THE EFFECT OF EXAMINATION STRESS ON
PHAGOCYTIC IMMUNE FUNCTIONING

THESIS

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MASTER OF SCIENCE

By

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The purpose of this study was to determine whether psychological stress, specifically examination stress, would decrease immune system functioning. Twenty-five first-year master's and doctoral students who volunteered to participate in the study were psychologically and immunologically assessed during two high- and two low-stress periods. Immunological assessments included a white blood cell differential count and nitroblue tetrazolium test (NBT) to measure neutrophil functioning. Psychological instruments administered at each assessment period included Clinical Analysis Questionnaire (CAQ), Bender Gestalt Test, State-Trait Anxiety Inventory (STAI) and a Brief Stress Questionnaire.

Stepwise discriminant function analysis of data revealed five variables which contributed significantly to change under stress and yielded an average canonical correlation of .79 (p < .002) providing evidence of support for the hypothesis that increased psychological stress will alter immune functioning and heighten psychological responses.
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THE EFFECT OF EXAMINATION STRESS ON PHAGOCYTIC IMMUNE FUNCTIONING

Many scholars, since the time of Plato, have believed that mind and body were of a different nature, and that the mind could influence and was dominant over the body (Benson, 1979). The modern separation of mind and body has been traced to Rene Descartes, the seventeenth-century mathematician and philosopher who believed the mind was not the master of the two and gave the body equal influence. According to Descartes, mind and body were more easily conceived of as being separate. Each was responsible for its own functions, and interacted in a purely machine-like fashion. While Descartes' arguments seemed somewhat dated, no one since has entirely succeeded in rejecting the concept that mind and body are quite distinct. Attempts have been made to eliminate this distinction by the formation of disciplines such as psychosomatic medicine which provides a theoretical framework to relate mind and body (Benson, 1979).

The roots of the idea that psychological factors can precipitate or cause disease date back to antiquity (Plaut & Friedman, 1981). More recently, the work of Alexander (1950) and Dunbar (1943) related specific psychological factors and personality types to the onset and course of disease. Alexander believed a specific psychological conflict to be a
causative factor in illness, whereas Dunbar believed a definable personality type was of etiologic importance. Conversely, Engel (1954, 1977) proposed a biopsychosocial disease model which considered the etiology of disease to be multifactorial.

Feuerstein and Schwartz (1977) have described humans as complex psychological systems who can be understood from an integrative perspective emphasizing biological, social, and psychological aspects. Dubos (1955, 1961) recognized the possible role of psychosocial factors in infectious disease, as well as other factors such as prior exposure, nutritional status, and genetic factors. Psychosocial factors have not influenced disease or health in a mystical way—rather, it has been known that psychological factors influence a wide range of physiological, hormonal, and biochemical responses as well as the natural history of many disease states. However, the mechanisms of their influence on the etiology of disease has been largely speculative (Plaut & Friedman, 1981). Perhaps the least understood are the interrelationships between the immune process and behavior (Adher & Cohen, 1974).

Much work was stimulated by the animal studies of Hans Selye (1946) which linked environmental stress to an adrenocortical response. His work on stress distinguished between nonspecific and specific features. Objective experiments have shown that every event or stimulus such as injury, interpersonal problems, or drugs, produces nonspecific stress
in addition to specific effects. Nonspecific stressors may have been the primary causes of many common diseases (Selye, 1974).

Selye called the response to noxious agents or stressors "The Biological Stress Syndrome," or "General Adaptation Syndrome." The first stage of the syndrome was termed the alarm reaction when the body shows the physical changes characteristic of the first exposure to a stressor. Resistance has been diminished and death could result if the stressor has been sufficiently severe. After the initial alarm reaction during which the secretion of corticoids has increased, the body becomes adapted and begins to resist (Stage 2). The body's adaptability or "adaption" energy is finite however, and eventually exhaustion ensues (Stage 3). During this third stage, the signs of the initial alarm reaction will appear once again, but this process has not been shown to be reversible and the organism dies. Increased adrenocortical function has been thought to be the primary mechanism by which stress and disease were linked (Pelletier, 1977). Corticoids have been considered to elicit thymus shrinkage, atrophy of the lymph nodes, inhibition of the inflammatory reactions, and sugar production.

Potentially, one of the negative results of excessive levels of stress has been the effect on the immune system (Pelletier, 1977). Research on the relationships between the human brain, behavior, and immunity has been fairly recent, most studies having been completed since 1977. These studies
have seemed to support the belief that a combination of excessive stress and inadequate coping skills may increase host susceptibility to illness and affect recovery periods (Locke, 1982).

Roessler, Cato, Lester, and Couch (1979) conducted a study in which they examined the relationship of coping ability and life-change stress to immune response. They found that antibody response to influenza vaccination was decreased among high life-change stress subjects with low ego strength, which was used as the measure of coping ability. Locke (1982) reported in a review paper that he found similar results in a study of natural killer cell activity. Those subjects who had poor coping skills, inferred from the self-reporting of large numbers of psychiatric symptoms, had significantly diminished natural killer cell activity in comparison to those with better coping skills.

Pelletier (1979) referred to the work of stress researchers Appley and Trumbell (1967) and discussed the differential effects of psychological stress on individuals. Different individuals have responded to the same conditions in different ways. Further, the same individual could have entered into a stress state in response to a given stress condition and may not have become stressed to the same stressor at another time.

Bartrop, Lazarus, Luckhurst, Kiloh, and Penny (1977) found depressed lymphoblast formation in bereaved spouses, as did Schliefer, Keller, McKegey and Stein (1980) in their
study of husbands of women who had died with cancer, demonstrating the link between the central nervous system and the immune system. The work of Stein, Schiavi, and Camerino (1976) demonstrated that psychosocial stressors may influence humoral and cell-mediated responses as well as susceptibility to infections and neoplastic processes.

McClelland, Floor and Davidson (1980) found that individuals high in the need for power, if it is inhibited and stressed, leads to chronic sympathetic overactivity characterized by higher rates of epinephrine excretion which were significantly associated with reports of more frequent illnesses. Monjan and Collector (1977) daily subjected rats to an auditory stressor for varying lengths of time and found that the stressor could enhance immune system functioning as well as depress it.

Holmes and Rahe (1967) have demonstrated through their research that increased incidence of stressful life changes has seemed to be related to increased incidence of disease. They developed the Social Readjustment Rating Scale to measure stressful life changes and their relations to disease susceptibility. Numerous studies have shown that generally, the more life stresses a person has experienced, the higher the probability that some physical or psychiatric symptom will develop (Borysenko, 1984).

One of the leading researchers in the area of stress and immunity is George Solomon (1985) who has begun to
develop a model of the links between the central nervous system and immunity. He noted several analogies between the immune system and central nervous system. Both serve functions of defense and adaption and have the capacity for memory. Pathological syndromes may have occurred in each as a result of inadequate defenses in each system. Prior experience of a stimulus or noxious agent could have led to tolerance or sensitivity in each system.

Solomon (1985) has listed 14 hypotheses which represent possible connections between the two systems. The following two hypotheses have been considered of particular importance to the present research.

1. Enduring coping style and personality factors (trait characteristics) should influence the susceptibility of an individual's immune system to alteration by exogenous events, including reactions to events.

2. Emotional upset and distress (state characteristics) should alter the incidence, severity, and/or course of diseases that are immunologically resisted (infectious and neoplastic diseases) or are associated with aberrant immunologic function (allergies, autoimmune disease, AIDS).

The human body has the capacity to resist almost all types of organisms or toxins that tend to damage tissues and organs. This capacity has been called immunity. The immune system has been shown to be the ultimate defense of vertebrates against disease. Without a functioning immune system, the
host organism has eventually succumbed to infection (Kimball, 1982). Immunologic competence has been defined by Palmblad (1981) as the capacity to identify and reject foreign materials and accept those with markers of self.

Immunity may either be (a) specific, involving humoral (antibody) and cell-mediated (specialized T-lymphocyte responses), or (b) nonspecific, such as complement activation or phagocytosis. Phagocytosis has been shown to be the ability of certain cells to ingest and digest particulate substances (Klein, 1982).

Neutrophils (polymorphonuclear leukocytes or PMNs) have been known as the most common blood leukocytes as well as the most common cells in the bone marrow. Their primary role has been the prevention of invasion by pathogenic microorganisms or in the localization and killing of them after they have invaded. Neutrophils adhere to and emigrate between the endothelial cells of blood vessels to reach the tissues where they perform their duties. Decreased adherence in vitro has been shown to correlate with decreased accumulation of PMN's in vivo with increased severity of infections. Impaired chemotaxis (migration toward chemo-attractants) has resulted in slower or decreased accumulation of PMN's at sites of infection, again correlated with increased frequency or severity of infections. After the microbe has attached to the surface of the neutrophil, it will be engulfed and killed. Killing has been dependent upon the generation of oxygen radicals and upon the release
of several lysosomal enzymes and cationic proteins. Many congenital and acquired pathologic conditions have led to disorders of uptake and killing and patients usually exhibited an increased susceptibility to bacteria and fungi (Boggs & Winkelstein, 1983).

Relatively few studies have assessed the relationship between stress and neutrophil functioning. Those that have reported conflicting results. Palmblad, Cantell, Strander, Froberg, Karlsson, Levi, Granstrom, & Unger (1976) found that blood neutrophil granulocytes exhibited decreased ability to phagocytize Staphylococcus aureus during sleep deprivation. After the stressor was removed, phagocytosis was even higher than before. In a second study, Palmblad, Petrini, Wasserman, and Akerstedt (1979) found again that granulocyte functions may be reduced during sleep deprivation. Measurements in both studies of adrenomedullary and adrenocortical hormone revealed increases in serum cortisol and urinary catecholamine output in the first study, but decreased in the second suggesting that these hormones are not important mediators.

Harmsen and Turner (1985) subjected rats to inescapable electric foot shock and found that fewer neutrophils accumulated at the inflammatory site of stressed rats as compared to nonstressed rats. However, phagocytosis of zymosan was higher in stressed rats. Glasser, Heustis and Jones (1977) used intravenously administered dexamethasone to determine whether steroids exert a direct inhibitory effect on
neutrophil functioning. Results revealed the functional competence of neutrophils was not altered after short-term exposure of healthy donors to steroid medication.

Clearly, more research is needed in this area of immune-system functioning, particularly to assess the long-term effects of psychological stress. The purpose of the present study was to determine if psychological stress, defined as examination stress, can systematically produce negative changes in immune parameters and psychological functioning. It is hypothesized that as (examination) stress occurs, there will be a decrease in immune-system functioning with heightened psychological activity.

Method

Subjects

Subjects were 25 first-year masters' and doctoral students in psychology at North Texas State University who volunteered for the research project. There were five men and 20 women aged 22 to 43 years with a mean age of 30 years. All subjects signed an Informed Consent Form (Appendix A) allowing for blood samples to be drawn and psychological tests to be administered to them. Prospective subjects completed a Health Questionnaire (Appendix B) to gather demographic data as well as screen for excessive alcohol intake, use of medications, normal blood pressures (below 140/90), and height and weight ratios.
Instruments

Social Readjustment Rating Scale (SRRS). A list of 43 life events with different weighted scores for each event was developed by Holmes and Rahe (1967), and designed to predict the onset of illness based upon the magnitude of life change experienced the preceding year.

Health Attribution Test (HAT). A questionnaire of 22 items designed to measure attitudes and perceptions of health was designed by Achterberg and Lawlis (1980). Scores reveal the percentage of locus of control attributed to self, powerful others, or chance. Validity is reported to range from .29 to .49 (Achterberg & Lawlis, 1980), and the reliability from .86 to .92.

State-Trait Anxiety Inventory (STAI). Separate self-report scales to measure state anxiety (a transitory emotional state) and trait anxiety (a relatively stable individual difference in anxiety proneness) was developed by Spielberger, Gorsuch, and Lushene (1970). Retest reliabilities are reported in the high .70s for A-trait. A-State correlations range from .27 to .54 (Anastasi, 1982).

Clinical Analysis Questionnaire (CAQ). The CAQ (Cattell, 1973) was developed to measure both pathological and normal personality factors. The validity of this instrument was determined by eight major factor analytic studies, all of which reported significant results (Krug, 1980). The validity of the CAQ is reported to range from .45 to .86. Reliability ranges from .51 to .90.
**Bender-Gestalt Test.** The Bender-Gestalt Test is used to assess perceptual-motor functioning as well as for the detection of brain damage. It was developed by Lauretta Bender in 1938. The Embree-Butler scoring system (1967), which has a reliability of .97, was utilized.

**Brief Stress Questionnaire.** This questionnaire (Appendix C) was designed to obtain a self-report by subjects regarding whether or not they were experiencing psychological stress and the nature of the stress.

**WBC Differential Count.** A differential white blood cell count was utilized using techniques described in Diggs (1976). Slides were stained with Wrights stain and one-hundred cells were counted to determine the percentage of each cell type.

**Quantitative Nitroblue Tetrazolium Test (NBT).** The quantitative NBT Test is used as a screening test for disorders of phagocytosis (the ability of leukocytes to engulf foreign materials and seal them with phagosomes) and microbial killing which is the ability to digest or destroy the engulfed material (Maderazo & Ward, 1980). The NBT (a water-soluble dye) is ingested with immune complexes and is reduced by lysosomal enzymes as the immune complex is digested. The amount of dye reduced is an index of phagocyte activity (Baehner & Nathan, 1968).

**Procedure**

Data were collected over a period of 7 months, during two final examination periods defined as high-stress periods.
and two nonexamination periods defined as low-stress periods. These data collection points occurred in May (high-stress final examination period), September (low stress), October to November (low stress) and December (high stress final examination period). Blood samples were drawn and psychological tests were administered in the biology building at North Texas State University. Some data collection points were missed by some students, therefore grouped data analyses are on uneven samples.

For each of the four assessment periods, subjects were asked to refrain from taking medications or consuming alcohol for 24 hours. No subjects reported extensive exercise immediately prior to blood sampling. Blood was not drawn from women during menses as neutrophil activity is reported to be at a decreased level during that time (Berger, 1982). Blood samples (7 ml.) were obtained by venous puncture using a vacutainer by trained phlebotomists under the supervision of a licensed physician. The time blood samples were drawn was held constant for each subject within 1 hour. After blood samples were obtained, the Bender-Gestalt test was administered. Subjects were then asked to complete the Clinical Analysis Questionnaire, the State-Trait Anxiety Inventory, and the Brief Stress Questionnaire. The Health Attribution Test and the Social Readjustment Rating Scale were administered at the first assessment period only.
Immediately after blood samples were drawn, slides were prepared for WBC differential count; however, staining and counting were completed at a later date. The quantitative Nitroblue Tetrazolium Test was completed within 4 hours after blood was drawn. Blood was allowed to settle 45 minutes to 1 hour each time, the time been held constant to reduce variability of results. The plasma which contained leukocytes was decanted into 5 ml. culture tubes using a Pasteur pipette and diluted with 0.9 ml. white blood cell diluting fluid. Cells were counted and leukocytes were then adjusted to a concentration of 5 X 10^5 cells/ml. using Hanks Balanced Salt Solution (HBSS). To each of duplicate 5 ml. glass culture tubes the following were added: 5 X 10^5 polymorphonuclear leukocytes in a volume of 0.1 ml. tissue culture medium (0.025 ml. of immune complex) and 0.1 ml. of 0.01 M potassium Cyanide. This mixture was then incubated at 37° C for 10 minutes with gentle shaking. To one tube was added 0.1 ml. of NBT (2 ng/ml. in saline), and to the other was added 0.1 ml. EDTA. Both were incubated at 37° C with gentle shaking for 15 minutes. The reaction was then stopped with 4.0 ml. of 0.5 N hydrochloric acid. The mixture was transferred into 30 ml. glass centrifuge tubes using a Pasteur pipette, and centrifuged for 10 minutes at 1,000 g at 4° C. The fluid was then discarded and NBT extracted by adding 4.0 ml. pyridine in a boiling water bath for 10 minutes. The optical density of the extract in each tube was determined at 515 mm. in a Perkin-Elmer Lambda 3A
spectrophotometer using pyridine as the blank control. The optical density of the control sample (EDTA) was subtracted from the optical density of the experimental sample (NBT). The results of the dual samples were averaged for each individual, and was reported as a percentage of the group NBT. \( N \) varied with each assessment period as some of the 25 subjects were not present each time.

**Results**

The data were treated statistically by stepwise discriminant function analysis. There were five variables which contributed significantly to change under stress. These variables in order of significance levels were the Nitroblue Tetrazolium Test (NBT), Clinical Analysis Questionnaire (CAQ) Factors I Sensitivity, Ps Psychological Inadequacy, State-Trait Anxiety Inventory I, state anxiety, and CAQ Factor D₁ Hypochondriasis (see Table 1).

**Table 1**

Factors Predictive of Stress Condition

<table>
<thead>
<tr>
<th>Variable</th>
<th>Partial ( R^2 )</th>
<th>( F )</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitroblue Tetrazolium Test (NBT)</td>
<td>.71</td>
<td>26.93</td>
<td>.0003</td>
</tr>
<tr>
<td>CAQ I Sensitivity</td>
<td>.66</td>
<td>21.78</td>
<td>.0007</td>
</tr>
<tr>
<td>CAQ Ps Psychological Inadequacy</td>
<td>.50</td>
<td>10.97</td>
<td>.00069</td>
</tr>
<tr>
<td>State-Trait Anxiety Inventory I - State Anxiety</td>
<td>.47</td>
<td>9.59</td>
<td>.0102</td>
</tr>
<tr>
<td>CAQ D₁ Hypochondriasis</td>
<td>.36</td>
<td>6.24</td>
<td>.0296</td>
</tr>
</tbody>
</table>

\( N = 69 \), Average Canonical Correlation .79, \( p < .002 \).
Discussion

Results provide clear evidence of support for the hypothesis that increased psychological stress will alter immune functioning and heighten psychological responsiveness. Students reported greater stress during examination periods as well as increased anxiety. The data point to several contributing psychological and immune variables indicative of heightened response patterns.

However, the examination periods and the self-report of stress may have also been compounded by such unmeasured factors as loss of sleep and decrease of essential nutrients, both by improper diet and loss from increased stress. For example, stress is known to promote a discharge of B vitamins which are necessary for stability in both physical and psychological functioning. Another possible contributor may be lack of exercise during examination periods due to time constraints.

The prime result may be seen in the decrease in immune functioning during the (examination) stress period. It seems likely that this conceptual relationship, i.e., that increased stress produces decreased immune functioning, can be generalized to other kinds of (external) psychological stress producing (internal) immune dysfunctioning. It also seems likely that internal biological stress could produce both psychological and physical symptoms.
Psychological activity during times of stress can be seen in the response patterns of the students. These patterns indicate a heightened sensitivity to interpersonal interaction involving sentiment, a sense of dependency and feelings of more need for protection which suggest the occurrence of a certain degree of regressive phenomenon and a concomitant reduction in coping skills. Conversely, the student may respond in a practical "get down to business" manner by focusing on present tasks and excluding extraneous or interfering stimuli, thereby coping with and reducing stress.

Variations in the belief of psychological adequacy and worth are also apparent which would include self-concept and image percept. These variables are seen to change under stress. The obvious interpretation is that the higher the sense of psychological adequacy, the less the effect of stress. The converse is also likely to be true, that is, the lower the sense of psychological adequacy, the greater the impact of stress.

Anxiety as a factor of change with stress is clearly seen in the response of these students and is inclusive with the factors of sensitivity and psychological adequacy. For example, anxiety is not likely to occur if a person believes himself/herself equal to the task at hand and becomes focused on the task, which will reduce both anxiety and stress. But, as stress and anxiety occur together, they will be accompanied by feelings of inadequacy and lowered sense of worth and
self-concept, as well as increasing dependency feelings and needs for protection, perhaps even associated with escape fantasy.

Obsessive/depressive somatization patterns are also in evidence as stress related factors. Stress can serve as both a stimulator of activity by heightening awareness to bodily (dys)functioning and to precipitating stimuli. However, as stress continues, adaptation occurs and body/mind adjustments take place to accomodate the stressor. The increase of obsessiveness is a psychological defense strategy, usually triggering compulsive behavior which can serve to block (but not dispel) anxiety through repetitive action. The compulsive behavior can also serve as a coping mechanism to reduce stress by serving as a motivator for working at problem solution, returning a measure of balance in functioning to the individual.

The body/mind reactive process can also be seen in the somatization of responses through a demonstration of stronger concern for the present state of health, which in fact may be somewhat diminished by lowered immune capability in reaction to stress. In addition, as obsessive/compulsive patterns ensue and somatization with anxiety occurs, depressive behavior will become evident.

The total body/mind function under stress will be bi-phasic, from stimulation to depression. It becomes individually adaptive with increased defenses or becomes healthful as stress is reduced.
This study has demonstrated that increased stress lowers the immune system's ability to resist harmful agents and increases psychological activity through changes in sensitivity, psychological adequacy, anxiety, obsessive somatization, and depressive patterns. Psychological stress may be considered as a stimulator of bio-psychological interactions, all related to the same kind of stress event—individual differences in susceptibility and reaction patterns notwithstanding. The specific consequences, however, are very probably individualistic in terms of a specific reaction syndrome.
Appendix A

Informed Consent

I, ________________________________, wish to participate in a research project being conducted by Nancy A. Didriksen. I understand that the primary purpose of this research is to demonstrate the existence or nonexistence of a relationship between perceived psychosocial stress, various immune system parameters and personality factors. I understand that this research is being conducted in cooperation with the biology department and that I will be required to give from 4 to 6 blood samples during the course of this project. I understand that all blood will be drawn by a trained phlebotomist and that J. R. Toledo, M.D., will provide medical supervision. I understand that I will be required to take paper and pencil psychological tests each time a blood sample is taken.

I understand that all test results both psychological and biological, will be coded to ensure confidentiality and that feedback will be provided upon completion of the study. I understand that my participation in this study is completely voluntary and that I may withdraw at any time without jeopardy. I understand that the investigator may drop me from the study as long as this action is not detrimental to me.

This research project has been fully explained to me and I have read and fully understand this agreement. Therefore, I voluntarily agree to participate in this research project.

Signed ___________________________ 
Participant

Signed ___________________________
Witness

Date ________________________________
Appendix B

Name ___________________________ Today's date ________________

Address ________________________________

Residence phone no. ( ) ________________
Business/other phone ( ) ________________

Date of birth ________________ Sex ______ Race ______
Marital status ________________ Other employment ________________

Where employed __________________________

Program at NTSU _______ Do you feel any pronounced stress at this time? ______ If yes, describe the nature of the stress __________________________

Do you consider yourself generally optimistic ________ pessimistic ________

What are your primary foods and drink (please list) ______

Do you consider your diet nutritionally sound? Explain ______

Do you take nutritional supplements? List them. How often? How much? ______


Describe the exercise __________________________

Average number of drinks daily ______ Weekly ______ Do not drink alcohol at all ______ (no alcohol is permitted 24 hours before blood samples are taken) What is your height? ______

Weight? ______ Last taken blood pressure ______

Have you ever had: Anaphylaxis ______ Arthritis ______

Emphysema ______ Paralysis ______ Peptic ulcer ______ Stroke ______
Appendix B—Continued

Tuberculosis____ Convulsions____ Diabetes____

Heart attack____ Severe dizzy spells____ High blood pressure
Laryngeal edema___ Loss of consciousness___ Psychiatric
care___ Pneumonia___ Severe reactions to allergy tests or
allergy injections____ What is the worst allergic reaction
you have ever had?________________________________________

Have you ever had a severe exposure to chemicals, for example,
to pesticides?_______ If so, describe. When, where, etc.

___________________________________________________________________

Are you chronically being exposed to any chemicals now?____
If so, describe____________________________________________________

DRUG HISTORY

Check drugs taken on a regular basis:

Cortisone____ Phenobarbital____ Tranquilizers____
Penicillin____ Demerol____ Digitalis____
Marijuana____ Sleeping Pills____ Sulfa Drugs____
Insulin____ Street Drugs____ Paregoric____
Nose Drops____ Mycin Drugs____ A.C.T.H.____
Hormones____ Aspirin____ Antihistimines____
Adrenalin____ TYLENOL____ Dilantin____
Cough Medicine____ Blood Pressure Med____ Laxatives____
Antibiotics____ Codeine____ Birth Control
Metaprel____ Susphrine____ Pills____
Brondecon____ Decadron____ Alupent____
Theokin____ Aminodur____ Potassium Iodine____
Deconamine____ Theophylline____ Bronkephrine____
Aminophylinn____
Appendix B--Continued

Bronkodyl_____ Elixophyllin_____ Vanceril_____
Verequad____  Bronkometer____  Ephedrine_____
Aerosols____  Prednisone____
Others

Do you require: normal____ low____ high____ doses of drugs as a rule? Explain__________________________

Do you require frequent use of antibiotics? Yes____ No____

Which__________________________

Do you get colds or other upper respiratory ailments frequently?

Explain__________________________

Indicate your choice of day and time when you would be able to take part in this study for each of the 4 scheduled intervals: First choice______ 2nd____  3rd____
Appendix C

Stress-Immune Study

Code Number ___________________________ Date ___________________________

Are you experiencing any significant psychological stress at this time? YES NO If you answered yes, describe the stress. ______________________________________________________________

Are you taking any medications at this time? YES NO If you answered yes, list the medications. ______________________________________________________________

Have you changed your health habits in any way since the last assessment period? Explain: ______________________________________________________________

PLEASE COMPLETE BOTH SIDES OF THE STATE-TRAIT ANXIETY INVENTORY AND BOTH PARTS 1 AND 2 OF THE CAQ. PLEASE RETURN THIS QUESTIONNAIRE AND ALL TEST MATERIALS TO DR. BUTLER'S MAILBOX IN TERRILL HALL. THANKS.
References


