PSYCHOIMMUNOLOGICAL ASPECTS OF ANGER:
T-CELL CORRELATES

THESIS

Presented to the Graduate Council of the
University of North Texas in Partial
Fulfillment of the Requirements

For the Degree of

MASTER OF SCIENCE

By

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Denton, Texas
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Immunological correlates of anger conditions were investigated. Participants were 33 females and 36 males, ranging from 25 to 55 years old. Percentages of total T-lymphocytes, suppressor-T, helper-T, and ratio of helper-T to suppressor-T cells were measured. Differences were found between males and females for Anger Control and Anger Expression. For females, total T-cell percentages correlated with State Anger, Angry Temperament, Anger Out, and the combination of State Anger/Angry Reaction. Suppressor-T cell percentages correlated with State Anger, Trait Anger, Angry Temperament, Anger Out, Anger Expression, and the combination of Angry Temperament/Anger In. Helper-T cells correlated with State Anger, Angry Temperament, Angry Reaction, Anger Out, and Anger Control. Mindbody appears to function in a unified fashion.
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## PSYCHOIMMUNOLOGICAL ASPECTS OF ANGER: T-CELL CORRELATES

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Psychomununological Aspects of Anger:
T-Cell Correlates

An abundance of research has emerged from the areas of behavioral medicine and psychoneuroimmunology implicating the significance of psychological factors in the etiology and progression of disease (Ader, 1981). This has led to the proposal that specific personality types may be selectively prone to illness. The primary basis used for distinguishing those most at risk appears to be factors related to emotionality. Particularly, negative emotions such as depression and anger have seemed to predominate as deleteriously affecting health. (Eysenck, 1988, and Friedman & Booth-Kewley, 1987)

In a longitudinal study of 500 adults, Barefoot, Siegler, Nowling, Peterson, Haney, and Williams (1987) found that the presence of negative emotions and attitudes successfully predicted general health and mortality status for both men and women. Johnson and Broman (1987) investigated the relationship between anger and health problems in a large sample of black adults and determined that those most at risk had higher levels of anger expression. In a subsequent analysis of this population, Broman and Johnson (1988) found that anger was significantly correlated with the experience of negative life events and that these two variables were independently associated with health problems. In their reviews of research linking personality variables and cardiac heart disease, Booth-Kewley and Friedman (1987) and Diamond (1982)
concluded that a mixture of negative emotions, including anger and depression, were strongly associated with this illness. Recent research, however, has identified anger and hostility as the components of the Type A personality that are most predictive of cardiovascular disease (Hearn, Murray, & Luepler, 1989, Koskenuvu, Kaprio, Rose, Kesaniemi, Sarna, Heikkila, & Langenvaino, 1988, and Krantz, Contrada, Hill, & Friedler, 1988).


According to Spielberger, Johnson, Russell, Crane, Jacobs, and Worden (1985), much ambiguity in the conceptualization and measurement of anger and its expression can be noted throughout the literature. Often, no distinctions have been made between anger, hostility, and aggression (Biaggio & Maiuro, 1985), which may account for inconsistency in results. Siegal (1986) noted that measures of hostility reflect an attitudinal component of anger. Aggression is described by Averill (1982) as a behavioral component of anger. Most researchers have agreed that the anger response involves activity of the autonomic nervous system (Diamond, 1982).

In his review of the literature, Diamond (1982) reported that anger has most often been associated with increases in sympathetic nervous system activity, and the observed changes in physiology due to resultant increases in epinephrine and norepinephrine. Activity of the sympathetic nervous system potentiates release of these catecholamines by direct innervation of nerve fibers into the adrenal medulla from the intermediolateral horn cells of the spinal cord (Tecoma & Huey, 1985). Epinephrine's effects on peripheral physiology include an increase in cardiac outflow, heart rate, contractile force, and vasodilation. Norepinephrine primarily acts as a vasoconstrictor. Goldstein and McDonald (1988) have suggested that prolonged autonomic arousal
precedes cardiovascular health risks. Furthermore, there is evidence that high levels of emotional control produce excessive autonomic arousal (Anderson, 1981). Henry (1988) suggested that emotional arousal coupled with suppression of emotions are factors mediating essential hypertension. Although most researchers seem to attribute cardiovascular risk to catecholamine increases (Diamond, 1982), Herd's (1986) review indicated that catecholamines do not appear as involved as cortisol in mediating these effects. Cortisol is a glucocorticoid that is important in the regulation of fat, carbohydrate, sodium, potassium, and protein metabolism. Activity in the limbic system, particularly the hippocampus, initiates activity of the limbic-hypothalamic-pituitary-adrenal system in which adrenocorticotropic hormone released from the pituitary promotes secretion of glucocorticoids from the adrenal cortex (Holsboer, 1988).

Studies of the effects of the products of sympathetic nervous system activity and limbic-hypothalamic-pituitary-adrenal system activity on various immune parameters provide strong evidence that arousal processes (such as those thought to occur during anger) may have significant effects on immune functioning. The immune system plays a major role in combating disease by recognition and destruction or elimination of substances recognized as foreign. This accomplished largely through the actions and interactions of two major system: cell-mediated immunity and humoral immunity. T lymphocytes are involved in
cell-mediated immunity and may be divided into several subsets based on function and identified by surface markers. Subsets include suppressor-T cells, helper-T cells, cytotoxic-T cells and delayed-type hypersensitivity-T cells. Helper T cells and suppressor T cells help regulate the immune response by controlling the initiation and termination of T and B cell activity. Some T cells release substances known as lymphokines which aid in the destruction process and may include macrophage migration factor, chemotactic factors, cytotoxic factors, and interferon. B cells are involved in the humoral immune system, and following activation, become sensitized, proliferate, and differentiate into plasma cells, which then synthesize immunoglobulins (IgG, IgM, IgA, IgE, and IgD). Other cells involved in the immune system are monocytes, neutrophils, macrophages, mast cells, and natural killer cells. Macrophages play a role in the activation of lymphocytes by production of interleukin-1, inducing helper-T cells to produce growth factors such as interleukin-2. Natural killer cells are considered a subpopulation of lymphocytes that are important in recognition and destruction of cancer cells and some virally infected cells. (Schliefer, Scott, Stein, & Keller, 1986).

Responses of the immune system to catecholamines are complex and difficult to delineate (Dienstbier, 1989). Enhancement of immune parameters as well as suppression have been demonstrated. Catecholamines have been shown, in vitro, to suppress the immune
system through such effects as diminished chemotactic and phagocytic activities of monocytes and neutrophils, and inhibition of lymphocyte proliferation, cytotoxic activity, and secretory activity, including immunoglobulin and interferon synthesis (Fuchs, Campbell, & Munson, 1988, Jemmott, 1985, and Tecoma & Huey, 1985). These effects appear to be produced when intracellular cyclic adenosine monophosphate is elevated in response to β-adrenergic agonists such as epinephrine and norepinephrine (Tecoma & Huey, 1985). Leukocyte β-adrenergic receptors have been identified, and in vivo exposure to β-agonists has been shown to affect β-receptor density of neutrophils and lymphocytes, decrease numbers of helper-T cells, increase numbers of suppressor-T cells, and reduce lymphocyte proliferation (Fuchs et al., 1988, and Jemmott, 1985).

It appears that cortisol may play an important role in modulating immunosuppressive effects of catecholamines. One role of cortisol in suppression of neutrophil functioning is derived from its ability to increase the number of β-agonist receptors (Tecoma & Huey, 1985). In their review of the literature, Guyre, Girard, Morganelli, and Manganiello (1988) proposed that glucocorticoids mediated the effects of catecholamines by inhibition of various cytokine production. These include tumor necrosis factor and interleukin-1 from macrophages and interleukin-2, interleukin-3, and interferon-gamma from T lymphocytes. Thus lymphocyte proliferation, differentiation, and cytotoxicity may be reduced, and inflammatory reactions may be diminished.
Evidence was presented relating negative emotions to disease states that are influenced by immunological competence; however, research investigating the relationship between anger and its expression and the immune system is sparse. Buetler, Engle, Oro-Buetler, Daldrup, and Meredith (1986) cited evidence that emotional suppression occurring in conjunction with prolonged stress may result in decreased natural killer cell activity and differentiation of lymphocyte subpopulations. Minimization of negative emotions was also determined to be associated with decreased levels of circulating monocytes (Jamner, Schwartz, & Leigh, 1988). Pettingale, Philalithis, Tee, and Greer (1981) found higher levels of serum IgA in those women with breast cancer who suppressed anger. Kiecolt-Glaser and Glaser (1988) investigated the effects of emotional expression through psychotherapy on immune functioning. Their results indicated that writing about traumatic experiences increased the responsiveness of T cells to mitogen stimulation. They also found a decrease in visits to a health center by the psychotherapy group as compared to a control group, as well as a decline over time in measures of autonomic arousal (blood pressure and heart rate.)

The purpose of the present study was to investigate the association of anger and its expression with immune parameters. The experience and expression of anger was determined by a self-report questionnaire. Immune parameters measured include T lymphocyte subtypes.
(suppressor-T cells and helper-T cells). It was hypothesized that anger would be associated with increased strain on immunological competence which would be reflected by differences in the circulating percentages of total T cells and T cell subtypes (suppressor-T and helper-T cells), as well as helper-T cell to suppressor-T cell ratios.

**METHOD**

**Subjects**

There were 69 subjects drawn mainly from a psychology graduate student population, but also included some from staff, faculty, and others outside the university. There were 36 males and 33 females. Ages ranged from 25 to 55 years old. Prospective subjects completed a health questionnaire to gather demographic information and screen for the presence of general health problems.

**Instruments**

*State-Trait Anger Expression Inventory (STAXI)*. The STAXI, developed by Spielberger (1988), was used to determine the experience and expression of various components of anger. Subscales include State Anger, Trait Anger, Angry Temperament, Angry Reaction, Anger In, Anger Out, Anger Control, and Anger Expression. Validation studies have indicated State Anger and Trait Anger are highly valid and reliable measures (Spielberger, 1988). Anger expression subscales reliabilities have been reported to range from .70 to .73 and validities from .22 to .52 (Knight, Chisholm, Paulin, & Waal-Manning, 1988).
Technicon H*1 Analyzer (Technicon, 1985). The Technicon H*1 is an automated flow cytometer that provides complete blood counts and white blood cell differential counts. The complete blood count was performed to assay the general health of each subject. The instrument also provides lymphocyte subset determination utilizing a direct cytometry option following manual preparation of blood samples. Marion, Auclair, and Jasmin (1987) have demonstrated good correlation between the Technicon H*1 and the Coulter Epics for lymphocyte subset determination.

Procedure

All subjects were administered the STAXI according to standard procedures. Three to four days later, early morning blood samples were drawn, including 5 ml in EDTA and 7 ml in a serum separator tube. All subjects were instructed to refrain from taking medications or consuming alcohol for 24 hours prior to blood sampling. Samples were transported and complete blood counts and lymphocyte subset assays were begun within four hours of collection.

Complete Blood Count. Following confirmation of instrument reliability, each sample was adequately mixed and aspirated through the Technicon H*1 Analyzer, producing complete blood counts. All subjects were within the normal range for white blood cell counts, thus ruling out the presence of infection.
Lymphocyte Subset Analysis. For the enumeration of lymphocyte subtypes, each blood sample was prepared manually prior to aspiration according to standard techniques (Hickey & Gannon, 1987, and Technicon, 1985). Each sample was divided and reacted with the appropriate monoclonal antibody. Lysis of red blood cells and fixation of lymphocytes and other white blood cells were then produced by addition of specified diluents. Enzyme labeling followed by using a biotinylated antibody to bridge the tagged lymphocytes with peroxidase-labelled avidin. Following peroxidase staining, samples were aspirated through the Technicon H*1 using the direct cytometry option. The antibody tagged cells were then detected and quantified in the tungsten-based optics channel.

RESULTS

Data were analyzed by Pearson product moment correlation and multiple regression analysis. Partial correlations were also performed to control for the significant association of recent chemical exposure with pan-T, suppressor-T and helper-T cells. The ratio of helper-T cells to suppressor-T cells was calculated and included in all analyses as well. Analyses were performed for males and females, n = 36 and 33 respectively.

Differences between males and females for immunological and anger data were determined by t-tests. Results indicate no differences in immunological responding between males and females. This appears to be true for all immunological parameters measured (pan-T, suppressor-T
and helper-T) as well as the calculated ratio of helper-T cells to suppressor-T cells.

Table 1

<table>
<thead>
<tr>
<th></th>
<th>Total-T</th>
<th>Suppressor-T</th>
<th>Helper-T</th>
<th>Helper-T/Suppressor-T Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>65.801</td>
<td>16.797</td>
<td>37.578</td>
<td>0.2695</td>
</tr>
<tr>
<td>Females</td>
<td>71.873</td>
<td>16.141</td>
<td>40.718</td>
<td>0.2821</td>
</tr>
<tr>
<td>t test</td>
<td>-1.82</td>
<td>0.48</td>
<td>-1.40</td>
<td>-0.33</td>
</tr>
</tbody>
</table>

T tests revealed significant differences between males and females for two anger conditions, Anger Control: $t(67) = -3.18, p < .01$, and Anger Expression: $t(67) = 4.06, p < .01$.

Table 2

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th>Females</th>
<th>t-test</th>
</tr>
</thead>
<tbody>
<tr>
<td>State Anger</td>
<td>55.395</td>
<td>52.057</td>
<td>1.63</td>
</tr>
<tr>
<td>Trait Anger</td>
<td>53.789</td>
<td>50.400</td>
<td>1.29</td>
</tr>
<tr>
<td>Angry Temperament</td>
<td>55.711</td>
<td>53.257</td>
<td>1.03</td>
</tr>
<tr>
<td>Anger Reaction</td>
<td>50.895</td>
<td>50.943</td>
<td>-0.02</td>
</tr>
<tr>
<td>Anger In</td>
<td>53.658</td>
<td>54.771</td>
<td>-0.49</td>
</tr>
<tr>
<td>Anger Out</td>
<td>54.868</td>
<td>51.600</td>
<td>1.25</td>
</tr>
<tr>
<td>Anger Control</td>
<td>43.763</td>
<td>53.000</td>
<td>-3.18*</td>
</tr>
<tr>
<td>Anger Expression</td>
<td>57.947</td>
<td>45.743</td>
<td>4.06*</td>
</tr>
</tbody>
</table>

* $p < .01$
For the female group, results indicated that, depending upon the particular anger conditions, significant relationships exist with total-T, suppressor-T, and helper-T cells. State anger was significantly correlated with total-T cells, $r(32) = .69$, $p < .01$, suppressor-T cells, $r(32) = .54$, $p < .01$, and helper-T cells, $r(32) = .40$, $p < .05$. Trait anger was significantly correlated only with suppressor-T cells, $r(32) = .46$, $p < .01$. Total-T, suppressor-T, and helper-T cells were significantly associated with Angry Temperament, $r(32) = .45$, $p < .01$, $r(32) = .67$, $p < .01$ and .36, $p < .05$, respectively. Angry Reaction was significantly correlated with only helper-T cells, $r(32) = .43$, $p < .01$. Anger Out was also significantly associated with total-T, suppressor-T, and helper-T cells, $r(32) = .45$, $p < .01$, .31 and .34, $p < .05$, respectively. Anger Expression was significantly correlated with only suppressor-T cells, $r(32) = .47$, $p < .01$, and Anger Control with only helper-T cells, $r(32) = .35$, $p < .05$.

Multiple regression analysis indicated that the combination of State Anger and Angry Reaction were significantly associated with total-T cells, $r(32) = .77$, $p < .01$ and that Angry Temperament and Anger In were together significantly correlated with suppressor-T cells, $r(30) = .71$, $p < .01$. 
### Table 3

**Correlation of Anger Scores and Immunological Measures**

<table>
<thead>
<tr>
<th></th>
<th>Pan-T Males</th>
<th>Pan-T Females</th>
<th>Suppressor-T Males</th>
<th>Suppressor-T Females</th>
<th>Helper-T Males</th>
<th>Helper-T Females</th>
<th>Helper-T Suppressor-T Ratio Males</th>
<th>Helper-T Suppressor-T Ratio Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>State Anger</td>
<td>.11</td>
<td>.69**</td>
<td>.11</td>
<td>.54**</td>
<td>.11</td>
<td>.40*</td>
<td>-.04</td>
<td>-.06</td>
</tr>
<tr>
<td>Trait Anger</td>
<td>.09</td>
<td>-.18</td>
<td>.05</td>
<td>.46**</td>
<td>.06</td>
<td>-.14</td>
<td>-.29</td>
<td>.08</td>
</tr>
<tr>
<td>Angry Temperament</td>
<td>.11</td>
<td>.45**</td>
<td>.02</td>
<td>.67**</td>
<td>.06</td>
<td>.36*</td>
<td>-.28</td>
<td>.09</td>
</tr>
<tr>
<td>Angry Reaction</td>
<td>.02</td>
<td>-.27</td>
<td>-.01</td>
<td>.05</td>
<td>-.04</td>
<td>.43**</td>
<td>-.15</td>
<td>.04</td>
</tr>
<tr>
<td>Anger In</td>
<td>.10</td>
<td>-.12</td>
<td>-.09</td>
<td>-.07</td>
<td>.17</td>
<td>-.20</td>
<td>-.03</td>
<td>.10</td>
</tr>
<tr>
<td>Anger Out</td>
<td>.11</td>
<td>.45**</td>
<td>.13</td>
<td>.31</td>
<td>.06</td>
<td>.34*</td>
<td>-.18</td>
<td>-.02</td>
</tr>
<tr>
<td>Anger Control</td>
<td>-.12</td>
<td>.05</td>
<td>-.02</td>
<td>.28</td>
<td>-.09</td>
<td>.35*</td>
<td>.31</td>
<td>-.06</td>
</tr>
<tr>
<td>Anger Expression</td>
<td>.21</td>
<td>.29</td>
<td>.00</td>
<td>.47**</td>
<td>.15</td>
<td>.18</td>
<td>-.21</td>
<td>.10</td>
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*p < .05,  **p < .01

**DISCUSSION**

Results indicate firm support for the hypothesis that anger would be associated with differences in circulating percentages of total T cells and their subtypes (suppressor-T and helper-T cells) for the female group only. There appears to be a clear relationship between the emotional state of anger and immunological disruption in women. No changes in this measure of immunocompetency were found for the male population. It is possible that the anger responding of males does not exert an appreciable effect on those physiological processes responsible for
adequate numbers of circulating suppressor- and helper-T cells. It may be, however, that some other aspect of immunocompetence is associated with anger expression and experience in males. The existence of differential changes between males and females in immunocompetence associated with various aspects of anger indicates that males and females will likely be differentially affected with respect to the types of illnesses mediated through these changes. Interestingly, a higher incidence of autoimmune disease (rheumatoid arthritis and systemic lupus erythmatosis) exists in the female population.

Those women who express anger as the situation demands, who express anger with little provocation, or who tend to express anger outwardly have associated increases in both suppressor- and helper-T cells. These subjective feelings are typically accompanied by autonomic arousal, the extent of which may depend on variations in anger intensity and individual differences in terms of physiological response. No significant change appears to have occurred in the ratio of helper- to suppressor-T cells for this group, thus qualitative changes in immune competency are difficult to predict. This may represent a compensatory mechanism by which the body maintains a balance in immune integrity, however, this may occur at the expense of a higher expenditure of energy for maintenance of adaptation in the presence of increased stressors. Constant disruption and consequent balancing could produce enough strain on the system to jeopardize appropriate responding under certain
conditions. This might then be reflected by decreased immune competency or health status in susceptible individuals.

The implications for health when helper-T cells are elevated are less clear. Increases in percentages of these cells appear to occur in those women who tend to respond with anger when criticized or when they perceive unfair treatment directed toward them, and in those who frequently attempt to control their expression of anger. These changes may be an immunological representation of psychological anticipation of a stress condition, or could be the result of an appropriate coping response for these individuals during such situations.

More indicative of immunological disruption are those changes in single T-cell subtypes associated with particular components of anger. The effects of such a change appear more evident with suppressor-T cell involvement than with helper-T cell changes. Increases in only suppressor-T cells appear to occur for those women in which anger constitutes the typical response to a wide variety of situations, presumably due to a long-standing propensity to perceive such situations as annoying and frustrating, and those who frequently express their anger, regardless of the direction of that expression. The association of suppressor-T cells in those women who typically respond to a wide array of situations with anger and also suppress or hold in their responses is particularly significant. The combination of these two types of anger seems more clearly indicative of deleterious health consequences. This effect may be
mediated by the associated increase in suppressor-T cells which diminish the ratio of helper to suppressor cells below its optimum, thus potentially having an important effect on other immune parameters whose function is dependent on the maintenance of an appropriate ratio. In particular, the functioning of the B cell response for production of antibodies seems to be the most likely parameter directly affected by such a change.

The operation of a balanced immune system is integral to health and is also dependent on a number of other factors and their individual interactions within the system. Enough disruption within the immune system in association with these various components of anger and in combination with an appropriately susceptible environment could result in a diminished ability to effectively combat a variety of illnesses. This could be of vital importance in the progression of a number of disease states, as well as possibly influencing the timely success of treatment regimens. The acceptance, particularly by physicians, of the influence of psychological states on physical health will have a positive influence on the traditional way of viewing the patient and their presenting symptoms as well as changing standard methods of patient care.

The implications of these results also have significance for the mental health practitioner. Mental processes and subsequent bodily reactions are much the same for the remembrance of a situation as the actual situation. Thus, the reinvestment of anger during the reliving of an event is likely to adversely affect the physical health of some clients.
As suggested by these results, traditional psychotherapeutic techniques are placing some clients at risk for certain types of illnesses. Future research should be directed toward identifying those who may be at risk as well as developing alternate techniques that may eliminate or decrease such risks, so that the physical health of these clients is no longer jeopardized.

It appears that all health-care professionals must necessarily become increasingly aware that the conceptualization of the mind and body as distinct entities with no influence on one another is an assumption not based upon scientific evidence. Mental and physical health care encompass more than what has been previously thought and professionals in these fields have increasingly important and extended responsibilities to their clients. Acceptance of this responsibility and incorporation of these ideas into practice would reconceptualize health care and greatly improve its efficacy. This would best be accomplished through a team approach to health in which the concern is focused on all aspects of an individual's functioning.
APPENDIX A

INFORMED CONSENT
Appendix A
Informed Consent

I, __________________________ wish to participate in a research project being conducted under the supervision of Dr. J. R. Butler. I understand that the primary purpose of this research is to demonstrate the existence or nonexistence of a relationship between certain psychological factors (e.g., hostility, tension, etc.), various immune system parameters, and endocrinological functions. I understand that all blood will be drawn by a trained phlebotomist and that J. R. Toledo, M.D., or other licensed physician, will provide medical supervision. I understand that I will be required to take paper and pencil psychological tests 3 or 4 days prior to the blood sample being 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.

I, __________________________, hereby release the University of North Texas, the Department of Psychology, and the Psychology Clinic from all claims, demands, damages, actions, or causes of action, costs, loss of services
and expenses resulting from research that will include blood draws to be taken to measure immune system parameters and endocrinological functions and the administration of psychological questionnaires. It is understood that I may withdraw from participation in this research at any time.

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

Signed ____________________________

Participant

Signed ____________________________

Witness

Date ____________________________
APPENDIX B
HEALTH INVENTORY
Appendix B
Health Inventory

Name (ID number) ____________  Today’s date ____________
Address ____________________________________________
Residence Phone no. ________________________________
Business/other phone ________________________________
Date of birth ________________  Sex _____  Race __________
Marital Status ________________  Other employment ______________________
Where employed ______________________________________
Program at UNT ____________  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 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 ______ 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 ______ Sufa Drugs ______
Insulin ______ Street Drugs ______ Paregoric ______
Nose Drops ______ Mycin Drugs ______ A.C.T.H. ______
Hormones ______ Aspirin ______ Antihistamines ______
Adrenalin ______ Tylenol ______ Dilantin ______
Cough Medicine ______ Blood Pressure Med ______ Laxatives ______
Antibiotics______  Codeine______  Birth Control Pills______
Metaprel______  Susphrine______  Alupent______
Brondecon______  Decadron______  Potassium Iodine______
Theokin______  Aminodur______  Bronkephrine______
Deconamine______  Theophylline______  Aminophyllin______
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.

First choice: Day____________ Time:____________
2nd choice: Day____________ Time:____________
3rd choice: Day____________ Time:____________
REFERENCES


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