower range (even though still scored within a range of 0-10) than those (e.g. frustrated, anxious, enthusiastic, excited) at the higher end, and have therefore been more difficult to judge accurately on a scale of 0-10. A resulting increase in error may have lowered their correlation with moods at the opposite pole.

(5) It may not be valid to equate reversed low pole scores with non-reversed high pole scores. Thus a score of 3 for "calm" when reversed ( $0-3 = -3$ ) may not equate with a score of 7 on frustrated. Scores derived by subtraction of the low pole scores from 10 may have been more appropriate. Then, a low score of 3 for calm (low pole) would have been converted to a high pole score of 7 ( $10 - 3$ ), which may have been more congruent, given that scores on moods from the high and low poles respectively are likely to be inversely related. Another problem is that the quantity of affect involved in "calm" may not be the same as that involved with "frustrated" as discussed in (4) above.

(6) The mood descriptors used may not have adequately sampled the range of affect experienced by the subjects. Thus in the attempt to keep the mood questionnaire to a reasonable size its power to sample mood may have been sacrificed. Many more moods during the day were recorded as not experienced than had been expected. Perhaps if other descriptors had been made available they would have been used.

(7) The subjects may not have been sufficiently aware of their moods even when their physiology had changed enough to influence S-IgA levels, at times when their focus was on external events and tasks involving them, rather than on their feelings. (For instance, cognitive tasks such as mental arithmetic have been shown to alter hormonal states, (Williams *et al.*, 1982)). This is a basic problem with questionnaires requiring awareness and recall of experiences, and may be important in relation to S-IgA measurement if both mood and S-IgA response are different results of the same underlying process.

(8) In some cases subjects may have been confused between behaviour and affect, and this differentiation may not always have been made by them, even though it was strongly emphasised at the start of the study that the questionnaire was for measurement of moods. For instance, people who felt a high degree of frustration but consciously restrained themselves and remained behaviourally passive may have recorded a high score on the mood "calm". In other words they may have attributed calmness to themselves on the basis of observation of their own behaviour. This may explain why the mood "calm" loaded on both the positive and negative factors in the factor analysis.

#### Relationship between Affect and S-IgA

No association between affect and S-IgA levels could be detected in the present study either by correlation of individual mood scores with S-IgA, or by regression of scores from the positive and negative scales respectively, for average mood intensity, peak mood intensity, and mood duration, against S-IgA. Although the regressions were all significant, the control variable "Day of Study" proved to be the source of significance in all cases, and the negative association between this variable and S-IgA indicated that over the course of the study S-IgA levels underwent a linear decline. This phenomenon has been noted by Stone et al.(1987) and is thought by them to be an effect of practice at saliva production on S-IgA concentration, reflecting a progressively increasing salivary flow rate, and consequently decreased S-IgA concentration as the number of saliva collections increased. It should be noted that this interpretation is not supported by recent findings discussed above, which showed the relationship between S-IgA concentration and flow rate to be weak or non-existent (Jemmott & McClelland, 1988; Mouton et al., 1989).

In case the mood scales did not represent an association of like variables, as suggested by the low correlations between items in the reliability analysis, correlation of individual moods against S-IgA was conducted. A failure to detect significant relationships between the individual mood variables and S-IgA suggests that the lack of significance of association between mood scale scores and S-IgA may not have been solely due to heterogeneity within the scales, but also because the individual mood item scores did not relate to S-IgA either.

In view of the problems associated with measurement of affect, discussed above, and the low reliabilities of the affect variables derived from the TML, it is not surprising for these reasons alone that no association between S-IgA and mood was detected. However, the fact that Chamberlain and Spicer (1991) obtained good reliabilities with their mood data, yet found no relationship between mood and S-IgA, suggests that there may be factors other than reliability which should be considered as having contributed to the absence of relationships in the present study:

(1) Mood may have affected S-IgA over a much shorter time period than one day. Levels of S-IgA might then have been influenced more by mood immediately prior to S-IgA sampling than by overall daily mood. Such within-day changes may have been confounded with overall daily mood measured at days end, in this and the study of Chamberlain and Spicer. If this is so, S-IgA in saliva collected during the day, as in Stone *et al.*'s study, would be more likely to correlate with mood measured during the day than that collected late at night, because of the probability of fewer changes in mood before S-IgA sampling earlier in the day. This does not rule out a cumulative affect on S-IgA concentrations, but points to the superimposed within day variation making an association difficult to measure.

Green and Green (1987) detected responses to relaxation within 20 minutes. It does not therefore seem surprising that daily moods did not correlate well with S-IgA collected at the end of the day. An in depth analysis of proximity of mood to S-IgA sampling, relative to S-IgA levels, could be conducted to shed some light on the interactive effects of mood and time on S-IgA levels, using a questionnaire such as the TML.

(2) The relationship between properties of stimuli and their effects on the immune system is more complex than originally thought. The same stimuli may enhance or suppress the immune response depending on other concurrent conditions (Dantzer and Kelley, 1989). It is not practicable to take into account such detail in the design of naturalistic studies such as this, and those of Stone *et al.* (1987) and of Spicer and Chamberlain (1991), in which the capacity of the respondents to take part is a limiting factor. Inclusion of a number of concurrent variables, as in the present research, provides some degree of control.

(3) Treatment of saliva samples after collection was more difficult to control in this and the Chamberlain and Spicer study than in Stone et al.'s investigation, in which samples were collected on site during student classes, and could be immediately treated in a standardized manner. Differences in storage conditions may have contributed to unmeasured systematic variation in S-IgA measurements. A direct comparison of the raw data for S-IgA concentrations from Stone et al.'s and this study was not possible from the data given by Stone et al.

(4) The range of affect experienced by the subjects may not have been extreme enough to have influenced physiological systems to the extent required to alter the immune response. This may have been true even of subjects who scored extreme mood responses, because the scores will have been relative scores and therefore subject to the range of experience they had experienced.

It is an unfortunate characteristic of the present and similar studies in which participation was voluntary, that potential subjects who felt themselves to be under stress, or susceptible to stress, would have chosen not to take part. In fact, the only subject to withdraw from the study did so because she was under stress.

In their study of daily mood and S-IgA Stone et al. (1987) used classes of dental students, and may therefore have been less affected by this possible "auto-culling" of potential responders. Perhaps this is why he was able to measure an association between mood and antigen-specific (though not total IgA) S-IgA response.

#### Present Results in Relation to Previous Studies with S-IgA

The inability to demonstrate an association between mood and S-IgA in this study is consistent with the findings of Stone et al., that daily mood was not significantly related to total S-IgA. This lack of association between mood and total S-IgA does not support the supposition that daily changes in antigen-specific S-IgA synthesis should be reflected in total S-IgA levels, if there is to be any connection between daily mood and upper respiratory tract infection. It is however relevant to the question of the source of significant effects detected by Stone et al., that the only variable to correlate with S-IgA levels in the present study was a control variable, "Day of study", because Stone et al.(1987) did not include control variables in their study, leaving a good deal

of uncertainty as to the reasons for the apparent association between mood and S-IgA which they detected. Stone et al., discarded their first eight saliva samples to eliminate conditioning effects. But the significant "Day of study" effect noted for data collected over 28 days in the present study suggests either that the eight day delay was not long enough, or that other day-related factors were operating.

Although Stone's results with antigen-specific S-IgA demonstrated a relationship between S-IgA concentration and mood, his results with total S-IgA, like those of the present study were not significant. This discrepancy needs to be considered in relation to actual figures presented by Stone. When Stone converted his data to Z scores, carried out a median split, and compared antigen-specific S-IgA levels associated with relatively high vs low negative, and relatively high vs low positive days respectively, the differences he obtained, although significant, were very small. Thus for high negative vs low negative days S-IgA levels expressed as Z scores were -0.133 and 0.138 and for high positive vs low positive days they were .138 and -.07. In the former case the mean S-IgA antibody scores were only 0.271 and in the latter only 0.208 of a standard deviation apart.

Given the uncontrolled naturalistic nature of the above study it would indeed be surprising if such small differences could be replicated in a study of total S-IgA, and conversely it would also not be surprising that some extraneous variable or sampling error could exert a sufficient effect on the data to achieve the degree of effect measured by Stone et al.

#### Results in Relation to Theory of Mood.

In the introduction it was argued that there were firm grounds for regarding mood as being expressed within two substantive domains relating to function of the Behaviour Inhibition and Behaviour Facilitation systems respectively. On this basis it was deemed appropriate to choose mood descriptors representing these domains identified as the positive and negative affect dimensions of Tellegen and Watson.

The results of this study did not show that there was any advantage from adopting this strategy given the low reliability of the positive and negative mood scales and the failure to detect an association of mood with S-IgA levels. The factor analysis results

nonetheless were generally consistent with the existence of the positive and negative mood dimensions.

The affect dimensions of Tellegen and Watson were established as orthogonal, through factor analysis. The schematic representation of activities in the PACS and SAMS, shown as axes at right angles by Henry and Meehan (1981) should not perhaps be regarded as representing activities which are expressed as orthogonal response sets, particularly as the two systems exhibit modulatory interactions. It should also be remembered that our knowledge of the patterned alterations in activity of these and numerous other immunomodulatory systems which operate to maintain homeostasis in response to environmental changes is as yet rudimentary.

The questionnaire items were based on well established findings, so that the lack of positive results in this work appears to be not so much due to inadequate mood theory as to the requirement for subjects to recall mood intensity and time of mood accurately, to the nearest hour. Perhaps there were also problems in the detail of the design with respect to the sampling of moods. There may have been a need for more extensive preliminary research to establish the suitability of the mood descriptors to the study population before the final choice of words was made, although this was not possible given the time constraints on this thesis.

Relating moods assessed by questionnaire to S-IgA will be difficult because of the semantic problems associated not only with the meaning of particular moods, but also with the meaning of 10 as opposed to, say, 1, on a scale of mood intensity. In measuring the subjective experience of mood one is entering the private world of the individual, for which objective research has no direct measure, and in which methodology has little power to access variables. Even though there is considerable evidence for the reliability of mood scales subjects asked to indicate on a 0-10 scale how intensely they experienced certain moods to be, had no way of knowing how intense intensity 10 was on an external scale which allowed them to compare themselves with one another or with some standard. Each subject will have had her own internal scale determined to some extent by the range of experience that she was familiar with, so that what was scored 10 by one individual may have been associated with a low level of physiological activation compared with that associated with 10 on



another's scale. It should be noted however, that the statistical analysis was designed to measure pooled within subject variation and to purge the data of between subjects variation.

### Further Research

#### Relationship between Mood - S-IgA and Health

In the field of research concerned with the relationship between mental states - depression, stress etc. - and immune function, findings have been inconsistent and often inconclusive (Stein *et al.*, 1991). Relatively short term experiments with laboratory animals have often produced significant associations between stress and immunosuppression, but with humans in naturalistic situations over longer time periods, such associations, and in particular their extension to consequences for health, will remain difficult to establish for a number of reasons.

(1) Physiological systems have a great intrinsic capacity to return to set points. Indeed, it has been hypothesised that the suppressive effect of cortisol on inflammatory/immune system reactions is part of the process of readjustment/modulation of physiological systems to their base levels (Munck *et al.*, 1984). The cerebral, neuroendocrine, and immune systems are involved in extensive feedback loops involved in preserving this balance (Blalock and Smith, 1985; Dantzer and Kelley, 1989). Over an extended period it appears unlikely that mood would severely compromise the ability of the body to respond to infection in a study population such as that of the present study, given that none of the respondents, to the author's knowledge, were under the extremes of physical or psychological stress which would be necessary to prevent homeostatic readjustment toward set points.

(2) Infection is a complex process depending on a large number of variables, including intrinsic factors such as age, and extrinsic factors such as rate of exposure to infectious agents. When both of these vary, as in the present study population, which included students continually operating in high population density situations, mothers with snotty-nosed children, and others having less contact with infection, a relationship between psychological factors and health will be difficult to detect.

(3) The body has a battery of defences against infection, which may respond differentially to the effect of stress (Kiecolt-Glaser et al., 1984). S-IgA is only a first line of defence. The ability of individuals deficient in S-IgA to successfully resist infection suggests that the influence of a slight reduction in S-IgA on resistance to infection might be difficult to detect in studies such as the present one, involving relatively short times and small numbers of subjects.

(4) The time course of response to stress may vary widely between different components of the neuroendocrine/immune systems, so that any coupling of stress to infection will be blurred by the overlap in rate of response. For instance, ACTH production by the pituitary gland may be inhibited by glucocorticoid negative feedback within minutes, while the response of peripheral leucocyte secretion of ACTH may take 1-2 days (Delahunt, 1988).

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These considerations lead to the conclusion that it would be difficult to detect an association between S-IgA and health in free-living human populations, and that this study, and the area as a whole, is subject to many extraneous variables. In naturalistic studies the sample size required to establish the significance of an association, will often be prohibitive, given the number of influences operating. Relationships between S-IgA and health have been reported, but in confined populations such as prisoners (McClelland et al., 1982).

Possibly a greater measure of control could be incorporated into future studies such as the present, by making more detailed measurement of control variables. For instance an hour's gardening may be different from one hour's jogging, similarly one glass of wine may be different to half a bottle of whiskey.

S-IgA is most relevant to health if studied in a cumulative way, while examining mood patterns which may be the cause of this cumulative change. Thus a theory of mood patterning in relation to a rise or fall in the cumulative response of S-IgA may be ascertained.

### Application of TML to within-day dynamics of mood

The design of the TML, to allow measurement of actual duration of mood, also allows time of change in mood within days to be ascertained, and may therefore be used to determine how patterns of mood change within the day relate to S-IgA in further research. Although S-IgA levels can change from day to day (Stone *et al.*, 1987a) the minimum time required for S-IgA to respond to psychological state may be less than 24 hours. It is, therefore, of interest to know when changes in mood occur within a day in relation to S-IgA sampling, as it is possible that moods occurring only at a critical time before S-IgA sampling have a significant impact on S-IgA levels. Such detailed data might be useful in interpretation of results given the evidence that S-IgA levels show rapid responses to psychological states such as relaxation (Green and Green, 1987) and stress (McClelland *et al.*, 1985), and also to longer term states such as chronic stress (Martin and Dobbin, 1988). Within day fluctuations may occur over a changing base-line which reflects the cumulative effects of mood on S-IgA, and would reduce the accuracy of its measurement.

Stone *et al.* (1987) showed that the net balance (as measured by their mood adjective checklist) of positive and negative mood within a day could influence S-IgA, but they did not provide any data on the impact on S-IgA of the relationship between moods within the day. This relationship could however strongly influence S-IgA, and the fact that Stone *et al.* found that overall daily mood relates to S-IgA may be simply a result of the increased probability of a predominant daily mood (compared with a non-predominant mood) coinciding with the critical time before S-IgA sampling at which a mood is influential. If a pattern of moods within the day is measured, it creates the possibility of grouping scores by time of mood in relation to a fixed time of S-IgA sampling, thereby increasing the power of data analysis. Such data may be easier to obtain in experimental (frustrating computer programmes), than in observational studies.

The TML may provide the researcher with sensitivity to the effects of circadian rhythms on measurement of mood. In terms of the relationship of mood to S-IgA, this is highly desirable, because evidence indicates that there is an association between circadian mood rhythms and cortisol levels, (Riley *et al.* 1981) and that cortisol in turn, has an immunosuppressive action. Furthermore, the position of peaks within the day

differs between individuals. It is possible that elevated cortisol levels due to circadian rhythmic variation could have a dampening effect on the cortisol response to mood, since cortisol levels are under feedback inhibition. Thus, the influence of a negative mood-inducing event on S-IgA may be lower if the event occurs in the morning than if it occurs in the afternoon.

Mood sequence within days can be determined only with an instrument such as the TML which relates mood to time, throughout the day. As in the case of the temporal relationship between mood and S-IgA change, there is no information on the way in which mood sequence (e.g. positive after negative versus positive before negative) may affect the S-IgA response to a particular mood. On a "Bad" day there is more chance that a relationship between negative mood and S-IgA will be detected, simply because more of the time during that day is negative, so there is a greater chance that any mood preceding another will be negative. The effects of less frequently occurring positive moods may be swamped where days have been grouped according to predominant mood, for analysis. The effects of time and sequence variations will be lost unless the questionnaire (instrument) provides for measurements to enable groupings of subjects, days, and moods to be made according to patterns and times of moods during the day. For instance, a positive after a negative on a negative day, may negate the negative affect and yet not be recorded when a single mood rating for the whole day is elicited, since the predominant mood for the day is negative. A daily time/mood log will provide the ability to use mood timing and mood interrelationship as independent variables (with S-IgA the dependent variable).

## Conclusion

In view of the large number of uncontrolled variables operating in this type of research, it will be difficult to demonstrate an effect of mood on S-IgA unless tight control is aimed for. Experimental designs provide the greatest opportunity for control, but often lack the external validity of naturalistic studies. If experiments produce positive results, as a number involving psychological measures other than mood and aspects of immune function other than of salivary IgA have, there will be strong support for mood-S-IgA research in naturalistic situations. At present researchers on mood in relation to S-IgA have to cope with a mass of uncertainties, which may be reduced by some systematic laboratory research, identifying critical variables and involving intensive examination of a small number of subjects, in parallel with the naturalistic studies of relevance to the "real world".

Such considerations apply to the field of psychoimmunology as a whole. As Stein *et al.* (1991) state, "It may be most meaningful in research concerned with the role of the immune system in relation to behavioural states and physical health and illness to first demonstrate an association between psychiatric disorders such as depression and/or stress and specific immune related disorders prior to considering mechanisms".

The challenge is now to formulate the research designs, naturalistic and/or experimental, under which such associations can be demonstrated.

## REFERENCES

Axelrod, J., and Reisine, T.D. (1984). Stress hormones: Their interaction and regulation. Science 224, 452-459.

Blalock, J.E., and Smith, E.M. (1985). A complete regulatory loop between the immune and neuroendocrine systems. Federation Proceedings 44, 108-111.

Bolger, N. DeLongis, A., and Kessler, R.C., and Schilling, E.A. (1989). Effects of daily stress on negative mood. Journal of Personality and Social Psychology. 57, 808-818.

Booth, R.J. (1990). The psychoneuroimmune network: expanding our understanding of immunity and disease. N.Z. Medical Journal. 103, 314-316.

Bowry, T.R. (1984). Immunology Simplified. (2nd edition) Oxford University Press.

Calbrese, J.R., Kling, M.A., & Gold, P.W. (1987). Alterations in immunocompetence during stress, bereavement and depression: focus on neuroendocrine regulation. American Journal of Psychiatry. 144 1123-1134.

Carlson, N.R. (1986). Physiology of Behaviour. 3rd edition. Allyn & Bacon Inc., London.

Casey, V.T. and Dywer, J.T. (1987). Premenstrual syndrome: theories and evidence. Nutrition Today. November/December 4-12.

Chamberlain, K. and Spicer, J. (1991). Assessing the effects of short term changes in negative affect on salivary IgA (paper presented at New Zealand Psychological Society Conference, Palmerston North, New Zealand).

Cohen, S. and Williamson, G.M. (1991). Stress and infectious disease in humans. Psychological Bulletin. 109, 5-24.

Dantzer, R. and Kelley, K.W. (1989). Stress and immunity: An integrated view of relationships between the brain and the immune system. Life Sciences. 44, 199-208.

Delahunt, J.W., (1988). Hormonal changes in stress. Patient Management, April 23-30.

Delongis, A., Folkman, S. and Lazarus, R.S. (1988). The impact of daily stress on health and mood: Psychological and social resources as mediators. Journal of Personality and Social Psychology. 54, 486-496.

Depue, R.A. and Iacono, W.G. (1989). Neurobehavioural aspects of affective disorders. Annual Review of Psychology. 40, 457-492.

Diener, E. Larsen, R.J. Levine, S. & Emmons, R.A. (1985). Intensity and frequency: dimensions underlying positive and negative affect. Journal of Personality and Social Psychology. 48, 1253-1265.

Eysenck, H.J. (1980)(Ed). A Model for Personality, Springer-Verlag, New York.

Goldstein, D. (1987). Stress-induced activation of the sympathetic nervous system. In: Bailliere's Clinical Endocrinology and Metabolism. 1, 253-278. Neuroendocrinology of Stress. A. Grossman (Ed.).

Gray, J.A. (1981). A critique of Eysenck's theory of personality. In: A Model for Personality. H.J. Eysenck (Ed.). Springer-Verlag, New York. 246-276.

Gray, J.A. (1982). Precipit of The neuropsychology of anxiety: An enquiry into the functions of the septo-hippocampal system. The Behavioural and Brain Sciences. 5 469-534.

Gray, J.A. (1987). The Psychology of Fear and Stress. (2nd edition). Cambridge University Press.

Green, R.G. and Green, M.L. (1987). Relaxation increases salivary immunoglobulin A. Psychological Reports. 61, 623-629.

Guyatt, G.H., Berman, L.B., Townsend, M., and Taylor, W.D.(1985). Should study subjects see their previous responses? Journal of Chronic Disease. 38, 1003-1007.

Harre, R. (1983). Personal Being. Blackwell, Oxford.

Hedges, S.M., Jandorf, L., and Stone, A.A. (1985). Meaning of daily mood assessment. Journal of Personality and Social Psychology. 48, 428-434.

Henry, J.P., and Meehan, J.P. (1981). Psychosocial stimuli, physiological specificity, and cardiovascular disease. In: Brain, Behaviour and Bodily Disease. H.Weimer, M.A. Hofer and A.J. Stunkard. (Eds) Raven Press, New York, 305-333.

Jemmott, J.B.III, Borysenko, J.Z., Borysenko, M., McClelland, D.C., Chapman, R., Meyer, D., and Benson, H. (1983). Academic stress, power motivation, and decrease in salivary secretory immunoglobulin A secretion rate. Lancet. 1; 1400-1402.

Jemmott J.B. and McClelland, D.C. (1988). Secretory IgA and resistance to infection. Behavioural Medicine.

Jemmott, J.B., and Magliore, K. (1988). Academic stress, social support, and secretory immunoglobulin A. Journal of Personality and Social Psychology. 55, 803-810.

Kiecolt-Glaser, J.K., Garner, W., Speicher, C.E., Penn, G.M., Holliday, J.W., and Glaser, R. (1984). Psychosocial modifiers of immunocompetence in medical students. Psychosomatic Medicine. 46, 7-14.

Kiecolt-Glaser, J.K., and Glaser, R. (1988). Methodological issues in behavioural immunology research with humans. Brain, Behaviour and Immunity. 2, 67-78.



Lammers, C.J. Dengerink, H.A., Wright, J., Whipple, J. and McGuire, T. (1987). Psychological stress and progressive muscle relaxation: Effects on secretory immunoglobulin A. Cited Jemmott, J.B., and McClelland D.C. (1988).

Lipinski, J.F., Cohen, B.M., Zubenko, G.S. and Waterman, C.M. (1987). Adrenoreceptors and the pharmacology of affective illness: A unifying theory. Life Sciences. 40, 1947-1963.

Locke, S.E. (1982). Stress, adaptation and immunity: Studies in humans. General Hospital Psychiatry. 4, 49-58.

Lundberg, U. and Frankenhaeuser, M. (1980). Pituitary-adrenal and sympathetic-adrenal correlates of distress and effort. Journal of Psychosomatic Research. 24, 125-130.

McClelland, D.C., Floor, E., Davidson, R.J. and Sarson, C. (1980). Stressed power motivation, sympathetic activation, immune function and illness. Journal of Human Stress. 6, 11-19.

McClelland, D.C., and Jemmott, J.B.III (1980). Power motivation, stress and physical illness. Journal of Human Stress. 6, 6.15.

McClelland, D.C., Alexander, C., and Marks, E. (1982). The need for power, stress, immune function, and illness among male prisoners. Journal of Abnormal Psychology. 91, 61-70.

McClelland, D.C., Ross, G., and Patel, V. (1985). The affect of an academic examination on salivary norepinephrine and immunoglobulin levels. Journal of Human Stress. 11, 52-59.

McClelland, D.C. and Kirshnit, C. (1988). The effect of motivational arousal through films on salivary immunoglobulin A. Psychology and Health. 2, 31-52.

McCormack, H.M., de L.Horne, D.J. and Sheather, S. (1988). Clinical applications of visual analogue scales: A critical review. Psychological Medicine. 18, 1007-1019.

Martin, R.A. and Dobbin, J.P. (1988). Sense of humor, hassles, and immunoglobulin A: Evidence for a stress-moderating effect of humor. International Journal of Psychiatry in Medicine. 18, 93-105.

Matthews, G., Jones, V.M., and Chamberlain, A.G. (1990). Refining the measurement of mood; the UWIST mood adjective checklist. British Journal of Psychology. 81, 17-42.

Morley, J.E., Kay, N.E., Solomon, G.F. & Plotnikoff, N.P. (1987). Neuropeptides: conductors of the immune orchestra. Life Sciences. 41, 527-544.

Mouton, C., Fillion, L., Tawadros, E., and Tessier, R. (1989). Salivary IgA is a weak stress marker. Behavioural Medicine. 15, 179-185.

Mower-White, C.J. (1982). Consistency in Cognitive Social Behaviour. An Introduction to Social Psychology. Routledge and Kegan Paul Ltd., London.

Munck, A., Guyre, P.M. and Holbrook, N.J. (1984). Physiological functions of glucocorticoids in stress and their relation to pharmacological actions. Endocrine Reviews 5, 25-44

Neveu, P.J. (1988). Cerebral neocortex modulation of immune functions. Life Sciences. 42, 1917-1923.

Norsusis, M.J. (1985). Advanced Statistics Guide. Chicago, USA.

O'Leary, A. (1990). Stress, emotion and human immune function. Psychological Bulletin. 108, 363-382.

Plutchik, R. (1980). Emotion: A Psychoevolutionary Synthesis. Harper Rowe, New York.

Riley, V., Fitzmaurice, M.A., and Spackman, D.H. (1981). Psychoneuroimmunologic factors in neoplasia: Studies in animals. In: Psychoneuroimmunology. R. Ader (Ed.), Academic Press, London. 31-102.

Rossi, A.S. and Rossi, P.E. (1977). Body time and social time: Mood patterns by menstrual cycle phase and day of the week. Social Science Research. 6, 273-308.

Shweder, R.A., and D'Andrade, R.G. (1980). The systematic distortion hypothesis. New Directions for Methodology of Social and Behavioural Science. 4, 37-58.

Stein, M., Miller, A.H., Trestman, R.L. (1991). Depression, the immune system, and health and illness: findings in search of meaning. Archives of General Psychiatry. 48, 171-177

Stenhouse, D. (1974). The Evolution of Intelligence: A general theory and its implications. Allen and Unwin, London.

Stone, A.A., Cox, D.S., Valdimarsdottir, H., Jandorf, J. and Neale, J.M. (1987)a. Evidence that secretory IgA is associated with daily mood. Journal of Personality and Social Psychology. 52, 988-993.

Stone A.A., Hedges, S.M., Neale, J.M. and Satin, M.S. (1985). Prospective and cross-sectional mood reports offer no evidence of a "Blue Monday" phenomenon. Journal of Personality and Social Psychology. 49, 129-134.

Tecoma, E.S. & Huey, L.Y. (1985). Psychic distress and the immune response. Life Sciences. 36, 1799-1812.

Tomasi, T.B. (1976). The Immune System of Secretions. Englewood Cliffs, NJ: Prentice-Hall. Cited in Jemmott & McClelland (1988). Secretory IgA and resistance to infection.

van Kammen, D.P. and Antelman, S. (1984). Impaired noradrenergic transmission in schizophrenia? Life Sciences. 34, 1403-1412.

Watson, D. and Tellegen, A. (1985). Toward a consensual structure of mood. Psychological Bulletin. 98, 219-235.

Watson, D. (1988). The vicissitudes of mood measurement: Effects of varying descriptors, time frames, and response formats on measures of positive and negative affect. Journal of Personality and Social Psychology. 55, 128-141.

Watson D., Clark, L.A. and Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. Journal of Personality and Social Psychology. 54, 1063-1070.

Williams, R.B., Lane, J.D., Kuhn, C.M., Melosh, W., White, A.D., Schanberg, S.M. (1982). Type A Behaviour and elevated physiological and neuroendocrine responses to cognitive tasks. Science. 218, 483-485.

APPENDIX A.

Mood Assessment Questionnaire, including Instructions

## DAILY MOOD RECORD

Fill in a daily mood record sheet for each day, between 9 and 11pm in the evening if possible.

Do not refer back to previous days when filling out your record sheet.

### (a) Mood time and duration.

A 24 hour time scale is given on each mood record sheet. Draw a line down the appropriate mood columns to show which moods you experienced on the day, when, and for how long they lasted. It is acceptable to record different moods as occurring at the same time.

If a peak intensity for the day occurred for any mood place a cross on its mood line to show when.

### (b) Mood intensity.

At the bottom of each mood column are two boxes; in the upper score the average intensity, and in the lower score the peak intensity (if it peaked) of the mood which you recorded in the column above.

Use the following scale to score daily average and peak intensities of mood, where zero indicates total absence, and 10 extreme experience of mood.

0    1    2    3    4    5    6    7    8    9    10



APPENDIX B.

Information about the study given to prospective subjects



## THE INFLUENCE OF MOOD CHANGE ON THE IMMUNE SYSTEM

I am conducting a study which aims to explore how daily moods influence the antibodies which reduce the risk of infectious diseases. This risk can be measured by taking a small sample of saliva and finding out how much secretory immunoglobulin A (s-IgA) antibody is present. The project is being run by Jan Binnie, research student, under the supervision of Dr J. Spicer, Psychology Department, Massey University.

### AM I ELIGIBLE TO TAKE PART?

You are eligible to participate if you are female, and if you are not pregnant. You should also not be suffering from any continuing illness which requires you to take regular medication.

### WHAT WOULD I HAVE TO DO?

You would be asked to take part in the study for a continuous four week period. At the beginning you will be asked a few questions about your physical health and shown how to take a saliva sample, and how to fill in the forms which are used during the study period. You will be required to do two sorts of things for a few minutes each evening for the four weeks: first would be to produce a small sample of saliva, place it in a container which we provide, and then store it in your refrigerator until it is collected once or twice a week at a pre-arranged time; the second would be to complete a form which records your moods during the day and another form which records changes in your health, any medication you have taken, your alcohol and tobacco intake, whether you have taken any strenuous exercise. At the end of the four weeks I would like to interview you again to review your mood patterns during the time.

### WHAT CAN I EXPECT FROM THE RESEARCHER?

All participants:

- have the right to refuse to answer any particular questions, and to withdraw from the study at any time.
- provide information on the understanding that it is confidential to the researcher. All forms and samples are identified only by code number, and are seen only by the researcher. It will not be possible to identify individuals in any published reports.
- have the opportunity to discuss their own results with the researcher at the completion of the study.

If you are interested in participating and would like to discuss it further with me, please fill in your name and phone number on the attached form and place it in the envelope labelled "Mood-IgA study" pinned to the notice board in the Postgrad. coffee room.  
Thank you for your assistance.

Jan Binnie

THE EFFECTS OF MOOD CHANGE ON THE IMMUNE SYSTEM

I am interested in this study, and am willing to be contacted to discuss my participation in it.

Name: -----

Phone: -----

APPENDIX C.

Instruction Sheet for Subjects

THE EFFECTS OF MOOD CHANGE ON THE IMMUNE SYSTEM

## INSTRUCTIONS

## SEQUENCE

Each day of the study you should complete the tasks between 9 & 11pm in the following order: take the saliva sample; complete the health record for the day; complete a daily mood record; note any memorable events on the reverse side of the sheet. Make sure that the day number coincides on the saliva sample and on both records.

## TAKING A SALIVA SAMPLE

Select the container with the same numbers as the forms for that day and any pipette. Hold head slightly down with jaw slack, and place pipette under tongue with bulb depressed.

When some saliva has formed, manipulate the bulb and transfer the saliva to the container.

Repeat this process until you have gone beyond the container mark. Seal the container, place it in the fridge & throw away the pipette.

## IMPORTANT

\* Producing the saliva sample could take up to ten minutes. To get accurate measurements it is important that the saliva is not forced out by "pumping". Try not to hurry the process by forcing out the saliva.

\* Often air bubbles will form in the pipette and the container. Ensure that there are no bubbles in the area up to the mark on the container. It does not matter if there are bubbles above the mark.

\* It is important to keep the container and pipette clean, so do not remove it from the plastic bag until you are ready to collect the sample.

\* If you have any problems discuss them with me as soon as possible.

## COMPLETING THE RECORDS

Instructions for these are given in each booklet. As we want YOUR records, it is important that these are completed on your own. Do not discuss your answers with anyone else.

## MISSED DAYS

If for some reason you cannot avoid missing a day DO NOT complete the records for that day, but move on to the next days sheets. But if you feel that something significantly affected your mood on the missing day still note it on the reverse side of the mood record sheet that you would have filled in. Under no circumstances should you complete the tasks the following morning for the previous day.

If you have any problems at all please contact: Jan Binnie, 82699